Supplemental Risk Assessment of Potential Air Emissions from the Confined Disposal Facility for the Indiana Harbor and Shipping Canal Sediment Dredging and Disposal Project

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EXECUTIVE SUMMARY

In order to maintain channel depths in the Indiana Harbor and Shipping Canal (IHSC), the United States Army Corps of Engineers (USACE) must periodically dredge the bottom sediment. The harbor was last dredged in 1972. Because the sediment contains a variety of pollutants, it must be handled and disposed of properly. USACE is currently constructing a confined disposal facility (CDF) for the sediment in East Chicago, Indiana. The site consists of about 168 acres of land and was formerly operated as an oil refinery for many years. Dredging is expected to begin in 2009 and continue for 30 years.

In September 1998, USACE submitted a Comprehensive Management Plan for the proposed construction of a CDF in East Chicago, Indiana for disposal of dredged sediments from the IHSC. This included a Feasibility Study (FS) and Environmental Impact Statement (EIS). Due to the concern of risks posed by construction and operation of a CDF, the United States Environmental Protection Agency (USEPA) conducted a risk assessment for USACE that was finalized in 1995 and included in the EIS. The 1995 risk assessment reported that the total cancer and noncancer hazard risks due to inhalation of potential emissions from the CDF are within the risk range that USEPA considers acceptable.

Because the public raised concerns about the 1995 health risk assessment and because risk assessment methods have evolved and advanced since 1995, USEPA agreed to perform additional risk study and calculations to provide additional information regarding potential exposures from the CDF. USEPA met with concerned residents and the public on several occasions to discuss the supplemental risk assessment and describe the approach that was used.

The purpose of the supplemental risk assessment (SRA) is to estimate potential health risks from the incremental emissions from the CDF. It does not attempt to estimate cumulative health risks from other existing background exposures. The methodology of the study generally followed the 2005 EPA *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.* As described in detail in the report, the SRA approach included several components, in the following general sequence:

- Estimation of sediment contaminant concentrations
- Estimation of contaminant releases (emission rates)
- Air dispersion and deposition modeling of contaminants
- Contaminant toxicity assessment and toxicity factor selection
- Selection of exposure pathways and exposure scenarios
- Estimation of environmental media concentrations and human exposures
- Quantified risk and hazard.

Information utilized in the study included:

- 53 chemicals of potential concern
- Site-specific CDF operating parameters and schedules
- Five years of meteorological data from three nearby meteorological towers, including hourly wind speed, wind direction, and temperature to account for seasonal and climatic changes

- Compliance limits on emissions that the facility will be required to meet (25 tons per year volatiles and 25 tons per year particulate matter), which were not in effect at the time of the 1995 study.
-

The following exposure scenarios were considered for cancer and noncancer health effects:

- Adult residents in six nearby neighborhoods
- Child residents in six nearby neighborhoods
- Students at local schools
- Local fishers (adults and children)
- Nursing infants.

Specifically, the SRA evaluated the following:

- Chronic inhalation exposures
- Acute inhalation exposures
- Chronic home-grown produce ingestion exposures
- Chronic incidental soil ingestion exposures
- Chronic fish consumption exposures
- Chronic dermal absorption exposures
- Early life stage differences.

Conclusions

The results of the SRA provide an estimate of cancer risks, noncancer hazard index results, early life-stage differences in exposure and toxicity, lead exposure, dioxin exposures and acute inhalation risks.

The risk of developing cancer is expressed as the chance or probability that an individual will develop cancer sometime during their lifetime from exposure to pollutants from the CDF. Using high-end exposure scenarios, the risk of developing cancer for an individual adult or child resident living near the CDF is estimated not to exceed 1.4 people in 100,000 people (expressed mathematically as 1.4×10^{-5}). The high-end cancer health risk found in the SRA is considered relatively low and below EPA's established risk level (less than 1.0×10^{-4}).

For non-cancer health effects, the study's results show that no individual chemical displays a "hazard quotient" above a level of 1.0. A hazard quotient indicates the extent to which an estimated level of chemical exposure is expected to cause adverse health effects. The hazard quotient is a ratio obtained by comparing the estimated chemical exposure level to a safe or "no effect" exposure level that should not cause serious illness even over the long term. A hazard quotient is determined for each chemical through each exposure pathway. The hazard quotients for all chemicals are then added, and the combined hazard quotients are called the "hazard index" for that exposure pathway.

For an estimated hazard index of less than 1.0, EPA will generally recommend no further action; for an estimated hazard index greater than 1.0, EPA will generally recommend some sort of action to cut down on the possible exposure to pollutants and reduce health risk. The total

hazard index for all the CDF chemicals combined is below 1.0 (highest HI Scenario, Adult Fisher HI = 0.265).

EPA evaluated possible lead exposure from the CDF. Lead can act as a developmental neurotoxin; therefore, lead exposure in young children is a significant health concern. Potential exposure to lead releases from the CDF could occur as the result of wind-blown particles landing on soil in the vicinity of the site. Exposure to lead emissions cannot be evaluated the same way that cancer and non-cancer risks are estimated. Instead, EPA uses a computer model that estimates possible increases in blood lead levels for children living near a lead source. The model showed that potential lead releases from the CDF will not cause significant increases in child blood lead levels.

EPA evaluated dioxin exposure from the CDF. For local adult and child residents, dioxin exposure is assumed to occur from consumption of locally caught fish, consumption of garden vegetables, incidental soil ingestion and dermal absorption. For infants up to 1 year of age, dioxin exposure is assumed to occur through breast milk consumption. The dioxin exposure estimates from the CDF were compared with expected U.S. average background exposure levels for adults and breast-feeding infants. The dioxin exposures estimated to occur from CDF emissions are much lower than the national background averages. Therefore, the estimated dioxin emission released from the CDF is not expected to significantly increase health risks compared to the existing background situation. In addition, potential cancer risk from dioxin exposure was also evaluated for adult and child residents.

EPA conducted a limited evaluation of potential early lifestage exposure and effects. This study attempts to quantify how much more sensitive exposed children are to pollutants than exposed adults. The study presents one exposure pathway (soil ingestion) to give a quantitative illustration showing the extent to which estimated cancer risk could increase under assumptions of increased childhood susceptibility to a particular type of carcinogenic agent. The study used age-dependent adjustment factors for sediment contaminants that have a mutagenic mode of carcinogenicity action. The early lifestage exposure study results show estimated cancer risk for exposure to these chemicals during ages zero to six years through the soil ingestion pathway produces approximately a three-fold increased cancer risk (from 1.6×10^{-6} to 5.2×10^{-6}).

Finally, the SRA examined acute inhalation exposure from CDF contaminants. When compared with levels of pollutants that can cause illness, the study shows the health risks from breathing short-term concentrations of contaminated dust and vapor from the CDF are relatively low.

The Indiana Department of Environmental Management has set limits on the amount of air pollution that can be released from the site. The Army Corps of Engineers will conduct modeling in order to show that CDF emissions conform to these limits. The Corps will also operate monitors around the site to measure contaminants coming from the CDF. If the site does not exceed these air pollution limits, the health risks should not increase.

1 INTRODUCTION

1.1 Background

The United States Army Corps of Engineers (USACE) is authorized to operate and maintain a navigation project at Indiana Harbor in East Chicago, Lake County, Indiana, to allow for passage of ship traffic in the harbor and shipping canal. Northwest Indiana is part of a highly industrialized urban area, which is one of the nation's foremost locations for integrated steel production and a wide range of other manufacturing and petroleum refining activities. Sediments, many of which are contaminated from previous industrial discharges, have entered the Grand Calumet River/Indiana Harbor and Shipping Canal (GCR/IHSC) waterway and have been deposited in the channel, reducing depth and restricting the movements of navigational traffic. In order to maintain authorized channel depths, these sediments must be dredged periodically. Because these sediments are contaminated with a variety of pollutants, they must be disposed of properly. Local residents and others in the area have expressed concern about air pollutant emissions and potential health consequences from the USACE-operated disposal site that is under construction.

Indiana Harbor – View from Lake Michigan

In September 1998, USACE submitted a Comprehensive Management Plan for the proposed construction of a confined disposal facility (CDF) in East Chicago, Indiana for disposal of dredged sediments from the IHSC. This included a Feasibility Study (FS) and Environmental Impact Statement (EIS). Due to the concern of risks posed by construction and operation of a CDF, the United States Environmental Protection Agency (USEPA) conducted a risk assessment for USACE that was finalized in 1995 and included in the EIS. This report, titled *Inhalation Risk Analysis for Potential Air Emissions from the Proposed Confined Disposal Facility in the Recommended Alternative for the Indiana Harbor and Canal Sediment Dredging and Disposal Project* (USEPA 1995b), was finalized on September 1, 1995.

The study had three objectives:

1) To compare the proposed CDF particulate and volatile toxic air contaminant emissions to emissions reported in the Toxics Release Inventory (TRI) and reported in two previous air pollution studies in Northwest Indiana.

2) To compare the expected particulate and volatile air contaminant emissions from the CDF to those expected from the site in the absence of the CDF.

3) To assess the human health risks posed by the inhalation of potential airborne contaminants released from the proposed CDF.

The following is a summary of results from the 1995 study.

1) In comparison to air contaminant emissions reported in the TRI and in two previous air pollution studies conducted by USEPA Region 5 for the area, the projected emissions from the CDF are small—less than one percent of those reported in the inventory and from both of the air pollution studies.

2) Exposing dredged material to air following disposal in the CDF results in emissions of air toxics to the atmosphere that are greater than those that would occur with no activity at the site. However, some volatile and particulate emissions from the soil at the site will be eliminated by the construction of the CDF, because the CDF will cover a portion of the site and prevent emission of existing soil contaminants. It was not possible to determine if the CDF air emissions are significantly different from those posed by the existing site.

3) The cancer risk assessment reported that, using conservative input parameters, the total cancer risk due to inhalation of emissions from the CDF are smaller than the risks attributable to inhalation of existing air pollutants. The cancer risk due to inhalation exposure to CDF emissions is estimated to be 2.3×10^{-6} (2.3 in 1,000,000). Based on air monitoring data, the total estimated cancer risk due to air toxics inhalation exposure from other sources in the area (i.e., without including CDF emissions) for 30 years is estimated to be 3.1×10^{-4} (3.1 in 10,000 or 310 in 1,000,000). The noncancer assessment showed that adverse health effects from lifetime exposure to noncancer compounds emitted from the proposed CDF are unlikely.

1.2 Purpose

USEPA completed the risk assessment (described above) for the proposed CDF for dredged sediments from the IHSC in March 1994 and revised it in 1995. Most assumptions used in the 1995 risk assessment were attempts to err on the side of overestimating, rather than underestimating, pollutant concentrations and resulting risk from the CDF. However, to respond to public concerns, USEPA agreed in 2001 to perform additional risk assessment work to supplement the existing study. This Supplemental Risk Assessment (SRA) is intended to:

1) Provide additional information regarding potential exposures from the CDF
2) Be used as a predictive tool to help evaluate potential risks from proposed future CDF operations
3) Serve as a basis for recommending additional CDF emission controls and mitigation measures, if necessary.

Partly due to these limited objectives and scope (see below), SRA results are likely to be most informative when used in conjunction with other important information, such as CDF air monitoring data, future USACE sediment sampling and analysis; and controls and contingency plans to mitigate CDF emissions

1.3 Scope

The SRA will estimate incremental human health risks from CDF air contaminant exposure under certain emission, transport and exposure assumptions. There are uncertainties associated with those assumptions, as discussed in the report. The SRA will not evaluate "background" air pollution, nor is it designed to provide a prediction of cumulative human health risk associated with CDF air emissions and background air contamination from other local sources.

In keeping with the purpose and intent described in Section 1.2 above, the SRA will address the following:

- Inhalation, ingestion and dermal exposures. In addition to inhalation, the SRA addresses the concern that airborne particles could be deposited on the ground and absorbed through the skin or ingested from consuming locally grown produce.

- Potential exposures and potential health effects to children. The SRA addresses the concern that children are more sensitive to chemical exposures than adults.

- Additional contaminants. USEPA agreed to re-evaluate the list of chemicals of concern to assess whether additional chemicals of concern should be included.

- Potential exposures from dredging and transport of sediments. There was concern that contaminants could be released from the dredging and transport operations, as well as, the operation of the CDF. However, USACE has modified its dredging and transportation operations since the time that these concerns were raised. It was originally proposed that sediments be dredged, loaded into vehicles, and transported over road surfaces to the CDF. It is currently proposed that the dredged sediments be slurried and hydraulically placed directly into the CDF from a barge on the canal adjacent to the facility. This eliminates the concern of emissions from vehicular transportation. In addition, the surface area of the canal affected by dredging is very small relative to the surface area of the CDF, and therefore, relatively insignificant in terms of emissions.

- Use of more site-specific information. The SRA uses site-specific operational, meteorological and geographic information, to the extent that it is available.

Peer Review

In accordance with the USEPA Peer Review Handbook (USEPA 2005g), this SRA report and supporting documents have been subjected to an internal peer review by individual independent experts from within the Agency. Appendix 1-1 identifies the peer review panel members and provides copies of their credentials, Appendix 1-2 provides the charge to the peer review panel, and Appendix 1-3 provides the response to peer review comments report.

2 CDF DESCRIPTION

2.1 Facility Setting

The CDF site consists of about 168 acres of land in East Chicago (Lake County) formerly occupied by an oil refinery, which was owned and operated by Sinclair Oil Corporation and subsequently acquired by Energy Cooperative Industries (ECI). Sinclair Oil Corporation was later purchased by Atlantic Richfield Company (ARCO). ECI operated for approximately 20 years at the site, which is located just north of the Lake George Canal. The site is bordered on the east by Indianapolis Boulevard, on the north by the Cline Avenue extension, and to the west by the former Baltimore and Ohio (now CSX) railroad. ECI filed for Chapter 11 bankruptcy reorganization in 1987 and went into liquidation in 1989. In response to a bankruptcy court order in 1990, operations ceased at the site and the refinery, including oil tanks, pipelines, hazardous waste storage areas, a hazardous waste incinerator, and buildings, were completely demolished above ground. The site was leveled, cleared of debris, covered with topsoil and seeded; however, there was no remediation of sub-surface on-site contamination.

2.2 Facility Description

The CDF, when constructed, will cover about 95 percent of the site north of the Lake George Canal. It will be constructed of earthen dikes using material brought in from off site. Dikes will be constructed to a final height of approximately 33 feet above the existing ground surface. The entire dike will be constructed of compacted clay. A soil bentonite slurry wall has been constructed beyond the outside toe of the dike to a clay strata approximately 33 feet below ground surface. See Figure 2-1.

A groundwater collection system will be constructed on the inside of the bentonite slurry wall. This groundwater collection system, composed of perforated drain pipes along three sides of the CDF site, will be connected to sump pits and an on-site wastewater treatment system.

Approximately 4.8 million cubic yards of sediment will be dredged using a mechanical (clamshell) dredge with modifications to the bucket to minimize resuspension of contaminated sediments into the water column. The dredged material will be lowered into barges or scows and transported to a location near the CDF site, where water will be added to create a desired consistency that will enable it to be slurried into the CDF disposal cells. The CDF facility will be divided into two disposal cells and a smaller water equalization basin.

Dredged sediment slurry deposited in the CDF will be offloaded at the north side of the CDF and gravitationally flow toward the south. Trenches will be dug in the dredged material along the dikes to facilitate dewatering. An adjustable weir will be installed in each cell to control the distribution of water. A pump in each decant structure will pump the water from the decant structure to the equalization basin. The effluent from the equalization basin will also be treated at the on-site wastewater treatment system.

The wastewater treatment system effluent will be discharged to the Lake George Branch of the IHSC, pursuant to a National Pollutant Discharge Elimination System (NPDES) permit issued by the Indiana Department of Environmental Management (IDEM). After the CDF is filled (i.e., in approximately 30 years), it will be capped with three feet of clay, six inches of sand drainage layer, and two feet of clean fill; overlain by six inches of topsoil; and seeded. Post-closure

groundwater monitoring will be conducted to ensure that the integrity of the CDF is maintained to prevent releases to the environment.

View from the South of the CDF Site (North of Canal) Prior to Construction; Lake Michigan in Background

3 GENERAL STUDY DESIGN AND METHODOLOGY

The SRA consists of a combination of several procedural steps and methodologies to derive quantitative estimates of health risks from potential exposure to contaminants released from the CDF. The following sections provide a preview and summary of the key steps, methodologies, and assumptions used in the SRA.

3.1 Contaminant Releases and Contaminant Emission Rates

Chemical contaminants available for release after dredging and disposal to the CDF are currently located in the IHSC (referred to as the "project area") in the form of buried sediments. Since the dredging project has not yet been initiated, data on contaminant identification and concentration levels in buried sediments were used as the starting point for predicting the contaminant concentration levels in the CDF sediments after the project starts. USEPA reviewed the available data record (i.e., "historical data") on sediments in the project area to select data sets that were judged to most appropriately represent the chemical identity, location, and characteristics of sediments planned for dredging and placement in the CDF.

After sediments are placed in the CDF, contaminant releases could occur in the form of volatile emissions and particulate emissions. Published theoretical models (supported by limited empirical data) indicate that volatilization occurs primarily from submerged sediments and wet exposed sediments, and that volatilization becomes negligible as the sediments become fully dry. By contrast, particulate emissions from soils/sediments are known to be practically negligible from wet sediments and to be maximized from dry sediments.

For development of the SRA, available published models for predicting volatile and particulate emission rates from sediments were reviewed. USEPA, USACE, and independent scientists who have developed and published the models, reviewed and discussed the major advantages, disadvantages, and uncertainties inherent in the theoretical models. During these discussions, it was determined that, as part of the operating requirements of the CDF, the emission levels for both volatile and particulate emissions would need to comply with a specific total annual limit designated by a State of Indiana air emission regulation. Based on this regulation, emission rates of contaminants were derived by setting the annual emissions of volatile contaminants and particulate matter to 25 tons for each category. In this way, the most advantageous aspects of emission rate models and regulatory emission limits were combined to devise strategies for modeling the emission rates of volatiles and particulates for use in the SRA.

3.2 Air Dispersion and Deposition Modeling of Contaminants

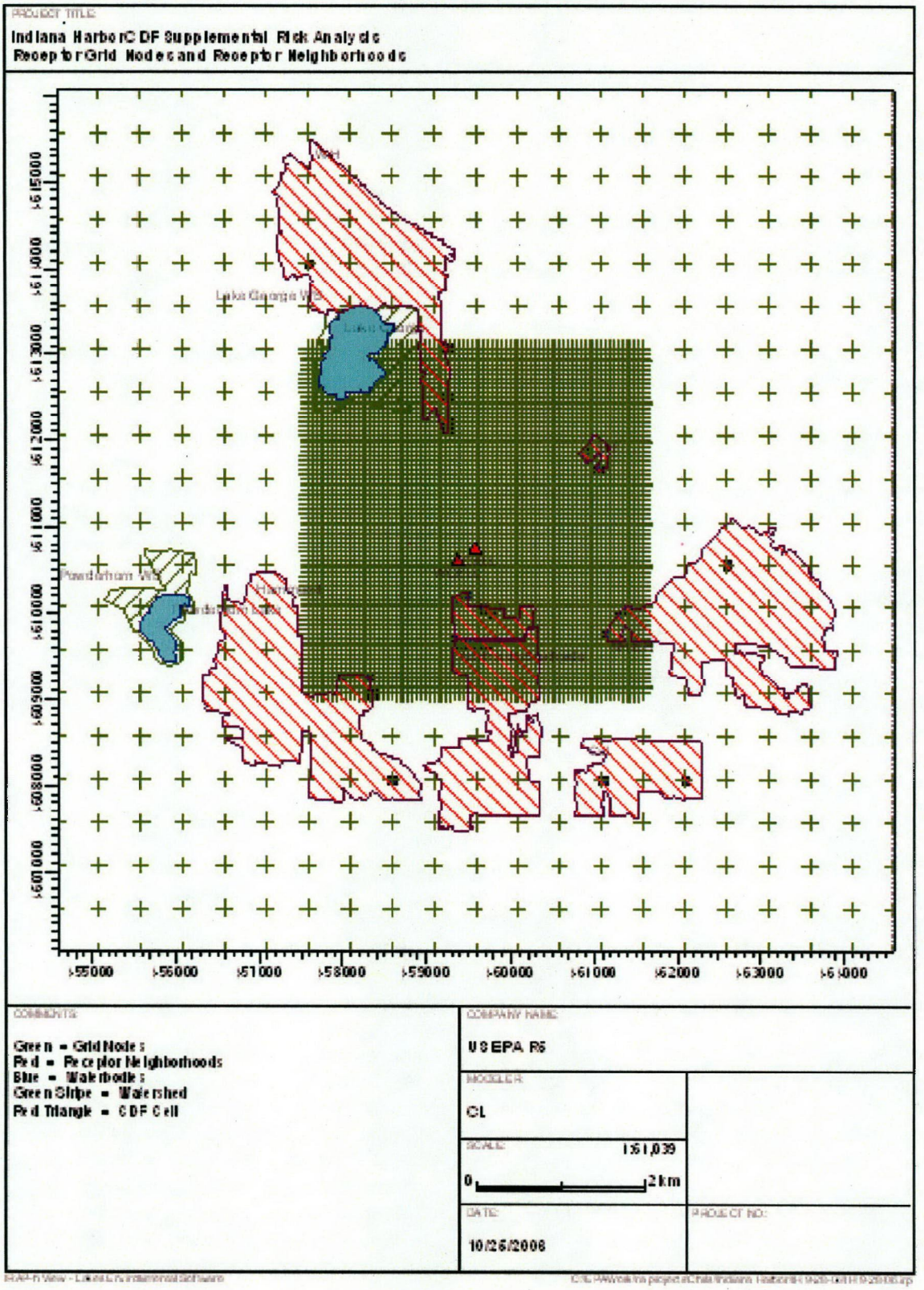
Chemical contaminants that become airborne from the surface of the CDF after release as volatiles or particulate matter will disperse in the ambient air and become available for deposition to the ground surface through a number of processes including dry deposition, wet deposition, and vapor deposition. At any given time, the direction and distance of contaminant transport will be governed by several meteorological factors including wind speed, wind direction, air temperature, solar radiation level, and precipitation events. Since disposal of sediments to the CDF has not started, direct monitoring (i.e., trapping and measurement) of contaminants cannot be performed. Instead, the air dispersion, transport, and deposition of contaminants must be predicted by a model system that accounts for the meteorological factors described above, and also incorporates local (i.e., site-specific) meteorological data and local

data on terrain elevations (i.e., land heights and land shape) to the extent possible. Air dispersion models are mathematical constructs that simulate the physical processes occurring in the atmosphere that directly influence the dispersion of vapor phase and particulate emissions from a point source or an area source. These mathematical simulations are coded into computer programs to facilitate the computational process.

The USEPA peer-reviewed Industrial Source Complex Short-Term 3 (ISCST3) Model ("Model") was employed for this project. This computerized software model estimates air concentration levels of contaminants and the rates of contaminant deposition onto local soil and local water bodies. ISCST3 was originally designed for estimating air dispersion and transport from point sources such as stack emissions from waste combustion facilities, area sources, and line and volume sources. For this application, the Model was adapted to treat the CDF as an "area source" containing two distinct sections, or "cells," with the actual dimensions planned for the CDF. In the basic Model configuration, the CDF was placed at the center of ten kilometer by ten kilometer square grid space, which serves as the study area within the Model software program. The Model then used the available data on contaminant emission rates, local meteorology, and local terrain as input data to predict the contaminant air concentrations and deposition rates at each receptor grid point for a large array of grid points spaced at specific intervals within the study area. The Model output data, in the form of digitized (numerical) air concentration and deposition rates at each grid point, were stored as a set of computer files that were utilized in the exposure pathway analysis procedure of the SRA.

The guidance for evaluating risk from combustion sources recommends using an array of receptor grid nodes covering the area within ten kilometers of the facility, with the origin at the centroid of a polygon formed by the stack emission sources (USEPA 2005c). The guidance gives an example wherein a Cartesian grid with nodes spaced 100 meters apart extends three kilometers from the origin, and grid nodes spaced 500 meters apart extend from three kilometers to ten kilometers from the origin. Consistent with this guidance, the centroid of the two CDF sediment cells was set as the origin, and a Cartesian receptor grid was extended from the origin. However, there was concern about the variability of the dispersion modeling in close proximity to the CDF, due to the large size of the cells and the presence of several critical receptor areas. Thus, a more densely spaced inner tier of receptors was generated with 50-meter spacing. After obtaining confirmation that the grid was sufficient to capture all critical receptor areas, the inner tier was limited to two kilometers from the origin and the outer tier was limited to five kilometers from the origin to compensate for the computational increase associated with the denser inner grid. See Figure 3-1 for an illustration of the receptor grid.

Figure 3-1: Indiana Harbor CDF Supplemental Risk Analysis Receptor Grid Nodes and Receptor Neighborhoods



Another feature of the Model is that it can be set up to make multiple runs that utilize different time durations of the local meteorological data. For example, to simulate the long-term average air concentrations of volatile and particulate contaminants, five years were modeled and annual average air concentrations were calculated from dispersion results of this entire period. To simulate short-term air concentrations of contaminants, the Model utilized meteorological data associated with short-term (e.g., hours, days) weather conditions and estimated the highest expected one-hour or one-day average. These results are useful for evaluating long-term and short-term concentrations of air contaminants and the corresponding potential inhalation health risks due to chronic (i.e., long-term) and acute (i.e., short-term) duration periods.

To enhance the utility of the ISCST3 Model output results for risk analysis, the SRA employed Geographic Information System (GIS) technology to aid in visualizing the relationship between the Model output and actual local features of concern in the vicinity of the CDF. For example, since the exact latitude-longitude coordinates of the CDF boundaries are known, the study area's computerized grid was placed ("overlaid") onto maps and/or photographs which also have verified latitude-longitude coordinates. The maps and photographs can display many local features of interest, in particular, land use designations—residential, industrial, and agricultural zoning; school locations; parkland locations; and water body boundaries.

3.3 Contaminant Toxicity Assessment and Toxicity Factor Selection

The toxicity assessment portion of a risk assessment combines the chemical contaminant identification with a dose-response assessment. The primary objectives of this step of the risk assessment process are to identify the types of toxic effects associated with each contaminant of potential concern (COPC), characterize the conditions (e.g., route, duration) of exposure under which these effects might occur, and assign a quantitative relationship between the amount of exposure and the magnitude of adverse health effects. The quantitative relationship is represented through the use of toxicity values, usually referred to in USEPA risk assessments as "toxicity factors." Toxicity factors are developed for individual chemicals and represent the quantitative relationship between an exposure level and a specific adverse health effect. USEPA derives toxicity factors for two categories of health effects—cancer and noncancer endpoints.

USEPA develops toxicity factors for specific chemical constituents after conducting reviews of the available toxicological and health effects information contained in the scientific literature or found in appropriate government and non-government studies. The preferred information sources are verifiable reports on human health effects (e.g., epidemiological studies of workers) from which adverse health effects can be correlated to dose levels and routes of exposure. If verifiable human studies are not available, then dose-response studies of chemical administration to experimental animals are used when the adverse effects observed in animals are determined to be relevant to potential human health effects.

USEPA has already conducted and published toxicity assessments on many of the most frequently occurring environmental chemicals and has developed toxicity factors for general use in risk assessment. Cancer and noncancer toxicity factors for chemical substances are published by USEPA and appear in an Internet database called the *Integrated Risk Information System* (IRIS), which is maintained by USEPA (USEPA 2005d). IRIS contains chemical profiles that summarize USEPA's assessment on the critical toxic effects of a chemical and explain the basis for deriving toxicity factors. IRIS profiles receive both internal and external peer review before publication. In addition, for several chemical substances with wide environmental distribution and/or complex toxicity assessments, USEPA also publishes internally and

externally peer-reviewed documents known as "Toxicological Reviews." When a Toxicological Review exists for a chemical, the IRIS profile will be developed from this document. The toxicity factors published in IRIS are routinely applied in USEPA risk assessments to promote consistency and transparency across the various USEPA programs, and because they represent a source of scientifically peer-reviewed information.

For certain substances or group of chemicals, the methodology that USEPA uses to assess toxicity or derive toxicity factors is somewhat different from that described above for the IRIS database. USEPA has adopted alternative procedures for addressing the cancer and/or noncancer dose-response assessment for the following chemicals and chemical groups addressed in the SRA: (1) polychlorinated dioxins and furans (dioxins), (2) polycyclic aromatic hydrocarbons (PAHs), (3) Lead, and (4) Particulate Matter.

The SRA provides an explanation of the alternative toxicity assessment and toxicity factor procedures that USEPA has developed for these chemicals and how the procedures were applied.

The toxicity factors described above apply under the assumption that long-term (i.e., chronic) exposure will be the primary scenario contributing to potential exposure for a given contaminant. For a risk assessment, this assumption is valid when the potential site-specific exposure is expected to occur over multiple years. This assumption is valid for the SRA since potential releases from the CDF could occur over many years of operation. The situation is evaluated in the SRA by using annual average air concentrations and deposition rates calculated from the entire modeled period as inputs to the risk assessment.

However, it is recognized that fluctuations (i.e., peaks and valleys) in the actual air concentration of a given contaminant could occur over shorter time frames within the annual average due to variations in meteorological conditions (e.g., wind speed, temperature, cloud cover, precipitation rates). As explained in Section 3.2, the ISCST3 Model utilized meteorological data corresponding to short-term (e.g., hours, days) variations in weather to estimate the highest expected one-hour or one-day average. These results are useful for evaluating the predicted range in short-term air concentration levels, and the corresponding potential inhalation health risks attributable to acute (short-term) duration periods. To evaluate the potential for adverse health effects due to short-term air concentration levels of contaminants, USEPA and other organizations have developed air concentration levels intended to provide protection for the general population from the acute effects of many commonly encountered air contaminants. These air concentration levels are commonly referred to as "acute inhalation exposure criteria" or "emergency response planning guidelines." The SRA uses these air concentration guidelines for comparison to the short-term air concentration levels predicted by the ISCST3 Model.

3.4 Exposure Pathways and Exposure Scenarios

Chemical contaminants that are emitted from the surface of the CDF, become airborne, and disperse beyond the boundaries of the CDF could subsequently be found in three environmental media of primary interest: (1) ambient air as airborne vapors and airborne particulates; (2) local soil due to wet deposition of vapor, and wet and dry deposition of particle and particle-bound contaminants; and (3) local surface water bodies due to direct deposition of contaminants from air and runoff of contaminants from land to surface water (see Figure 3-2 in Section 3.6). Human contact with one or several of these environmental media could be a potential source of exposure and intake of contaminants.

In keeping with typical USEPA risk assessment methodology, complete exposure pathways for potential CDF contaminants are assumed to exist for some individuals in the vicinity of the CDF. In some USEPA risk assessments, including this one, an individual who could be exposed to contaminants is called a receptor. A complete exposure pathway exists if the receptor has contact with a contaminated medium and an exposure route is present that results in contaminant intake. These receptors are assumed to have exposure to contaminants through one or several exposure pathways that constitute an exposure scenario.

Depending on the number of contaminated media and the expected receptor behaviors in the vicinity of the contaminant source, there could be a number of exposure scenarios selected for a risk assessment. The following exposure scenarios were selected for the SRA after USEPA evaluated the potential contaminant exposures expected in the vicinity of the CDF, conducted site visits to the CDF locale, and held discussions with local citizens:

- **Local Area Resident** – An adult or child who lives within a known residential area in the vicinity of the CDF. Residential areas are verified by evaluating zoning maps, United States Geological Survey (USGS) Quadrant maps, local land use maps, and site visits. The applicable exposure pathways are:

    - Inhalation of volatile contaminants and particulate contaminants
    - Incidental ingestion of soil containing deposited contaminants
    - Dermal contact with soil containing deposited contaminants
    - Ingestion of contaminants incorporated into produce from a typical home garden.

    See Figure 3-3 (in Section 3.6) for a conceptual model.

- **Local Area Student** – A school-age child or teenager who attends school in the vicinity of the CDF. Specific schools were selected by evaluating local maps and through site visits. The applicable exposure pathways are:

    - Inhalation of volatile contaminants and particulate contaminants during routine school attendance
    - Incidental ingestion of soil during routine school attendance or as a student-athlete
    - Dermal contact with soil during routine school attendance or as a student-athlete.

    See Figure 3-4 in Section 3.6 for a conceptual model.

- **Local Area Fisher** – An adult or child who consumes fish obtained from a local water body and is also a local area resident. A candidate local water body is one that is located partially or completely within the air model study area and is known to support the habits of a recreational fisher or the nutritional needs of a subsistence fisher (high-end fish consumer). The applicable exposure pathways are:

    - High-end or subsistence consumption of fish fillets harvested from a local water body
    - Exposure pathways expected for a Local Area Resident (as described above).

    See Figure 3-5 in Section 3.6 for a conceptual model.

After the exposure scenarios and applicable exposure pathways are selected, several numerical values are assigned to quantify the level or magnitude of the receptor's exposure to contaminants through a specific exposure pathway. These values are commonly known as exposure factors or exposure parameters. Examples of exposure factors include rates of soil ingestion (e.g., amount/day), frequency of soil ingestion (e.g., days/year), exposure duration (e.g., years), and body weight. For any given exposure pathway, the applicable exposure factors are combined with the estimated concentration of the chemical in a specific medium (e.g., air, soil) to construct an intake equation. The equation is used to calculate an intake dose of the chemical contaminant, and the calculation is repeated until an intake dose is calculated for each applicable chemical in the medium.

For the SRA, exposure factors and intake equations were selected from guidance documents that USEPA routinely applies for risk assessments. These guidance documents have undergone both internal and external peer review, and have become the standard sources that USEPA risk assessors employ for developing risk assessments. In many cases, the guidance documents recommend specific default exposure factor values and intake equations that should be applied when evaluating a specific exposure pathway. The following documents comprise the primary USEPA guidance on exposure factors and intake equations:

1) *Exposure Factors Handbook* (USEPA 1997a)

2) *Child-Specific Exposure Factors Handbook* (USEPA 2002a)

3) *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A)* (USEPA 1989)

4) *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part E: Supplemental Guidance for Dermal Risk Assessment)* (USEPA 2004a)

5) *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (USEPA 1998b, 2005c)

Another important concept inherent to the SRA is that it is a prospective or screening-level risk assessment. Namely, it is an evaluation of potential health risk from exposures to contaminants that have not been released and to exposure intakes that have not yet occurred. For contaminant releases that actually occur from the CDF, a wide range or distribution of actual contaminant exposure/intake levels would be possible within the entire population in the vicinity of the CDF. The distribution of exposure will be due mainly to variability in the contaminant concentrations (i.e., contaminant variability by location) and the variability in human exposure factors and behaviors (e.g., variability in body weight, ingestion rate, exposure duration).

An analysis of the full range of exposure distribution in the vicinity of the CDF is beyond the scope of the SRA. Consequently, the SRA adopted a simplified approach to modeling or estimating contaminant exposure, which is realistic and also protective in nature. The approach is the application of the concept that USEPA calls reasonable maximum exposure (RME). The RME is the highest exposure that is reasonably expected to occur under the exposure scenario that applies to a given situation. The concept of the RME was developed within the USEPA Superfund remediation program, where the goal is to protect an individual at the high-end level of exposure, but not at the highest possible level of exposure that could be envisioned (USEPA 2004b). Thus, the RME is intended to represent an exposure level at the high end but within the realistic range of exposure. The high end is usually defined as that part of the exposure

distribution that is at or above the 90th percentile, but below the 99.9th percentile. In practice, the RME estimate for a specific scenario is constructed by setting one or more sensitive exposure factors to their near-maximum values and employing other factors at their known or expected mean (i.e., average) values (USEPA 1992a).

The SRA contains detailed lists and tables of exposure factors and intake equations that were used in this risk assessment; guidance documents from which they were derived; and justifications for factor selection, such as whether a factor is set at a high-end or mean value. In a limited number of situations, a recommended numerical value for a specific exposure factor was not available from published USEPA guidance. For those cases, professional judgment was applied to select a value considered reasonable and appropriately conservative for the exposure scenario in question.

3.5 Estimation of Environmental Media Concentrations and Receptor Exposure Doses

Individuals residing in the vicinity of the CDF facility may be exposed to chemical substances released from the CDF through two primary mechanisms—direct and indirect exposure. The direct exposure pathway occurs through inhalation of vapors and particles that are released to, and remain in, ambient air. Indirect ingestion and dermal exposure pathways occur as a result of dry and wet deposition of particles and vapor onto soil and vegetation, and subsequent migration of these chemicals into other environmental media. For example, chemical constituents released from the CDF that are deposited onto soil can be incorporated into vegetation, which can then contribute to human exposure through ingestion of vegetables grown in home gardens. Additionally, deposition of CDF chemical constituents on local water bodies and runoff from watershed soils will result in concentrations of contaminants in surface water in the vicinity of the facility. Deposition and runoff of chemicals to surface water could result in uptake by fish and contribute to human exposure through ingestion of fish.

In order to assess the risk of exposure to chemical constituents emitted from the CDF, an estimate of the concentrations of chemicals of concern in air, soil, vegetables, surface water, and fish is necessary. The modeling of contaminant transport in air is determined through the air dispersion and transport model (ISCST3) described earlier. For other environmental media, USEPA risk assessments estimate media concentrations using chemical "fate and transport models" designed to simulate the transport of substances in the environment over time. USEPA has issued several guidance documents that cover various aspects of environmental fate and transport processes. The SRA employs the fate and transport modeling presented in the guidance document titled *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (USEPA 2005c) ("HHRAP guidance"). USEPA Region 5 believes that this peer-reviewed guidance provides the current and comprehensive modeling procedures. Although originally developed for evaluating the fate and transport of chemical substances deposited from stack gas and fugitive emissions from combustion facilities, the procedures are applicable to a variety of situations in which chemical constituents could be released to the environment. The HHRAP guidance provides a detailed presentation of the fate and transport models, recommended default input values used in the models, and the basis for selecting default values. Whenever possible, the SRA applied site-specific data rather than relying only on conservative default values. In particular, site-specific parameters describing the hydrologic characteristics of local water bodies were used to model the deposition and transport of chemical contaminants into local water bodies in the vicinity of the CDF.

After the environmental media concentration estimates were completed for all the applicable chemicals of concern, the SRA estimated contaminant doses for receptors in each exposure

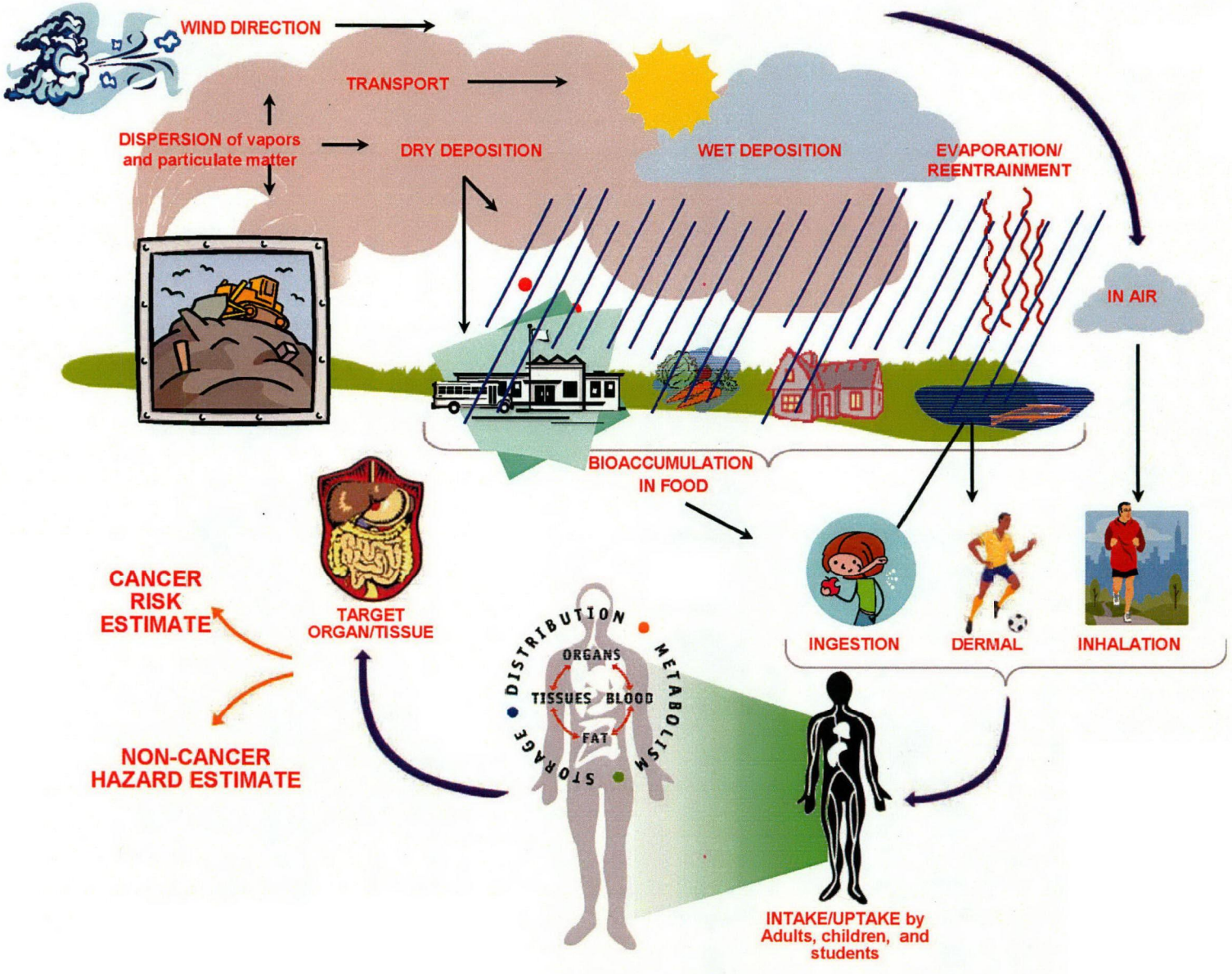
describing the hydrologic characteristics of local water bodies were used to model the deposition and transport of chemical contaminants into local water bodies in the vicinity of the CDF.

After the environmental media concentration estimates were completed for all the applicable chemicals of concern, the SRA estimated contaminant doses for receptors in each exposure scenario and through each applicable exposure pathway. This is the step in the SRA where environmental media concentrations are combined with exposure factors and intake equations.

3.6 Conceptual Models

The following conceptual models represent the SRA study design and methodology.

Figure 3-2: Generic Conceptual Model of How Air Toxics Releases May Result in Injury or Disease



Summary of Figure 3-2

Starting at the upper left hand side of this diagram, air toxics are released from the source (CDF) to the air and begin to disperse by the wind away from the point of release. Once released, the chemicals may remain airborne; convert into a different substance; and/or deposit out of the air onto soils, water, or plants. People may be exposed to air toxics by breathing contaminated air (inhalation) or through ingestion of chemicals that can accumulate in soils, sediments, and foods (the latter process is called bioaccumulation). People also can be exposed to deposited chemicals via skin (dermal) contact; however, this tends to be a less important risk factor than ingestion or inhalation. Inhalation, ingestion, and dermal absorption are called the routes of exposure.

This description of what happens to an air pollutant once it is released into the air is called fate and transport analysis. "Transport" evaluates how a toxic air pollutant physically moves (i.e., is transported) through the environment. "Fate" describes what ultimately happens to the chemical after it is released to the air (i.e., what is the "fate" of the chemical in the environment). The results of a fate and transport analysis is an estimate of the concentration of the air pollutant in the air, soil, water, and/or food at the point where it is contacted by a person. The exposure assessment is the process of evaluating how human contact with the contaminated media occurs.

In the case of an air pathway analysis, the metric representing the inhalation exposure is called the exposure concentration (EC). For example, if benzene is released from the source and all of this blows into a nearby neighborhood where people breathe it, the EC is the concentration of benzene in the air that they breathe.

Once an exposure occurs, air pollutants can enter the body and exert an effect at the point of entry (the "portal of entry") or move via the bloodstream to other target organs or tissues. The action of a pollutant on a target organ can result in no adverse effect or a variety of harmful effects, including cancer, respiratory effects, birth defects, and reproductive and neurological disorders. An overall risk assessment process evaluates what people are exposed to, how the exposure occurs, and, when combined with information about the toxic properties of the chemicals in question, estimates the likelihood that the exposure will result in injury or disease.

Modified from Air Toxics Reference Library, Vol. 3, Community Scale Assessment, Exhibit 3-1, p. 3-2;
http://www.epa.gov/ttn/fera/data/risk/vol_3/Chapter_03_April_2006.pdf

Figure 3-3: Conceptual Model for Local Area Resident

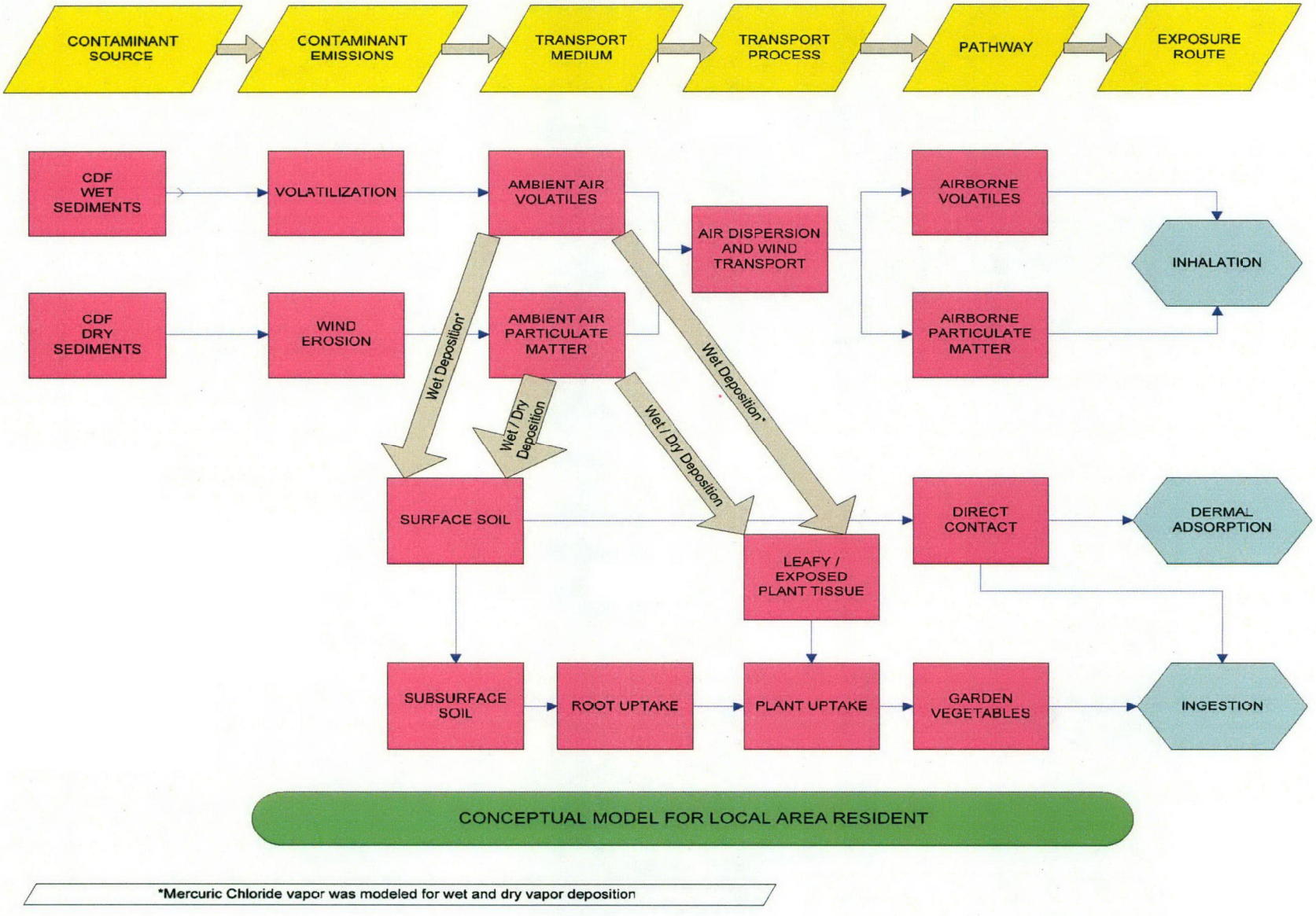
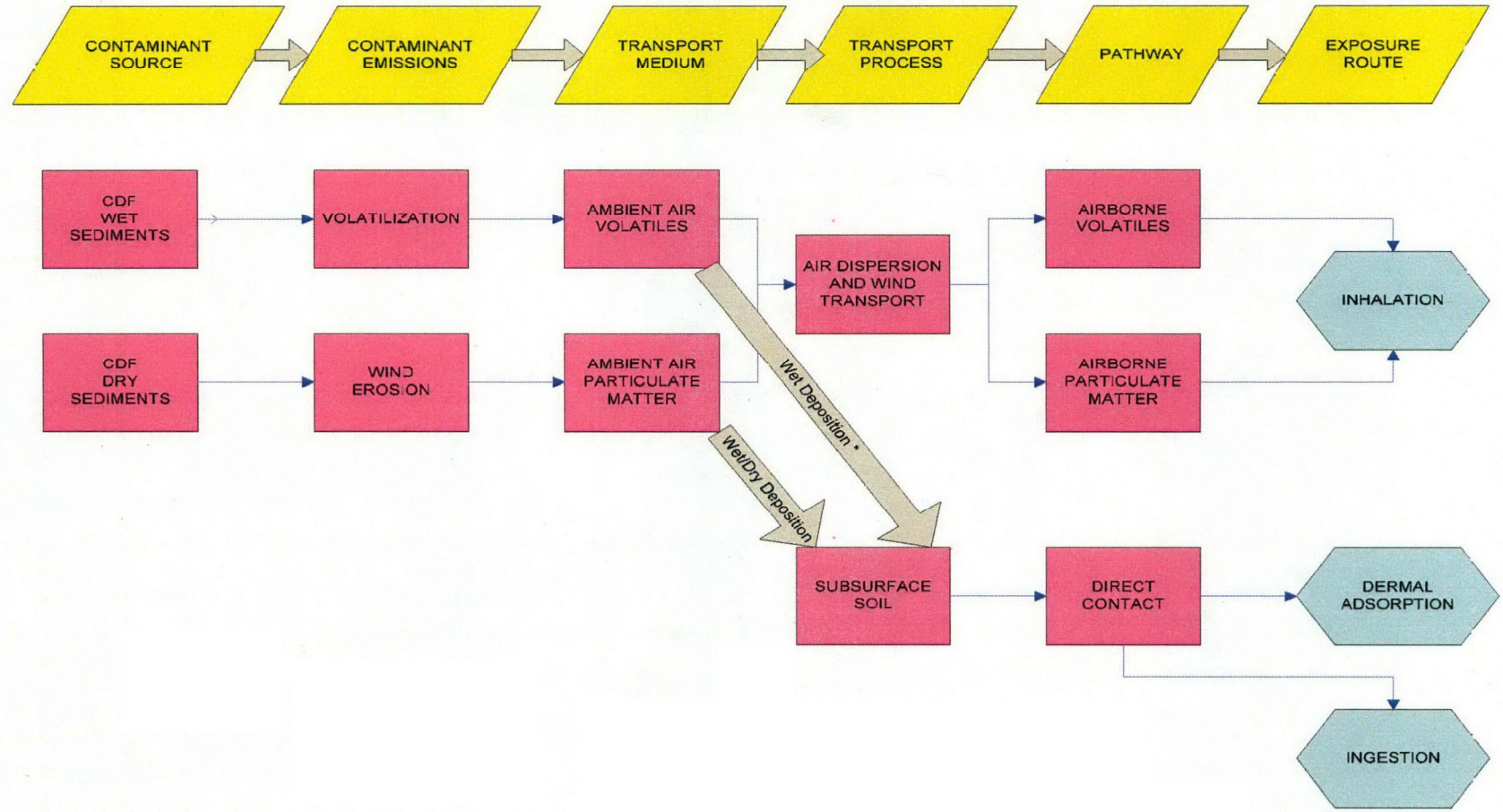
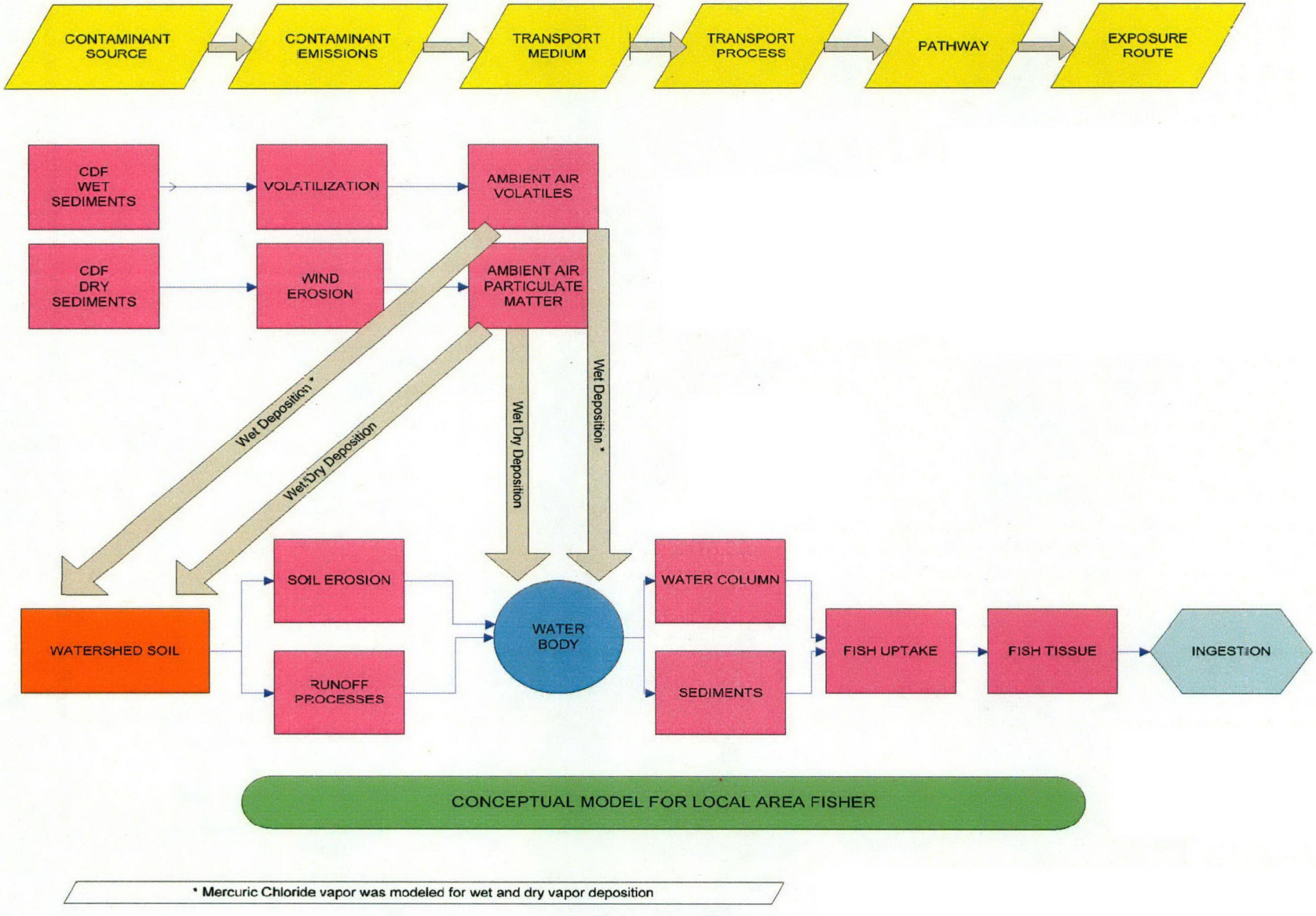


Figure 3-4: Conceptual Model for Local School Student



* Mercuric Chloride vapor was modeled for wet and dry vapor deposition

Figure 3-5: Conceptual Model for Local Area Fisher



3.7 Risk Characterization

The risk characterization integrates information from the preceding risk assessment components. In the risk characterization step of a risk assessment, the chemical toxicity factors are combined with dose estimates for each of the defined exposure scenarios and corresponding exposure pathways to make quantitative estimates of both potential carcinogenic risks and the potential for noncarcinogenic health effects.

Beyond the quantitative aspects of the risk assessment and the tabulation of risk results, the risk characterization is also intended to place the estimated risks in context through a discussion of the qualitative elements of the risk assessment, including a discussion of the major factors influencing the risk estimates, the underlying assumptions, and the rationale for these assumptions. In addition, the risk characterization generally includes a qualitative discussion of the uncertainty and variability associated with the quantitative results.

Because the evaluation of multiple chemicals, multiple exposure pathways, and multiple fate and transport processes is a very challenging computational exercise, the SRA utilized a computer software program to run the contaminant dispersion, exposure and risk modeling. For this project, the software system called Industrial Risk Assessment Protocol - Human Health (IRAP-h View™; abbreviated IRAP) was used. This is a commercial software package developed by Lakes Environmental Software (Waterloo, Ontario, Canada). IRAP was expressly designed to closely follow the recommendations, chemical-specific parameters, and fate and transport algorithms given in the 1998 and 2005 HHRAP guidance. IRAP is a Microsoft Windows application that can be run in the Windows 3.1, Windows 3.11, Windows 95, Windows 98, and Windows NT operating systems.[1]

The major features of the IRAP system that make it advantageous for conducting a risk assessment are its capability to perform the following tasks:

- Recognize and import ISCST3 plot files containing the output from the ISCST3 air dispersion/deposition model runs, and provide a graphical display of the ISCST3 receptor grid node locations

- Recognize and import GIS-generated land use/land cover data (e.g., residential, farming, and water body locations)

- Define the perimeter of important exposure assessment landmark areas (e.g., residential zones, water bodies, watersheds) using a polygon drawing tool, and identify all the ISCST3 receptor grid nodes that fall within a selected polygon

- Eliminate the need to perform hand calculations and write multiple interconnected computational spreadsheets

- Simultaneously calculate intake doses and risk values (i.e., cancer risks and hazard quotients) for multiple chemicals and multiple exposure pathways.

[1] USEPA does not endorse or require the use of the IRAP-h View™ computer software, but recognizes that the developers of IRAP-h View™ made a concerted effort to design a program that would closely follow the recommendations of the 1998 and 2005 HHRAP guidance documents.

Whether a COPC is emitted as a vapor, particle, or particle-bound contaminant is a primary input to dispersion modeling and deposition calculations. For stack emissions, many studies have concluded that stack sampling techniques can be unreliable for determining the partitioning of contaminant emissions among the three phases. Faced with a need for this information, USEPA turned to a growing body of research focused on the partitioning of certain contaminants in ambient aerosols and compared it to proposed partitioning models in literature. The analytical data from this area of study is based primarily on analysis of air samples collected from areas of similar land use (urban versus rural). Ultimately, USEPA recognized that significant uncertainty is still associated with both approaches. Partition coefficients, expressed as a fraction of the airborne contaminant in the vapor phase, have been developed from this research and adopted by the Agency for partitioning stack contaminants. An integral part of this approach is to run the dispersion model at the unit emission rate (1 gram per second [g/s] or 1 gram per second per square meter [$g/s \cdot m^2$]) for all emission phases so that the partitioning can be assigned to the node values after dispersion modeling using these partition coefficients (F_V). Accordingly, IRAP has been developed with this feature integrated in the calculation.

The nature of emissions from the Indiana Harbor CDF is expected to be episodic, such that the different emission phases (vapor, particle, and particle bound) may not occur at similar magnitudes or even simultaneously throughout the project. Thus, we believe it is computationally inaccurate to re-partition the emissions in the same manner as suggested in the guidance for stack emissions modeled with unit emission rates. Further investigation into the theoretical origins of the atmospheric partitioning revealed additional reasons why the F_V coefficients might not be applicable to CDF sediment emissions. The air sampling programs for partitioning estimates were not designed for the evaluation of any specific source of air contamination, but rather to assess overall conditions in areas of similar land use (usually urban). Furthermore, the theoretical basis for the partitioning always refers to an inexchangeable fraction of particle or particle-bound contaminants that are not available for conversion into the vapor phase. Unfortunately, very little research has been conducted into techniques for estimating these phenomena. Sediment contaminants weathered over many decades could very well have a significant amount of inexchangeable contaminant mass that is not accounted for at all in the F_V partitioning values. Moreover, key parameters used for the theoretical development of the partitioning coefficients are representative of particles that are a lot smaller in diameter than much of those expected to be released from the CDF.

Although some amount of Indiana Harbor sediment contaminants probably do partition after release, the current approach in IRAP likely overestimates the partitioning because of the uncertainty regarding inexchangeable contaminant fractions. This disparity between the sizes of particles considered and the independent nature of how the vapors and particles are emitted from the CDF leads us to disable the contaminant partitioning in IRAP in order to obtain the most appropriate results. Accordingly, we defined the F_V value of contaminants primarily emitted as vapors as 1.0 and that of contaminants emitted primarily as particles or particle bound as 0.

Risk characterization also serves as the starting point for evaluating the significance of the risk results. There are a variety of different benchmarks or health risk criteria that USEPA uses when determining the significance of a cancer risk or a noncancer hazard index, depending on the context and purpose of the risk assessment and potential risk management decisions that may need to be made. This section discusses several criteria that may be helpful in interpreting the results of the SRA. The SRA does not specify any single set of health risk criteria that should be regarded as conclusive or absolute for determining the significance of the identified

risks. This is because each set of criteria was developed in a different USEPA statutory or programmatic context that may not exactly match with the regulatory requirements or compliance limits that apply to operation of the CDF.

4 CONTAMINANTS OF POTENTIAL CONCERN AND CONTAMINANT EMISSION RATES

4.1 Contaminant Identification and Contaminant Levels in Buried Sediments

Since the dredging project has not yet been initiated, existing data on contaminant identification and concentration levels in buried sediments were regarded as the logical starting point for predicting the contaminant concentration levels that would be expected in the CDF sediments. Because a number of factors are related to the operation plan for the CDF, the use of data on chemical contaminants in buried sediments should be regarded as an approximation of the contaminant concentrations that will exist when the CDF dredging/disposal is under way. For example, the dredging/disposal operation will actually occur in various stages based on geographic locations, channel geometry, and water depth within the project area. Concentrations of chemical contaminants disposed to the CDF at any given time may vary by the location in the IHSC where dredging occurs and the depth profile of the sediments removed. Consequently, the SRA uses data on chemical contaminants in buried sediment to provide an estimation of reasonable long-term average concentrations of contaminant levels that could be expected within the CDF over the life of the project.

USEPA reviewed the available data record on chemical contaminant identification and concentration levels in IHSC sediments. The data record contains many reports and compilations on chemical analysis of harbor sediments collected over a span of decades based on work by USACE, USEPA, and other organizations. The analytical reports vary in regard to the range of selected chemical contaminants, the geographic location of sampling points within the project area and the type of sample collected. A summary of the data record is presented in Table 4-1. The summary provides general descriptive information on the date of the sampling project, the type of samples, and the identity/class of targeted chemical contaminants.

The goal of the review was to identify the available sediment data reports that were best able to partially or fully meet the following criteria:

1) Data reports on chemical contaminants that would be expected to result from sources such as industrial discharges, combined sewer overflows, and urban runoff.

2) Data reports on chemical contaminants that would serve as potential contaminants of concern (PCOCs) for evaluation in a risk assessment. Examples include known toxic or hazardous chemical contaminants from the following classes: volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), and metals.

3) Data reports that include sampling locations providing comprehensive coverage of the geographic areas of the Harbor and Canal that are targeted for inclusion in the dredging/disposal project area for the CDF.

4) Data reports that include adequate descriptions of sampling locations, sample collection methodology, and quality assurance/quality control (QA/QC) parameters (e.g., analytical method procedures, constituent detection limits).

Based on the review, four data reports were identified that reasonably met at least three of the four criteria set out above.

Table 4-1: Summary of Chemical Constituent Data from Buried Sediments – Indiana Harbor and Canal

Year Collected	Agency or Entity	Sample Number / Type	Chemical Constituents/Groups	Comments on Usability for SRA
1966	USEPA	7 / Surface grab	Organic nitrogen, total nitrogen, phenol, sulfide, iron	Surface samples only, not constituents for SRA, QA/QC unknown or outdated; not useful
1971	Unknown	3 / Surface grab	9 metals, phosphorus	Surface samples only, QA/QC unknown or outdated; not useful
1977	USEPA	13 / Surface grab	11 metals, total PCBs	Surface samples only, QA/QC unknown or outdated; not useful
1979	USACE	13 / Core	11 metals, total PCBs	QA/QC unknown or outdated; not useful
1980	USACE	7 / Surface grab	22 metals	Surface samples only, QA/QC unknown or outdated; not useful
1983	USACE	5 / Core	Total PCBs	QA/QC unknown or outdated; not useful
1984	USACE	6 / Core	11 metals, total PCBs	QA/QC unknown or outdated; not useful
1987	USACE	22 / Surface grab	7 metals, total PCBs	Surface samples only, QA/QC unknown or outdated; not useful
1988	USACE	9 / Core	8 metals	QA/QC unknown or outdated; not useful
1988	USACE	9 / Core	20 metals, PCBs	QA/QC unknown or outdated; not useful
1989	USEPA-ARCS	7 / Surface grab	Metals, PAHs, PCBs, pesticides, dioxins	Surface samples only, known locations, QA/QC is well documented, important constituents for SRA; potentially useful
1990	USEPA-ARCS	37 / Core	7 metals	Samples not collected by water depth measurement, known locations, QA/QC is well documented, sample locations relevant to dredging plan for CDF; potentially useful
1992	USEPA	14 / Core	Metals, PAHs, pesticides, VOCs	Samples collected by water depth measurement, known locations, QA/QC is well documented, important constituents for SRA, sample locations relevant to dredging plan for CDF; potentially useful
1993	USACE	4 / Core	PCBs, metals, PAHs	QA/QC is well documented, most relevant data on PCBs, sample locations relevant to dredging plan for CDF; potentially useful
2003	USACE	5 / Surface grab	Dioxins, pesticides, metals	Composite grab sample from five locations, QA/QC is well documented, sample locations relevant to dredging plan for CDF; potentially useful

ARCS – Assessment and Remediation of Contaminated Sediments
PCB – Polychlorinated biphenyl

1992 USEPA "Sediment Core Study"

In this study, USEPA conducted targeted sampling across the IHSC project area for the purpose of characterizing chemical contaminants in sediments. A sampling methodology was designed through the use of IHSC bathymetry (water depth) maps of the project area. Bathymetry data indicated that the greatest accumulations of sediment within the project area occur along the boundaries of the navigation channel and at the near-shore portions of the outer harbor.

Greater accumulations of sediment occur in the more quiescent areas of the channel and harbor that are the most distant from the turbulence caused by surface water flow, outfalls and ship traffic. In addition, the quiescent areas with the most sediment accumulation will also accumulate the finer-grained sediment material. The finer-grained material will generally contain higher concentrations of chemical contaminants than coarse-grained material for a variety of reasons, including: higher percentage of clay/silt sized materials; a higher fraction of organic carbon to retain organic contaminants; and higher surface area-to-mass ratios (ratios of particle sample surface area to particle sample mass). Consequently, sediments from accumulation zones were assumed to be associated with higher concentrations of chemical contaminants than elsewhere in the IHSC.

Bathymetry maps were used to select 14 sampling locations that represented local maximum levels of sediment accumulation thickness. These samples were well distributed geographically across the project area. At each sample location, a sediment core sample was collected, and a five-foot length of core was homogenized for chemical constituent analysis. The chemical constituent list included chemicals from the following classes: eight metals, 16 PAHs, six pesticides/herbicides, and six volatile hydrocarbons. The sampling methodology was subsequently published (Petrovski 1995). A diagram depicting the approximate sampling locations in the IHSC project area is shown in Figure 4-1.

1993 USACE "Sediment Core Study"

In 1993, USACE performed a small characterization study of sediment samples from a limited geographic zone of the IHSC. In this study, sediment core samples were collected and analyzed from four locations within the "Calumet River Branch" of the project area. (Bathymetry data apparently were not used to select sampling locations). The primary chemical constituent targeted in this sampling was PCBs. Additional target constituents included heavy metals and PAHs. A diagram depicting the approximate sampling locations in the IHSC project area is shown in Figure 4-1.

1989/1990 USEPA ARCS Study

In 1989 and 1990, USEPA conducted two sediment characterization surveys of IHSC sediments under the Agency's ARCS Program (USEPA 1996a). The purpose of the program was to characterize sediment quality and chemical contamination at five areas of concern around the Great Lakes region.

In the 1989 sampling survey, only surface grab samples of sediments were obtained. The samples were collected at seven locations (called Master Stations) distributed geographically over the entire length of the IHSC, including the inner Harbor, the main channel, the Calumet River Branch, and the Lake George Branch. The chemical constituent list included chemicals from the following classes: metals, PAHs, PCBs, pesticides/herbicides, and polychlorinated dioxins/furan congeners. A diagram depicting the approximate sampling survey locations in the IHSC project area is shown in Figure 4-1.

In the 1990 sampling survey, sediment core samples of approximately 14 feet in length were collected at 37 locations that were distributed over the entire length of the IHSC. The sample locations were well distributed geographically but did not represent areas associated with known sediment accumulation, as in the 1992 bathymetry study. The sediment cores were each divided into four depth segments on which chemical analysis was performed. However, seven

metals (i.e., cadmium, chromium, copper, iron, lead, nickel, and zinc) were the only constituents analyzed in these sediment core samples; no organic constituents were analyzed.

2003 USACE "Surface Sediment Samples"

In 2003, USACE performed a small characterization study of sediment samples from a limited geographic zone of the IHSC. In this study, sediment surface grab samples were collected from five locations in the project area and homogenized into a single sample for analysis. The primary chemical constituents targeted in this sampling were polychlorinated dioxins/furan congeners. Additional target constituents included heavy metals, pesticides, and PAHs.

4.2 Sediment Contaminant Identification and Contaminant Levels in the CDF

The sediment data reports described above contain information on sample collection and chemical constituent identity that were determined to meet a set of basic criteria for potential use in the SRA project. However, these data reports exhibit variation in their capacity to appropriately represent the distribution of contaminant levels geographically across the project area and by the depth of sediments to be dredged.

PCOCs selected for the SRA are chemicals that display the following criteria: (1) expected byproducts of industrial/commercial/urban discharges to waterways; (2) potentially toxic or hazardous to humans; and (3) persistent in the environment, and therefore a concern for long-term exposure and for uptake or bioaccumulation in the food chain.

Consequently, a rationale was developed to prioritize and select chemical constituent data from the above reports to serve as an appropriate representation of chemical constituent identity and concentration levels in the sediments after disposal of buried sediments in the CDF. The following rationale was adopted:

1) The 1992 USEPA "Sediment Core Study" provides the most appropriate data set on buried sediments to use for representing the long-term sediment profile in the CDF after disposal. The data from this study on the following classes of chemical constituents should be used to represent the identity and concentration levels of constituents in the CDF: metals, PAHs, pesticides/herbicides, and volatile hydrocarbons.

2) The 1993 USACE "Sediment Core Study" and the 2003 USACE "Surface Sediment Samples" provide the most appropriate source for data on PCBs in buried sediments, and seven additional pesticides/herbicides not available from rationale number 1 above.

3) The detection of dioxins (i.e., polychlorinated dioxins/furan congeners) in sediments is considered potentially significant for the SRA, even though they were verified only as a surface sediment contaminant. The 1989 USEPA ARCS Study and the 2003 COE "Surface Sediment Samples" of surface sediments should be utilized for this constituent.

The list of constituents and summary data on sediment concentration levels in buried sediments is shown in Table 4-2. This table presents the PCOCs for the SRA, which will be discussed further in Section 6.

Table 4-2: Identification and Summary of Analytical Data on PCOCs in Buried Sediments

Constituents	Data Set [a]	Frequency of Detection (# Detects/ #Samples)	Arithmetic Mean (mg/kg)	Standard Deviation	Comments
Metals					
Antimony	1993 CORE	18/18	23.5	8.4	
Arsenic	1992 CORE	16/16	75.4	40.1	
Barium	1992 CORE	14/16[b]	159	75.6	
Cadmium	1992 CORE	16/16	13.6	5.39	
Chromium (total)	1992 CORE	16/16	705	369	
Copper	1993 CORE	18/18	336	81.3	
Lead	1992 CORE	16/16	1022	470	
Manganese	1993 CORE	18/18	3374	939	
Mercury					
Mercury (Total)	1992 CORE	16/16	1.06	0.70	
Methylmercury	1989 SURFACE	3/7[b]	6.0E-04	7.8E-04	
Nickel	1993 CORE	18/18	165	102	
Selenium	1992 CORE	16/16	3.7	1.2	
Silver	1992 CORE	0/16[c]	< 6.5	[2.7]	Not detected in any sample
Zinc	1993 CORE	18/18	6973	2021	
PAHs					
Acenaphthene	1992 CORE	10/16[b]	21.6	36.0	
Acenaphthylene	1992 CORE	5/16[b]	54.9	158	
Anthracene	1992 CORE	10/16[b]	35.0	77.5	
Benzo[a]anthracene	1992 CORE	16/16	44.1	65.7	
Benzo[a]pyrene	1992 CORE	16/16	35.3	50.6	
Benzo[b]fluoranthene	1992 CORE	15/16[b]	35.4	52.9	
Benzo[k]fluoranthene	1992 CORE	15/16[b]	18.5	24.1	
Benzo[g,h,i]perylene	1992 CORE	6/16[b]	25.3	48.5	
Chrysene	1992 CORE	16/16	60.7	102	
Dibenz[a,h]anthracene	1992 CORE	0/16[c]	10.6	[23.1]	Not detected in any sample
Fluoranthene	1992 CORE	16/16	88.1	160	
Fluorene	1992 CORE	13/16[b]	42.7	94.5	
Indeno[1,2,3-cd]pyrene	1992 CORE	1/16[b]	94.6	228	
Naphthalene	1992 CORE	16/16	478	1467	
Phenanthrene	1992 CORE	16/16	171	346	
Pyrene	1992 CORE	16/16	93.4	145	
PCBs (total)	1993 CORE	16/16	35.6	25.3	
Dioxin/Furan Congeners[d]					
2,3,7,8-TetraCDD	1989/2003 SURFACE	1/8[b]	4.7E-05	4.0E-05	
1,2,3,7,8-PentaCDD	1989/2003 SURFACE	4/8[b]	4.8E-05	1.9E-05	
1,2,3,4,7,8-HexaCDD	1989/2003 SURFACE	7/8[b]	8.6E-05	1.3E-04	

Constituents	Data Set [a]	Frequency of Detection (# Detects/ #Samples)	Arithmetic Mean (mg/kg)	Standard Deviation	Comments
1,2,3,6,7,8-HexaCDD	1989/2003 SURFACE	8/8	1.4E-04	1.7E-04	
1,2,3,7,8,9-HexaCDD	1989/2003 SURFACE	8/8	2.0E-04	1.9E-04	
1,2,3,4,6,7,8-HeptaCDD	1989/2003 SURFACE	8/8	3.1E-03	3.6E-03	
OctaCDD	1989/2003 SURFACE	8/8	1.7E-02	1.8E-02	
2,3,7,8-TetraCDF	1989/2003 SURFACE	8/8	2.7E-04	2.4E-04	
1,2,3,7,8-PentaCDF	1989/2003 SURFACE	8/8	2.4E-05	1.6E-05	
2,3,4,7,8-PentaCDF	1989/2003 SURFACE	8/8	5.7E-05	4.5E-05	
1,2,3,4,7,8-HexaCDF	1989/2003 SURFACE	8/8	7.8E-05	7.3E-05	
1,2,3,6,7,8-HexaCDF	1989/2003 SURFACE	6/8[b]	4.5E-05	3.4E-05	
1,2,3,7,8,9-HexaCDF	1989/2003 SURFACE	8/8	2.8E-05	2.1E-05	
2,3,4,6,7,8-HexaCDF	1989/2003 SURFACE	3/8[b]	1.9E-05	2.7E-05	
1,2,3,4,6,7,8-HeptaCDF	1989/2003 SURFACE	7/8[b]	8.2E-04	1.1E-03	
1,2,3,4,7,8,9-HeptaCDF	1989/2003 SURFACE	7/8[b]	2.0E-04	3.0E-04	
OctaCDF	1989/2003 SURFACE	8/8	5.1E-03	6.4E-03	
Pesticides and Phenols					
Aldrin	1993 CORE	1/1	0.045	NA	Reported detection is from a single sample; therefore, this is only value that can be used for SRA
d-BHC	1993 CORE	2/2	0.004	0.004	Apparently this constituent is "delta-lindane", one of several isomers of lindane
Dieldrin	1993 CORE	16/16	0.019	0.006	
DDD (dichlorodiphenyl-dichloroethane)	1993 CORE	18/18	0.103	0.073	
DDE (dichlorodiphenyl dichloroethylene)	1993 CORE	16/16	0.038	0.013	
DDT (dichlorodiphenyl-trichloroethane)	1989/2003 SURFACE	4/8 [b]	0.068	0.031	
Endosulfan II	1993 CORE	2/2	0.038	0.004	Isomer of endosulfan; could be evaluated in SRA using the chemical-physical

Constituents	Data Set [a]	Frequency of Detection (# Detects/ #Samples)	Arithmetic Mean (mg/kg)	Standard Deviation	Comments
					parameters and toxicity factors for endosulfan
Endrin	1992 CORE	0/16[c]	< 0.05	NA	Not detected in any sample
Heptachlor	1992 CORE	0/16[c]	< 0.05	NA	Not detected in any sample
Heptachlor Epoxide	1992 CORE	0/16[c]	< 0.05	NA	Not detected in any sample
Lindane	1992 CORE	0/16[c]	< 0.25	NA	Not detected in any sample
Phenol	1992 CORE	0/16[c]	< 28.1	[61.7]	Not detected in any sample
Toxaphene	1992 CORE	0/16[c]	< 0.5	NA	Not detected in any sample
VOCs					
Benzene	1992 CORE	16/16	3.09	7.11	
Ethylbenzene	1992 CORE	7/16[b]	0.74	0.96	
Tetrachloroethene (PCE)	1992 CORE	0/16[c]	< 0.17	[0.04]	Not detected in any sample
Toluene	1992 CORE	9/16[b]	4.95	14.1	
Trichloroethene (TCE)	1992 CORE	0/16[c]	< 0.25	[0.06]	Not detected in any sample
Xylene (meta-para)	1992 CORE	9/16[b]	6.65	15.4	
Xylene (ortho)	1992 CORE	10/16[b]	2.18	4.39	

Footnotes:

[a] 1993 CORE - Data from sediment core samples collected from 4 locations by the USACE in November 1993.
1992 CORE - Data from sediment core samples collected from 16 locations by USEPA in June 1992.
1989 SURFACE - Data from sediment surface grab samples collected by USEPA in 1989.
1989/2003 SURFACE - Combined data from sediment surface grab samples collected by USEPA and USACE in 1989 and 2003.

[b] If the constituent was detected in at least one sample, the non-detects were included in the calculation at the full reported detection limit.

[c] If the constituent was not detected in any sample, the non-detects were included in the calculation at one-half of the reported detection limit.

[d] CDD - Chlorinated dibenzo-para-dioxin
CDF - Chlorinated dibenzofuran

NA - Not applicable because the constituent was not detected in any sample and the detection limit was identical for each sample.

[ ] - Apparent Standard Deviation calculated when the constituent was a "non-detect" in every sample and the detection limits were not identical for each sample.

4.3 CDF Operating Parameters Needed as Modeling Inputs for the SRA

According to USACE, the CDF will comprise two sediment dewatering and containment cells and an equalization basin to facilitate dewatering and water treatment (Thai 2005a, 2005b). Dredged sediments will arrive at the CDF by barge and be slurried into one of the two disposal cells, which will each be as small as 36 acres when empty and 47 acres in size when full (due to

the sloping design of the cell walls). The cells were each modeled as 41-acre emission sources for the SRA. The CDF cell walls will be constructed and operated in two phases. During the first phase, the cell walls will have a constructed height of 20 feet above grade (approximately 6 meters), which will be raised to 30 feet (approximately 9 meters) for the second phase of dredging. The 6-meter release height was used for SRA emission modeling.

The cells will be operated on a two-year cycle, receiving sediments every other year. Figure 4-2 presents a schematic of the two-year operating cycle of the CDF. According to USACE, a cell would receive dredged sediments starting in May of a given year and it would remain or be kept wet through August 1 of the following year, at which point it would become dry enough to emit particles. Particle emissions would then be possible until May 1 of the third year when active placement of dredged sediment resumes in that cell. Volatile emissions were assumed to occur primarily during the saturated or wet periods, when the cell is receiving dredged sediment as a slurry and when sediments dewater and dry.

Note that the period selected for Figure 4-2 is from the middle of the CDF operating cycle, such that there would be sediments and potential emissions from both cells. This is also the way the cells were modeled for air dispersion, except that the five years of dispersion modeling began at the start of the calendar year.

4.4 Chemical Contaminant Emission Rates as Modeling Inputs for the SRA

During and after sediment placement in the CDF, the release of chemical contaminants can be described by essentially two mechanisms: release of volatile contaminants into ambient air as vapor phase contaminants, and release of low volatility and non-volatile contaminants into ambient air as particles (i.e., "particulate matter"). Contaminants could be released as particulate matter because the contaminants themselves are expected to be particles (e.g., metals), or because the contaminants are low volatility organic chemicals (e.g., PAHs, pesticides, PCBs) that will primarily be adsorbed onto the surface of particulate matter.

To proceed with the SRA, release rates of contaminants must be defined as inputs for the air dispersion model. Because the dredging/disposal operation for the CDF has not yet started, release rates of volatile and particulate contaminants were assigned based on a modeling procedure combined with applicable information regarding state regulatory emission limits, the CDF operating parameters and climatic conditions.

4.4.1 Volatile Emissions from Submerged Sediments and Drying Sediments

The current operating scenario calls for the dredged sediments to be placed in the CDF in the form of a slurry. The sediments will likely be submerged in water during periods of active dredging and exposed as wet sediments during inactive periods. Mechanisms that govern volatile emissions include desorption of contaminants from sediment to water and air, diffusion of dissolved contaminants through sediment pores and free water, diffusion of contaminant vapor through sediment pores, and mass transfer through evaporation at the air/water interface.

The mechanistic and quantitative aspects of the available volatilization model for sediments have been described in detail by Dr. L.J. Thibodeaux and co-workers (Thibodeaux et al. 2004, Thibodeaux 2005). After a review of the available models (including discussions with

developers of published models[2]), it was determined that application of the full volatilization model to operation of the CDF would be exceedingly complex. The mechanisms for volatilization described above are dependent on time, weather, chemicals, and operations. These consequences create significant difficulty in defining a reasonable set of conditions for estimating volatilization rates of Indiana Harbor sediment constituents. USEPA did not possess a ready means to simultaneously evaluate all of these variables in a way that would allow detailed volatile emission estimates. Furthermore, while the application of these theoretical mechanisms to actual pilot laboratory observations of sediment emissions was successful in predicting volatilization trends, the predicted volatile chemical emission rates did not match the actual laboratory measured rates (Thibodeaux et al. 2004, Thibodeaux 2005). Due to these challenges, complexities and uncertainties, a basic understanding of these models was combined with a regulatory compliance limit of 25 tons per year (TPY) in preparing the volatile emissions estimate for the SRA (discussed further below).

4.4.2 Particulate Emissions from Exposed Sediments

Particulate emissions from the surface of the CDF can be regarded as a variant of the typical process for wind erosion of soils. Soil erosion by wind is initiated when the local wind exceeds a "threshold" speed that allows soil particles to become airborne and be carried to a new location. The properties of the soil (Indiana Harbor sediment in this case), local weather, and operational factors could all influence what the "threshold wind speed" will be for the CDF. After initiation, the severity of an erosion event depends primarily on the wind speed duration and magnitude. The threshold wind speed, severity, and duration of an erosion event can be altered by modifying the roughness of the terrain, deploying different types of vegetative cover, and other forms of mitigation to reduce or eliminate windblown particle emissions.

From a historical standpoint, the evaluation of soil erosion has been considered to be a major challenge in the context of food crop production, land management, and soil conservation. The U.S. Department of Agriculture - Agricultural Research Service (USDA-ARS) has been involved in direct measurement and modeling studies of the soil erosion process for many years. USDA-ARS developed an empirical model called the Wind Erosion Equation to predict soil erosion in simple situations, such as a single soil type and a fixed wind speed and direction (Woodruff and Siddoway 1965). To supplement the empirical model and to address the fact that soil erosion is a more complex non-linear process over space and time, USDA-ARS developed a more robust model known as the Wind Erosion Prediction System (WEPS) (Hagen et al. 1995). WEPS is a continuous, daily time-step computer model that simulates the basic wind erosion process and also accounts for parameters that modify a soil's susceptibility to wind erosion such as vegetation type, surface roughness, surface crust, and soil water content. In addition, WEPS has the ability to incorporate important site-specific information such as the basic size/shape of the erodible source area, local meteorological data (i.e., precipitation rate, wind speed, wind direction, humidity, solar radiation), and the addition of mitigation controls (e.g., vegetative cover, hedgerows, fences).

[2] The developers of this SRA, in cooperation with USACE, held a number of discussions with Dr. Louis Thibodeaux and co-workers at Louisiana State University on the application of models to simulate the volatilization of chemical contaminants from sediments. For example, in March 2005, Dr. Thibodeaux led a two-day workshop in Chicago at which a detailed analysis of the mechanistic and quantitative aspects of the volatilization model for sediments was provided. In addition, the workshop explored the problems, complexities, and uncertainties expected if the complete model were to be applied to the SRA project. (Dr. Thibodeaux and co-workers provide consultation on the IHSC dredging-disposal project through a contract issued by the USACE).

After a review of the available models for soil erosion (including discussions with developers of the WEPS Model), it was determined that application of the WEPS Model for soil erosion would be valid for simulating sediment erosion within the CDF. USEPA began by reviewing annual particle emissions as estimated by USDA-ARS for USACE as the basis for the emissions estimate for dispersion modeling (Hagen 2005a). Dr. Lawrence Hagen of ARS recommended against using a constant particle emission rate derived from the annual average because windblown erosion events at the CDF were expected to be limited to only a few days per year. If the wind direction during those few days is different than the average wind direction over time, the location of impacted receptors based on dispersion modeling using a constant emission rate will be incorrect.

The WEPS Model was modified to use hourly meteorological data to generate hourly particle emission estimates. In order to increase confidence in the proper use of WEPS, representatives of USDA-ARS collaborated with USEPA on the application of WEPS to the SRA project, including defining the project area, the sediment surface characteristics, and the incorporation of the site-specific meteorological data. USEPA tested whether the additional sophistication of an hourly variable particle emission was necessary by comparing particle concentration and deposition results from dispersion modeling trials using constant emission rate inputs and preliminary variable rates from USDA-ARS. The constant year-long emission rate was scaled so that total annual emissions equaled the losses predicted by the hourly variable modeling from USDA. Air concentrations and deposition rates from the constant emission flux were subtracted at each receptor node from the air concentrations and deposition rates from the hourly variable emission estimate from USDA-ARS. These differences between modeling results are graphed in Figures 4-3 and 4-4. The pink areas show where the air concentration or deposition values would be underestimated by annualizing the particle emission and the green areas show where they would be overestimated. Thus, ignoring the episodic nature of the particle emissions as predicted by the modified WEPS would have significantly underestimated impacts south of the CDF and overestimated them elsewhere. Strong winds combined with favorable soil conditions for erosion were usually associated with winds from the north. Due to the advanced development of the modified WEPS model and the fact that some of the most critical receptor locations (i.e., schools and residences) are located directly south of the CDF, USEPA decided to incorporate the modified WEPS predicted variable emissions to the greatest extent possible.[3]

Five years of local meteorological data, including hourly records of wind speed/wind direction and other significant weather data, were compiled and processed as a computer input file to the WEPS Model to determine which hours possessed the required combination of wind speed and surface conditions to cause the release of particulate matter from the CDF. USDA-ARS provided USEPA with hourly particle emission estimates for the five-year period using a worst case emission scenario for the CDF (Hagen 2004a, 2004b). To generate a conservative particle emission estimate, the following conditions were used:

[3] The developers of the SRA collaborated with USDA-ARS to apply the WEPS Model to simulate erosion and particulate matter release from sediment disposal in the CDF. The developers of the WEPS Model (Dr. Lawrence Hagen and co-workers of USDA-ARS, Manhattan, KS) were provided with site-specific information on the CDF (e.g., geographic location, area dimensions, local meteorological data) that was used to run the WEPS Model. USDA-ARS developed a written report that summarizes the application of the WEPS Model to the IHSC CDF project (Appendix 4-1). (USDA-ARS provides consultation on the IHSC dredging-disposal project through a contract issued by USACE.)

1) A single large containment cell comprising the entirety of the CDF was assumed to be filled to capacity (minimizing the height of the potentially wind-blocking cell wall above the sediment surface)
2) Sediments were assumed to be placed hydraulically to dry in a smooth, flat configuration (the worst case for windblown emissions)
3) The single large cell was modeled for WEPS in its second year of drying, after a season of freeze/thaw cycles had already served to break up the dried sediment to enhance erodibility.

Particle emissions were not estimated from the equalization basin, barges, or dredging areas because these are wet operations. The resulting hourly particle emissions estimate was placed into a large text file with lines of data corresponding to each hour of the five-year meteorological data used for the dispersion model (see Appendix 4-5). The file was prepared in the format required by the dispersion model for the hourly variable emission option.

4.4.3 Volatile and Particulate Emission Rates Based on Regulatory Limits

In the course of developing the SRA, it was determined that the CDF operation would be subject to specific regulatory compliance limits on allowable emissions from the CDF. The compliance limits are spelled out in a June 2002 New Source Registration issued by the Indiana Department of Environmental Management (IDEM) to the East Chicago Waterway Management District (Appendix 4-2).[4] The basic annual emission limits of the Registration may be summarized as follows: total volatile organic compounds and total particulate matter during construction and operation of the CDF may each not exceed 25 tons; a single hazardous air pollutant (HAP) may not exceed 10 tons; and combined HAPs may not exceed 25 tons. The Registration also outlines requirements for on-site sediment analysis and air monitoring to provide demonstrations that the emissions limits would not be exceeded during the actual operation of the CDF.[5]

The application of regulatory-based emission compliance limits offered a reasonable and practical approach for setting emission rate inputs for the SRA. This approach uses essentially maximized emission rates allowed by regulation in lieu of complex, theoretical and time-dependent emission models. The use of compliance limits as inputs for the SRA also allows the results of SRA to serve as a check on the range of potential health risks associated with operation of the CDF at the compliance limits. This should be important information for the operators of the CDF and the public.

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[4] The regulatory authority for requiring a New Source Registration is found in the Indiana Administrative Code (IAC) at 326 IAC 2-5.1-2; this citation stands for Title 326: "Air Pollution Control Board"; Article 2: "Permit Review Rules"; Rule 5.1: "Construction of New Sources"; Part 2: "Registrations."

[5] USEPA interpreted the emission limits in the Registration to apply to the entire operation of the CDF, including the dredging and transport of project sediments to the CDF. Consequently, a separate analysis of contaminant emissions from the dredging/transport operation was not performed for the SRA. This is considered justified for the following additional reasons: 1) The time required for dredging and transport is a small fraction of the total time that sediments will spend in the CDF where volatile emissions could occur over several months during any dredging season; 2) the surface area of the transport barge (< 0.5 acre) is minimal compared to the total surface area from which volatile contaminants could be released in the CDF (82 acres); and 3) particulate matter release during dredging/transport is predicted to be negligible because sediments will remain saturated with water and no dry sediments will be generated that could be subjected to wind erosion.

The regulatory compliance limit approach to the SRA was proposed to USACE in May of 2005 (USEPA 2005e; Appendix 4-3) and incorporated into the CDF emissions estimate as described in the following Sections.

4.4.3.1 Regulatory Limit-Based Volatile Emissions

Since a mixture of volatile contaminants is found in the sediment, a mixture of volatile contaminants would likely be emitted after placement in the CDF. The SRA uses naphthalene as a surrogate volatile organic pollutant to represent all volatile PCOCs. The toxicity characteristics of naphthalene represent the highest Cancer Slope Factor (CSF) and lowest reference concentration compared to all other volatile PCOCs selected for this study. This approach presumes that all of the allowable 25 TPY of volatile emissions from the CDF would be composed of naphthalene. Although the compliance limit does not allow an emission of greater than 10 TPY (TPY) of a single HAP, for analysis purposes we took this conservative approach and assessed the risk of a single HAP (naphthalene) at the higher limit of 25 TPY for combined volatile HAPs. This is a simpler method to implement than the multiple chemical, full volatilization emission model, and is conservative. In reality, the 25 TPY volatile maximum will include other less toxic compounds, thereby the actual risk and hazard levels will be below the estimated naphthalene level. The annual 25 tons of naphthalene emissions were uniformly apportioned throughout the CDF operating cycle based on operating parameters provided by USACE, basic knowledge derived from the extensive modeling efforts of Thibodeaux et al., and the available climatological information.

The volatile organic emissions estimate of 25 TPY is based on the knowledge that volatile emissions are likely to be continuous, at least when the sediment is wet. Thus the 25 TPY was divided between the two cells based on the wet or saturated periods of the cell cycle and did not include winter months when declining temperatures, a frozen surface, and/or snow cover could significantly inhibit volatilization. The result is a period between May 1 and July 31 when both cells are sources of volatile emissions coinciding with the active dredging season. One cell actively receives dredged sediments as a slurry while the other cell, containing the previous year's dredged sediment, continues to dry. After July 31, the inactive cell is assumed to be too dry to continue emitting significant volatiles, while the cell with the fresh sediment continues to emit volatiles as it begins to dewater. This continues until November 30 when the surface of the sediment is assumed to be effectively frozen over. This assumption is consistent with the latest first soil freeze reported as December 2 for the top 2.5 centimeters of soil in West Lafayette, Indiana (approximately 85 miles south of East Chicago) measured between 1965 and 1980 (Dale et al. 1981).

The 25-ton annual volatile emission limit was divided into these active periods and a constant emission rate flux (emissions per unit area) was calculated uniformly for each cell. Thus the site-wide hourly volatile emissions are twice as high during the active dredging period from May 1 through July 30 as they are during the single dewatering cell period from August 1 through November 30. Volatile emissions from the dredge and barges were considered as part of the 25 TPY limited emission from the CDF. USEPA decided to prepare the volatile emission as an hourly variable emission file for the dispersion model, even though the volatile flux rate did not vary from hour to hour when a cell was assumed to be emitting volatiles. Rather, preparing hourly emission files was deemed the most appropriate way to incorporate the cyclic nature assumed to govern volatile emissions as well as the alternating year operation of each cell. Hourly volatile emission rates for the dispersion modeling period were developed as a text file for each cell and can be found in Appendix 5-2 of this report.

4.4.3.2 Regulatory Limit-Based Particulate Emissions

The regulatory limit approach sets the total allowable loss of particulate matter from the CDF due to wind erosion to 25 TPY.[6] USEPA modified the original hourly variable conservative particle emission estimate from USDA-ARS in order to evaluate the impact of particle emissions at the 25 TPY limit. Based on recommendations from USDA-ARS and USACE, the particle emission estimate was restricted to August 1 of a given year through May 1 of the following year (since the cells will be wet or kept wet during the dredging season) and individual hourly emissions from the modified WEPS model were scaled proportionally such that the annual total for each year studied was exactly 25 tons. USACE estimated that a cell operated within a two-year cycle would be dry enough to emit particles approximately one year after sediments had last been placed in the cell (late summer of the second year). Particle emissions would terminate the following spring (the third year) when new wet sediments were placed in the cell.

One of the findings of the initial conservative particle emissions estimate from WEPS was that the 25 TPY limit may be exceeded. USACE subsequently tasked USDA-ARS to recommend particle emission mitigation techniques and to use WEPS to evaluate their potential effectiveness. USDA-ARS ultimately provided updated hourly particle emission data, applying the revised two-cell operation scheme as well as various emission mitigation techniques (Hagen 2005b). The newly estimated particle emissions were generally lower, reflective of the mitigation techniques added to the simulation. The average annual emission for the five-year period with mitigation techniques was 11 TPY, with one year exceeding the 25-ton annual limit. Since USEPA does not presently know how USACE will actually modify operations to prevent exceeding the annual emission limit, USEPA decided to continue using the modified particle emission data for the SRA wherein the annual emissions were proportionally set equal to the annual emission limit of 25 tons. This approach is more conservative and is in keeping with the intention to evaluate exposure and risks at the compliance limit. Hourly particle emission rates for the dispersion modeling period were developed as a text file for each cell with a line for each hour's particle emission flux corresponding to each hour of the five-years of meteorological data. The hourly particle emission file can be found in Appendix 5-1of this report.

The particle emission estimate coupled with the subsequent air dispersion modeling is used to predict the impact of bulk particle emissions within the air model receptor grid. The impact of specific Indiana Harbor chemical contaminants that migrate as part of the particle emission will be taken into account in the SRA based on their chemical-specific concentrations in the bulk CDF sediments.

4.5 Mercury Emissions

While the concentration of mercury in the sediments is relatively low in comparison to other Indiana Harbor sediment contaminants, mercury has a high potential to bioaccumulate in the environment and the type of mercury present (i.e., elemental, divalent, or methylated) has a large impact on the fate and transport of mercury from the CDF to potential receptors. Due to the Agency's ongoing focus on the speciation, fate, and transport of mercury from a variety of

[6] Particulate matter derived from sediments should be composed predominantly of naturally occurring or inert materials (e.g., sand, clays, organic detritus) with relatively small concentrations of chemical contaminants adsorbed to the particulates. Contaminants on airborne particulate matter could be available for direct inhalation exposure or for indirect exposure after deposition to soil. Therefore, it was not appropriate to model the allowable 25 tons of particulate matter as a single "most toxic" contaminant for cancer risk and noncancer hazard as was done for the volatile emission modeling. The concentrations of chemical contaminants on airborne particles and deposited particles were assigned based on the data reports on chemical contaminant concentrations in the project sediments.

sources, we have the ability to evaluate different chemical species of mercury (this level of detail is generally not available for other contaminants). However, actual speciation data for mercury in Indiana Harbor sediments is minimal.

In order to evaluate possible risks from the different possible species of mercury, a number of scenarios were run with different mercury species in a manner that likely exceeded the mass balance of mercury present while maximizing the pathway specific health threats of mercury. For example, the greatest risk from a vapor emission of elemental mercury is from direct inhalation. To evaluate direct inhalation, elemental mercury vapor was emitted based on all available mercury in the sediment dredged within a single year. However, other mercury species were evaluated simultaneously (with different pathway outcomes) so that the amount of mercury emitted was greater than the amount of mercury actually available.

Table 4-3 summarizes the modeling approach for the different species of mercury possible from the CDF. Elemental mercury was run as a vapor emission by taking the vapor emission scaled to 25 tons (originally run for naphthalene) and using the emission rate entry in the risk assessment program to scale the emission to the total tons of mercury present in a heavy dredging year. At a maximum dredge removal rate of 240,000 cubic yards per year of wet in-situ sediment, a dry sediment in wet in-situ volume density of 0.67 tons per cubic yard, and an average concentration of 1.1 mg/kg mercury in Indiana Harbor sediments, USEPA estimates that 0.18 tons of mercury could be placed in the CDF annually. Assuming that all 0.18 tons emit from the CDF each year, the 25-ton vapor emission must be adjusted by a factor of 0.007 (the ratio of 25 tons to 0.18 tons).

Elemental, divalent (as mercuric chloride), and methylmercury potentially emitted as particle contaminants were run by applying the average concentration of these species to the results of the 25-ton erodible particle emission. Actual data were available for average sediment concentrations of elemental and methylmercury. The concentration of mercuric chloride was obtained by adjusting the average sediment concentration of elemental mercury to include the molecular weight of two additional chlorine atoms.

Mercuric chloride potentially emitted as a vapor was run by taking the maximum possible amount of mercuric chloride present in a heavy dredging year and using the 1993 Air/Superfund National Technical Guidance Study Series – Models for Estimating Air Emission Rates from Superfund Remedial Actions (USEPA 1993a) to estimate a volatile flux from the ponded CDF (modeled as a lagoon). This flux was used for all periods of volatile flux for mercuric chloride. Please see Appendix 4-9 for a detailed description of the flux calculation.

Table 4-3: Modeling Approach for Mercury Species

Mercury Species	COPC Name	Vapor or Particle Phase	Emission - Dispersion Model Run Used	Annual Mass of Species Emitted	Source for Emission Rate	IRAP Options	
						Emission Rate Scalar	F_V
Hg^O (elemental)	mercury (v)	vapor	25-ton vapor emission with wet gas deposition	0.18 tons	mass balance	0.007	1
Hg^O (elemental)	mercury	particle	25-ton bulk erodible sediment particle emission	25 grams	average concentration in sediment	1.1×10^{-6} (1.1 mg/kg)	0
$HgCl_2$ (divalent)	mercuric chloride	vapor	unit vapor emission with wet and dry gas deposition	89 grams	estimated flux from lagoon water	2.04×10^{-11}	1
$HgCl_2$ (divalent)	mercuric chloride (p)	particle	25-ton bulk erodible sediment particle emission	34 grams	estimated average concentration in sediment	1.49×10^{-6} (1.49 mg/kg)	0
CH_3Hg^+	methyl-mercury (b)	particle	25-ton bulk erodible sediment particle emission	0.0136 grams	average concentration in sediment	6.0×10^{-10} (0.6 µg/kg)	0
CH_3Hg^+	methyl-mercury	Derived from fate and transport and methylation of other species					1

4.6 Uncertainties

The selection of site PCOCs carries with it some uncertainty. There could be contaminants present in bulk sediments that have not been analyzed in historical sampling efforts. However, the uncertainty in PCOC selection is reduced because the selected PCOCs are among the most common and toxic contaminants USEPA typically evaluates for risk assessment. Furthermore, the selected PCOCs are representative of a wide variety of contaminant types (e.g., pesticides, metals, volatile organics, PCBs, dioxins) such that each evaluated exposure pathway likely includes PCOCs that could have a significant impact in that pathway due to variations in physical, chemical, and toxicological parameters.

The concentration levels assigned to PCOCs in bulk sediment are also a source of uncertainty. Based on a review of appropriate historical analytical sample data, mean concentration levels were calculated for PCOCs detected in bulk sediments. The use of mean contaminant concentration levels obtained from the analysis of the in-situ Indiana Harbor sediment was deemed appropriate and conservative for the following reasons:

1) The processes of dredging, slurrying and placement of the sediments in the CDF will entail significant mixing. This will significantly decrease the spatial variation for the sediments in the CDF compared to the spatial variation exhibited by the in-situ sediments. Consequently, selecting a measure of central tendency for the particle-bound contaminant concentrations is regarded as appropriate.

2) The contaminant data sets selected for use in the SRA commonly contain a small number of measured concentration levels which are notably higher in magnitude than

the rest of measurements. As such, the mean will be a more conservative estimate of central tendency than other estimates such as the median, which would not reflect the limited number of elevated contaminant concentrations.

3) A large portion of the in-situ contaminant data (USEPA Sediment Core Study) selected for use in the SRA was obtained from sediment depositional areas in the Federal navigation project. These areas are generally more quiescent and therefore associated with finer-grained deposits. For a variety of physical and geochemical reasons, finer-grained sediments are generally associated with elevated contaminant concentration values. Consequently, a mean obtained from these data should be both biased high and a conservative estimate of sediment contaminant concentrations in the CDF.

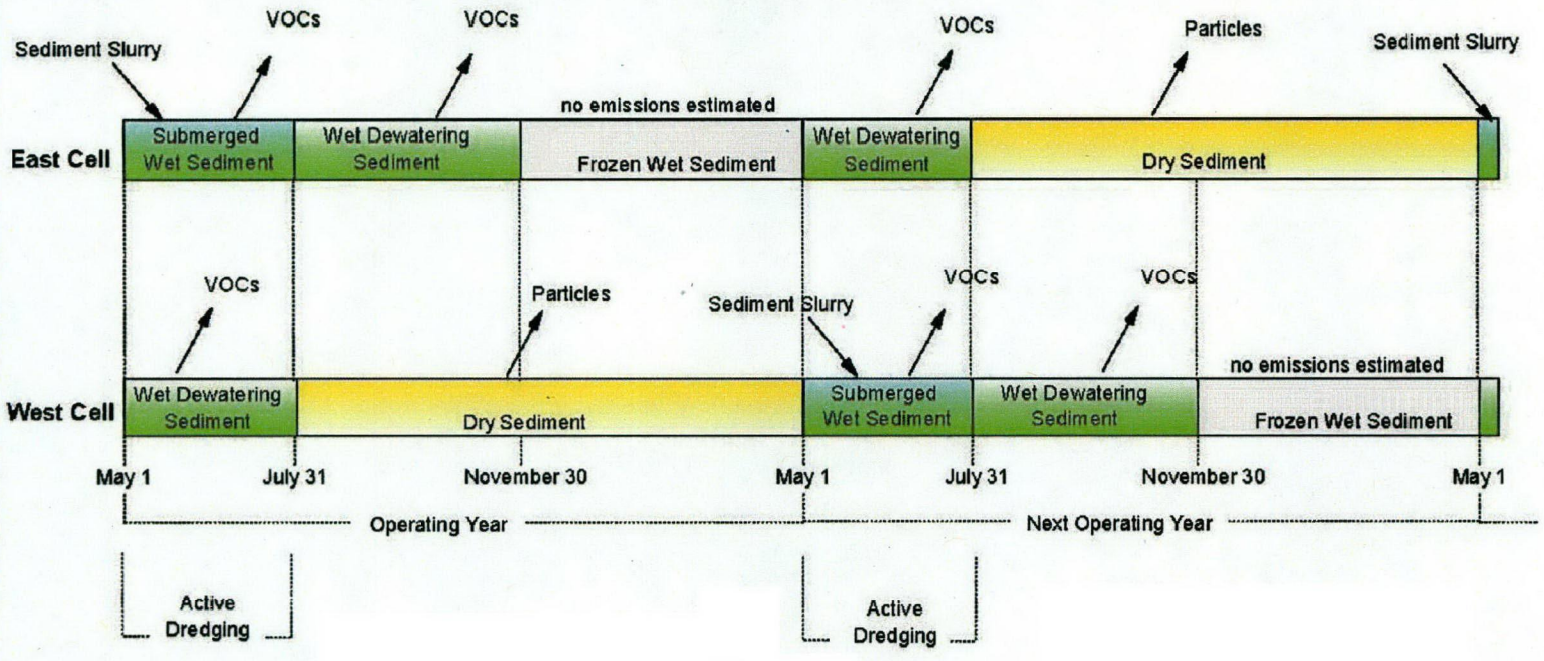
4) For PCOCs which had some bulk sediments samples reported as "non-detect" values, the non-detect samples were included in the calculation of the mean at a value equal to the full analytical detection limit, instead of a value of zero. As the actual concentration should in most instances be lower than one-half of the limit of detection, the contaminant mean values obtained in this manner should again be skewed high and would consequently be a conservative estimate of sediment contaminant concentrations in the CDF.

Volatile emissions are driven primarily by concentration-gradient evaporation from dredge slurry and could essentially occur continuously. Use of a constant emission rate for the SRA is regarded as reasonable in the absence of more detailed information. Subsequent iterations of the work undertaken by Thibodeaux and his colleagues have shown that certain meteorological inputs such as wind speed could significantly impact the volatile emissions estimate. However, the long-term chronic risks from volatile emissions evaluated at the 25 TPY maximum using naphthalene, the most toxic volatile, should still represent a conservative estimate of risk. By contrast, the evaluation of acute (short-term) risk could include greater uncertainty because of the potential impact of day-to-day variations in operations and weather which are not accounted for in a constant emission rate.

Relatively few individual particle emission events are expected from the CDF in a given year because of the episodic nature of the elevated wind velocities needed to initiate particle erosion. Consequently, use of five years of meteorological data may not have been an adequate sampling to reveal the true worst-case year for the CDF location. To compensate for this uncertainty, the annual emission level was scaled up to the 25 TPY compliance limit, instead of using the most recent estimate (i.e., average of 11 TPY) for the long-term emissions. This should introduce a degree of conservatism that is expected to offset the meteorological uncertainty of an unevaluated worst year. Since one year of the updated particle emission scenario (including mitigation techniques to limit emissions) was estimated to exceed the 25 TPY limit, the acute particle emission scenario may be underestimated.

Figure 4-2 Two-Year Operating Cycle and Emissions

Title Figure 4-2 2-Year Operating Cycle and Emissions



Project Supplemental Risk Analysis of Potential Air Emissions from the Confined Disposal Facility for the Indiana Harbor and Shipping Canal Sediment Dredging and Disposal Project

Date March 7, 2006 Author Todd D. Ramaly

Comments
1) Dredged sediment placed in each cell alternating every other year
2) Modeled period from the middle of the operating life such that there are sediments and potential emissions from both cells at all times.
3) Modeled period began at the beginning of a calendar year

Figure 4-3 - Difference in Total Particle Deposition in for Constant Versus Variable Emission Rates

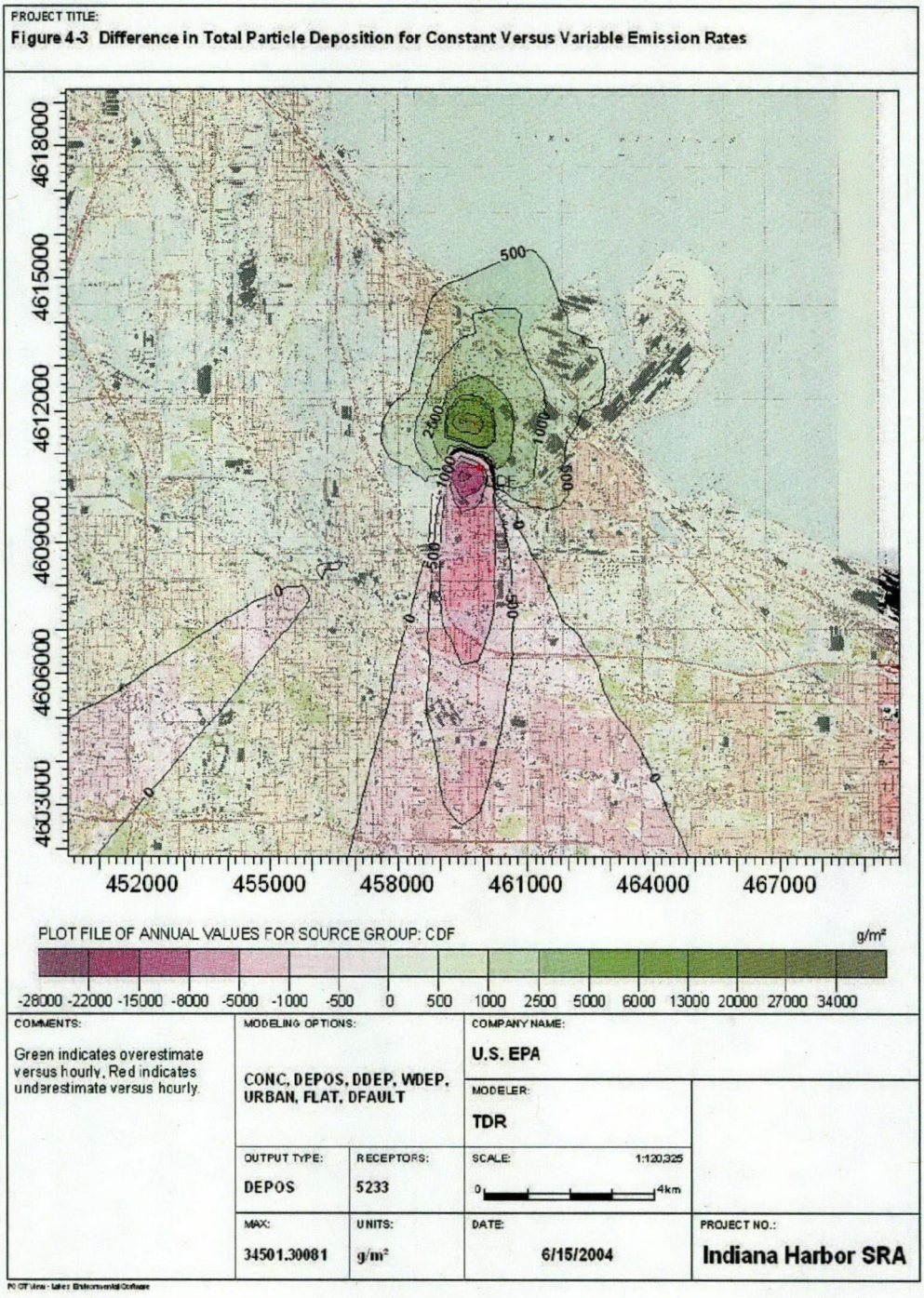
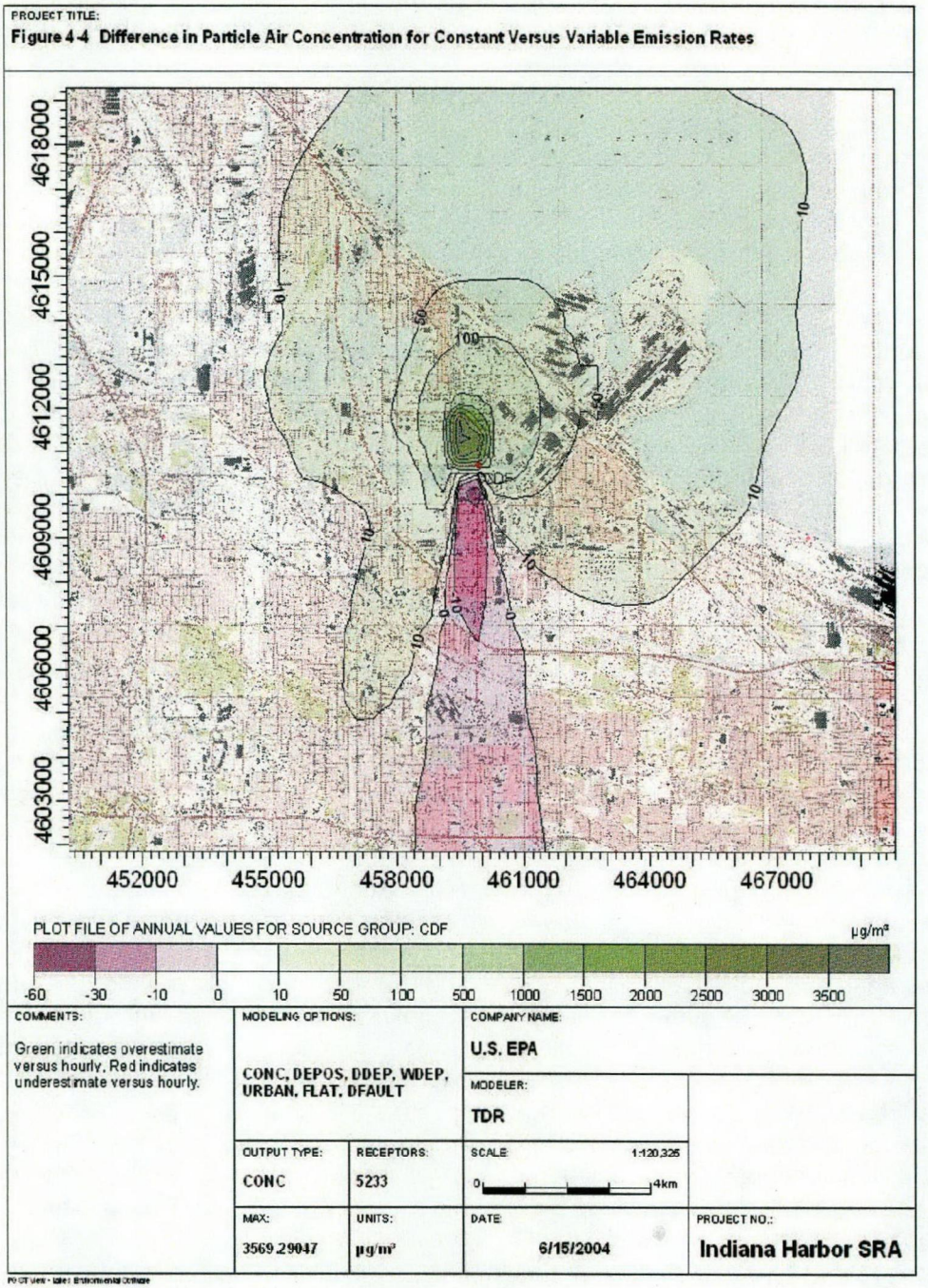


Figure 4-4 - Difference in Particle Air Concentration for Constant Versus Variable Emission Rates



5 ATMOSPHERIC DISPERSION AND DEPOSITION MODELING OF EMISSIONS

5.1 Dispersion Model

In order to assess the potential risks posed from exposure to VOCs and particulate matter (PM) from the contaminated sediments from the dredging and disposal operations, dispersion modeling was conducted to estimate the potential ambient air concentrations and deposition flux values from the CDF.

The USEPA-approved ISCST3 (version 02035) was used to generate short term averages (one-hour average) and annual average values over a five-year period (1987-1991). The model estimates ambient air concentrations and total (wet and dry) deposition flux values at each receptor. Prior to November 2005, ISCST3 was identified in 40 CFR Part 51, Appendix W, as the recommended model for most regulatory modeling analyses (USEPA 1994e).

On November 9, 2005, a new dispersion model called American Meteorological Society/ Environmental Protection Agency Regulatory Model Improvement Committee (AERMIC) Dispersion Model (AERMOD) was approved and published in the Federal Register (FR Vol. 70, No. 216). This Federal Register became effective December 9, 2005 at which time AERMOD replaced the ISCST3 model as the recommended tool for most regulatory uses. The rule allows a one-year transition period during which "timely applications of ISCST3 may still be approved at the discretion of the reviewing authority". Due to this and because most of the modeling work had been completed using the ISCST3, USEPA decided to continue using ISCST3 model for this analysis.

The ISCST3 model applies the steady-state Gaussian plume equation to model emissions released from the CDF. Even though the model was set up to include actual terrain elevation, the area source algorithm in ISCST3 is limited to flat terrain. Additional discussion can be found in Section 5.5. Urban dispersion coefficients were used and the model was set up to produce wet and dry deposition flux values. The regulatory default parameters which include buoyancy-induced dispersion, default wind profile exponents, default vertical potential temperature gradients, etc. were also applied.

For each source, the model conducts two scenarios. Scenario one is the long-term average which calculates annual average. Scenario two estimates the one-hour average. In addition, two specific pollutants were also calculated: 24-hour average concentrations for particulate matter less than or equal to 10 μm in diameter (PM10), and annual average for dry and wet gas deposition for mercuric chloride. Details of the model runs are discussed in Section 5.2. The model will provide output in the plot file format. These plot files were later utilized in the exposure model (IRAP). For scenario one and two, the ISCST3 model was run in three phases; particle, particle bound, and vapor. For particle and particle bound phases, the model produces concentrations, wet deposition flux values, dry deposition flux values, and total deposition flux values. For the vapor phase, the model produces concentrations and wet deposition flux values. The dry deposition flux values for gas phase were not calculated for scenario one and two. These values would require pollutant-specific modeling with ISCST3 using different physical parameters such as molecular diffusivity, the solubility enhancement factor, the pollutant reactivity parameters, and the Henry's law coefficient. Also individual IRAP runs would need to apply for each specific pollutant if dry gas deposition was calculated for all pollutants. Table 5-3 lists the details of model runs for scenario one and scenario two.

5.2 Source Information

Emissions from the proposed CDF were represented as two area sources: west cell and east cell sediment dewatering and containment units, to be operated in alternating years. The west cell area source was represented in the model using ten vertices and the east cell area source using four vertices. Both cells were assumed to be approximately 41 acres in size and oriented in the north-south direction.

The sediment containment cells were manually drawn into ARCMap version 8.3, a geographic information system (GIS) software package developed by Environmental Systems Research Institute, Inc. (ESRI), using USACE drawings (Thai 2005a). USEPA used the North American Datum 1927 (NAD27) as the geodetic datum for the Indiana Harbor site and the Universal Transverse Mercator Projection (UTM) for identifying coordinates. This datum was chosen because it corresponded to digital elevation maps needed to import terrain heights. Accordingly, the two cells can be defined by the following vertices.

Vertex	X	Y
	East Cell	
1	459584.75	4610765.58
2	459586.16	4611470.92
3	459858.32	4611309.53
4	459859.72	4610765.58
	West Cell	
1	459369.37	4610617.91
2	459369.38	4611405.88
3	459372.73	4611433.65
4	459382.58	4611455.45
5	459399.10	4611475.84
6	459416.68	4611491.31
7	459437.08	4611500.46
8	459462.74	4611506.08
9	459552.06	4611482.17
10	459555.21	4610617.91

Each cell was modeled separately using two different hourly emission files. The assumed maximum of 25 TPY for each category of emissions (VOCs and PM) was applied while developing the hourly emission files. For particle emissions, the hourly emission file was developed based on the wind events represented by actual local meteorology data. For a 25 TPY VOC emission, an emission rate of 5.2×10^{-6} grams per second per square meter ($g/s/m^2$) was applied on days when VOCs were expected to be emitted. The hourly files for PM and VOC can be found in Appendix 5-1 and Appendix 5-2, respectively. Section 4 of the SRA Report discusses details of the 25 TPY VOC and 25 TPY particulate emissions, assumptions, and how the CDF emissions were estimated. The release height of the CDF emissions was assumed to be 6 meters based on USACE assumption on current design height of the containment cell berm.

The particle information was based on the wind erosion model and assumptions performed by Larry Hagen, PhD, which are also described and referenced in Section 4. Table 5-1 lists the

particle size categories, particle densities, percent of mass provided by USDA (Hagen 2004c), and fraction of total mass assigned to each size category which were calculated using Table 3-1 in the draft HHRAP (USEPA 1998b). USDA determined the particle size distribution by dry sieving. This approach is consistent with ASTM Method D422-63 (2002), Standard Test Method for Particle-Size Analysis of Soils for fractions at 75 µm and above. The ASTM-recommended sedimentation process for particles less than 75 µm was not used for the following reasons:

- The samples contained a degree of small particle aggregation
- Wet sedimentation testing has the potential to significantly alter the properties of the small aggregates
- The samples were taken from outdoor-weathered Indiana Harbor sediments and were considered to be consistent with dry surface particles likely to occur at the CDF.

Accordingly, USDA employed methods that are least likely to alter the sample in order to get the best estimate of wind erosion-based emissions. The dry sieving at sizes less than 75 µm was conducted using high-precision electroformed micro-mesh sieves manufactured to ASTM standards for sieves and processed in a sonic sifter. USDA supplied an additional study in support of using electroformed sieves in sonic sieve devices for high accuracy particle analysis (Rideal 1996).

Table 5-1: Selected Characteristics of Particle Size Categories

Particle Size Categories (µm)	Percent of Mass (use for particle phase)	Fraction of Total Surface Area (use for particle bound phase)	Particle Density (g/cm3)
5.0	0.05	0.24	2.0
16.0	0.26	0.40	1.94
35.0	0.34	0.24	1.8
61.0	0.20	0.08	1.7
87.0	0.15	0.04	1.68

The wet scavenging coefficients for liquid and frozen precipitation were derived from Figure 1-11 in the ISCST3 User's Guide Volume II (USEPA 1995a). These values were required for calculation of wet deposition flux values and are listed in Table 5-2.

Table 5-2: Wet Scavenging Coefficients by Particle Size

Particle Size Categories (μm)	Liquid	Ice
5.0	3.6 E-04	1.2 E-04
16.0	6.6 E-04	2.2 E-04
35.0	6.6 E-04	2.2 E-04
61.0	6.6 E-04	2.3 E-04
87.0	6.6 E-04	2.2 E-04

The wet vapor scavenging coefficients for liquid and ice are required for modeling vapor phase wet deposition. The values listed below are recommended for vapor emissions in the HHRAP (USEPA 1998b).

Liquid $1.7E^{-4}$
Ice $0.6E^{-4}$

Table 5- 3 lists the details of model runs for scenario one and scenario two.

Table 5-3: Details of Model Runs for Scenarios One and Two

Phases/Emission	Scenario One (Annual Average)	Scenario Two (One-Hour Average)
Gas (VOC emission)	Concentrations and wet deposition flux values	Concentrations
Particle (PM emission)	Concentrations and wet and dry deposition flux values	Concentrations
Particle Bound (PM emission)	Concentrations and wet and dry deposition flux values	Concentrations

For each cell, 24-hour average PM10 concentrations were calculated using the same hourly particle emission file. Other modeling inputs are listed below. In order to ensure that all receptor node particle concentrations are PM10 only, the entire particle emission was rerun assuming a mean particle diameter of 10 microns. The ISCST3 model provided the high sixth high (H6H) concentrations at each receptor over a five-year period. The final 24-hour average for PM10 at each receptor is actually only 5 percent of these H6H concentrations based on the particle size distribution given by Dr. Hagen, which showed particle sizes from 1 to 10 micrometers (μm) representing 5 percent of the total emissions emitted from the CDF. Therefore, a correction factor of 0.05 was applied to the results at each node.

Additional PM10 modeling inputs:

Particle diameter	10 μm (for the entire particle emission)
Particle density	2 gram per cubic centimeter (g/cm^3)
Fraction mass	1
Scavenging coefficients (liquid)	6.6E-04
Scavenging coefficients (frozen)	2.2 E-04

In addition, annual average wet and dry gas deposition for mercuric chloride was also modeled using the ISCST3 toxics option. The ISCST3 model was set up to produce the annual plot files for wet and dry gas deposition for each cell separately. These plot files were then utilized in the IRAP model. Below is the list of assumptions and inputs that were used in the ISCST3 Toxics option for the wet and dry gas deposition mercuric chloride runs.

1) Gaseous dry deposition velocity is 0.029 m/s

2) Model option in this new run was set up as: MODELOPT CONC DEPOS DDEP WDEP DRYDPLT WETDPLT URBAN TOXICS

3) Hourly VOC emission files, which have constant emission rate of 5.2 X 10^{-6} g/s/m^2, were modified by replacing the value of 5.2 X 10^{-6} g/s/m^2 to 1 g/s/m^2.

4) The meteorological data file was also reformatted to include columns for Incoming Short-wave Radiation, and Leaf Area Index, which are required for modeling dry gas deposition. However, since we used constant dry deposition velocity, these parameters were not applied.

5.3 Meteorological Data

The study used meteorological data for the years 1987-1991 that were collected from three local towers within 2 miles of the CDF. The three nearby meteorological data stations are identified as *Hammond*, *Amoco Tower*, and *Amoco Water Treatment*. The Amoco Tower data is the primary meteorological data set. Missing data was substituted by the Hammond and Amoco Water Treatment sites. A table of missing meteorological data can be found in Appendix 5-3. The hourly surface data are from the Amoco Tower (Aerometric Information Retrieval System [AIRS] number 180892014) located about one mile west-southwest of the CDF center. The missing wind speed and wind direction data were substituted from the Hammond station (AIRS number 180892008), located about one mile southwest of the CDF center. The missing wind direction deviation data was substituted from the Amoco Water Treatment station (AIRS number 180892005), located about 1.8 miles northeast of the CDF center (USEPA 2006). Precipitation data was also taken from the Amoco Water Treatment station. The upper air (mixing height) data from the National Weather Service (NWS) in Peoria, Illinois were used, since that site is the nearest NWS station which collects upper air data. The meteorological data for the years 1987 to 1991 were used because it is the most recent readily available, representative data that include on-site precipitation information (USEPA 1987). Five years of processed meteorological data can be found in Appendix 5-4.

The wind rose plots of five years of meteorological data (1987-1991) were made using the WRPLOT view program from Lake Environmental Software. The wind rose plot from five years of local wind speed and wind direction data was compared with the plot from five years of NWS data from Chicago/O'Hare International Airport. Both wind rose plots show similar prevailing winds from the southwest. However, the wind rose plot for the local data shows more northerly winds from the Lake than the plot with NWS data. Additionally, the local data is characterized by somewhat lighter wind speeds overall than the NWS data. This indicates that using the local meteorological data would better address Lake effects than using data from Chicago/O'Hare Airport. Figures 5-1 and 5-2 are wind rose plots for the local data and the NWS data respectively.

The Meteorological Processor for Regulatory Models (MPRM) version 04048 was used to process five years (1987 to 1991) of on-site meteorological data (Bailey 2004). This preprocessor model was necessary to prepare on-site meteorological data for use with the ISCST3 model. This new MPRM model allows us to process all three stages using one executable file (USEPA 2003c). Table 5-4 below lists the surface characteristics at the CDF site that were used as the inputs for the MPRM model (USEPA 1996c, 1999a). An example of MPRM input file for one year of meteorological data can be found in Appendix 5-5.

Table 5-4: Surface Characteristics at the CDF Site

	Winter (Dec, Jan, Feb)	Spring (Mar, Apr, May)	Summer (Jun, Jul, Aug)	Fall (Sep, Oct, Nov)
Albedo	0.35	0.14	0.16	0.18
Bowen Ratio	1.5	1.0	2.0	2.0
Roughness (MS)	1.0	1.0	1.0	1.0
Roughness(AS)	1.0	1.0	1.0	1.0
M-O length	50	50	50	50
Surface Heat Flux	0.27	0.27	0.27	0.27
Anthropogenic Heat Flux	75	50	25	50
Leaf Index	0.0	0.0	0.0	0.0

MPRM uses cloud cover, station pressure, and global radiation to calculate friction velocity and estimate the stability classes (Bailey 2004). These data, however, were not available at the Hammond, Amoco Tower, or Amoco Water Treatment stations. The NWS surface data from O'Hare Airport were substituted for these parameters. For the years from 1987 to 1990, the NWS data were available in Solar and Meteorological Surface Observation Network (SAMSON) format and included global radiation. For the year 1991, however, the NWS data were in Hourly United States Weather Observations (HUSWO) format, which does not include global radiation. Extra effort was made to develop Global radiation for the year 1991 using the new version of MPRM. O'Hare Airport is located approximately 35 miles northwest of the CDF site. These data are the best available and should provide a reasonable estimate of the cloud cover, station pressure, and global radiation conditions in South Chicago/Northwest Indiana region.

Figure 5-1: Wind Rose Plot for Five Years (1987-1991) of On-Site Meteorological Data, AMOCO TOWER Monitoring Site, IN

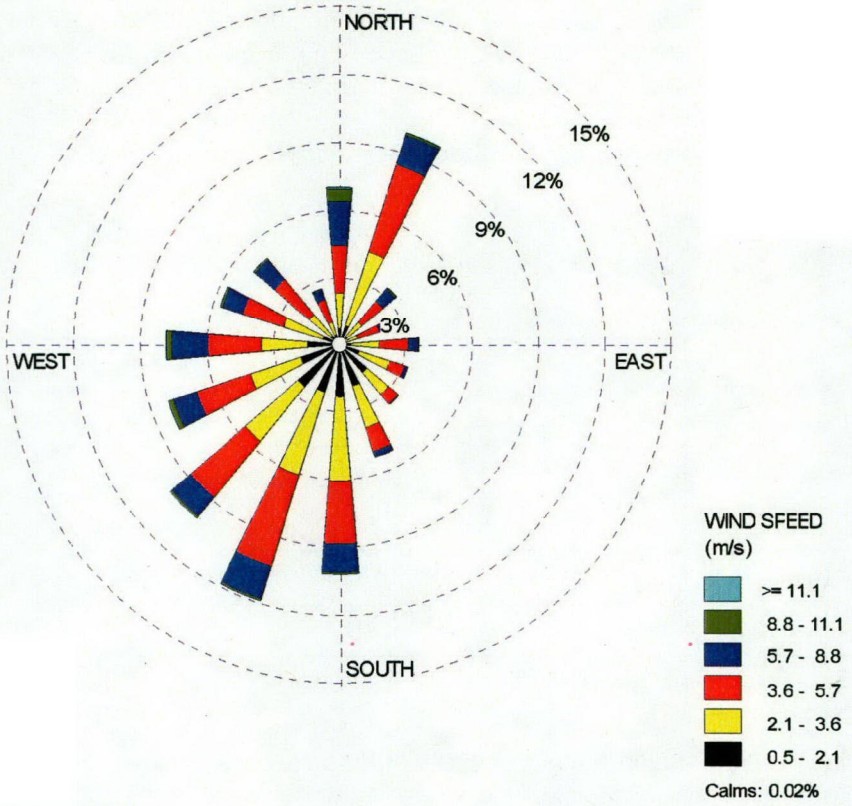
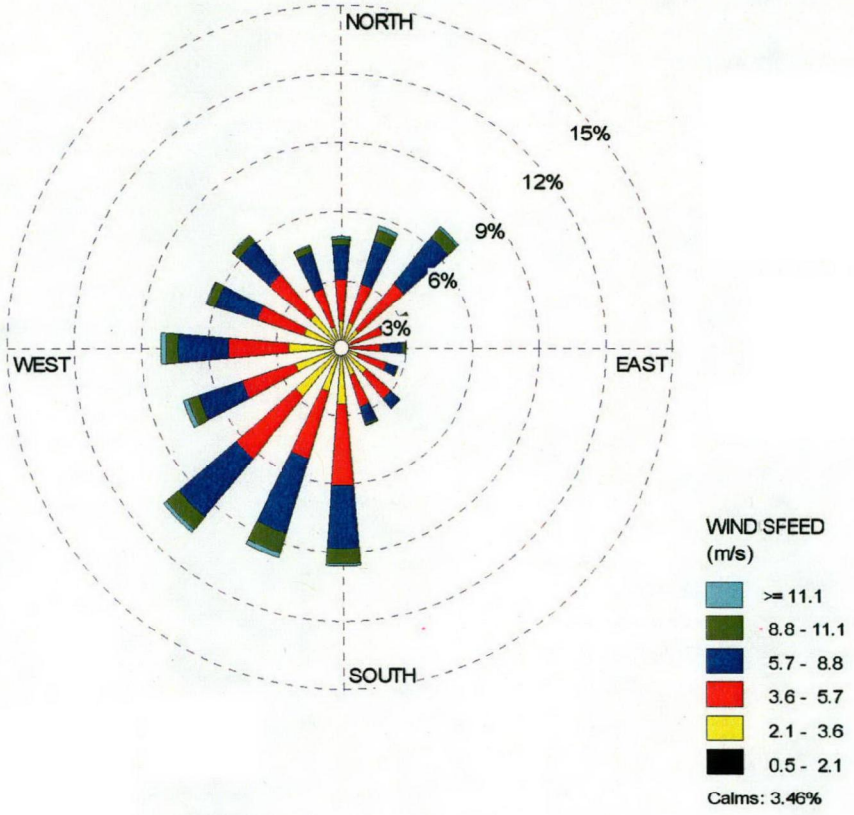


Figure 5-2: Wind Rose Plot for Five Years (1987-1991), NWS Data, Chicago/O'Hare INT'L ARPT, IL



5.4 Source Characterization Inputs

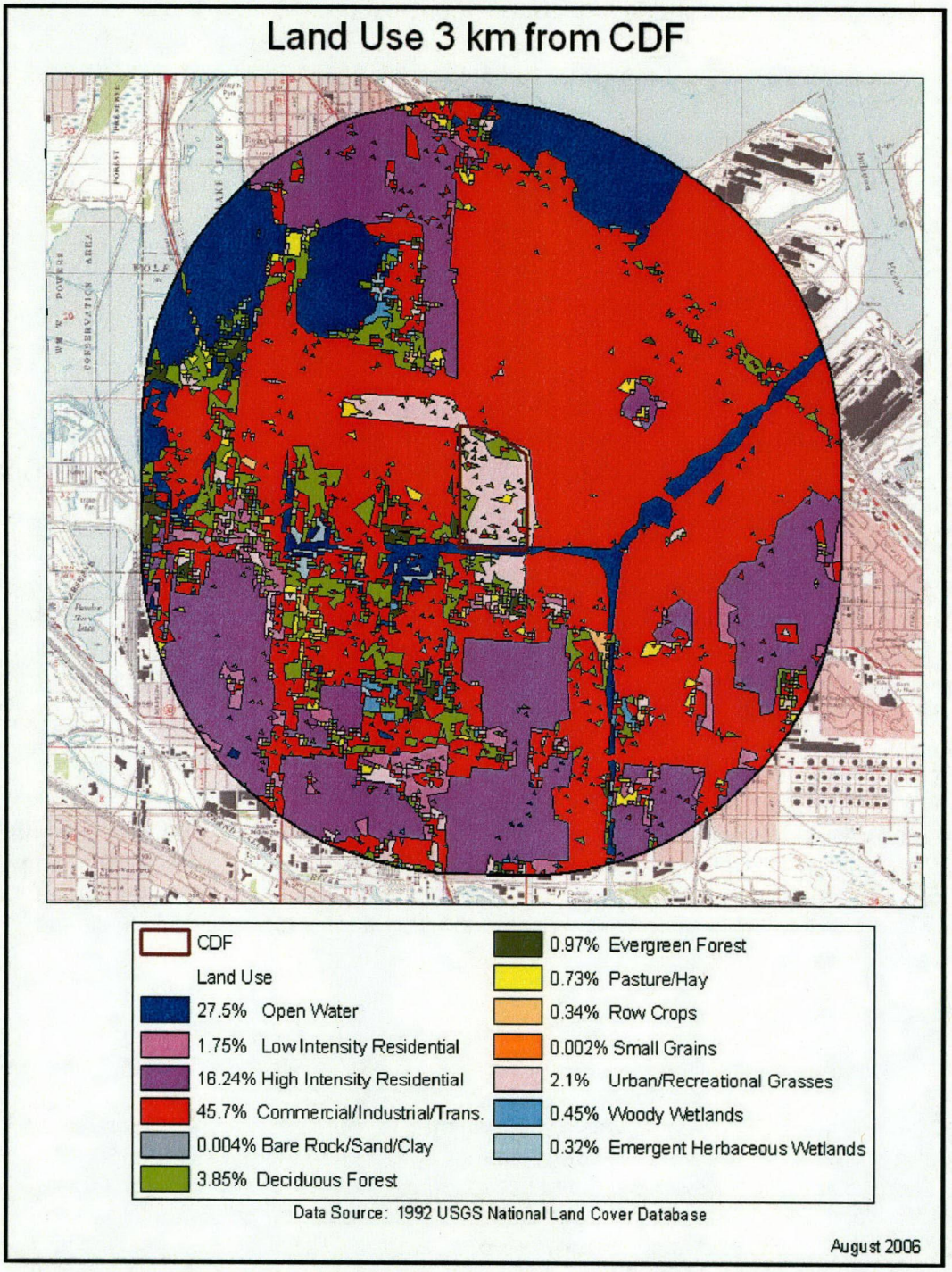
Source parameters required by the ISCST3 model for this analysis include hourly pollutant emission rates, East and West Cell locations and dimensions, number of vertices (or sides) of each cell, and release height above the ground. The hourly emission rates for PM and VOCs for each cell were modeled separately. The operation plans for each cell were used to develop the PM and VOC hourly emission files. On-site meteorological conditions were also incorporated while developing the hourly emission rates for PM, as explained in Section 4.

The urban dispersion coefficient was selected based on the application of the Auer method as specified in USEPA guidance on air quality models. The Auer method defines an area as urban if industrial, commercial, and residential land use types within a 3-kilometer (km) radius of the facility comprise 50 percent or more of land use by area. The 1992 National Land Cover Classification Database showed that more than 50 percent of the land use within 3 km of the CDF is industrial, commercial, and residential. Table 5-5 and Figure 5-3 provide percentages of land use within 3 km of the CDF.

Table 5-5: Land Use Within 3 km of the CDF (1992 Data)

Land Use Code Definition	Percentages
High intensity commercial	45.749
Low intensity resident	1.748
High intensity resident	16.245
Total Urban	**63.742**
Open water	27.438
Bare Rock/Sand/Clay	0.004
Deciduous Forest	3.850
Evergreen Forest	0.969
Pasture/Hay	0.733
Row Crops	0.337
Small Grains	0.002
Other Grasses Urban	2.104
Woody Wetland	0.454
Emergent Herbaceous	0.322
Total Rural	**36.213**

Figure 5-3: Percentages of Land Use Within 3 km of the CDF



Source input data for the SRA are summarized in Table 5-6.

Table 5-6: Source Input Data

Source Parameter	Values
Dispersion Coefficient	Urban
Terrain Height (above sea level)	180 meters
Anemometer Height	10.0 meters
Release Height	6.0 meters

5.5 Terrain Heights

Terrain heights were imported automatically for receptor grid nodes using AERMOD-View version 4.8 (a Windows-based interface for running USEPA-approved dispersion models developed by Lakes Environmental Software of Waterloo, Ontario) from the following 7.5 minute USGS Digital Elevation Model (DEM) files obtained from the WebGIS Web site (http://www.webgis.com): Lake Calumet, IN; Whiting, IN; Calumet City, IN; and Highland, IN.

The terrain heights above sea level at the CDF site and within the receptor grid are around 180 meters. Terrain heights were assigned for all vertices of the East and West cells and for all receptor locations. This information was included to address the actual land uses around the CDF site but has no effect on the overall results since the ISCST3 area source algorithm assumes flat terrain.

5.6 Receptor Grid

A Cartesian receptor grid was used in the modeling analysis. The center of the Cartesian grid is located at the center of the CDF, which has the UTM Easting (459592.06) and UTM Northing (4611066.99). Receptor nodes were spaced at 50-meter intervals within 2 kilometers of the origin and 500-meter intervals within 5 kilometers of the origin. The receptor grid file can be found in Appendix 5-6.

5.7 Output

5.7.1 Long-Term Average (Annual)

For each CDF cell, the ISCST3 model was run three times to calculate the vapor, particle, and particle bound pollutant phases using two separate hourly emission input files (one for PM emissions and one for VOC emissions). A total of six output files were produced for annual average; three files for the west cell and three files for the east cell. Air concentrations and deposition flux values were estimated at each receptor location. The ISCST3 input files and plot files for annual average (three phases) can be found in Appendix 5-7 and Appendix 5-8 respectively.

5.7.2 Short-Term (One-Hour) Averages

Short-term, one-hour concentrations were calculated from each cell. The one-hour deposition flux values were not included. Similar to the annual average, the ISCST3 model was run three times to calculate the vapor, particle, and particle-bound pollutant phases using two separate hourly emission files. A total of six files were produced for short term averages (three files for west cell and three files for east cell). For each cell, the three files represent three possible phases: vapor, particle, and particle bound. Concentrations were estimated at each receptor location. The ISCST3 input files and plot files for one-hour average (three phases) can be found in Appendix 5-9 and Appendix 5-10, respectively.

The above ISCST3 results (long term average and short term average) were not pollutant specific. For each run, ISCST3 was set up to produce the plot files to represent the dispersion of a generic pollutant These plot files were then used in the IRAP exposure model to determine the potential risks.

5.7.3 PM10 (24-hour Average)

Generally, at least one year of sequential hourly meteorological data is used to ensure that worse-case seasonal conditions are addressed in the analysis. The National Ambient Air Quality Standards (NAAQS) for 24-hour average PM10 is 150 microgram per cubic meter and it is not to be exceeded more than once per year. The Allowable increments are defined as maximum allowable increases in ambient air concentrations that are also not to be exceeded more than once per year for other than an annual time period (USEPA 1990c). It was understood that for PM10 modeling, one exceedance per year is permitted for the 24-hour NAAQS as well as for the 24-hour Class II PSD.

For this analysis, five years (1987-1991) of meteorological data were used to model 24-hour average concentration for PM10; therefore, five exceedances are permitted. This allows us to look at the sixth highest concentration (H6H) for 24-hour average. A correction factor of 0.05 was then applied at each receptor to account for the assumption that 5 percent of the total particle emission was comprised of PM10. After applying the 0.05 correction factor, the H6H maximum 24-hour concentration within study area of interest was compared to the 24-hour NAAQS standard and the 24-hour class II PSD for PM10. Additional discussion on PM10 modeling results and its impact on short -term inhalation exposure to respirable particle matter can be found in Section 6.4.8.2.

24-hour average concentrations were estimated from each cell. Both wet and dry depositions were selected in the model to account for plume depletion. The ISCST3 model was run and produced the plot file of H6H concentrations at each receptor location. The ISCST3 input files and plot files can be found in Appendix 5-11 and Appendix 5-12, respectively.

5.7.4 Mercuric Chloride Vapor (Annual Average)

We calculated annual average of total, wet and dry gas deposition fluxes for mercuric chloride from each cell. The ISCST3 toxics option was run and produced two annual plot files. For mercuric chloride, the model was set up to run for vapor phase only. These two vapor plot files were then utilized in the IRAP model to estimate the impact of mercury on fish consumption. The input files and plot files are in Appendix 5-13 and Appendix 5-14, respectively.

Other forms of mercury were also evaluated for annual and acute exposures but did not require additional dispersion modeling. We evaluated particle form of divalent (as mercuric chloride) and methylmercury from the CDF by applying their proportional sediment concentrations to the 25-ton particle plot files. We also evaluated elemental mercury emitted as a vapor by using the 25-ton vapor plot files and modifying it to account for the maximum mass of elemental mercury expected in the sediment.

Additional information on mercury speciation, IRAP options, and results can be found in Section 4.5 and Section 6.1.4.5.

5.8 Uncertainties

Meteorological conditions influence how pollutants will transport, disperse, and deposit at downwind distances. Use of local meteorological data reduces the uncertainties to some extent. Using cloud cover, station pressure, and Global radiation information from the NWS at O'Hare Airport instead of local data has some disadvantages but was chosen as the best approach at the time of the analysis. Some surface characteristics such as albedo, bowen ratio, and surface roughness length are estimates based on seasons and land use classifications. These values are sensitive to other factors, such as the reflection of radiation and the ratio of sensible heat, and therefore will also add to the model uncertainties.

In addition, the ISCST3 model does not simulate chemical transformations or reactivity in the plume. It assumes that the pollutant is either emitted as a vapor, as a particle, or as particle bound and disperses in the same manner. Using such a generic pollutant approach adds uncertainty to the overall model results. The lack of dry gas deposition may underestimate deposition flux values and overestimate the concentrations. Because ISCST3 is a steady-state model, it does not account the effects of varying winds on plume as it travels downwind. This adds some uncertainties to the model predicted values as well as the locations of the impacts. The ISCST3 model did not calculate values for hours with calm wind conditions which could add some uncertainties. However, for this analysis, calm wind conditions over a five-year period occurs only about 2 percent of the time. If calm wind conditions were significantly higher, stagnation conditions should be included in the ISCST3 model.

Mercury modeling is a very complicated task due to the complex environmental behavior of this chemical. The ISCST3 model with toxics option might not be able to address all of the assumptions and uncertainties relevant to mercury characterization.

The predicted air concentrations and deposition flux values are calculated based on a selected dispersion model. Dispersion modeling, like any model, has inherent uncertainties. Users should take all uncertainties into account when interpreting the model results.

6 HUMAN HEALTH RISK ASSESSMENT

6.1 Toxicity Assessment

The purpose of the toxicity assessment portion of the SRA is to combine the chemical identification and dose-response assessment. The primary objectives of this step of the risk assessment process are: to identify the types of toxic effects associated with each PCOC, characterize the conditions of exposure (e.g., intake route, time-duration) under which these effects might occur, and determine the relationship between the magnitude of the exposure dose and the extent of adverse health effects. This relationship is represented through the use of toxicity values related to cancer or noncancer health endpoints.

6.1.1 Toxicity Associated with Chronic Exposure

USEPA has conducted toxicity assessments on many frequently encountered environmental contaminants, and has developed toxicity factor values for use in risk assessment, based on these analyses. These assessments and the toxicity factors generally apply to chronic, long-term exposure that could potentially occur over an extended time duration, such as multiple years or a significant fraction of the lifespan.

6.1.2 Carcinogenic Effects

Substances classified by USEPA as potentially carcinogenic are considered by many scientists to pose a finite cancer risk at all exposure levels. Therefore, in evaluating cancer risks, a "no-threshold" assumption is applied. A "no-threshold" assumption means that there is not an established exposure level below which no cancer risk is assumed. It should be noted that the no-threshold assumption may not apply for some classes of carcinogens that act through a mechanism that requires a threshold dose to be exceeded prior to initiation of the carcinogenic process. For purposes of this assessment, the no-threshold assumption is conservatively assumed for all chemical carcinogens.

A two-step evaluation is used in assessing the carcinogenic potential of a chemical. The first step involves evaluating the likelihood that the substance is a human carcinogen (i.e., a weight-of-evidence assessment), and the second step involves defining the quantitative relationship between dose and response (i.e., development of a slope factor or IUR). In the first step, USEPA classifies a chemical into one of five groups that indicate the likelihood that the chemical is a human carcinogen, based on the weight of evidence from human and animal investigations as follows (USEPA 2005b):

- Carcinogenic to Humans
- Likely to Be Carcinogenic to Humans
- Suggestive Evidence of Carcinogenic Potential
- Inadequate Information to Assess Carcinogenic Potential
- Not Likely to Be Carcinogenic to Humans

Those chemicals showing evidence of being known, likely, or suggestive human carcinogens are further evaluated.

The second step is a dose-response assessment for each tumor type identified in the relevant data sets. The assessment occurs in two parts: (1) examination of observed data on the dose-

response relationship for tumor formation in order to derive a point of departure (POD). The POD is an estimated dose (expressed in human-equivalent terms) near the lower end of the observed dose range from epidemiological or laboratory animal studies; and (2) extrapolation from the POD to lower exposure doses below the observable range. The outcome of the second part of the evaluation is the development of a CSF or an inhalation unit risk (IUR), which is an estimate of the potency of the carcinogen. The CSF represents the upper 95 percent confidence limit on the linear component of the slope of the tumorigenic dose-response curve in the low-dose (low-risk) region. The CSF is an upper bound estimate of the likelihood that a response will occur per unit intake of a chemical over a 70-year lifetime, and is derived by applying a mathematical model to extrapolate from the relatively high doses administered to experimental animals or experienced by persons in the workplace to the lower exposure levels expected for human contact in the environment. A number of low-dose extrapolation models have been developed. USEPA generally uses the linear multistage model in the absence of adequate information to support some other model. The linear multistage model is regarded as conservative, meaning that it is likely to over predict the true CSF for a chemical. The concepts and procedures summarized above are discussed in detail in USEPA's *Guidelines for Carcinogen Risk Assessment* (USEPA 1986a, 2005b).

In practice, the CSF may be regarded as the cancer risk (proportion of affected individuals) per unit of dose. In USEPA's derivation, the slope factor is expressed on the basis of chemical weight: milligrams of substance per kilogram body weight per day (mg/kg/day, mg/kg-day). The magnitude of the CSF can be used to compare the relative potency of one chemical substance to another.

Quantitative cancer risk estimates have several uses, and the expression employed should be tailored to each use. For example, to estimate risk from a given chemical via exposures by the oral route, the slope factor (risk per mg/kg-day) is multiplied by the long-term daily dose (mg/kg-day) from each oral pathway of exposure (e.g., soil, food, water). The total oral risk is found by summing risks across all oral intake pathways.

For evaluating risks from chemicals by the inhalation route, the dose-response slope factor is expressed as the risk per air concentration unit. This factor is called the IUR and is usually expressed as risk per micrograms per cubic meter (risk per $\mu g/m^3$) of air. To estimate risk from a given chemical via exposures by the inhalation route, the IUR (risk per $\mu g/m^3$) is multiplied by the long-term daily air concentration ($\mu g/m^3$).

6.1.3 Noncarcinogenic Effects

The basic approach used by USEPA in developing toxicity values for noncarcinogenic effects of substances is based on the concept that some minimum (threshold) exposure level must be reached before the effect will occur, i.e., that protective mechanisms exist that must be overcome before an adverse health effect can occur. The estimated level of daily human exposure below which it is unlikely that deleterious effects will result is known as the Reference Dose (RfD). RfD values apply to the oral route of exposure and are reported in milligrams of chemical per kilogram body weight per day (mg/kg-day). If adequate human data are available from epidemiological studies (e.g., occupational exposure; environmental media exposure), the RfD can be based on human effects data. In the absence of adequate human data, an RfD value is based on data from experimental animals. If data from several animal studies are available, USEPA first seeks to identify the animal model that is most biologically relevant to humans (e.g., similar metabolism of the substance). In the absence of information that identifies a given animal model as clearly most relevant, an assumption is made that humans are at least

as sensitive to the substance as the most sensitive animal species tested. Accordingly, USEPA selects the study using the most sensitive species tested and the most sensitive endpoint measured as the critical study upon which the RfD is based.

From this critical study, the experimental exposure representing the highest tested dose level at which no adverse effects were demonstrated (the no-observed-adverse-effect level, NOAEL) is identified. In selecting the NOAEL as the basis for the RfD, the assumption is made that if the critical toxic effect is prevented from occurring, then all toxic effects are prevented. The NOAEL is distinguished from the no-observed-effect level (NOEL), which corresponds to the exposure level at which no effect at all is observed; whereas, the NOAEL is the level at which no effect considered to be of toxicological significance is observed. In some studies, only a lowest-observed-adverse-effect level (LOAEL) is available. The use of a LOAEL in deriving an RfD requires the use of an additional uncertainty factor as described below.

The RfD is derived from the NOAEL or LOAEL for the critical toxic endpoint by dividing the NOAEL or LOAEL by one or more uncertainty factors. These factors are generally multiples of 10, with each factor representing a specific area of uncertainty in the extrapolation from the available study data. For example, a 100-fold uncertainty factor is typically used when the RfD is based on results from long-term animal studies. This factor of 100 incorporates an uncertainty factor of 10 to account for variation in sensitivity in the human population and another uncertainty factor of 10 to account for interspecies variability between humans and experimental animals. Additional modifying factors ranging from 1 to 10 may be applied to reflect qualitative judgments about limitations or uncertainties in the critical study or in the available data base. Since the RfD is intended to be adequately protective of sensitive individuals, such as children and the elderly, application of the RfD to the general population is considered to be appropriate.

For the assessment of noncarcinogenic effects from inhalation exposures, USEPA has developed values known as Reference Concentrations (RfCs). An RfC value is reported as an air concentration in units of mass of chemical per unit volume of air (e.g., mg/m^3). In analogy with the RfD value, an RfC is also based on the concept of minimum threshold exposure and the analysis of sensitive endpoints from critical studies. An RfC may also be developed from toxicological data on humans or animals.

The concepts and procedures summarized above for the dose-response of noncarcinogenic effects are discussed in detail in the document *Review of the Reference Dose and Reference Concentration Processes* (USEPA 2002b).

6.1.4 Special Substances or Groups of Compounds

For certain substances or group of compounds, the methodology used to assess toxicity and dose-response effects is somewhat different from that described above. In addition, there are specific issues with respect to the toxicity of certain substances that should be noted. Therefore, a separate discussion of the toxicity assessment approach is provided for the following substances: dioxins, PAHs, PCBs, lead, mercury, chromium, and particulate matter.

6.1.4.1 Chlorinated Dioxins and Furans (Dioxins)

There are 210 individual forms or "congeners" of chlorinated dioxins and furans. A congener is a single member of a chemical family (e.g., there are 75 congeners of chlorinated dibenzo-para-dioxins). USEPA has developed procedures for assessing the cancer risks associated with

exposure to the many forms of dioxins and furans based on the relative toxicity of these compounds to the toxicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin (2,3,7,8-TCDD), which is generally believed to be the most toxic form (USEPA 2003a).

Each congener is assigned a value, referred to as a toxicity equivalency factor (TEF), corresponding to its toxicity relative to 2,3,7,8-TCDD (i.e., 2,3,7,8-TCDD has a TEF of 1.0 and other dioxin and furan congeners have TEFs between zero and 1.0). Although various groups and organizations have developed TEF approaches, the most recent USEPA-recommended approach (USEPA 2003a) is used in this risk assessment. This approach is the same as the internationally accepted approach adopted by the World Health Organization (WHO 1998). Of the 210 possible congeners of the chlorinated dioxins and furans, only the 17 congeners having chlorine in the 2, 3, 7, and 8 positions are generally regarded as displaying dioxin-like toxicity. The names of these congeners and their corresponding TEF values are shown below.

PCDD/PCDF TOXICITY EQUIVALENCY FACTOR VALUES

Dioxin Congener	TEF (unitless)	Furan Congener	TEF (unitless)
2,3,7,8-Tetrachlorodibenzo(p)dioxin	1.0	2,3,7,8-Tetrachlorodibenzofuran	0.1
1,2,3,7,8-Pentachlorodibenzo(p)dioxin	1.0	1,2,3,7,8-Pentachlorodibenzofuran	0.05
1,2,3,4,7,8-Hexachlorodibenzo(p)dioxin	0.1	2,3,4,7,8-Pentachlorodibenzofuran	0.5
1,2,3,6,7,8-Hexachlorodibenzo(p)dioxin	0.1	1,2,3,4,7,8-Hexachlorodibenzofuran	0.1
1,2,3,7,8,9-Hexachlorodibenzo(p)dioxin	0.1	1,2,3,6,7,8-Hexachlorodibenzofuran	0.1
1,2,3,4,6,7,8-Heptachlorodibenzo(p)dioxin	0.01	1,2,3,7,8,9-Hexachlorodibenzofuran	0.1
1,2,3,4,6,7,8,9-Octachlorodibenzo(p)dioxin	0.0001	2,3,4,6,7,8-Hexachlorodibenzofuran	0.1
		1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.01
		1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.01
		1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.0001

The CSFs for dioxin and furan congeners other than 2,3,7,8-TCDD are derived by the assignment of TEF values which compare the toxicity of the toxic congeners to that of 2,3,7,8-TCDD. For 2,3,7,8-TCDD, the CSF is based on actual experimental evidence in rodents. For the other 16 toxic congeners, the toxic potency compared to 2,3,7,8-TCDD is based on receptor binding studies or a sensitive measure of receptor binding, namely induction of aryl hydrocarbon hydroxylase (AHH) enzyme activity. These TEFs have been developed based on the activity of these compounds in short-term toxicity assays that are considered predictive of their ability to cause cancer in long-term carcinogenicity studies in experimental animals. Consequently, the CSFs (derived from the TEF values) for these 16 toxic congeners are less certain than the cancer slope factor for 2,3,7,8-TCDD.

In accordance with USEPA guidance (USEPA 2005c), the individual dioxin and furan congeners are assessed separately throughout the exposure assessment process. This involves the estimation of congener-specific emission rates (Section 4), followed by the evaluation of each congener for partitioning into various environmental media. This approach is adopted due to the

important differences between dioxin and furan congeners in the fate and transport properties needed to estimate exposures through the food chain pathways (USEPA 2003a, 2005c; Washburn 1991; McLachlan 1993). The TEFs are applied in the final step of the risk assessment process, for estimating potential health risks.

In addition to the potential for dioxins to cause cancer, there is also concern for the potential noncancer effects from these chemicals. USEPA has concluded that adequate evidence exists to suggest that exposure to 2,3,7,8-TCDD and related dioxin-like compounds results in a broad spectrum of effects in animals, some of which may occur in humans (USEPA 2003a).

This conclusion is based on results from epidemiology studies in human populations, experiments in laboratory animals, and ancillary experimental studies. The induced effects will likely range from adaptive changes at or near background levels of exposure, to adverse effects with increasing severity as exposure levels increase above background levels. Enzyme induction, alterations in hormone levels and indicators of altered cellular function are examples of effects of currently unknown significance; they may or may not be early indicators of toxic response. Because the threshold levels for exposure to 2,3,7,8-TCDD and dioxin-like compounds below which toxic effects are not observed has not been established, USEPA does not currently list RfD or RfC values for dioxin-like compounds.

The deduction that humans could respond to exposures to 2,3,7,8-TCDD and dioxin-like compounds with adverse noncancer effects is based on the fact that these compounds impact cellular regulation at a fundamental molecular level in a diverse variety of animal species, which have been shown to respond with adverse effects. In addition, similar impacts on cellular regulation have been demonstrated in human cells in experimental cell culture. It is well known that individual animal species vary in their sensitivity to exhibit different effects due to exposure to 2,3,7,8-TCDD. The available evidence indicates that humans most likely fall in the middle of the range of sensitivity for individual effects among animals rather than at either extreme (USEPA 2003a). Thus, humans do not appear to be either extremely sensitive to or extremely insensitive to the individual effects of 2,3,7,8-TCDD and dioxin-like compounds.

In general, biochemical, cellular and organ-level effects have been observed in experiments in which only 2,3,7,8-TCDD was studied. Specific data on the effects of other dioxin-like homologues such as the pentachlorinated and hexachlorinated dioxins and furans are generally not available. However, as mentioned previously, dioxin-like compounds exhibit the common property of binding to the intracellular AHH receptor. Based on differences in receptor binding capacity, TEFs have been developed for the 17 dioxin and furan congeners with chlorine substituents in the 2,3,7, and 8 ring positions. The TEF for 2,3,7,8-TCDD is one; the TEFs for the 16 other congeners are a fraction of this value (i.e., between zero and 1.0). Greater uncertainty exists with respect to the extent of noncancer effects of the 16 other congeners as compared to those of 2,3,7,8-TCDD due to the very limited amount of toxicology testing of these congeners. Hence, greater uncertainty is associated with the TEFs of these congeners as compared with the TEF for 2,3,7,8-TCDD.

USEPA's draft "Dioxin Reassessment" documents (USEPA 2000, 2003a) concluded that it would be inappropriate to develop an RfD for dioxins. This is because dioxins are persistent compounds in the environment and because pre-existing background exposures to dioxins are not necessarily low compared to incremental dioxin exposures arising from a single source under investigation. Most compounds for which RfDs are derived are not persistent, and background exposures are generally very low and not taken into account. Therefore, the draft

Dioxin Reassessment concluded that it is not appropriate to use the reference dose approach in evaluating incremental exposures to dioxins.

Since the reference dose approach is considered inappropriate for evaluating the potential noncancer effects of dioxins, USEPA Office of Research and Development (USEPA-ORD) and Office of Solid Waste (USEPA-OSW) have recommended using a "Margin of Exposure" (MOE) approach. This is an approach for estimating the potential for noncancer health effects arising from incremental exposures to dioxins (USEPA 2003a, 2005c). To apply this approach, one determines the ratio of the estimated daily dose of dioxins from a particular source (in this case, intake of dioxin emissions from the CDF) to the average daily intake of dioxins in the general U.S. population from existing sources, which is between 1 and 3 picograms per kilogram per day (pg/kg-day) (USEPA 2000). The ratio of this incremental dose to the background dose of 1 - 3 pg/kg-day represents the margin of exposure to dioxins. A low ratio indicates that the incremental source under investigation does not contribute a significant addition to the expected background exposure to dioxin.

6.1.4.2 Polycyclic Aromatic Hydrocarbons (PAHs)

The compounds benzo[a]anthracene, benzo[a]pyrene (BaP), benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene, and indeno[1,2,3-cd]pyrene are considered to be known or likely human carcinogens by USEPA. With the exception of chrysene, these PAHs are all known animal carcinogens. USEPA has derived an oral CSF for BaP; however, the remaining carcinogenic PAHs have not been assigned CSF values because of the limitations in the dose-response cancer studies performed on these compounds. Until individual CSF values are assigned, USEPA recommends an interim relative potency approach to determining carcinogenic potential based on results in a group of carcinogenicity studies in animals (USEPA 1993b). The toxicity of each carcinogenic PAH is evaluated relative to the toxicity of BaP. The potency of BaP has been assigned a value of 1.0, which is equivalent to an oral CSF of 7.3 per mg/kg-day. The other PAHs have been assigned a relative potency factor (RPF) between zero and 1.0, as shown in the table below. Each of the PAHs considered in this SRA is evaluated separately with regard to estimating CDF emissions, modeling fate and transport in the environment, and quantifying human dose. The relative potency factors are applied in the final step of the risk assessment process for estimating potential health risks.

Compound	RPF
Benzo(a)pyrene	1.0
Benz(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenz(a,h)anthracene	1.0
Indeno(1,2,3-cd)pyrene	0.1

6.1.4.3 Polychlorinated Biphenyls (PCBs)

PCBs are mixtures of synthetic chlorinated organic chemicals. Different mixtures can take on forms ranging from oily liquids to waxy solids. Although their chemical properties vary widely, different mixtures can have many common components. Because of their inflammability, chemical stability, and insulating properties, commercial PCB mixtures had previously been used in many industrial applications, especially in capacitors, transformers, and other electrical equipment. They were also used as additives in some paint products and as lubricants for heavy machinery. These chemical properties also contributed to the persistence of PCBs after release into the environment. The use of PCBs in the U.S. was banned in 1977, as a result of evidence that PCBs persist in the environment and have significant potential for inducing adverse human health and ecological effects. However, PCBs continue to be encountered in the environment as the result of historical releases. The table below shows the chemical composition of several commercially manufactured PCB mixtures (USEPA 1996b):

	Aroclor					Clophen		Kanechlor		
	1016	1242	1248	1254	1260	A 30	A 60	300	400	500
Mono-CBs	2	1	—	—	—	—	—	—	—	—
Di-CBs	19	13	1	—	—	20	—	17	3	—
Tri-CBs	57	45	21	1	—	52	—	60	33	5
Tetra-CBs	22	31	49	15	—	22	1	23	44	26
Penta-CBs	—	10	27	53	12	3	16	1	16	55
Hexa-CBs	—	—	2	26	42	1	51	—	5	13
Hepta-CBs	—	—	—	4	38	—	28	—	—	—
Octa-CBs	—	—	—	—	7	—	4	—	—	—
Nona-CBs	—	—	—	—	1	—	—	—	—	—
Deca-CB	—	—	—	—	—	—	—	—	—	—

Columns may not total 100% due to rounding; "—" signifies less than 1%.

PCB mixtures manufactured in the United States carried the trademark "Aroclor" followed by a four-digit number; the first two digits are "12," and the last two digits indicate the percent chlorine content by weight. For example, Aroclor 1260 contains approximately 60 percent chlorine by weight. Aroclor 1016 is an exception to this scheme; it contains approximately 41 percent chlorine. ("Clophens" and "Kanechlors" are PCB mixtures manufactured in Germany and Japan, respectively; these series have their own numbering schemes). Each PCB molecule consists of two six-carbon rings, with one chemical bond joining a carbon from each ring. Chlorine can attach to any of the other ten carbons; these positions are said to be substituted. There are 209 possible arrangements, called congeners; congeners with the same number of chlorines are called isomers. The number and position of the chlorine atoms determine a molecule's physical and chemical properties.

In earlier guidance (before 1996b), USEPA recommended that risk assessments treat all 209 PCB congeners as a mixture having a single carcinogenic potency. This recommendation was based on the Agency drinking water criteria for PCBs (USEPA 1988), and used available toxicological information with the following limitations:

- Aroclor 1260 was the only PCB for which a CSF had been developed; there was no consensus procedure for applying this CSF for PCB mixtures with lower chlorine content.

- Available physical, chemical, fate-and-transport, and toxicological information on individual PCB congeners was limited (primarily because separation and synthesis of pure congeners is technically difficult).

- The number of tests conducted with various PCB mixtures and specific congeners to demonstrate similar toxicological effects was very limited.

In the time since the drinking water criteria were published, USEPA has collected and re-evaluated all of the accumulated research on the carcinogenic potential of PCB Aroclors. The most significant findings were the availability of separate carcinogenesis studies of Aroclors 1016, 1242, 1254, and 1260 in appropriate animal models and a number of studies of the transport and bioaccumulation of various congeners (USEPA 1996b). USEPA used this information to derive three new CSFs to replace the former single CSF for PCBs. These new CSFs became effective in IRIS in 1996. The CSFs and the criteria for their use are as follows (USEPA 1996b):

Slope Factor (milligrams per kilogram-day)$^{-1}$	Criteria for Use
2	Food chain exposure Sediment or soil exposure Early-life (infant and child) exposure by all routes to all PCB mixtures Congeners with more than four chlorines comprise *more than* 0.5 percent of the total PCBs
0.4 (Not Typically Used)	Ingestion of water-soluble (less chlorinated) congeners Inhalation of evaporated (less chlorinated) congeners
0.07	Congeners with more than four chlorines comprise *less than* 0.5 percent of the total PCBs

For evaluation of PCB cancer risk in the SRA, only the highest available CSF is employed for the following reasons:

1) Potential exposures through the food chain and to children are being evaluated
2) The available analytical data on PCBs in Indiana Harbor sediments is reported only as the total PCB Aroclor content; therefore the assumption is made that a significant level of chlorination is present in the PCB sediment mixture (i.e., congeners containing more than four chlorine atoms comprise at least 0.5 percent of the total PCB mixture).

In addition to the CSF associated with PCBs, USEPA recommends application of a noncancer RfD for PCBs. IRIS specifies RfD values for Aroclor 1254 and Aroclor 1016. The RfD for Aroclor 1254 is lower (i.e., more conservative) than the RfD for Aroclor 1016. Therefore, the RfD for Aroclor 1254 is typically used for a risk assessment, such as this SRA, where analytical information is available only for the total Aroclor content. This approach is considered valid and conservative because approximately 77 percent of Aroclor 1254 is composed of PCB congeners with more than four chlorine atoms (USEPA 1996b). In contrast, Aroclor 1016 is composed of only about one percent of PCB congeners with more than four chlorine atoms (Huntzinger et al. 1974).

6.1.4.4 Lead

USEPA does not currently list an RfD or RfC for lead because an absolute threshold level for exposure to lead below which toxic effects are not observed has not been established.

Additionally, based on findings that neurobehavioral effects have been observed in children with blood lead levels well below those that have caused carcinogenic effects in laboratory animals, a CSF has not be derived by USEPA. The Agency relied upon the well-characterized neurological effects observed in children as the sensitive endpoint for evaluating lead toxicity. To apply a protective reference exposure level for lead in children, the goal of the Agency is to limit exposure to lead levels in soil and air such that a typical (or hypothetical) child or group of similarly exposed children would have no more than a five percent probability of exceeding a 10 micrograms per deciliter (µg/dL) blood lead level. This 10 µg/dL blood lead level is based on analyses conducted by the Centers for Disease Control and Prevention (CDC) and USEPA that associate blood lead levels above 10 µg/dL with neurological health effects in children; and this blood lead level is below a level that would trigger medical intervention (USEPA 1994a, 1998a). This strategy is usually employed as part of determining a soil remediation goal for lead at hazardous waste sites (e.g., Superfund, RCRA, Brownfields). But it can also be used to determine an allowable limit for long-term air emission and deposition of lead onto soil in the vicinity of a lead-emitting combustion unit or other lead-emitting source. USEPA has developed an approach called the Integrated Exposure Uptake Biokinetic (IEUBK) Model, which evaluates potential risks by predicting blood lead levels associated with exposure to lead. The IEUBK Model for lead was originally developed by USEPA and collaborators from academia (Kneip et al. 1983; USEPA 1990a). The IEUBK Model integrates a number of characteristics reflecting the complex exposure pattern and physiological handling of lead by the body, and has been validated at several sites where lead exposure data and human blood lead levels are available. The IEUBK Model has been reviewed and recommended by the USEPA Science Advisory Board (USEPA 1992b).

The Agency has now developed a computerized version of the IEUBK Model that predicts blood lead levels and percentage distributions for children ranging in age from infancy to seven years (USEPA 2001a). The IEUBK Model accounts for the major characteristics that influence the uptake and absorption of lead from the environment, including the ability to incorporate default or site-specific values for background levels of lead in air, soil, water, and diet. At present, it is not possible to apply the computer model to predict potential blood lead levels in adults. In general, however, children are more susceptible to lead exposures than adults as a result of higher soil ingestion rates, higher absorption from the gut, nutritional variables and lower body weight. Consequently, in the SRA, environmental concentrations of lead resulting from CDF particulate emissions are used as inputs to the IEUBK Model to predict if child blood lead levels could be significantly affected by lead emissions from the CDF.

6.1.4.5 Mercury

Mercury can exist in either organic or inorganic forms in the environment. Although potential emissions of mercury from CDF particulates are expected to be in an inorganic form (as ionic mercury), it is possible that some portion of the mercury is converted to organic forms (e.g., methylmercury) in the environment after deposition to land or water. The bioaccumulation potential of methylmercury may be as much as ten times greater than that of inorganic forms of mercury. Based on empirical measurements of mercury behavior in water bodies and recommendations from USEPA's combustion risk guidance, the fate and transport modeling in the SRA will assume that 15 percent of the total mercury predicted to be found in the aquatic environment is in the form of methylmercury. The potential health risk for exposure to methylmercury is evaluated using the specific RfD for methylmercury.

6.1.4.6 Chromium

The oxidation state of chromium is important for evaluating the toxicity of this metal and the risks associated with exposure. Hexavalent chromium (Cr^{+6}) is the most toxic valence state of chromium and has been shown to be a respiratory toxicant and a human carcinogen through inhalation exposure. Consequently, the inhalation cancer potential for Cr^{+6} in air is evaluated through an established IUR value, and the inhalation noncancer adverse effect of Cr^{+6} in air is evaluated through an established RfC value. Trivalent chromium (Cr^{+3}) is the most commonly occurring form of chromium in the environment as the naturally-occurring metal (ATSDR 2000). Cr^{+3} has not been shown to be carcinogenic via inhalation or oral exposure in either humans or laboratory animals; it has some potential for inducing noncancer adverse effects via oral exposure, but intake levels must be relatively high.[7] Therefore, the noncancer risk potential for Cr^{+3} is evaluated through an established RfD value.

The analytical data on chromium in the buried sediments associated with the CDF project are reported only as total chromium. No information on the distribution of valence states is available. Consequently, to assess potential health risks in the SRA, two separate procedures are used to evaluate chromium risk. In the first procedure, the total chromium released from the CDF is treated as 100 percent Cr^{+6} for evaluating inhalation cancer risk, inhalation noncancer hazard potential, and oral noncancer hazard potential. In the second procedure, the total chromium released from the CDF is treated as 100 percent Cr^{+3} for evaluating oral noncancer hazard potential. The combination of these procedures should prevent underestimation of health risks due to chromium exposure.

6.1.4.7 Particulate Matter

Particle pollution is a mixture of solid particles and liquid droplets found in the air. Some particles are emitted directly from a source, while others are formed from complicated chemical reactions in the atmosphere. In general, particle pollution consists of a mixture of larger materials, called "coarse particles," and smaller particles, called "fine particles." Coarse particles have diameters ranging from about 2.5 µm to more than 40 µm, while fine particles, also known as known as PM2.5, include particles with aerodynamic diameters equal to or smaller than 2.5 µm. USEPA also monitors and regulates PM10, which refers to particles less than or equal to 10 µm in aerodynamic diameter. PM10 includes coarse particles that are "respirable"—particles ranging in size from 2.5 to 10 µm that can penetrate the upper regions of the body's respiratory defense mechanisms (USEPA 2004c).

Exposure to particles can lead to a variety of serious health effects. Scientific studies show links between these small particles and numerous adverse health effects. Long-term exposures to PM, such as those experienced by people living for many years in areas with high particle levels, are associated with problems such as decreased lung function, development of chronic bronchitis, and premature death. Short-term exposures to particle pollution (hours or days) are associated with a range of effects, including decreased lung function, increased respiratory symptoms, cardiac arrhythmias (heartbeat irregularities), heart attacks, hospital admissions or emergency room visits for heart or lung disease, and premature death (USEPA 1982; 2004c).

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[7] In humans and animals, Cr^{+3} is an essential nutrient that plays a role in glucose, fat, and protein metabolism by enhancing the action of insulin (ATSDR 2000). The biologically active form of chromium, called glucose tolerance factor (GTF), is a complex of chromium, nicotinic acid, and possibly amino acids (glycine, cysteine, and glutamic acid). Both humans and animals are capable of converting inactive inorganic chromium(III) compounds to physiologically active forms.

Respirable particles are those which can penetrate to the lower regions of the respiratory system and enter the lungs, and are generally assumed to be PM10. There are currently no RfD, RfC, or CSF values for either total particles or respirable particles. However, USEPA has established health-based criteria (i.e., the NAAQS) to provide protection against the adverse effects of particulate matter in ambient air. The NAAQS were mandated by the Clean Air Act, which established the following two types of national air quality standards:

1) Primary Standards are limits set to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. The 24-hour Primary NAAQS for PM10 is 150 $\mu g/m^3$.

2) Secondary Standards are limits set to protect public welfare, including protection against decreased visibility, damage to animals, crops, vegetation, and buildings.

Consequently, in the SRA, the predicted 24-hour average airborne concentration of particulate matter released from the CDF was evaluated in view of the current Primary NAAQS for PM10. To account for the episodic nature of particulate emissions from the CDF, the predicted 24-hour average concentration was compared to the 24-hour Primary NAAQS. It should be noted that the NAAQS pertains to monitored ambient air particulate matter concentrations that reflect contributions from all sources, not just the incremental particulate matter concentration attributable to individual sources such as the CDF.

6.1.5 Selection of Chronic Toxicity Factors

Cancer and noncancer toxicity factors for many commonly encountered chemical substances in the environment are published by USEPA in an Internet-accessible database known as IRIS. IRIS is a compilation of toxicity factors and supporting information for the chemical substances. Information in IRIS receives extensive internal and external peer review. At present, IRIS contains recommended toxicity factors for the oral and/or inhalation route of exposure for more than 500 chemical substances. As stated previously, the toxicity factors published in IRIS are routinely applied in USEPA risk assessments to promote consistency and transparency across the various USEPA programs, and because they represent a source of scientifically peer-reviewed information.

For some chemical substances encountered in the environment, IRIS profiles and toxicity factors are not available because the toxicological information is not adequate to assign toxicity factors or because USEPA has not completed the formal peer review process required for entry into the IRIS database. In cases where USEPA believes that a chemical substance not found in IRIS has a toxicological potential, the Agency will review the available information and assign "provisional" or "interim" toxicity factors for use in risk assessment. It is recognized that a higher degree of uncertainty could be associated with the use of these provisional toxicity factors compared to the IRIS-derived factors. In some cases, older toxicity information already contained in IRIS could benefit from updating to better reflect current knowledge. Due to the extensive resource requirements of adding and/or changing IRIS information, a process for developing or revising assessments is in place (see http://www.epa.gov/iris/process.htm).

In order to manage the task of assigning toxicity factors for use in risk assessment, USEPA has developed a recommended hierarchy or priority system for use in selecting toxicity factors for risk assessment. The hierarchy system was developed for the Superfund program but is generally applicable to other investigations where hazardous chemicals would be encountered.

The hierarchy is contained in a policy directive issued by the Superfund program (USEPA 2003b). The hierarchy is summarized as follows:

- Tier 1 – IRIS Toxicity Factors. IRIS remains in the first tier as the preferred source of human health toxicity values. IRIS contains the following preferred values for evaluating toxicity from chronic exposure:

  - Oral exposure toxicity factors
    Oral Reference Doses (RfDs) for effects other than cancer
    Oral CSFs for cancer

  - Inhalation exposure toxicity factors
    Inhalation RfCs for effects other than cancer
    IURs for cancer

- Tier 2 – Provisional Peer Reviewed Toxicity Values (PPRTVs). USEPA ORD National Center for Environmental Assessment (USEPA-NCEA) generates PPRTVs on a chemical specific basis when requested by the USEPA Superfund program. PPRTVs are derived after a review of the relevant scientific literature using the same methods and sources of data employed by the IRIS program. All provisional toxicity values receive internal review by two USEPA scientists and peer review by at least two non-USEPA experts. A third scientific review is performed if there is a conflict between the two original external reviewers. PPRTVs differ in part from IRIS values in that PPRTVs do not receive the multi-program consensus USEPA review and the extensive external peer review provided for IRIS values. This is because IRIS values are generally intended to be used in all USEPA programs, while PPRTVs are developed specifically for the Superfund program. The need for a PPRTV is eliminated once a corresponding IRIS value becomes available.

- Tier 3 – Other Toxicity Values. Tier 3 includes additional USEPA and non-USEPA sources of toxicity information. Priority should be given to those sources of information that are the most current, have a transparent and publicly available basis, and have been peer reviewed. Three primary sources of information have been identified under this Tier:

  - California EPA Reference Exposure Levels (RELs). The California Environmental Protection Agency (CalEPA) publishes toxicity profiles and toxicity values that are peer reviewed and address both cancer and noncancer effects (CalEPA 2002, 2005). The process for developing these profiles is similar to that used by USEPA to develop IRIS values and incorporates significant external scientific peer review. The noncancer information includes available inhalation health risk guidance values expressed as chronic inhalation and oral RELs. CalEPA defines the REL as a concentration level at (or below) which no health effects are anticipated—a concept that is substantially similar to USEPA's noncancer RfD/RfC values. CalEPA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the unit risk estimate (URE), defined similarly to USEPA's IUR.

  - The Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Levels (MRLs). These values are estimates of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse

noncancer health effects over a specified duration of exposure. The ATSDR MRLs are peer reviewed and are available at http://www.atsdr.cdc.gov/mrls.html on the ATSDR website.

- Health Effects Assessment Summary Tables (HEAST) Values (USEPA 1997b). These tables cite dose-response assessments and toxicity factor values for some substances that were prepared by USEPA-ORD but not submitted for internal USEPA consensus. The level of internal USEPA and external peer review is questionable or unknown. For some substances, older health effects assessment documents omitted from HEAST also exist and are generally cited in HEAST Table A-1 as USEPA-NCEA values. In 1989 and for several years thereafter, HEAST was updated on a quarterly basis. HEAST is no longer updated with new values and existing values are deleted as revised values become available on IRIS.

Based on the hierarchy discussed above for selecting toxicity factors for chronic exposure, the applicable toxicity factors (e.g., RfC, RfD, CSF, and IUR values) for the PCOCs in the SRA are presented in Table 6-1. Table 6-1 also presents information on the origin of the toxicity factor (e.g., IRIS, CalEPA, route-to-route extrapolation).

Table 6-1: Toxicity Factors for the PCOCs Identified in the SRA

Constituents	Oral CSF (mg/kg-day)$^{-1}$	IUR ($\mu g/m^3$)$^{-1}$	Oral RfD (mg/kg-day)	Inhalation RfC ($\mu g/m^3$)	Notes
Metals					
Antimony	NA	NA	4E-04 [a]	NA	[a]
Arsenic	1.5 [a]	4.3E-03 [a]	3E-04 [a]	3E-02 [b]	[a], [b]
Barium	NA	NA	0.2 [a]	NA	[a]
Cadmium	NA	1.8E-03 [a]	1E-03 [a]	2E-02 [b]	[a], [b]
Chromium:					
Chromium (+3)	NA	NA	1.5 [a]	NA	[a]
Chromium (+6)	NA	1.2E-02 [a]	3E-03 [a]	1E-01 [a]	[a]
Copper	NA	NA	NA	NA	
Lead	NA	NA	NA	NA	
Manganese	NA	NA	1.4E-01 [a]	5E-02 [a]	[a]
Mercury:					
Mercury (Elemental)	NA	NA	NA	3E-01 [a]	[a]
Mercury (+2)	NA	NA	3E-04 [a]	1.05 [e]	[a], [e]
Methylmercury	NA	NA	1E-04 [a]	NA	[a]
Nickel		2.0E-04 [a]	2E-02 [a]	9E-02 [c]	[a], [c]
Selenium	NA	NA	5E-03 [a]	NA	[a]
Silver	NA	NA	5E-03 [a]	NA	[a]
Zinc	NA	NA	0.3 [a]	NA	[a]
PAHs					
Acenaphthene	NA	NA	6E-02 [a]	2.1E+02 [e]	[a], [e]
Acenaphthylene	NA	NA	NA	NA	
Anthracene	NA	NA	3E-01 [a]	1.05E+03 [e]	[a], [e]
Benzo[a]anthracene	0.73 [f]	1.1E-04 [i]	NA	NA	[f], [i]
BaP	7.3 [a]	1.1E-03 [b]	NA	NA	[a], [b]
Benzo[b]fluoranthene	0.73 [f]	1.1E-04 [i]	NA	NA	[f], [i]
Benzo[k]fluoranthene	0.073 [f]	1.1E-05 [i]	NA	NA	[f], [i]
Benzo[g,h,i]perylene	NA	NA	NA	NA	
Chrysene	0.0073 [f]	1.1E-06 [i]	NA	NA	[f], [i]
Dibenz[a,h]anthracene	7.3 [f]	1.1E-03 [i]	NA	NA	[f], [i]
Fluoranthene	NA	NA	4E-02 [a]	1.4E+02 [e]	[a], [e]
Fluorene	NA	NA	4E-02 [a]	1.4E+02 [e]	[a], [e]
Indeno[1,2,3-cd]pyrene	0.73 [f]	1.1E-04 [i]	NA	NA	[f], [i]
Naphthalene	NA	3.4E-05 [b]	2E-02 [a]	3.0 [a]	[a], [b]
Phenanthrene	NA	NA	NA	NA	
Pyrene	NA	NA	3E-02 [a]	1.05E+02 [e]	[a], [e]
(PCBs) (total)	2.0 [a]	5.7E-04 [d]	2E-05 [a]	7E-02 [e]	[a], [d], [e]

Constituents	Oral CSF (mg/kg-day)$^{-1}$	IUR ($\mu g/m^3$)$^{-1}$	Oral RfD (mg/kg-day)	Inhalation RfC ($\mu g/m^3$)	Notes
Dioxin/Furan Congeners					
2,3,7,8-TCDD	1.5E+05 [c]	3.3E+01 [c]	NA	NA	[c]
1,2,3,7,8-PeCDD	1.5E+05 [g]	3.3E+01 [h]	NA	NA	[g], [h]
1,2,3,4,7,8-HxCDD	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,6,7,8-HxCDD	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,7,8,9-HxCDD	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,4,6,7,8-HpCDD	1.5E+03 [g]	0.33 [h]	NA	NA	[g], [h]
OctaCDD	1.5E+01 [g]	0.0033 [h]	NA	NA	[g], [h]
2,3,7,8-TCDF	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,7,8-PeCDF	7.5E+03 [g]	1.65 [h]	NA	NA	[g], [h]
2,3,4,7,8-PeCDF	7.5E+04 [g]	16.5 [h]	NA	NA	[g], [h]
1,2,3,4,7,8-HxCDF	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,6,7,8-HxCDF	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,7,8,9-HxCDF	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
2,3,4,6,7,8-HxCDF	1.5E+04 [g]	3.3 [h]	NA	NA	[g], [h]
1,2,3,4,6,7,8-HpCDF	1.5E+03 [g]	0.33 [h]	NA	NA	[g], [h]
1,2,3,4,7,8,9-HpCDF	1.5E+03 [g]	0.33 [h]	NA	NA	[g], [h]
OctaCDF	1.5E+01 [g]	0.0033 [h]	NA	NA	[g], [h]
Pesticides and Phenols					
Aldrin	1.7E+01 [a]	4.9E-03 [a]	3E-05 [a]	1.05E-01 [e]	[a], [e]
d-BHC	NA	NA	NA	NA	
Dieldrin	1.6E+01 [a]	4.6E-03 [a]	5E-05 [a]	1.75E-01 [e]	[a], [e]
DDD	2.4E-01 [a]	6.9E-05 [d]	NA	NA	[a], [d]
DDE	3.4E-01 [a]	9.7E-05 [d]	NA	NA	[a], [d]
DDT	3.4E-01 [a]	9.7E-05 [a]	5E-04 [a]	1.75 [e]	[a], [e]
Endosulfan II	NA	NA	6E-03 [a]	2.1E+01 [e]	[a], [e]
Endrin	NA	NA	3E-04 [a]	1.05 [e]	[a], [e]
Heptachlor	4.5 [a]	1.3E-03 [a]	5E-04 [a]	1.75 [e]	[a], [e]

Constituents	Oral CSF (mg/kg-day)$^{-1}$	IUR ($\mu g/m^3$)$^{-1}$	Oral RfD (mg/kg-day)	Inhalation RfC ($\mu g/m^3$)	Notes
Heptachlor Epoxide	9.1 [a]	2.6E-03 [a]	1.3E-05 [a]	4.55E-02 [e]	[a], [e]
Lindane	1.8 [a]	5.1E-04 [a]	3E-04 [a]	1.05 [e]	[a], [e]
Phenol	NA	NA	3E-01 [a]	1.05E+3 [e]	[a], [e]
Toxaphene	1.1 [a]	3.2E-04 [a]	NA	NA	[a]
VOCs					
Benzene	5.5E-02 [a]	7.8E-06 [a]	4E-03 [a]	3.0E+1 [a]	[a]
Ethylbenzene	NA	NA	1E-01 [a]	1E+03 [a]	[a]
PCE	5.4E-01 [b]	5.9E-06 [b]	1E-02 [a]	3.5E+01 [e]	[a], [b], [e]
Toluene	NA	NA	8E-02 [a]	5E+03 [a]	[a]
TCE	1.3E-02 [b]	2E-06 [b]	NA	6E+02 [b]	[b]
Xylene (meta-para)	NA	NA	0.2 [a]	100 [a]	[a]
Xylene (ortho)	NA	NA	0.2 [a]	100 [a]	[a]

Notes:
[a] – From USEPA IRIS database
[b] – USEPA Tier 3 chronic toxicity factor value as published by CalEPA
[c] – USEPA Tier 3 chronic toxicity factor values
[d] – IUR Factor value determined by route-to route extrapolation from the Oral CSF
[e] – Inhalation Reference Concentration factor value determined by route-to route extrapolation from the Oral Reference Dose
[f] – Oral CSF is based on the Relative Potency Factor for this PAH and the listed Oral CSF for BaP
[g] - Oral CSF is based on the TEF for this dioxin/furan congener and the listed oral CSF for 2,3,7,8-TCDD
[h] – IUR is based on the TEF for this dioxin/furan congener and the listed IUR for 2,3,7,8-TCDD
[i] – IUR is based on the RPF for this PAH and the listed IUR for BaP
NA – Not Available; no recommended value is available for this exposure route.

6.1.6 Toxicity Associated with Short-Term Exposure

As stated previously, the primary focus of the SRA is on long-term operating emissions from the CDF, which is scheduled to be open for disposal of project sediments for 30 years until the CDF site will be capped. Consequently, for the long-term operation of the CDF, emission limits of volatiles and particulate matter were modeled based on the annual air emission limits imposed by the CDF New Source Registration issued by the State of Indiana. The annual air emission limits were used to calculate volatile and particulate matter emission rates from the CDF. These rates were incorporated into the ISCST3 air dispersion model to predict average air concentrations for modeling chronic inhalation risk for volatiles and particulate matter and for predicting the average annual deposition rates of volatiles and particulate matter onto soil.

In addition to long-term annual emission rates and long-term exposure scenarios, the potential for short-term or "acute" exposure to contaminants is recognized. This is because fluctuations in the meteorological conditions (e.g., wind speed, wind direction, calm conditions) will cause periodic deviations from the average annual predictions for air concentrations of contaminants. From the standpoint of potential health risk due to CDF emissions, short-term deviations above the average exposure will essentially be limited to a concern for the inhalation exposure pathway. For indirect pathway exposures (e.g., ingestion, dermal), the level of exposure is

based on the long-term deposition and accumulation of contaminants, which is much less dependent on fluctuations from average meteorological conditions.

The site-specific meteorological data combined with the ISCST3 Model output can be used to identify time periods associated with short-term air concentrations of volatiles, total particulate matter, and particulate matter contaminants that are higher than the average case. The typical procedure is to examine the ISCST3 Model output to identify the highest predicted air concentrations of contaminants occurring over time periods ranging from one to 24 hours. These concentrations are subsequently compared to health-based benchmarks for chemical constituents that are commonly referred to as Acute Inhalation Exposure Criteria (AIEC).

AIEC values are air concentration guidelines designed to protect a variety of potentially exposed groups including the general public, occupational workers, and military personnel. The values are based on varying exposure durations up to 24 hours in length, and are intended to protect against a variety of adverse health endpoints ranging from slight discomfort to mild, reversible health effects up to serious, debilitating, and potentially life-threatening effects.

AIEC values are developed by several health research and regulatory organizations in the United States. USEPA has recommended a general hierarchy for identifying AIEC values that are appropriate to apply as short-term guideline values for protection of the general population, including sensitive groups (e.g., children, the elderly). The hierarchical approach is recommended because AIEC values are chemical-specific and no uniform methodology has been devised for developing criteria values or benchmarks for all of the potential environmental and industrial chemicals. The hierarchy appears in the USEPA HHRAP guidance for hazardous waste combustor emissions, and is summarized below (USEPA 2005c):

1) Cal/USEPA Acute RELs – the concentration of a chemical in air at or below which no adverse health effects are anticipated in the general population, including sensitive individuals, for a specified short duration exposure period (CalEPA 1999). http://www.oehha.ca.gov/air/pdf/acuterel.pdf

2) Acute Inhalation Exposure Guidelines – Level 1 (AEGL-1) – defined as "the airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure" (NOAA 2001; USEPA 2001b). http://www.epa.gov/oppt/aegl/chemlist.htm

3) Emergency Response Planning Guidelines – Level 1 (ERPG-1) – defined as "the maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor" (USDOE 2001; SCAPA 2001). http://www.bnl.gov/scapa/scapawl.htm

4) Temporary Emergency Exposure Limits (TEEL-1) – defined as "the maximum concentration in air below which it is believed nearly all individuals could be exposed without experiencing other than mild transient adverse health effects or perceiving a clearly defined odor" (USDOE 2001; SCAPA 2001). http://tishq.eh.doe.gov/web/Chem_Safety/teel.html

5) AEGL-2 values – defined as "the airborne concentration 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." (An AEGL-2 value should only be used if a lower ERPG-1 or TEEL-1 value is not available for a given chemical). (NOAA 2001; USEPA 2001b) http://www.epa.gov/oppt/aegl/chemlist.htm

The guidelines listed above are those which have received the most extensive scientific peer review as applicable guidelines for protection of the general population. The hierarchy is presented in order of preference, from 1 (most preferred) to 5 (least preferred).

6.1.7 Acute Inhalation Exposure Criteria Values for Potential Chemicals of Concern from CDF Emissions

Based on the selection hierarchy discussed above, relevant AIEC values for the PCOCs for the SRA are presented in Table 6-2. This table presents the compilation of all published AIECs. Since the hierarchy is a general recommendation, if a chemical has multiple AIEC values, all values are listed along with the applicable exposure duration time. For respirable particulate matter (i.e., PM10) there are no AIEC values since PM10 is not a specific chemical constituent. To address short-term exposure to PM10, USEPA has promulgated a Primary Standard for short-term exposure. The Standard requires that PM10 should not exceed 150 $\mu g/m^3$ for a 24-hour period.

Table 6-2: Acute Inhalation Exposure Criteria Values for PCOCs in the SRA

Constituents	Cal USEPA Acute REL	AEGL-1	ERPG-1	TEEL-1
Metals				
Antimony	NA	NA	NA	1.5E+03 µg/m
Arsenic[8]	1.9E-01 µg/m^3 4-hour value	NA	NA	3.0E+01 µg/m^3
Barium	NA	NA	NA	1.5E+03 µg/m^3
Cadmium	NA	NA	NA	3.0E+01 µg/m^3
Chromium:				
Chromium (+3)	NA	NA	NA	1.5E+03 µg/m^3
Chromium (+6)	NA	NA	NA	3.0E+01 µg/m^3
Copper	1.0E+02 µg/m^3 1-hour value	NA	NA	3.5E+03 µg/m^3
Lead	NA	NA	NA	1.5E+02 µg/m^3
Manganese	NA	NA	NA	7.5E+02 µg/m^3
Mercury:				
Inorganic Mercury	1.8E+00 µg/m^3 1-hour value	NA	NA	1.0E+02 µg/m^3
Methylmercury	NA	NA	NA	3.0E+01 µg/m^3
Nickel	6.0E+00 µg/m^3 1-hour value	NA	NA	7.5E+02 µg/m^3
Selenium	NA	NA	NA	6.0E+02 µg/m^3
Silver	NA	NA	NA	3.0E+02 µg/m^3
Zinc	NA	NA	NA	1.5E+04 µg/m^3
PAHs				
Acenaphthene	NA	NA	NA	1.25E+03 µg/m^3
Acenaphthylene	NA	NA	NA	2.0E+02 µg/m^3
Anthracene	NA	NA	NA	4.0E+02 µg/m^3
Benzo[a]anthracene	NA	NA	NA	3.0E+02 µg/m^3
BaP	NA	NA	NA	6.0E+02 µg/m^3
Benzo[b]fluoranthene	NA	NA	NA	6.0E+02 µg/m^3

[8] CalEPA only lists a four-hour REL value.

Constituents	Cal USEPA Acute REL	AEGL-1	ERPG-1	TEEL-1
Benzo[k]fluoranthene	NA	NA	NA	6.0E+02 µg/m^3
Benzo[g,h,i]perylene	NA	NA	NA	3.0E+04 µg/m^3
Chrysene	NA	NA	NA	6.0E+02 µg/m^3
Dibenz[a,h]anthracene	NA	NA	NA	3.0E+04 µg/m^3
Fluoranthene	NA	NA	NA	1.0E+01 µg/m^3
Fluorene	NA	NA	NA	2.5E+04 µg/m^3
Indeno[1,2,3-cd]pyrene	NA	NA	NA	5.0E+02 µg/m^3
Naphthalene	NA	NA	NA	7.86E+04 µg/m^3 (15 parts per million [ppm])
Phenanthrene	NA	NA	NA	1.0E+03 µg/m^3
Pyrene	NA	NA	NA	7.5E+03 µg/m^3
PCBs				
(PCBs) (total)	NA	NA	NA	3.0E+03 µg/m^3
Dioxin/Furan Congeners				
2,3,7,8-TCDD[9]	NA	NA	NA	2.5 µg/m^3
1,2,3,7,8-PeCDD	NA	NA	NA	2.5 µg/m^3
1,2,3,4,7,8-HxCDD	NA	NA	NA	1.25 µg/m^3
1,2,3,6,7,8-HxCDD	NA	NA	NA	15 µg/m^3
1,2,3,7,8,9-HxCDD	NA	NA	NA	15 µg/m^3
1,2,3,4,6,7,8-HpCDD	NA	NA	NA	500 µg/m^3
OctaCDD	NA	NA	NA	10 µg/m^3
2,3,7,8-TCDF	NA	NA	NA	2.0 µg/m^3
1,2,3,7,8-PeCDF	NA	NA	NA	7.5 µg/m^3
2,3,4,7,8-PeCDF	NA	NA	NA	0.075 µg/m^3
1,2,3,4,7,8-HxCDF	NA	NA	NA	7.5 µg/m^3
1,2,3,6,7,8-HxCDF	NA	NA	NA	2.5 µg/m^3
1,2,3,7,8,9-HxCDF	NA	NA	NA	12.5 µg/m^3

[9] A TEEL-1 for 2,3,7,8-TCCD is not published. The published TEEL-1 value for 1,2,3,7,8-PeCDD is used as the surrogate value because these two congeners have the same TEF value.

Constituents	Cal USEPA Acute REL	AEGL-1	ERPG-1	TEEL-1
2,3,4,6,7,8-HxCDF	NA	NA	NA	1.5 µg/m^3
1,2,3,4,6,7,8-HpCDF	NA	NA	NA	150 µg/m^3
1,2,3,4,7,8,9-HpCDF	NA	NA	NA	250 µg/m^3
OctaCDF	NA	NA	NA	7.5 µg/m^3
Pesticides and Phenols				
Aldrin	NA	NA	NA	7.5E+02 µg/m^3
d-BHC	NA	NA	NA	1.5E+03 µg/m^3
Dieldrin	NA	NA	NA	7.5E+02 µg/m^3
DDD	NA	NA	NA	3E+04 µg/m^3
DDE	NA	NA	NA	3E+04 µg/m^3
DDT	NA	NA	NA	3E+03 µg/m^3
Endosulfan II	NA	NA	NA	3E+02 µg/m^3
Endrin	NA	NA	NA	3E+02 µg/m^3
Heptachlor	NA	NA	NA	1.5E+02 µg/m^3
Heptachlor Epoxide	NA	NA	NA	1.5E+02 µg/m^3
Lindane	NA	NA	NA	1.5E+03 µg/m^3
Phenol	NA	NA	NA	3.84E+04 µg/m^3 (10 ppm)
Toxaphene	NA	NA	NA	1E+03 µg/m^3
VOCs				
Benzene[10]	1.3E+03 µg/m^3 6-hour value	1.7E+05 µg/m^3 (52 ppm) – 1 hour 2.9E+04 µg/m^3 (9 ppm) – 8 hour	1.6E+05 µg/m^3 (50 ppm)	1.6E+05 µg/m^3 (50 ppm)
Ethylbenzene	NA	NA	NA	5.42E+05 µg/m^3 (125 ppm)
PCE	2.0E+04 µg/m^3 1-hour value	2.37E+05 µg/m^3 (35 ppm) – 1 hour 2.37E+05 µg/m^3 (35 ppm) – 8 hour	6.78E+05 µg/m^3 (100 ppm)	2.37E+05 µg/m^3 (35 ppm)
Toluene	3.7E+04 µg/m^3 1-hour value	7.52E+05 µg/m^3 (200 ppm) – 1 hour 7.52E+05 µg/m^3 (200 ppm) – 8 hour	1.88E+05 µg/m^3 (50 ppm)	7.52E+05 µg/m^3 (200 ppm)
TCE	NA	6.98E+05 µg/m^3 (130 ppm) – 1 hour	5.37E+05 µg/m^3 (100 ppm)	6.98E+05 µg/m^3 (130 ppm)

[10] CalEPA lists only a 6-hour REL value.

Constituents	Cal USEPA Acute REL	AEGL-1	ERPG-1	TEEL-1
		4.13E+05 µg/m^3 (77 ppm) – 8 hour		
Xylene	2.2E+04 µg/m^3 1-hour value	5.64E+05 µg/m^3 (130 ppm) – 1 hour 5.642E+05 µg/m^3 (130 ppm) – 8 hour	NA	5.64E+05 µg/m^3 (130 ppm)

NA = Value not available

6.1.8 Toxicology Uncertainties and Limitations

In the majority of risk assessments, as in this SRA, available scientific information is insufficient to provide a complete understanding of all the toxic properties of chemicals to which humans are potentially exposed. Therefore, it is often necessary to infer these properties by extrapolating them from data on the toxicological effects of chemicals in laboratory animals under controlled conditions. Experimental animal data have been relied upon for many years by regulatory agencies and other expert groups for assessing the hazards and safety of human exposure to chemicals. This reliance has been supported in general by empirical observations. There may be differences in chemical absorption, metabolism, excretion, and toxic response, however, between humans and the species for which experimental toxicity data are available. Uncertainties associated with the characterization of chemical toxicity in humans are also introduced as a result of the following (USEPA 1989):

- Using dose-response information from effects observed at relatively high exposure levels to predict effects that may occur following exposure to the much lower exposure levels expected from contact with the chemical in the environment
- Using data from one route of exposure to predict effects from exposure via other routes
- Using dose-response data from short-term or subchronic exposures to predict the effects following longer-term exposure
- Using dose-response information from homogeneous animal populations or healthy human populations to predict effects that may occur in the general population, including sensitive subpopulations.

The methods for addressing these uncertainties in the toxicological assessment for cancer and noncancer effects are discussed below.

6.1.8.1 Uncertainties in the Characterization of Carcinogenic Effects

For many substances that are carcinogenic in animals, there is uncertainty as to whether they are also carcinogenic in humans. The USEPA Office of Science and Technology Policy (USEPA 1985) has stated the following:

> ...known human carcinogens, with the single exception of arsenic, are carcinogenic in appropriately conducted studies in some animal system (arsenic has recently been reported to produce carcinomas of the respiratory tract in hamsters). This does not mean that all chemicals found carcinogenic in animals will turn out to be carcinogenic in humans. Because of differences in the production of critical metabolites and because of

other differences between species, a given carcinogen may not produce cancer in all species or in all strains of rodents.

The finding that relatively few substances are known human carcinogens may be due in part to the difficulty in conducting adequately designed epidemiologic investigations in exposed human populations. The available data in humans are derived mainly from retrospective epidemiology studies of workers exposed to multiple chemicals and at dose levels that cannot be confirmed with a high degree of reliability.

All CSFs in IRIS are accompanied by a weight-of-evidence classification, which is an indication of the likelihood that the agent is a human carcinogen. This classification is based on the completeness of the evidence that the agent causes cancer in experimental animals and humans. The strength of the evidence that an animal carcinogen is a potential human carcinogen is enhanced by such factors as the following: (1) a carcinogenic response in more than one species, strain, and sex, and by multiple routes of exposure; (2) evidence of a clearly definable dose-response relationship; (3) a high level of statistical significance of the increased tumor incidence in treated compared to control groups; (4) a dose-related shortening of the time-to-tumor occurrence or time to death with tumors; (5) a dose-related increase in the proportion of tumors that are malignant; (6) a plausible biological mechanism for tumorigenicity; (7) similar carcinogenic properties exhibited by structurally-related compounds; and (8) evidence of an association between exposure to the chemical of concern and an increased tumor incidence in human populations (USEPA 1986b, 1989).

Because of uncertainties associated with the measure of carcinogenic potency of a chemical in humans, USEPA has adopted procedures in the calculation of CSFs that are generally conservative. For example, USEPA uses the biologically acceptable data set from long-term animal studies showing the greatest sensitivity. There are several mathematical models available to derive low-dose unit risks from high exposure levels used in experimental studies. No single model is recognized as the most appropriate for low-dose extrapolation. The model generally used by USEPA is the linear multistage model, which provides the most conservative estimate of risk at low doses (i.e., highest risk per unit dose). The procedure employed by USEPA is also to use the 95 percent upper confidence limit on the slope of the dose-response curve estimated by the linear multistage model. According to USEPA, use of the 95 percent upper confidence limit value provides an estimate of the upper boundary on the actual risk (USEPA 1989).

6.1.8.2 Uncertainties in the Characterization of Noncancer Effects

To adjust for uncertainties, USEPA and other regulatory agencies typically base the RfD or RfC for noncancer effects on the most sensitive animal species—the species that experiences adverse effects at the lowest experimental dose. This experimental dose is then adjusted downward by the use of uncertainty and modifying factors to compensate for various sources of uncertainty in the underlying toxicity data. The resulting toxicity factor incorporates a substantial margin of safety, although the actual magnitude of this safety margin cannot be quantified with certainty.

For all verified RfD and RfC values, USEPA provides in IRIS a qualitative statement of the confidence that the evaluators have in the following: the RfD or RfC, the critical study upon which the RfD or RfC is based, and the overall database.

6.1.8.3 Uncertainties Associated with Route-to-Route Extrapolation

USEPA HHRAP guidance for emissions from hazardous waste incinerators (USEPA 1998b, 2005c) suggests that when a verified oral RfD has been developed for a given chemical by USEPA, but there is no verified RfC, the RfD value should be extrapolated to a provisional inhalation RfC value. And conversely, where a verified inhalation RfC has been developed but there is no verified RfD for that chemical, the RfC should be used to extrapolate a provisional RfD value. This extrapolation approach may introduce significant quantitative uncertainties in the estimate of noncancer effects from inhalation or ingestion of these chemicals. While this is not an optimal approach for risk assessment practice, the alternative would be to omit the inhalation or oral route of exposure from the quantitative risk estimate, and cause a potential underestimation of the risk from the omitted exposure route. Therefore, using route-to-route extrapolation of oral dose-response or inhalation information is considered preferable when no toxicity factor value is available in the peer reviewed data sources. However, assumptions and uncertainties involved when using toxicity factors calculated based on route-to-route extrapolation should limit their use to screening-level or priority type risk assessments (USEPA 2005c).

As discussed in USEPA guidance for derivation of RfCs (USEPA 1994b), the ability to perform quantitative route-to-route extrapolations is critically dependent on the availability of chemical-specific data on both the capabilities of the chemical to reach the target site for toxicity and the nature of the toxic effect. In cases where these data are not available, the use of default assumptions to perform the route-to-route extrapolation results in increased uncertainty associated with the derived RfC value. The magnitude of the uncertainty will be chemical-specific, and determined by the level of understanding provided by the supporting database as to the response of the human body to exposure to the chemical.

There may be several explanations for differences in toxicity when the route of administration differs, but the primary reason is likely to be related to the pharmacokinetics (i.e., absorption, distribution, metabolism, and excretion) of the chemical. Different routes of exposure may influence the factors that affect absorption at the portal of entry, such as the chemical's physicochemical properties (e.g., dissociation state, solubility, reactivity), the nature of the exposure (e.g., concentration, duration, regimen), or the physiologic parameters of the exposed tissues (e.g., metabolic capabilities, cell types, pH). Similarly, factors that affect the distribution of the chemical to the various tissues in the body (e.g., solubility, chemical reactivity), the metabolism of the chemical (e.g., metabolic activation vs. metabolic detoxification, metabolic capabilities of exposed tissues), and the excretion of the chemical from the body (e.g., rate of clearance, site of excretion) all may be significantly affected by the route of exposure. Thus, different routes of exposure may have a strong impact on the delivered dose of the chemical at the target site of toxicity. For example, the portal of entry of the chemical may be exposed to relatively high concentrations of the chemical. If the chemical acts directly on the local tissue (e.g., stomach or respiratory tract), or if the local tissue can metabolize the chemical to an active form, and if that tissue is susceptible to the effect of the chemical or its metabolites, lesions may arise preferentially at the site of administration. Chemicals administered orally (either by gavage, in feed, or in drinking water), pass directly from the gastrointestinal tract via the portal system to the liver, and thus may be subject to "first-pass" metabolism. This in turn may either increase or decrease the toxic response, depending upon whether the liver detoxifies or activates the chemical, and what tissue is susceptible to the toxic effects of the chemical. A similar chain of events may occur for inhalation exposures, where metabolism of the inhaled chemical may occur at sites along the respiratory tract, thereby presenting the surrounding tissues, and, assuming systemic absorption, remote tissues with a metabolically-modified

chemical. Thus, one factor that must be considered in route-to-route extrapolations is the metabolic capabilities of the tissues (in terms of both quantity and type of metabolites produced) at the different portals of entry.

The chemicals of concern for which the procedure of route-to-route extrapolation is used to assign an RfC value for the SRA is noted in Table 6-1.

6.1.8.4 Uncertainties Associated With Substances Without Toxicity Factor Values

Where experimental data on the toxicity of a given contaminant are so limited that no valid toxicity factor value can be derived (i.e., either IRIS or provisional), potential cancer risks and noncancer health effects posed by the chemical through the applicable exposure routes are not evaluated quantitatively. However, most of the chemicals that are commonly encountered as environmental contaminants in sediments, and that also possess a significant potential for exposure and potential to cause cancer and/or noncancer toxic effects, have already been identified and studied. For example, the National Toxicology Program has studied the carcinogenic potential of approximately 450 chemicals to date and published the results in peer-reviewed, publicly available reports (Huff 1996). Other investigators have studied the carcinogenic potential of about 800 additional chemicals (Gold et al. 1995). The noncancer toxic effects of a much larger number of chemicals have been investigated. Information on these effects is published in a number of publicly available databases. Thus, the degree of underestimation of risks based on not evaluating the toxicities of chemicals with inadequate testing results, although not quantifiable, is expected to be relatively low.

6.2 Selection of Contaminants of Concern for the SRA

Based on the evaluation of sampling data on chemical constituents from Indiana Harbor sediments, a list of PCOCs was developed for the SRA (Table 4-2). This PCOC list includes all of the chemical constituents identified in the analytical data sets that were judged to be valid for use in the SRA (Section 4).

The next step in the chemical-specific evaluation is to select a set of chemical constituents that will be carried forward into the quantitative evaluation of cancer risk and noncancer hazard. This set will be designated as the contaminants of concern (COCs) for the SRA. The COCs represent the chemicals which could contribute most significantly to the cancer risk and toxic hazard due to a combination of the following factors: concentration levels in the buried sediments; level of cancer or toxic potency; and relative capacity to persist, biotransfer, and bioaccumulate in the environment if released from the CDF. The COCs are the chemicals that will be carried through all of the subsequent quantitative steps of the SRA.

The following criteria were used to develop the COC list. These criteria were judged to be consistent with: 1) the objectives for evaluating risk in the SRA; and 2) the procedures used for selecting COCs for risk assessment, as outlined in USEPA guidance documents for evaluating risks from chemical releases at hazardous waste sites and from combustion emission facilities (USEPA 1989, 1998b, 2005c).

1) PCOCs not detected in any sediment sample at a concentration level equal to or above their corresponding analytical sample detection/reporting limit are not carried forward to the COC list. PCOCs in this category are: silver, dibenzo[a,h]anthracene, endrin, heptachlor, heptachlor epoxide, lindane, phenol, toxaphene, PCE, and TCE.

2) If a PCOC was detected above the analytical sample detection/reporting limit but it possesses no verifiable toxicity factors from any database of acceptable toxicity factors, that PCOC is not carried forward to the COC list. PCOCs in this category are: copper, acenaphthylene, benzo[g,h,i]perylene, and d-BHC (delta-lindane).

3) For estimating the inhalation risk from volatile constituents, select from the COPC list the volatile constituent that possesses the highest IUR, and the volatile constituent that possesses the lowest inhalation RfC. Assume that these chemicals represent 100 percent of the allowable annual volatile emissions from the CDF for the purpose of estimating inhalation cancer risk and inhalation noncancer hazard (see Section 4). Based on a review of the toxicity factors for the volatile COPCs, naphthalene is the volatile constituent which possesses both the highest IUR and the lowest RfC. Therefore, naphthalene is the constituent carried forward to the COC list as the surrogate for volatile emissions.

4) All remaining PCOCs not addressed by criteria 1, 2, or 3 above are carried forward to the COC list.

After application of the above criteria, the COC list includes the following constituents:

Metals	Dioxin/Furan Congeners
Antimony	2,3,7,8-TetraCDD
Arsenic	1,2,3,7,8-PentaCDD
Barium	1,2,3,4,7,8-HexaCDD
Cadmium	1,2,3,6,7,8-HexaCDD
Chromium	1,2,3,7,8,9-HexaCDD
Lead	1,2,3,4,6,7,8-HeptaCDD
Manganese	OctaCDD
Mercury	2,3,7,8-TetraCDF
Nickel	1,2,3,7,8-PentaCDF
Selenium	2,3,4,7,8-PentaCDF
Zinc	1,2,3,4,7,8-HexaCDF
	1,2,3,6,7,8-HexaCDF
SVOCs	1,2,3,7,8,9-HexaCDF
Acenaphthene	2,3,4,6,7,8-HexaCDF
Anthracene	1,2,3,4,6,7,8-HeptaCDF
Benzo[a]anthracene	1,2,3,4,7,8,9-HeptaCDF
BaP	OctaCDF
Benzo[b]fluoranthene	
Benzo[k]fluoranthene	**Pesticides**
Chrysene	Aldrin
Fluoranthene	Dieldrin
Fluorene	DDD
Indeno[1,2,3-cd]pyrene	DDE
Naphthalene	DDT
Phenanthrene	Endosulfan II
Pyrene	
	Other
PCBs	Particulate Matter
PCBs (total)	

6.3 Exposure Assessment

The exposure assessment step of the risk assessment involves the identification of the potentially exposed population and the measurement or estimation of the magnitude of chemical contaminant exposure to individuals in that population. This section of the SRA describes the steps used in assessing exposure to the population residing in the vicinity of the SRA. Within the exposed population, the magnitude of exposure is expected to vary by individual, due primarily to differences in residential location, and differences in individual characteristics and activity patterns. Therefore, a distribution of exposures exists across the population. Direct measurement of this exposure distribution cannot readily be performed. Therefore, subgroups within the population are identified which are expected to have similar exposure because of similarities in activity and behavior patterns. A population subgroup which shares similar activity and behavior patterns and a similar combination of exposure pathways is described by a specific exposure scenario. This section also describes the methods used to: (1) develop a geographically-based "Study Area" for evaluating potential contaminant exposure in the vicinity around the CDF; (2) estimate Study Area chemical contaminant concentrations in environmental media based on the fate and transport properties of chemicals in the environment; and (3) estimate amounts of contaminant intakes within a population subgroup described by a specific exposure scenario.

6.3.1 Study Area

The Study Area map for the SRA project (Figure 6-1) was generated using ArcGIS 9.1™ software. In creating the map, sequential data layers were added in ArcGIS ArcMap™ application software.[11] Data layers were assembled in the following order: (1) adding a topographic map layer and Digital Ortho Quarter Quads (aerial photos); (2) drawing polygons to outline geographic areas of interest, such as the six residential and school zones; 3) adding line features; and (4) by adding point features. The U.S. Geological Survey (USGS) topographic map layer is displayed at 60 percent transparency so that aerial photography can be visible on the map.

Data layers included in the map were obtained from several sources. The Digital Ortho Quarter Quads (aerial photos) and the topographic map were obtained from USGS. The Indiana Harbor Navigational Channel and the CDF layers are Computer Aided Design (CAD) files from USACE. These CAD layers were converted to a shapefile in an ESRI data format. This was accomplished by adding the data layer to the ArcMAP application and then exporting the data layer as an ESRI shapefile. All other layers were generated and provided by USEPA staff.

The locations of the air monitoring sites were verified during a site visit where a Global Positioning System (GPS) instrument was used to determine latitude-longitude (LAT-LONG) coordinates. These coordinates were then imported into the ArcMap application software and generated into an ESRI event file for placement onto the map. The event file was exported as an ESRI shapefile for use in mapping. The neighborhood/school zone layers and the water body layers were generated by digitizing the polygons using ArcGIS ArcMap editing tools. The aerial photos and topographic map were used as reference layers in the digitizing of the neighborhood/school zone layers and the water body layers.

[11] ArcGIS 9.1™ and ArcMap™ application software are proprietary products from ESRI; Redlands, CA.

6.3.2 Exposure Scenarios

Based on the expected behaviors of populations in the vicinity of the CDF source and the environmental media that could be impacted by airborne chemical contaminant releases from the CDF, there could be a number of exposure scenarios selected for a risk assessment. After evaluation of the likely potential contaminant exposures expected in the vicinity of the CDF, and after conducting site visits to the CDF locale and discussions with local citizens, the following exposure scenarios were selected for the SRA:

6.3.2.1 Local Area Resident

A local area resident is defined as an adult or child who lives within a known existing residential area in the vicinity of the CDF. Specific residential areas were selected based on their distance and direction from the CDF site. The selected residential areas were assigned geographic boundaries determined by evaluating zoning maps, USGS Quadrant maps, local land use maps, and site visits. Based on the geographic analysis, the following neighborhoods or residential zones were selected for evaluation in the SRA. (These zones are illustrated in Figure 6-1).

Residential Zone or Neighborhood	Approximate Distance from Center of CDF	Approximate Direction from CDF	Municipality or Political Entity
Calumet	2.8 km	Southeast	East Chicago
Hammond	2.2 km	Southwest	Hammond
Marktown	1.5 km	Northeast	East Chicago
North Harbor / East Harbor	2.0 km	East/Southeast	East Chicago
Northside / Southside	1.4 km	South	East Chicago
Robertsdale	1.1 km	North/Northwest	Whiting

The exposure pathways that apply to any individual living within a residential area are:

- Inhalation of volatile contaminants and particulate contaminants
- Incidental ingestion of soil containing deposited contaminants
- Incidental dermal contact with contaminants in soil
- Ingestion of contaminants incorporated into produce from a typical home garden.

6.3.2.2 Local Area Student

A local area student is defined as a school age child or teenager who attends school in the vicinity of the CDF. Specific schools were selected by evaluating local maps and through site visits. Based on the evaluation, two schools were selected based on geographic proximity to the CDF[12]. The two schools are:

West Side Junior High – 4001 Indianapolis Blvd., East Chicago, IN
East Chicago Central High School – 1100 W. Columbus Dr., East Chicago, IN

[12] A total of ten public schools were identified in East Chicago. Based on proximity to the CDF and the prevailing wind direction for particle release predicted by the WEPS Model, the two selected school locations would be expected to have higher impacts of contaminants from air and soil than other school locations. In addition, these two schools are more likely to have the types of outdoor athletic facilities and athletic programs which would favor more significant contact time and contact opportunity with contaminants in air and soil.

Both schools are located at approximately the same distance and direction from the CDF; 1.2 km south of the CDF, at the intersection of Indianapolis Blvd. and Columbus Dr. (The school locations are illustrated by the area outlined in green color on Figure 6-1).

Due to the proximity of the two schools, they were combined into a single location for the purpose of evaluating potential contaminant exposure under the Student scenario.

The exposure pathways that apply to any student attending school are:

- Inhalation of volatile contaminants and particulate contaminants during school attendance
- Incidental ingestion of soil contaminants during school attendance or as a "student-athlete"
- Dermal contact with soil contaminants during activities typical of a "student-athlete" during school attendance.

6.3.2.3 Local Area Fisher

A local area fisher is defined as an adult or child who consumes fish obtained from a local water body and is also a local area resident. A candidate local water body is one which is located partially or completely within the 10 x 10-km air model study area and which is assumed to support the habits of a "subsistence fisher." A subsistence fisher is a term that is often applied to a high-end fish consumer who obtains a significant proportion of dietary protein through fish harvested from a local water body. Based on water bodies located in the study area and a review of available information on the likelihood for a significant level of fishing from local water bodies, Lake George and Powderhorn Lake were chosen as the water bodies for evaluating contaminant exposure through fish consumption.[13] Lake George is located northwest of the CDF, with a southern shoreline located approximately 1.9 km from the CDF. Powderhorn Lake is located west of the CDF (in the State of Illinois), with an eastern shoreline located approximately 3.7 km from the CDF. As part of the evaluation, several other water bodies were considered, but determined to be inappropriate for inclusion in the SRA. Specifically, there are several, relatively small water bodies located to the southwest and west of the CDF on property currently owned and operated by the BP Amoco Refinery. These include an inactive borrow pit, informally referred to as Lake Mary; a second inactive borrow pit used for catalyst disposal from refinery operations; and an inactive turning basin just north of the Lake George Branch Canal. All of these water bodies are within the boundary of the BP Amoco facility. It was confirmed by BP Amoco personnel that access by the public is restricted and controlled, and recreation or fishing in these water bodies by anyone from the public or refinery employees is strictly unauthorized. Some of these water bodies, such as the catalyst pond, will be undergoing remediation actions by the refinery. In addition, Lake Mary and adjacent wetlands are subject to

[13] Information sources on local water bodies were reviewed. The primary information sources were from the Indiana Department of Natural Resources (IDNR; http://www.in.gov/dnr) and the Illinois State Water Survey (ISWS; http://www.sws.uiuc.edu). IDNR and ISWS present information on water bodies that are recommended for fishing, and would therefore be candidate water bodies for supporting the needs of a high-end fish consumer. Other water bodies are located partially within the Study Area, including the GCR and the IHSC. Available information indicates that these water bodies could not likely support the fish harvest needs of a high-end fish consumer, and/or these water bodies have advisories for fish consumption because of historical problems with chemical contamination and poor water quality (http://www.epa.gov/glnpo/aoc/grandcal.html). In addition, since sediment currently in the IHSC is the source of the material which is being placed into the CDF, it is not appropriate that an incremental risk from emissions should be calculated, when in fact, the dredging and removal of the material will result in an overall net decrease of contaminants in those areas.

a federal consent decree entered pursuant to the Natural Resource Damage Act whereby BP Amoco will, at some future date, turn these properties over to the trustees for preservation. Therefore, it will be the responsibility of the trustees to determine the acceptable future use of these areas.

The exposure pathways that apply are:

- Consumption of fish fillets harvested from Lake George or Powderhorn Lake
- Exposure pathways expected for a Local Area Resident (as described above).

The exposure scenarios described above cover multi-pathway exposure situations. In addition, one other subgroup was identified as a special population segment who may receive contaminant exposure because of one particular pathway or activity pattern. On this basis, the following additional subgroup was evaluated: breast-feeding infants (children up to one year old) of local area residents.

Breast-feeding infants were assumed to reside in the same residential zones or neighborhoods described previously. Based on current USEPA methodology and guidance for risk assessment, the evaluation of contaminant uptake into breast milk is limited to the highly persistent and highly lipophilic contaminants, namely chlorinated dioxins and furans (USEPA 1998b, 1999a, 2005c).

6.3.3 Exposure Pathways

The potential pathways of exposure for the population subgroups that may be exposed to airborne chemical contaminants released from the CDF are reviewed below, including rationale for why a pathway was included or excluded from the exposure evaluation.

6.3.3.1 Air Exposure

Direct inhalation of emissions from the CDF is the primary route of exposure to airborne contaminants for all population subgroups.

6.3.3.2 Soil Exposure

A portion of the chemical contaminants emitted from the CDF is expected to deposit on local surface soils due to the processes of dry and wet deposition. Residents engaged in outdoor work and recreation may inadvertently ingest soil and absorb chemicals through the skin during soil contact. These exposure pathways also apply to local school children, who could engage in outdoor recreational activities on school property.

6.3.3.3 Home Garden Food Chain Exposure

Chemical contaminants deposited in the vicinity of the facility may accumulate in various parts of the food chain. Chemicals may be incorporated into vegetation and crops as a result of deposition on leaves, absorption from the soil through the roots, and uptake of vapors from the air. Vegetable produce grown in home gardens could become a source of contaminant exposure for local residents.

6.3.3.4 Fish Consumption Exposure

Chemical contaminants may enter water bodies through direct deposition from air or by transport and runoff from the land. After entering a water body, chemicals could be dissolved in surface water and/or adsorbed to sediments. Depending on their potential for uptake and bioaccumulation, chemicals may be transferred to and accumulate in fatty tissues of fish in local water bodies. Individuals who consume locally-caught fish may be indirectly exposed to chemical contaminants. For a typical resident in the vicinity of the CDF, fish harvested from a single local water body in the vicinity of the CDF are not likely to represent a significant source of food or a major portion of total dietary protein. However, it is possible that a subgroup of the local population may exist that derives a significant fraction of their dietary protein from locally caught fish ("subsistence fishing" as defined above). Although no information was obtained to confirm that subsistence fishing actually occurs from local water bodies in the Study Area, the exposure assessment assumes that subsistence fishermen reside in the vicinity of the CDF. This activity is used to represent or model the high-end consumption of fish.

6.3.3.5 Breast Milk Exposure

Local resident women of child-bearing age may be exposed to chemical contaminants through the exposure pathways described previously. Chemical contaminants that are persistent and lipophilic compounds such as chlorinated dioxins and furans may accumulate in body tissues, preferentially concentrating in adipose (fatty) tissue. Such compounds may then accumulate in the breast milk of nursing women in the study area. Based on current USEPA guidance, exposure of nursing infants to chlorinated dioxins and furans in breast milk is considered in the risk assessment.

6.3.3.6 Surface Water Exposure

Chemical emissions may enter surface water in the vicinity of the facility through deposition of emissions directly onto a water body or through runoff of contaminated soil that enters the water body. In the vicinity of the CDF, Lake Michigan is the only known and expected source of drinking water for residents living in the vicinity of the CDF. Lake Michigan is located a significant distance north of the CDF site and outside of the zone where significant contaminant deposition would occur. In addition, Lake Michigan surface water is subjected to several treatment steps before use, and water quality is regularly evaluated for compliance with regulations governing contaminant limits. Therefore, significant contaminant exposure through this pathway is unlikely, and is not evaluated further in the SRA.

6.3.3.7 Groundwater Exposure

It is possible that chemicals deposited onto surface soils could become dissolved in precipitation and percolate through surface soils to the groundwater. In a previous study, USEPA evaluated the potential for chemicals deposited onto soil from air deposition to become a source for groundwater contamination (USEPA 1990b). This potential route of chemical transport was evaluated using a worst-case assumption model for leaching and transport of organic and inorganic constituents from surface soils to groundwater. The study concluded that a very limited potential for contamination of groundwater exists, and that further evaluation of this pathway was unnecessary for airborne deposition of chemicals. In addition, in this highly urbanized area in the vicinity of the CDF, the drinking water source for all residents is surface water from Lake Michigan and the use of groundwater for drinking water is not allowed. Consequently, the groundwater exposure pathway is not evaluated further in the SRA.

6.3.4 Estimation of Contaminant Concentrations in Environmental Media

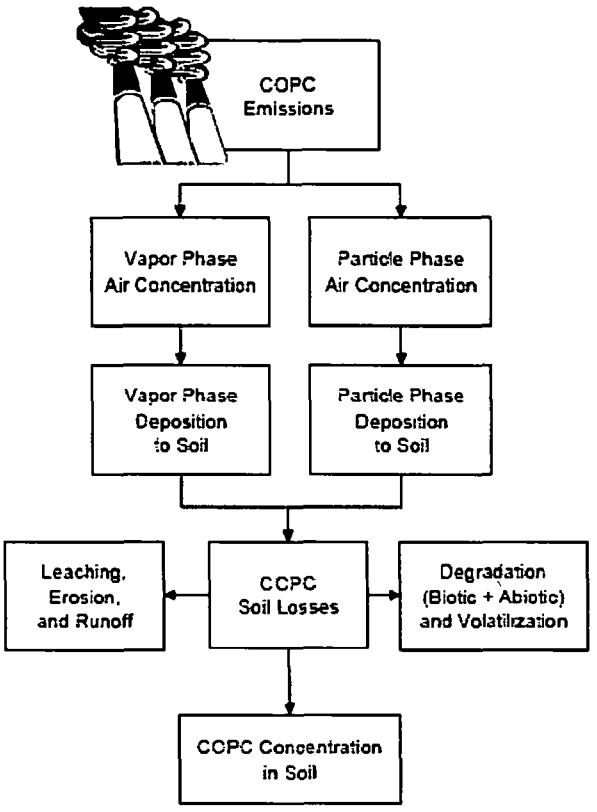
In order to assess the risk of exposure to chemical contaminants emitted from the CDF, an estimate of the concentrations of chemicals of concern in air, soil, vegetables, fish, and breast milk is necessary. The modeling of contaminant dispersion and transport in air is discussed in Sections 4 and 5 of this SRA Report.

For other environmental media, concentrations are estimated using fate and transport models designed to simulate the transport of substances in the environment over time. The models used in this assessment are based on USEPA guidance (USEPA 1990b, 1993c, 1998b, 2005c) and reflect the current understanding of environmental fate and transport processes for chemical contaminants which originate as air emissions. This section presents an overview of the models employed in the SRA. A very detailed presentation of the fate and transport models, the model algorithms (i.e., equations), many recommended input values needed for the models, and the basis for these values is found in USEPA's most recent guidance document for assessing the fate and transport of chemical emissions from combustion facilities (USEPA 2005c). Because of the expected similarity in the fate and transport characteristics of chemicals emitted from a combustion stack or emitted by the CDF area source, the fate and transport models developed for combustion emissions are considered as directly applicable and usable for assessing fate and transport in this SRA study.

Whenever reasonable, site-specific data and model inputs are applied in this assessment rather than using only default values. It is anticipated that use of site-specific values reduces the uncertainty associated with the modeled results. It should be noted, however, that several of the models applied in this assessment are based on limited data and, therefore, contribute to the uncertainty in the results. The uncertainties that result from applying these models are discussed qualitatively at the end of this section. To compensate for the uncertainty in the estimation process, conservative fate and transport assumptions are generally applied so that estimated environmental concentrations are likely to be higher than what would be actually found or measurable in the vicinity of the CDF.

6.3.4.1 Estimation of Contaminant Concentrations in Soil

Chemical contaminants emitted to the atmosphere from a point source or an area source such as the CDF may deposit onto local surface soils due to dry and wet deposition of particles and vapor. The general processes which could contribute to contaminants entering and accumulating in soil at given location are illustrated below.



The concentration of chemicals in surface soil is required to: (1) estimate potential human exposures through soil ingestion and dermal contact with the soil, (2) predict uptake into vegetation for human consumption, and (3) estimate concentrations in surface water due to runoff from contaminated soil. To estimate the chemical concentration in soil, the recommended equation has the following form:

$$Cs = \frac{100 \cdot (Dydp + Dyww + L_{dif}) \cdot [1.0 - \exp\ (-ks \cdot tD)]}{Z_s \cdot BD \cdot ks}$$

where

Cs	=	Average soil concentration over exposure duration (mg COPC/kg soil)
100	=	Units conversion factor (mg-m^2/kg-cm^2)
$Dydp$	=	Unitized yearly dry deposition from particle phase (s/m^2-yr)
$Dyww$	=	Unitized yearly wet deposition from vapor phase (s/m^2-yr)
L_{dif}	=	Dry vapor phase diffusion load to soil (g/m^2-yr)
ks	=	COPC soil loss constant due to all processes (yr^{-1})
tD	=	Time period over which deposition occurs (time period of combustion) (yr)
Z_s	=	Soil mixing zone depth (cm)
BD	=	Soil bulk density (g soil/cm^3 soil)

Soil contaminant concentrations are estimated for surface soil intervals (i.e., upper 1 centimeter) and root zone soil (i.e., upper 20 centimeters) (USEPA 1990b). In addition, average soil concentrations within the upper 20 centimeters are used as an input to estimating surface water concentrations (USEPA 1994c), assuming the presence of an agricultural watershed, which could have some tilled and some untilled soils. Estimated soil concentrations at these depths are based on deposition rates of constituents of concern assuming complete mixing within the soil layer of interest (1 centimeter or 20 centimeters) and continuous operation of the CDF over a period of 30 years. Dry and wet deposition rates for particles (D_{yd} and D_{yw}) are predicted by the ISCST3 air dispersion model.

The parameter values used in the above equation are presented in previous USEPA guidance along with the methodology and assumptions used in estimating soil concentrations (USEPA 1998b, 2005c). Site-specific information needed to estimate soil concentrations include: fraction of organic carbon in soil; bulk density of soil; annual precipitation, irrigation, runoff, and evapotransporation; soil volumetric content; universal soil loss equation (USLE) constants for erosivity and erodability; wind speed; and air temperature, viscosity and density.

Appendix 6-1 presents a detailed description of the equations and parameters needed to calculate contaminant concentrations in soil. The site-specific values used in this assessment for estimating contaminant concentrations in soil are presented in Appendix 6-2.

6.3.4.2 Estimation of Concentrations in Vegetation

The concentration of constituents in vegetation is necessary to estimate the exposure to chemicals through ingestion of vegetation (i.e., home-grown vegetables). Chemical contaminants may bioaccumulate in plants through three mechanisms: uptake by roots, direct deposition onto exposed plant tissues, and air-to-plant transfer of vapor-phase constituents.

The rate and amount of chemical uptake by produce is dependent on the type of vegetable and its potential for exposure to the atmosphere. For example, contaminant deposition is more likely to occur onto leafy vegetables (such as lettuce) than onto vegetables that are protected from the atmosphere (such as corn or root vegetables). Similarly, the uptake of chemicals from the soil will differ for belowground and aboveground vegetables. Therefore, because of the general differences in contamination mechanisms, garden produce is usually assigned into two broad categories: aboveground produce and belowground produce. In addition, aboveground produce is further subdivided into exposed and protected aboveground produce for consideration of contamination as a result of indirect exposure.

Aboveground Produce

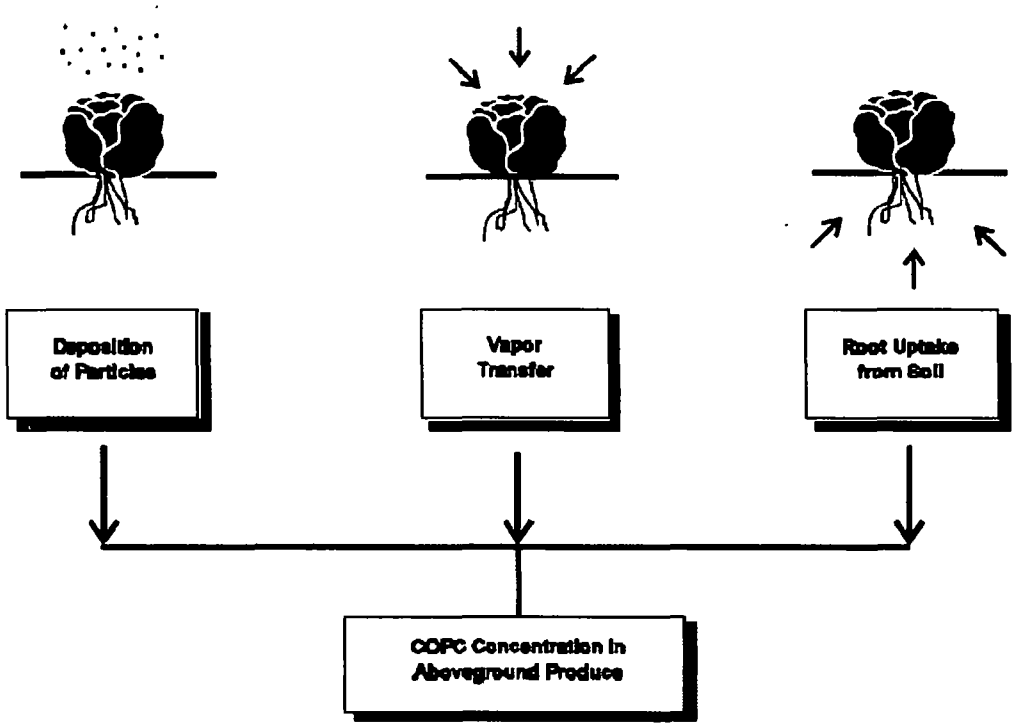
Aboveground produce is usually categorized in to the following types:

- Aboveground leafy produce (e.g., lettuce, broccoli)
- Aboveground protected produce (e.g., corn, peas, grain)
- Aboveground exposed produce (e.g., tomatoes, green peppers).

Aboveground exposed produce is assumed to be contaminated by three possible mechanisms:

- Direct deposition of particles – wet and dry deposition of particle phase contaminants on the leaves and fruits of plants

- Vapor transfer – uptake of vapor phase contaminants by plants through their foliage
- Root uptake – root uptake of contaminants available from the soil and their transfer to the aboveground portions of the plant.



The total contaminant concentration in aboveground exposed produce is calculated as a sum of contamination occurring through all three of these mechanisms.

$$CV = Pr_i + Pd_i + Pv_i$$

where:

CV = total concentration of constituent in the i^{th} plant group, mg/kg

Pr_i = concentration of constituent in i^{th} plant group due to root uptake, mg/kg

Pd_i = concentration of constituent in i^{th} plant group due to direct deposition, mg/kg

Pv_i = concentration of constituent in i^{th} plant group due to air-to-plant transfer, mg/kg

However, edible portions of aboveground protected vegetables and fruits, such as peas, corn, and melons, are covered by a protective coating (e.g., pods, sheaths, rinds); consequently, they are protected from contamination through deposition and vapor transfer. Therefore, root uptake of contaminants is the primary mechanism through which aboveground protected produce becomes contaminated.

Appendix 6-1 presents a detailed description of the equations and parameters needed to calculate contaminant concentrations in exposed and protected aboveground produce. The site-specific values used in this assessment for estimating contaminant concentrations in aboveground produce are presented in Appendix 6-2.

Belowground Produce

For belowground produce, contamination is assumed to occur only through one mechanism—root uptake of contaminants available from soil. Contamination of belowground produce via direct deposition of particles and vapor transfer from ambient air is not considered because the root or tuber is protected from contact with contaminants in the air phase.

For below ground vegetation (i.e., root vegetables), the parameter Pr_i can be estimated by the method developed by Briggs (1982) based on the following relationship:

$$Pr_i = \frac{(CS)\ (RCF_i)\ (VG_{bg})}{Kd_s}$$

where:

CS = soil concentration of contaminant after applicable period of deposition, mg/kg soil
RCF_i = root concentration factor for the ith plant group, L/kg
VG_{bg} = empirical correction factor, unitless
Kd_s = soil/water partition coefficient, L/kg

VG_{bg} is a factor introduced into the calculation of contaminant concentrations to reflect the reduced translocation of compounds into dense, belowground vegetables, such as carrots and potatoes (USEPA 1994d). In general, the contaminant concentrations measured in the barley roots of the Briggs experiments would be representative of the levels of contaminants in the outer few millimeters of below ground vegetation which could be much higher than the average concentration in interior of the whole vegetable. In particular, transfer of lipophilic contaminants (log K_{ow} greater than 4) to the interior of the produce is much less likely compared to non-lipophilic, readily water soluble contaminants[14] Thus, a VG_{bg} value of 0.01 is used as a correction factor for all lipophilic contaminants of concern in order to obtain a realistic estimate of whole vegetable concentrations of contaminants (USEPA 1998b, 2005c). For non-lipophilic contaminants (log K_{ow} less than 4), no correction is needed (i.e., VG_{bg} = 1).

Appendix 6-1 presents a detailed description of the equations and parameters needed to calculate contaminant concentrations in belowground produce. The site-specific values used in this assessment for estimating contaminant concentrations in belowground produce are presented in Appendix 6-2.

Generally, risks associated with exposure of highly volatile contaminants through food-chain pathways have not been found to be significant in previous USEPA exposure assessment evaluations, primarily because volatiles are typically low-molecular-weight (< 200 g/mole) contaminants that do not persist in the environment and do not bioaccumulate to any significant level (USEPA 1994d, 1998b).

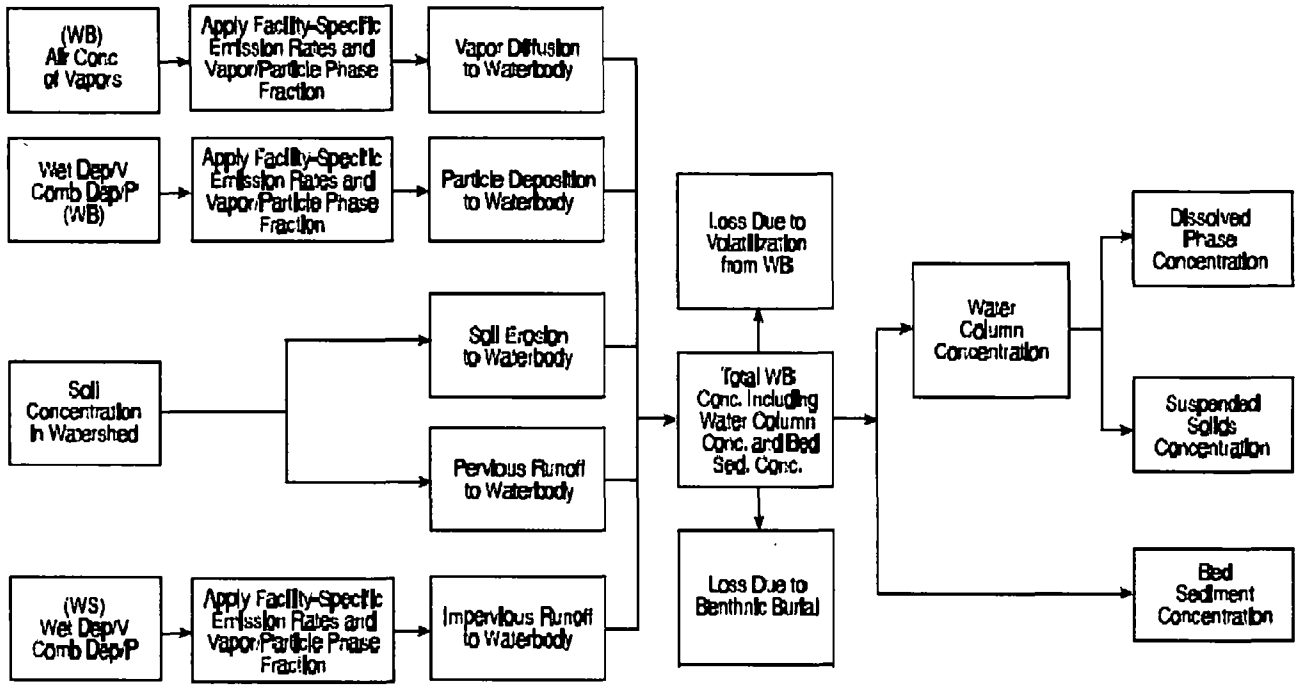
6.3.4.3 Estimation of Contaminant Concentrations in a Water Body

Surface water bodies may receive chemical contaminants from direct deposition, from runoff

[14] K_{ow} is a parameter called the "octanol-water partition coefficient." It is a unitless ratio that represents the tendency of a chemical to dissolve in or adsorb to organic material as compared to water.

of contaminated soils in the vicinity of the CDF facility, and from eroded soils. The concentration of contaminants in the surface water is required to estimate uptake of chemicals from the surface water by fish.

The processes by which chemical contaminants enter into a water body and the steps needed to estimate water body contaminant concentrations are illustrated in the following diagram:



The general mechanisms considered for determination of chemical contaminant loading of the water column are:

1) Direct deposition of particles
2) Runoff from impervious surfaces within the watershed
3) Runoff from pervious surfaces within the watershed
4) Soil erosion over the total watershed
5) Direct diffusion of vapor phase contaminants into the surface water.

The USLE and a sediment delivery ratio are used to estimate the rate of soil erosion from the watershed.

Surface water concentration algorithms include a sediment mass balance, in which the amount of sediment assumed to be buried and lost from the water body is equal to the difference between the amount of soil introduced to the water body by erosion and the amount of suspended solids lost in downstream flow. As a result, the assumptions are made that sediments do not accumulate in the water body over time, and that equilibrium is maintained between the surface layer of sediments and the water column. The total water column contaminant concentration is the sum of the contaminant concentration dissolved in water and the contaminant concentration associated with suspended solids. Partitioning between water

and sediment varies with the contaminant. The total concentration of each contaminant is partitioned between the sediment and the water column.

To evaluate the contaminant loading to a water body from its associated watershed, the contaminant concentration in the watershed soils needs to be calculated. As described previously, the equation for contaminant concentration in soil includes a loss term that considers the loss of contaminants from the soil after deposition. These loss mechanisms all lower the soil concentration associated with a specific deposition rate.

Appendix 6-1 presents a detailed description of the equations and parameters used for calculating contaminant concentrations in watershed soils and contaminant concentrations in the water bodies selected for evaluation in the SRA.

Site-specific values were needed for several parameters to characterize the water body and watershed area of Lake George and Powderhorn Lake within the Study Area (e.g., water body surface area, volume, average depth, watershed area, volumetric flow rate). These values are presented in Table 6-3. These values were obtained from the literature or from information supplied by IDNR (IDNR 2006). In cases where a site-specific watershed parameter was not available for Lake George or Powderhorn Lake, a reasonable default value was obtained from USEPA guidance on modeling of watershed characteristics.

Table 6-3: Hydrologic Parameters for the Water bodies Selected for Evaluation in the SRA

Lake George			
Water Body Parameters		**Watershed Parameters**	
Depth of Water Column	1.95 m	Impervious Area	23%
Current Velocity	1.4E-6 m/s	USLE Cover Management Factor	0.8
Average Volumetric Flow	71,000 m^3/yr	USLE Erosivity Factor	160 yr^{-1}
Surface Area	633,301 m^2	Impervious Area Receiving Deposition	326,728 m^2
		Area Receiving Fallout	1,420,556 m^2
Powderhorn Lake			
Water Body Parameters		**Watershed Parameters**	
Depth of Water Column	2.5 m	Impervious Area	23%
Current Velocity	1.4E-6 m/s	USLE Cover Management Factor	0.8
Average Volumetric Flow	35,170 m^3/yr	USLE Erosivity Factor	160 yr^{-1}
Surface Area	244,691 m^2	Impervious Area Receiving Deposition	111,094 m^2
		Area Receiving Fallout	483,018 m^2

6.3.4.4 Estimation of Contaminant Concentrations in Fish

The concentration of a contaminant in fish is calculated using either a contaminant-specific bioconcentration factor (BCF), a contaminant-specific bioaccumulation factor (BAF), or a contaminant-specific biota-sediment accumulation factor (BSAF). For a contaminant with a log K_{ow} less than 4.0, BCFs are used. For a contaminant with a log K_{ow} greater than 4.0 (except for extremely hydrophobic compounds such as dioxins, furans, and PCBs), which is assumed to have a high tendency to bioaccumulate, a BAF value is used. Extremely hydrophobic contaminants like dioxins, furans, and PCBs are also assumed to have a high tendency to bioaccumulate. However, field studies and actual field measurements of these contaminants in water bodies have shown that they exhibit a high tendency for adsorption to the bed sediments rather than association with the water phase. Therefore, for dioxins, furans, and PCBs, BSAFs are generally used to calculate concentrations in fish. A detailed discussion on the sources of the contaminant-specific BCF, BAF, and BSAF values, and the methodology used to derive them is presented in other USEPA guidance (USEPA 1998b, 2005c).

BCF and BAF values are generally based on dissolved water concentrations. Therefore, when BCF or BAF values are used, the COPC concentration in fish is calculated using dissolved water concentrations. BSAF values are based on benthic sediment concentrations. Therefore, when BSAF values are used, COPC concentrations in fish are calculated using benthic sediment concentrations.

Fish Concentration Estimated from Bioconcentration Factors Using Dissolved-Phase Water Concentration

USEPA (1998b, 2005c) recommends using the following equation to calculate fish concentrations for contaminants having a log K_{ow} less than 4.0.

$$C_{fish} = C_{dw} \cdot BCF_{fish}$$

where

C_{fish}	=	Concentration of COPC in fish (mg COPC/kg FW tissue)
C_{dw}	=	Dissolved phase water concentration (mg COPC/L)
BCF_{fish}	=	Bioconcentration factor for COPC in fish (L/kg)

The dissolved-phase water concentration (C_{dw}) is calculated by using the methods described previously. The contaminant-specific BCF_{fish} values used in this assessment are presented in Appendix 6-3.

Fish Concentration Estimated from Bioaccumulation Factors Using Dissolved-Phase Water Concentration

USEPA (1998b, 2005c) recommends using the following equation to calculate fish concentrations for contaminants having a log K_{ow} of 4 or higher (excluding chlorinated dioxins, furans, and PCBs).

$$C_{fish} = C_{dw} \cdot BAF_{fish}$$

Where

C_{fish}	=	Concentration of COPC in fish (mg COPC/kg FW tissue)
C_{dw}	=	Dissolved phase water concentration (mg COPC/L)
BAF_{fish}	=	Bioaccumulation factor for COPC in fish (L/kg)

The dissolved phase water concentration (C_{dw}) is calculated by using the methods described previously. The contaminant-specific BAF_{fish} values used in this assessment are presented in Appendix 6-3.

Fish Concentration Estimated from Biota-To-Sediment Accumulation Factors Using Bed Sediment Concentrations

USEPA guidance recommends the use of the following equation to calculate fish concentration from BSAFs using concentrations adsorbed to bed sediment for very hydrophobic contaminant, namely chlorinated dioxins, furans, and PCBs.

$$C_{fish} = \frac{C_{sb} \cdot f_{lipid} \cdot BSAF}{OC_{sed}}$$

where

C_{fish}	=	Concentration of COPC in fish (mg COPC/kg FW tissue)
C_{sb}	=	Concentration of COPC sorbed to bed sediment (mg COPC/kg bed sediment)
f_{lipid}	=	Fish lipid content (unitless)
$BSAF$	=	Biota-to-sediment accumulation factor (unitless)
OC_{sed}	=	Fraction of organic carbon in bottom sediment (unitless)

The recommended default value for the lipid content of fish (f_{lipid}) is 7 percent, based on previous USEPA guidance (USEPA 1993c) and other published data (Cook et al. 1991). For the fraction of organic carbon in bottom sediment (OC_{sed}), the recommended default value is 4 percent, which is the midpoint in the range of 3-5 percent measured in field studies (USEPA 1998b, 2005c). The 4 percent value is higher than the usual surface soil organic carbon estimate of 1 percent. Organic carbon content in bottom sediments is higher than the organic carbon content in soils because: 1) erosion favors lighter-textured soils with higher organic carbon contents; and 2) bottom sediments are partially comprised of decomposed aquatic organisms and other detritus material.

BSAFs are presented in Appendix 6-3.

6.3.4.5 Estimation of Contaminant Concentrations in Breast Milk

Based on current USEPA risk guidance and other published USEPA risk assessments (USEPA 1998b, 2005c), the evaluation of uptake of chlorinated dioxins and furans into breast milk is recommended for this SRA because of the highly lipophilic properties of these chemicals and their potential for bioaccumulation into breast milk.

In addition, there is a significant amount of published data and information which indicates that chlorinated dioxins and furans are present as detectable background contaminants in human breast milk. These chemicals could be incorporated into breast milk from a number of sources, with the diet being a primary source (USEPA 2003a).

USEPA guidance recommends use of the following relationship for estimating the concentration of chlorinated dioxins and furans in the lipid fraction of breast milk based on the model published by Smith (1987).

$$C_{milkfat} = \frac{m \cdot 1 \times 10^9 \cdot h \cdot f_1}{0.693 \cdot f_2}$$

Where,

$C_{milkfat}$ = Concentration of a dioxin congener in milkfat of breast milk (pg/kg)
m = Average daily intake of a dioxin congener from all adult exposure pathways (mg/kg-day)

h = Half-life of dioxin in adults (days)
f_1 = Fraction of ingested dioxin/furan congener stored in fat (unitless)
f_2 = Fraction of fat in maternal body weight (unitless)
$1x10^9$ = Units conversion factor (pg/mg)

The recommended values for the input parameters are the following (USEPA 2005c):

h = Half-life of dioxin in adults (2555 days)
f_1 = Fraction of ingested dioxins stored in fat (0.9)
f_2 = Fraction of fat in maternal body weight (0.3)

6.3.5 Estimation of Contaminant Exposure Doses

The next step in the exposure assessment is the calculation of contaminant-specific exposure rates or intake doses for each exposure pathway included in the selected exposure scenarios. This section describes the parameters that need to be evaluated and the exposure factors that need to be assigned for quantifying the exposure received under each exposure scenario.

The calculation of contaminant-specific exposure rates for each exposure pathway evaluated involves solving an equation that combines the following parameters: (1) estimated contaminant-specific media concentrations determined by the methods previously described; (2) an intake or consumption rate for the exposure medium; (3) a receptor body weight; and (4) values which account for the frequency and duration of exposure. The calculation is repeated as necessary for each contaminant and for each exposure pathway included in an exposure scenario.

The following sections describe a general exposure rate calculation and the pathway-specific variables that may affect this calculation.

6.3.5.1 Generic Intake Dose Equation

Exposure can occur over a period of time. In the calculation of an average exposure per unit of time, the total exposure can be divided by an appropriate exposure time period and body weight.

The following generic equation is used to calculate contaminant intake doses (USEPA 1989):

$$I = \frac{C_{gen} \cdot CR \cdot EF \cdot ED}{BW \cdot AT}$$

Where:

I = Intake—the dose of contaminant at the exchange boundary (mg/kg-day) which is available for absorption; for evaluating exposure to noncarcinogenic contaminants, the intake is referred to as average daily dose (ADD); for evaluating exposure to carcinogenic contaminants, the intake is referred to as lifetime average daily dose (LADD)

C_{gen} = Contaminant concentration in any media of concern (e.g., mg/kg for soil or $\mu g/m^3$ for ambient air)

CR = Consumption rate—the amount of contaminated medium consumed per unit of time or event (e.g., kg/day for soil and L/day for water)

EF = Exposure frequency (days/year)

ED = Exposure duration period (years)

BW = Average body weight of the receptor over the exposure period (kg)

AT = Averaging time—the period over which exposure is averaged (days); for carcinogens, the averaging time is 25,550 days, based on a lifetime of 70 years; for noncarcinogens, averaging time equals ED (years) multiplied by 365 days/year.

Variations of the above equation are used to calculate receptor-specific exposures to contaminants. The detailed exposure dose and pathway-specific equations used in the SRA are presented in Appendix 6-4.

The exposures calculated in a risk assessment are intended to represent RME conditions as further described in USEPA (1989). As explained in Section 3, the use of RME values is consistent with other USEPA guidance and analysis (USEPA 1989, 1994d, 2004b). The RME is meant to represent an exposure level at the high end, but within the realistic range of exposure. A study on the quantitative effects of conservative factor selection indicated that setting as few as two exposure factors at their RME level or at the high-end (e.g., near the 90th percentile), while setting the remaining variables at typical or "central tendency" values (e.g., near the 50th percentile) could result in a product equivalent to setting all input variables at an RME level (e.g., 99th percentile value) (Cullen 1994; USEPA 1998b).

USEPA guidance for assessing health risks of emissions from combustion stacks recommends that exposure factors set at RME values should include: (1) the highest modeled ISCST3 output values within the selected exposure scenario locations (e.g., residential zones; schools) in the study area; (2) the exposure frequency; and (3) the exposure duration (USEPA 1998b, 2005c). Other exposure factors are set at typical or average values. Since the SRA is an evaluation of contaminant emissions to air, the decision was made to follow this recommendation wherever reasonably possible in order to be consistent with the RME concept.

The exposure factors used in the SRA to estimate contaminant intake doses are listed according to exposure scenario in Tables 6-4A and 6-4B.

The following sections provide additional information on the necessary exposure factors and the rationale behind exposure factor selection.

Table 6-4A: Exposure Factors and Intake/Contact Rates for Environmental Media Local Area Resident and Local Area Fisher Scenarios

Exposure Factor or Intake Parameter	Selected Intake or Contact Rate	Origin of Value or Rationale for Selection
Inhalation Rate – Adult	0.63 m^3/hr	Recommended average inhalation rate for a residential setting and activity; based on data from USEPA 1997a
Inhalation Rate – Child	0.3 m^3/hr	Recommended average inhalation rate for a residential setting and activity; based on data from USEPA 1997a
Inhalation Exposure Time – Adult	24 hr/day	Conservative default to account for exposure at a single location
Inhalation Exposure Time – Child	24 hr/day	Conservative default to account for exposure at a single location
Exposure Duration – Adult	30 years	High-end default value for residence in a single location; based on recommendations from USEPA 1997a, 1998b, 2005c
Exposure Duration – Child	6 years	Subgroup specific; intake evaluation for children ages 1 to 7 years
Exposure Frequency – Adult	350 days/year	High-end default value to prevent underestimation of time spent at a single location
Exposure Frequency – Child	350 days/year	High-end default value to prevent underestimation of time spent at a single location
Soil Ingestion Rate – Adult	50 mg/day	Average long-term daily soil ingestion rate based on recommendations in USEPA 1997a, 1998b, 2005c
Soil Ingestion Rate – Child	100 mg/day	Average long-term daily soil ingestion rate based on recommendations in USEPA 1997a, 1998b, 2005c
Garden Produce Consumption Rate – Adult Aboveground exposed Aboveground protected Belowground	 21 g/day (dry weight) 39.9 g/day (dry weight) 9.8 g/day (dry weight)	Mean values based on data for consumption of home-grown produce as given in USEPA 1997a; derived from USDA Food Consumption Survey; adjusted for cooking losses
Garden Produce Consumption Rate – Child Aboveground exposed Aboveground protected Belowground	 6.3 g/day (dry weight) 11.6 g/day (dry weight) 3.3 g/day (dry weight)	Mean values based on data for consumption of home-grown produce as given in USEPA 1997a; derived from USDA Food Consumption Survey; adjusted for cooking losses; child values represent a time-weighted mean for data from USEPA 1997a
Fish Consumption Rate – Adult	82 g/day	Mean value based on data for consumption of home caught/consumed fish as given in USEPA 1997a; derived from USDA Food Consumption Survey; adjusted for cooking losses
Fish Consumption Rate – Child	11.4 g/day	Mean values based on data for consumption of home caught/consumed fish as given in USEPA 1997a; derived from USDA Food Consumption Survey; adjusted for cooking losses; child values represent a time-weighted mean for data from USEPA 1997a

Exposure Factor or Intake Parameter	Selected Intake or Contact Rate	Origin of Value or Rationale for Selection
Dermal Surface Area Exposed – Adult	5700 cm^2	From recommendation in USEPA 2004a; based on mean adult value for combination of skin area from head, forearms, hands, and lower legs
Dermal Surface Area Exposed – Child	2800 cm^2	From recommendation in USEPA 2004a; based on mean child value for combination of skin area from head, forearms, hands, lower legs, and feet
Dermal Adherence of Soil – Adult	0.07 mg/cm^2	From recommendation in USEPA 2004a; based on recommendation for home gardeners
Dermal Adherence of Soil – Child	0.2 mg/cm^2	From recommendation in USEPA 2004a; based on recommendation for children playing in moist soil
Dermal Event Frequency – Adult	1/day	Default value for contact on a given day
Dermal Event Frequency – Child	1/day	Default value for contact on a given day
Dermal Exposure Frequency – Adult	240 days/year	Professional judgment; based on 8 months of daily dermal exposure to soil from outdoor activity in northern Indiana climate
Dermal Exposure Frequency – Adult	240 days/year	Professional judgment; based on 8 months of daily dermal exposure to soil from outdoor activity in northern Indiana climate
Body Weight – Adult	70 kg	Recommendation from USEPA 1997a and other USEPA guidance (USEPA 1998b, 2005c) for average in general adult population
Body Weight – Child	15 kg	Recommendation from USEPA 1997a and other USEPA guidance (USEPA 1998b, 2005c) for average in general child population
Averaging Time – Cancer Risk; Adult and Child	25550 days	70 years x 365 days/year; time over which intake is averaged to calculate a life time average daily dose for estimating cancer risk
Averaging Time – Noncancer Hazard – Adult	10950 days	30 years x 365 days/year; total number of days in exposure duration period; time over which intake is averaged to calculate an average daily dose for estimating non-cancer hazard
Averaging Time – Noncancer Hazard – Child	2190 days	6 years x 365 days/year; total number of days in exposure duration period; time over which intake is averaged to calculate an average daily dose for estimating non-cancer hazard

Table 6-4B: Exposure Factors and Intake/Contact Rates for Environmental Media
Local Area Student Scenario

Exposure Factor or Intake Parameter	Selected Intake or Contact Rate	Origin of Value or Rationale for Selection
Inhalation Rate	0.63 m^3/hr	Same as adult inhalation rate; recommended average inhalation rate for adults; based on data from USEPA 1997a
Inhalation Exposure Time	10 hr/day	Exposure time in school setting; combination of school class time and additional on-site recreational/athletic activity time
Exposure Duration	6 years	School matriculation; combination of 2 years of junior high school and 4 years of senior high school
Exposure Frequency	180 days/year	Based on total number of days in published school calendar; inhalation, incidental soil ingestion and outdoor dermal exposure are assumed to occur on every school day[15]
Soil Ingestion Rate	100 mg/day	Same as soil ingestion rate for a child; average long-term daily soil ingestion rate based on recommendations in USEPA 1997a, 1998b, 2005c
Dermal Surface Area Exposed	5100 cm^2	From recommendation in USEPA 2004a; based on age-adjusted mean values of body part surface area for adolescents/teens of age 12-18 years; combination of skin area from head, forearms, hands, and lower legs
Dermal Adherence of Soil	0.1 mg/cm^2	From recommendation in USEPA 2004a; surrogate value for all athletic activities; based on recommended value for rugby players
Dermal Event Frequency – Adult	1/day	Default value for contact on a given day
Body Weight	57 kg	Based on data from USEPA 1997a; corresponds to mean age-adjusted body weight for adolescents/teens of age 12-18 years
Averaging Time – Cancer Risk;	25550 days	70 years x 365 days/year; time over which intake is averaged to calculate a life time average daily dose for estimating cancer risk
Averaging Time – Noncancer Hazard	2190 days	6 years x 365 days/year; total number of days in exposure duration period; time over which intake is averaged to calculate an average daily dose for estimating non-cancer hazard

6.3.5.2 Air Exposure

Direct inhalation of vapors and particulate emissions from the CDF is a potential pathway of exposure. Individual receptors residing or conducting activities in the Study Area could be directly exposed to contaminants in vapor, particulate, and particle-bound phases as a result of normal respiration. The factors that affect the amount of exposure include vapor and particulate contaminant concentrations, respiration rate during the period of exposure, and length of exposure.

[15] Based on published school calendar for East Chicago School District (http://www.ecps.org). For dermal contact to soil through outdoor athletic activity, another option was considered: if outdoor athletic activity is assumed to occur every weekday for 8 months of the year, excluding mid-winter and summer recess (i.e., from August – November and February – May), the total number of athletic days would be approximately 160 days/year. Since this value is less than the total number of school days, the total number of school days (i.e., 180) was selected to represent the estimate of the total number of outdoor dermal soil contact days.

As presented in Tables 6-4A and B, default inhalation rates are used for adult and child receptors in a given exposure scenario. It is recognized that inhalation rates could vary depending on the level of activity during a given day or exposure period. Therefore, the recommended inhalation rates from USEPA guidance reflect an averaging of inhalation rates expected for different levels of physical activity. USEPA has evaluated an extensive set of data and studies on inhalation rates in order to recommend reasonable inhalation rates for particular receptors, time periods, and activities (USEPA 1997a).

In addition, three significant assumptions are used which are regarded as conservative, and should lead to an overestimate of contaminant exposure for most individuals:

1) For a specific location and period of exposure (e.g., home day, school day), a receptor is assumed to be continuously exposed only to air which contains contaminants. There is no adjustment for activities or time periods spent outside the contaminated zone (e.g., shopping, dining, work location, field trips, etc.).

2) All inhalation exposure is assumed to result from inhalation of outdoor levels of contaminants. Although vapors entering buildings and residences as a result of air exchange could remain airborne and available for inhalation, particulates entering buildings are more likely to settle out and not be inhaled. Studies of human activity patterns indicate that for many individuals, the majority of time on most days is spent in indoor environments (USEPA 1997a).

3) The WEPS model for erosion of sediments in the CDF indicated that particulate matter released from the CDF should be composed of particles exhibiting a size of 100 µm or less in diameter. The particles transported downwind of the CDF at any location will exhibit a range of particle sizes. However, for assessing inhalation exposure, all particles transported to any receptor area location are assumed to be respirable particles (i.e., PM10). Respirable particles are those which can penetrate to the lower regions of the respiratory system and enter the lungs.

6.3.5.3 Soil Contaminant Ingestion

Receptor populations can be exposed to contaminants in soil by consuming soil that has adhered to the body, especially the hands, as a result of hand-to-mouth behavior. Factors that influence exposure by soil ingestion include soil concentration, the rate of soil ingestion during the time of exposure, and the length of time spent in the vicinity of contaminated soil. Soil ingestion rate estimates in children are based on studies that measure the quantities of non-absorbable tracer minerals in the feces of young children. Ingestion rate estimates for adults are based on assumptions about exposed surface area and frequency of hand-to-mouth contact. Indoor dust and outdoor soil may both contribute to the total daily ingestion (USEPA 1997a). Exposure levels are also influenced by the amount of time that the individual spends in the vicinity of soil exposed to deposition of emitted pollutants. Based on empirical measurements and expected differences in behavior patterns, children are assigned higher soil ingestion rates than adults.

6.3.5.4 Food Consumption

Plants and animals impacted by emission sources may take up contaminants from the air or after deposition onto soil or water bodies. Humans are exposed to contaminants through the

food chain when they consume edible plants and animals as a food source. Human intake of contaminants is determined from the following factors: (1) the types of foods consumed, (2) the amount of food consumed per day, (3) the concentration of contaminants in the food, and (4) the percentage of the diet derived from contaminated food sources. The other important variables and assumptions used for the assessment of food consumption in the SRA are described below.

Food Consumption Rate

The rate of food consumption varies within the population based on many factors including age, sex, body weight, and geographic region. For most risk assessments, resource and time constraints will preclude an investigation of site-specific consumption rates. Instead, food consumption rates are derived from published studies and surveys on food consumption rates for the U.S. population. USEPA recommends that data from U.S. Department of Agriculture USDA food consumption surveys should be used for the risk assessment process (USEPA 1990b). USEPA has conducted an extensive analysis of food consumption rates and behavior based on the USDA surveys and compiled the results into summary tables in the *Exposure Factors Handbook* (USEPA 1997a).

In cases where homegrown food consumption is being evaluated, current USEPA risk guidance recommends that food consumption rates should be derived from the *Exposure Factors Handbook* data on U.S. populations which reported consumption of "home produced" food items (USEPA 1998b, 2005c). These data cover consumption rates for persons who raise food in home gardens and farms or catch fish for home consumption. The consumption rates used in the SRA are summarized in Tables 6-4A and B and explained in detail in Appendix 6-3, based on the analysis presented in the guidance documents (USEPA 1998b, 2005c).

Percentage of Contaminated Food

Normally, it is not expected that an individual's diet will be composed entirely of homegrown or home-raised food items. The percentage of homegrown food consumed by the individual will affect contaminant exposure, because not all of an individual's dietary intake will originate from a contaminated source. For example, residents of highly populated urban areas will have a smaller portion of their diet supplied by their own homegrown food compared to persons living in rural or suburban areas, who can more readily raise food in home gardens or raise animals on farms.

Current USEPA guidance (1998b, 2005c) recommends the following assumptions regarding the percentage of contaminated food for the following scenarios:

- For the Local Resident (adult and child) living in the predominantly urbanized area of East Chicago, IN, it is assumed that 25 percent of aboveground and belowground produce are grown from within a source of contamination.

- For the local fisher, it is assumed that 100 percent of the fish consumed by the fisher is harvested from a local contaminated water body (i.e., Lake George). This is a conservative assumption because it means that no fish in the diet is supplied from a non-contaminated water body. This assumption is included in the recommendation that a screening-level analysis should evaluate a high-end consumer of potentially contaminated fish.

6.3.5.5 Exposure Frequency

The frequency at which repeated exposure occurs is specific to each scenario. Because exposure frequency is one of the parameters intended to be set for evaluation of RME, selection of the EF was simplified as follows for the SRA. For all residential scenarios, the EF is set to 350 days per year. This assumption is based on the conservative estimate that all residents spend a maximum of 15 days at a location outside of a residential zone selected for evaluation. For the exposure scenario at the school location, the conservative estimate is that exposure could occur on every school day based on the local school district calendar. There is no adjustment for days of absence, periods when outdoor recreational activity does not occur (e.g., mid-winter), or days on which weather would not actually permit outdoor recreational activity (e.g., snow, cold, heavy rain).

6.3.5.6 Exposure Duration

Exposure duration is the length of time that a receptor is exposed through the exposure pathways that are part of a specific exposure scenario. For the direct inhalation exposure pathway, exposure can continue as long as the emission source is in operation. Since the current plan for operation of the CDF calls for the CDF to receive sediments for 30 years before closing, the longest exposure duration for inhalation would be 30 years.

For exposure via indirect pathways that result after contaminants are deposited onto soil and transported to vegetation and water, an individual receptor could be exposed for as long as the receptor remains in a location or area being evaluated in the risk assessment. Consequently, USEPA guidance recommends using default RME values to estimate exposure duration for specified receptors.

Theoretically, an individual could be exposed to contaminants via some indirect pathways for an entire lifetime (approximately 70 years). However, U.S. census data and population demographic data indicate that the U.S. population has significant mobility and few Americans reside in the same small geographic area (e.g., home, neighborhood, census block) for an entire 70-year lifetime. Based on data for population mobility and residence time, a period of 30 years is recommended as the RME value for adult exposure duration (USEPA 1997a, 1998b, 2005c).

For other receptors, the recommended exposure duration will be determined for a selected population subgroup or for specific exposure scenario. For example, USEPA risk assessment guidance (USEPA 1989, 2002a) has generally defined childhood as being from one to seven years old, based on the concept that childhood represents approximately ten percent of the lifespan. The daily intake for an exposure pathway is expressed as the dose rate per body weight. Because children have lower body weights than adults, typical ingestion exposures normalized to body weight for items such as soil, milk, and fruits, can be significantly higher for children. This is the primary reason for evaluating a child resident scenario (USEPA 1998b, 2002a). Consequently, an exposure duration of six years is typically used for the child resident.

For non-resident exposure scenarios, USEPA guidance does not recommend an exposure duration that can be defined as an RME value. For such cases, the typical options are to select a value based on site-specific information, or to develop a value based on published data for specific activity patterns, such as the length of time spent working in a particular occupation (USEPA 1997a).

For this SRA Report, the non-resident scenario is for contaminant exposure to junior and senior high school students at the selected school location. Consequently, an exposure duration of six years was selected to represent students who would spend their entire junior/senior high school careers attending school the same location.

6.3.5.7 Dermal Exposure to Soil

Individuals could be exposed to chemical contaminants by absorption through the skin when it comes into contact with chemical contaminants in soil. The process of absorption into the bloodstream after a chemical crosses the skin barrier is often referred to as "percutaneous" absorption. Factors that affect dermal exposure include: (1) surface area exposed, (2) contact time, (3) capacity for soil adherence to skin, (4) amount of time spent near the contaminated source, and (5) the fraction of a contaminant absorbed through the skin. In general, an increased dose of a specific contaminant could potentially be absorbed through the skin as the surface area of the skin is increased. Surface area is affected by age and body weight—for example, children have less total surface area than adults. The amount of surface area available for exposure to soil is also affected by the amount of clothing worn. An adult working in a garden in long sleeves and long pants will have a smaller exposed surface than an adult working in shorts and a short-sleeved shirt. For dermal exposure from soil, the exposed surface area affects the amount of soil that can adhere to exposed skin.

As the time duration for which the contaminated soil stays in contact with the skin increases, so does the amount of a contaminant that can be absorbed. "Contact time" refers to the duration of time each day that contact with soil is possible. Dermal exposure is also affected by the amount of time, each day, spent in the vicinity of the contaminated soil.

Seasonal exposure to soil can also be considered, because regional climate will influence this variable. For example, in cold weather regions, dermal exposure to soil is reduced compared to warm weather climates because of factors such as fewer days spent outdoors, higher level of clothing coverage, and more days with snow/ice cover.

The amount of a given contaminant that can be absorbed through the skin depends on the chemical properties of the contaminant, the properties of the soil matrix, and dermal absorption pharmacokinetics. For example, if a contaminant cannot be readily absorbed through the skin, the daily intake of the contaminant may be small even if other exposure characteristics, such as contact time, are favorable.

For estimating dermal absorption of chemicals, the following algorithms are used. These are variations of the generic Intake equation shown previously.

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

where:

Parameter		Definition (units)
DAD	=	Dermal Absorbed Dose (mg/kg-day)
DA_{event}	=	Absorbed dose per event (mg/cm²-event)
SA	=	Skin surface area available for contact (cm²)
EV	=	Event frequency (events/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

$$DA_{event} = C_{soil} \times CF \times AF \times ABS_d$$

where:

Parameter		Definition (units)
DA_{event}	=	Absorbed dose per event (mg/cm²-event)
C_{soil}	=	Chemical concentration in soil (mg/kg)
CF	=	Conversion factor (10^{-6} kg/mg)
AF	=	Adherence factor of soil to skin (mg/cm²-event) (Referred to as contact rate in RAGS, Part A)
ABS_d	=	Dermal absorption fraction

USEPA has published a specific guidance document for evaluating dermal exposure that is followed in this SRA Report (USEPA 2004a). The guidance document provides a detailed description of the factors and variables affecting dermal exposure as well as recommended numerical values for exposure factors needed to evaluate dermal exposure.

There are two chemical-specific parameters needed for the dermal exposure assessment: the chemical contaminant concentration in soil and the dermal absorption fraction (ABS). Chemical concentrations at a given location are determined from the air dispersion/deposition modeling and the fate and transport processes described previously. The ABS represents the capacity of a chemical contaminant in soil to penetrate the skin barrier and become internally absorbed and available for metabolism, excretion, or transport to a sensitive organ or organ system.[16] The number of contaminants evaluated in the risk assessment for the dermal-soil pathway will be limited by the availability of dermal absorption values for chemicals in soil. In general, very limited data exist in the scientific literature for deriving verifiable dermal absorption fractions for chemicals from soil. USEPA guidance recommends dermal absorption factors for ten specific chemicals in soil based on well-designed studies. These chemicals include mainly persistent organic chemicals (e.g., dioxin, PCB, pesticides) and two metals (arsenic, cadmium). For other semi-volatile organic chemicals and metals, a default surrogate ABS value is selected to enable a screening level dermal evaluation to be performed. Based on these recommendations, the ABS values selected for use in the SRA are shown in Table 6-5.

[16] USEPA guidance on dermal exposure evaluates the systemic chronic health effects resulting from long-term exposure at relatively low doses of chemicals adsorbed to soil. Acute chemical injury directly to the skin (e.g., allergic responses, urticarial reactions, hyperpigmentation) is not evaluated since exposure to undiluted or high concentration forms of chemicals (e.g., coal tar, petroleum, metal fabrication, commercial pesticide products) will not occur as a result of releases from the CDF.

Table 6-5: Dermal Absorption Fraction (ABS) from Soil for the COCs in the SRA

Constituent	Dermal Absorption Fraction (ABS)	Recommendation and/or Rationale for Selection
Metals		
Antimony	0.03	USEPA 2004[1]; based on value for arsenic
Arsenic	0.03	USEPA 2004
Barium	0.03	USEPA 2004; based on value for arsenic
Cadmium	0.001	USEPA 2004
Chromium +3	0.03	USEPA 2004; based on value for arsenic
Chromium +6	0.03	USEPA 2004; based on value for arsenic
Manganese	0.03	USEPA 2004; based on value for arsenic
Mercury	0.03	USEPA 2004; based on value for arsenic
Nickel	0.03	USEPA 2004; based on value for arsenic
Selenium	0.03	USEPA 2004; based on value for arsenic
Zinc	0.03	USEPA 2004; based on value for arsenic
PAHs		
Acenaphthene	0.13	USEPA 2004; value for PAH constituents
Anthracene	0.13	USEPA 2004; value for PAH constituents
Benzo[a]anthracene	0.13	USEPA 2004; value for PAH constituents
BaP	0.13	USEPA 2004; value for PAH constituents
Benzo[b]fluoranthene	0.13	USEPA 2004; value for PAH constituents
Benzo[k]fluoranthene	0.13	USEPA 2004; value for PAH constituents
Chrysene	0.13	USEPA 2004; value for PAH constituents
Fluoranthene	0.13	USEPA 2004; value for PAH constituents
Fluorene	0.13	USEPA 2004; value for PAH constituents
Indeno[1,2,3-cd]pyrene	0.13	USEPA 2004; value for PAH constituents
Naphthalene	0.13	USEPA 2004; value for PAH constituents
Phenanthrene	0.13	USEPA 2004; value for PAH constituents
Pyrene	0.13	USEPA 2004; value for PAH constituents
PCBs		
PCBs (total)	0.14	USEPA 2004; value for PCBs/Aroclors
Dioxin/Furan Congeners		
2,3,7,8-TetraCDD	0.03	USEPA 2004; value for dioxin congeners
1,2,3,7,8-PentaCDD	0.03	USEPA 2004; value for dioxin congeners
1,2,3,4,7,8-HexaCDD	0.03	USEPA 2004; value for dioxin congeners
1,2,3,6,7,8-HexaCDD	0.03	USEPA 2004; value for dioxin congeners
1,2,3,7,8,9-HexaCDD	0.03	USEPA 2004; value for dioxin congeners
1,2,3,4,6,7,8-HeptaCDD	0.03	USEPA 2004; value for dioxin congeners
OctaCDD	0.03	USEPA 2004; value for dioxin congeners
2,3,7,8-TetraCDF	0.03	USEPA 2004; value for dioxin congeners
1,2,3,7,8-PentaCDF	0.03	USEPA 2004; value for dioxin congeners
2,3,4,7,8-PentaCDF	0.03	USEPA 2004; value for dioxin congeners
1,2,3,4,7,8-HexaCDF	0.03	USEPA 2004; value for dioxin congeners
1,2,3,6,7,8-HexaCDF	0.03	USEPA 2004; value for dioxin congeners
1,2,3,7,8,9-HexaCDF	0.03	USEPA 2004; value for dioxin congeners
2,3,4,6,7,8-HexaCDF	0.03	USEPA 2004; value for dioxin congeners
1,2,3,4,6,7,8-HeptaCDF	0.03	USEPA 2004; value for dioxin congeners
1,2,3,4,7,8,9-HeptaCDF	0.03	USEPA 2004; value for dioxin congeners
OctaCDF	0.03	USEPA 2004; value for dioxin congeners
Pesticides and Phenols		
Aldrin	0.04	USEPA 2004; based on value for chlordane
Dieldrin	0.04	USEPA 2004; based on value for chlordane

DDD	0.03	USEPA 2004; based on value for DDT
DDE	0.03	USEPA 2004; based on value for DDT
DDT	0.03	USEPA 2004;
Endosulfan II	0.04	USEPA 2004; based on value for chlordane

[1] USEPA (2004a)

Dermal contact with contaminants can result in systemic toxicity after percutaneous absorption. In an ideal situation, a route-specific (i.e., dermal) toxicity factor would consider portal-of-entry effects (i.e., direct toxicity) and would also provide dosimetry information on the dose-response relationship for systemic effects via percutaneous absorption. However, very few chemical contaminants have been adequately studied in humans or animals model systems for the purpose of defining percutaneous absorption characteristics or chemical-specific toxicity following dermal absorption. Therefore, USEPA and other health agencies have not developed dermal route-specific toxicity factors.

In the absence of dermal toxicity factors, USEPA has devised a simplified approach for making route-to-route (oral-to-dermal) extrapolations for systemic effects. This process is outlined in Appendix A of the *Risk Assessment Guidance for Superfund* (USEPA 1989). Primarily, the approach accounts for the fact that most oral RfDs and oral CSFs are expressed as the amount of substance administered per unit time and body weight, whereas exposure estimates for the dermal pathway are expressed as absorbed dose. The approach utilizes the dose-response relationship obtained from oral administration studies and makes an adjustment, if necessary, for absorption efficiency to represent the toxicity factor in terms of absorbed dose.

When USEPA derives an RfD or CSF for a chemical that is based on the oral route, the value is based on the administered dose, namely the external dose from the environmental medium (e.g., soil, food, water) before absorption into the body occurs. For estimating intake doses of the same chemical for other routes of exposure (i.e., dermal, inhalation), USEPA's risk methods yield an estimation that corresponds to an absorbed dose. Therefore, to characterize risk from the alternate exposure routes, adjustment of the oral toxicity factor to represent an absorbed rather than administered dose is necessary (USEPA 2004a). This adjustment accounts for the absorption efficiency in the "critical study," which forms the basis of the RfD or the CSF. For example, in the case where oral absorption in the critical study is essentially complete (i.e., 100 percent), the absorbed dose is equivalent to the administered dose, and therefore no toxicity adjustment is necessary. When gastrointestinal absorption of a chemical in the critical study is poor (e.g., one to ten percent), the absorbed dose is much smaller than the administered dose. Consequently, toxicity factors based on absorbed dose should be adjusted to account for the difference in the absorbed dose relative to the administered dose (USEPA 2004a).

In effect, the magnitude of the recommended toxicity factor adjustment is inversely proportional to the absorption fraction in the critical study. That means when absorption efficiency in the critical study is high, the absorbed dose approaches the administered dose, resulting in little difference in a toxicity factor derived from either the absorbed or administered dose. As absorption efficiency in the critical study decreases, the difference between the absorbed dose and administered dose increases. At some point, a toxicity factor based on absorbed rather than administered dose should be used to account for this difference in dose. An adjustment in the oral toxicity factor (RfD or CSF) is recommended when the following conditions are met: (1) the toxicity value derived from the critical study is based on an administered dose (e.g., delivery by water, diet, gavage) in the study design; and (2) documented evidence demonstrates that the gastrointestinal (GI) absorption of the chemical in question, from a medium (e.g., water, feed) similar to the one employed in the critical study, is significantly less than 100 percent. In

practice, a cutoff of 50 percent GI absorption is recommended to reflect the intrinsic variability in the analysis of absorption rates derived from different experimental studies and different species. Use of this cut-off level is preferable to making comparatively small adjustments in the toxicity value that would imply the existence of a level of accuracy that is not supported by the scientific literature.

The recommended GI absorption values (ABS_{GI}) for those chemical contaminants with chemical-specific dermal absorption factors from soil are presented in Table 6-6, as presented in the USEPA guidance on dermal risk assessment (USEPA 2004a). A review of the available literature indicates that organic chemicals are generally well absorbed (> 50 percent) across the GI tract. For those organic chemicals that do not appear on in table, the recommendation is to assume a 100 percent ABS_{GI} value. Absorption data for inorganics are also provided in Table 6-6, which indicates a wide range of absorption values for inorganics.

Table 6-6: Summary of Gastrointestinal Absorption Efficiency and Recommendations for Adjustments of Toxicity Factors for Specific Chemicals[1,2]

Compound	GI Absorption				IRIS Critical Toxicity Study			Adjust?
	Ref[3]	Species	Dosing Regimen	% Absorbed ABS_{GI}	Species	Dosing Regimen	Toxicity Factor	
Organics								
Chlordane	Ewing, 1985 Ohno, 1986	Rats	assume aqueous gavage	80%	Mice	diet	SF	No
					Mice	inhalation	RfD	
2,4-Dichlorophenoxyacetic acid (2,4-D)	Knopp, 1992 Pelletier, 1989	Rats	assume aqueous gavage	>90%	Rats	diet	RfD	No
DDT	Keller, 1980	Rats	vegetable oil	70-90%	Rats	dissolved in oil, mixed with diet	RfD	No
Pentachlorophenol	Korte, 1978	Rats	diet	76%	Rats	diet	RfD	No
	Meerman, 1983	Rats	water	100%				
Polychlorinated biphenyls (PCBs)	Albro, 1972	Rats	squalene	96%	Rats	diet	SF	No
	Muhlebach, 1981	Rats	emulsion	80%				
	Tanabe, 1981	Rats	corn oil	81%				
Polycyclic aromatic hydrocarbons (PAHs)	Chang, 1943	Rats	starch solution	58%	Mice	diet	SF	No
	Hecht, 1979	Rats	diet	89%				

Compound	GI Absorption				IRIS Critical Toxicity Study			Adjust?
	Ref[3]	Species	Dosing Regimen	% Absorbed ABS_{GI}	Species	Dosing Regimen	Toxicity Factor	
TCDD	Fries, 1975	Rats	diet	50-60%	under review			No
	Piper, 1973	Rats	diet	70%				
	Rose, 1976	Rats	corn oil	70-83%				
Other Dioxin/ Dibenzofurans	ATSDR, 1994a	multiple studies		>50%	under review			No
All other organic compounds	multuple references			generally >50%	multiple studies		RfD or SF	No
Inorganics								
Antimony	Waitz, 1965	Rats	water	15%	Rat	water	RfD	Yes
Arsenic (arsenite)	Beutley, 1975	Human	assume aqueous	95%	Human	water	SF	No
Barium	Cuddihy and Griffith, 1972 Taylor, 1962	Dog	water	7%	Human	water	RfD	Yes
Beryllium	Reeves, 1965	Rats	water	0.7%	Rat	water	RfD	Yes
Cadmium	IRIS, 1999	Human	diet	2.5%	Human	diet and water	RfD	Yes
		Human	water	5%				Yes
Chromium (III)	Donaldson and Barreras, 1996 Keim, 1987	Rats	diet/water	1.3%	Rat	diet	RfD	Yes

Compound	GI Absorption				IRIS Critical Toxicity Study			Adjust?
	Ref[1]	Species	Dosing Regimen	% Absorbed ABS_{GI}	Species	Dosing Regimen	Toxicity Factor	
Chromium (VI)	Donaldson and Barreras, 1996 MacKenzie, 1959 Sayato, 1980	Rats	water	2.5%	Rat	water	RfD	Yes
Cyanate	Farooqui and Ahmed, 1982	Rats	assume aqueous	>47%	Rat	diet	RfD	No
Manganese	Davidsson, 1989 IRIS, 1999 Ruoff, 1995	Human	diet/water	4%	Human	diet/water	RfD	Yes
Mercuric chloride (other soluble salts)	IRIS, 1999	Rats	water	7%	Rat	oral gavage in water: 2X/week	RfD	Yes
Insoluble or metallic mercury	ATSDR, 1994b	Human	acute inhalation of Hg vapor	74-80%	Human	Inhalation	RfC	No
Methyl mercury	Aberg, 1969	Human	aqueous	95%	Human	diet	RfD	No
Nickel	Elakhovskaya, 1972	Human	diet/water	4%	Rat	diet	RfD	Yes
Selenium	Young, 1982	Human	diet	30-80%	Human	diet	RfD	No
Silver	Furchner, 1968 IRIS, 1999	Dogs	aqueous	4%	Human	i.v. dose	RfD (based on estimated oral dose)	Yes

[1] This table is derived from USEPA (2004a)
[2] All literature references in Column #2 are listed in USEPA (2004a)

These ABS_{GI} values in Table 6-6 are recommended for the adjustment of toxicity values for the assessment of dermal absorption through contact of chemical contaminants in soil. The practical significance of the ABS_{GI} value on risk assessment is the following: as the ABS_{GI} value decreases, the greater is the contribution of the dermal pathway to overall risk relative to the ingestion pathway. Therefore, the ABS_{GI} can significantly influence the comparative importance of the dermal pathway in a risk assessment. The quantitative significance is illustrated by the following proportional relationship:

$$\frac{Dermal\ Risk}{Ingestion\ Risk} \propto \frac{1}{ABS_{GI}}$$

where:

Parameter		Definition (units)
ABS_{GI}	=	Fraction of contaminant absorbed in gastrointestinal tract (dimensionless) in the critical toxicity study

Once the criteria for adjustment have been defined and a specific ABS_{GI} value has been identified, a toxicity factor that reflects the absorbed dose can be calculated from the oral toxicity values by employing the following equations:

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

where:

Parameter		Definition (units)
RfD_{ABS}	=	Absorbed reference dose (mg/kg-day)
RfD_O	=	Reference dose oral (mg/kg-day)
ABS_{GI}	=	Fraction of contaminant absorbed in gastrointestinal tract (dimensionless) in the critical toxicity study

$$SF_{ABS} = \frac{SF_O}{ABS_{GI}}$$

where:

Parameter		Definition (units)
SF_{ABS}	=	Absorbed slope factor
SF_O	=	Oral slope factor $(mg/kg\text{-}day)^{-1}$
ABS_{GI}	=	Fraction of contaminant absorbed in gastrointestinal tract (dimensionless) in the critical toxicity study

Based on the approach for toxicity factor adjustment presented in USEPA guidance (USEPA) and the information in Table 6-6, toxicity factor adjustment was applied for the following contaminants of concern in the SRA: antimony, barium, cadmium, chromium, manganese, nickel, and zinc.

6.3.5.8 Breast Milk Consumption

Based on current USEPA guidance (USEPA 2005c), exposure of nursing infants to chlorinated dioxins and furans in breast milk is considered in this risk assessment.

The equation used to calculate the intake dose of an infant to a dioxin/furan congener is a refinement of the generic intake dose equation presented previously:

$$ADD_{infant} = \frac{C_{milkfat} \cdot f_3 \cdot f_4 \cdot IR_{milk} \cdot ED}{BW_{infant} \cdot AT}$$

Where:

ADD_{infant} = Average daily dose for infant exposed to a dioxin/furan congener in breast milk (pg/kg-day)

$C_{milkfat}$ = Concentration of a dioxin/furan congener in milkfat of breast milk (pg/kg) (calculated from the parameters given previously)

The recommended values for the input parameters are the following (USEPA 1998b, 2002a, 2005c):

f_3 = Fraction of fat in breast milk (0.04)
f_4 = Fraction of ingested congener that is absorbed (0.9)
IR_{milk} = Ingestion rate of breast milk by infant (0.8 kg/day)

ED = Exposure Duration (1 year)
BWi_{infant} = Body Weight of infant during feeding period (10 kg)
AT = Averaging time for intake period (1 year)

With regard to the value for exposure duration, many infants could be breast-fed for less than one year, while some may be breast-fed for more than one year. Available guidance and published survey data on breast-feeding patterns in the U.S. indicate that one year is a reasonable or typical value for breast-feeding duration (USEPA 2002a, 2005c).

6.3.5.9 Intake of Lead: IEUBK Model for Lead

USEPA and public health agencies (CDC, State agencies) have relied upon the well-characterized neurological effects observed in children as the sensitive endpoint for evaluating the potential for adverse health effects from lead exposure. To apply a protective reference exposure level for lead in children, the goal of the agencies is to limit lead levels from a combination of sources (e.g., soil, air, water, food) such that a typical child or group of similarly exposed children would have no more than a five percent probability of exceeding a 10 µg/dL blood lead level. This 10 µg/dL blood lead level is based on analyses conducted by CDC and USEPA that associate blood lead levels above 10 µg/dL with the on-set of neurological deficits in some children. This blood lead level is below a level that would require medical intervention (USEPA 1994a, 1998a).

USEPA has developed an approach called the IEUBK Model, which evaluates potential risks by predicting blood lead levels associated with exposure to lead. The IEUBK Model integrates a number of characteristics reflecting the complex exposure pattern and physiological handling of lead by the body, and has been validated at several sites where lead exposure data and human blood lead levels are available. The IEUBK Model has been reviewed and recommended by the USEPA Science Advisory Board (USEPA 1992b). The CDF is an example of an area source from which lead emissions are possible due to particulate emissions to air followed by air transport and deposition to soil.

The Agency has now developed a computerized version of the IEUBK Model that predicts blood lead levels and percentage distributions for children ranging in age from infancy to age seven (USEPA 2001a). The IEUBK Model accounts for the major characteristics that influence the uptake and absorption of lead from the environment, including the ability to incorporate default or site-specific values for background levels of lead in air, soil, water, and diet. At present, it is not possible to apply the computer model to predict potential blood lead levels in adults. In general, however, children are more susceptible to lead exposures than adults as a result of higher soil ingestion rates, higher absorption from the gut, nutritional variables and lower body weight. Consequently, in most risk assessments, children are identified as the subgroup of primary concern for lead exposure.

The IEUBK Model is used to predict blood lead levels for an individual child or population of children, and was specifically designed to evaluate lead exposure in young children because this age group is known to display enhanced sensitive to lead exposure. The IEUBK Model is a versatile assessment tool that allows the user to make rapid calculations from a complex array of intake, absorption, distribution, and elimination equations by building site-specific and age-dependent exposure scenarios. The IEUBK Model allows the user to input different media concentrations and dietary intake rates for lead for the set of consecutive years being modeled (i.e., different concentrations/ingestion rates can be entered for different years to reflect changing site conditions; the model does not allow a temporal resolution finer than a year). The

IEUBK Model then uses the input data to generate a yearly average blood lead level for the population being modeled. The IEUBK Model is comprised of four distinct components that work together in series:

- Exposure component – Determines how much lead enters the child's body over the exposure period. This component combines media-specific (e.g., air, soil, food, water) lead concentrations and age-dependent media intake rates to calculate age- and media-specific lead intake rates.

- Uptake Component – Calculates how much of the lead that enters the body through the exposure routes is actually absorbed into the blood.

- Biokinetic Component – Models the distribution of the lead from the blood to other body tissues and/or elimination from the body.

- Probability Distribution Component – Calculates a probability distribution of blood lead for a hypothetical child or population of children. The geometric mean blood lead concentration is calculated. This is combined with a prescribed Geometric Standard Deviation representing inter-individual variability in lead uptake to generate a blood lead distribution from which the probability (e.g., the estimated proportion) of the target population to exceed a blood lead level of 10 µg/dL is estimated.

The IEUBK Model uses standard age-weighted exposure parameters for consumption or intake rates of food, drinking water, soil, dust, and inhalation of air. These parameters are combined with the available site-specific information on the concentrations of lead in these media in order to estimate exposure for the child. The model inserts default values whenever site-specific information is not used. The default values (e.g., dietary lead concentrations, consumption values) are typical of a child's environment and were derived from research and published information on lead levels in environmental media and child-specific consumption and intake rates for U.S. children.

The IEUBK Model is generally applied to characterize lead exposure for sites or situations where the local background environmental media concentrations of lead have already been impacted or could be impacted by emissions of lead from a specific local source. Examples include lead emissions present at contaminated waste sites and lead emissions into air from point sources or area sources.

The CDF is an example of an area source from which lead emissions are possible due to particulate emissions to air followed by air transport and deposition to soil. Consequently, operation of the CDF could cause an incremental addition of lead to air and soil in the vicinity of the CDF. For the SRA, the IEUBK Model is used to evaluate the whether lead emissions from the CDF could have a significant impact on the predicted blood lead level of children assumed to reside in the vicinity of the CDF. The IEUBK Model evaluates a typical child resident assumed to live in each local neighborhood selected for evaluation within the risk assessment study area.

The input parameters needed to run the IEUBK Model are presented in Table 6-7. These parameters represent a combination of site-specific parameters, conservative default parameters, and child-specific exposure parameters. The table provides a rationale for the parameter selection and the origin of the selected values.

Table 6-7: Input Values for the IEUBK Lead Model

Medium	Parameter	Model Default Value(s)[17]	Value Used	Comment
Air	Background lead air concentration	0.1 µg/m^3	0.035 µg/m^3	Value used is site-specific; based on air monitoring at E.C. Central High School[18]
Air	Addition to lead air background concentration due to CDF emission		Site-specific	Site-specific value for each location determined by lead emission rate and air dispersion modeling
Air	Inhalation Rate	6 - 12 months: 2.0 m^3/day 12 - 24 months: 3.0 m^3/day 24 - 36 months: 5.0 m^3/day 36 - 48 months: 5.0 m^3/day 48 - 60 months: 5.0 m^3/day 60 - 72 months: 7.0 m^3/day 72 - 84 months: 7.0 m^3/day	Model default	
Soil	Background lead surface soil concentration	200 mg/kg	Model default	Model default is a reasonable estimate for an urban setting if verified site-specific data not available
Soil	Addition to lead soil background concentration due to CDF emission		Site-specific	Site-specific value for each location determined by lead emission rate and air dispersion/air deposition modeling
Soil	Soil ingestion rate	6 - 12 months: 85 mg/day 12 - 24 months: 35 mg/day 24 - 36 months: 35 mg/day 36 - 48 months: 135 mg/day 48 - 60 months: 100 mg/day 60 - 72 months: 90 mg/day 72 - 84 months: 85 mg/day	Model default	
Diet	Lead intake from food	6 - 12 months: 5.53 µg/day 12 - 24 months: 5.78 µg/day 24 - 36 months: 6.49 µg/day 36 - 48 months: 6.24 µg/day 48 - 60 months: 6.01 µg/day 60 - 72 months: 6.34 µg/day 72 - 84 months: 7.00 µg/day	Model default	
Water	Background lead drinking water concentration	4 µg/Liter	Model default	
Water	Water ingestion rate	6 - 12 months: 0.2 Liter/day 12 - 24 months: 0.5 Liter/day 24 - 36 months: 0.52 Liter/day 36 - 48 months: 0.53 Liter/day 48 - 60 months: 0.55 Liter/day 60 - 72 months: 0.58 Liter/day 72 - 84 months: 0.59 Liter/day	Model default	
Maternal blood lead	Maternal blood lead concentration at birth	2.5 µg/deciliter	Model default	

[17] Model default values are taken from USEPA (2001a)

[18] The value of 0.035 $\mu g/m^3$ is the highest quarterly mean value for all lead monitoring data reported for the air monitoring station located at East Chicago Central High School for the period April 2002 through October 2005. The data are from the USACE Long-Term Perimeter Air Monitoring Project for the Indiana Harbor CDF (https://web.ead.anl.gov/inharbor/data/)

6.4 Risk Characterization and Results

In the risk characterization step of the risk assessment process, the information from the preceding steps and procedures of the assessment are combined and integrated. In particular, the dose-response analysis and chemical toxicity factors for the selected chemical contaminants are combined with dose estimates representing potential chemical contaminant exposure from each of the applicable exposure pathways. Exposure pathways are combined to evaluate the health risks associated with specific exposure scenarios. Health risks are defined in terms of the potential for cancer risk and noncancer health effects, usually referred to as health hazards. In addition to the quantitative aspects of the risk assessment (i.e., "number crunching"), the risk characterization is also intended to place the estimated risks in context through a discussion of the qualitative elements of the risk assessment, including the major factors influencing the risk estimates, important underlying assumptions, and the rationale for these assumptions. In addition, the risk characterization generally includes a discussion of the uncertainty and variability associated with the quantitative results.

6.4.1 Modeling Methodology

As described earlier in Section 3, the risk assessment for the SRA employs a combination of "model" procedures based on previously developed and reviewed USEPA guidance for risk assessment:

- *Risk Assessment Guidance for Superfund* (USEPA 1989) outlines the general methodology that can be followed to define chemical exposure pathways and to evaluate health risks for chemical contaminants released to air, soil, and water. The methodologies are applicable for chemical releases from practically any source.

- *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (HHRAP; "combustion guidance") (USEPA 1998b, 2005c). The combustion guidance outlines a comprehensive procedure for calculating estimated environmental media (e.g., air, soil, vegetables, fish, meat) concentrations, human intake rates, and health risks due to the emission of chemicals from combustion stacks. While the guidance was written specifically for hazardous waste combustion facilities, the procedures are applicable to other sources that generate chemical emissions to ambient air (e.g., municipal waste combustors, boilers, area sources).

6.4.2 Methodology for Estimating Cancer Risk

A cancer risk is an expression of the probability that an individual will develop cancer, based on a unique set of exposure, model, and toxicity assumptions. For example, a risk of 2×10^{-5} is interpreted to mean that an individual has no more than a 2 in 100,000 chance of developing cancer over a lifetime from the exposure being evaluated. An alternative interpretation is that

no more than 2 cases of cancer would be expected in a population of 100,000 individuals who all received exposure at the same level of chemical intake. [19]

As described above, for carcinogenic chemicals, risk estimates represent the incremental probability that an individual will develop cancer as a result of a specific exposure to a carcinogenic chemical. These risks are calculated as follows:

$$Cancer\ Risk = LADD \times CSF$$

Where:

LADD = Lifetime average daily dose (mg/kg-day)
CSF = CSF $(mg/kg\text{-}day)^{-1}$

Within a specific exposure pathway, receptors may be exposed to more than one chemical. The total risk associated with exposure to all chemicals through a single exposure pathway is estimated as follows:

$$Cancer\ Risk_T = \Sigma_i\ Cancer\ Risk_i$$

Where:

$Cancer\ Risk_T$ = Total cancer risk for a specific exposure pathway
$\Sigma Cancer\ Risk_i$ = Sum of cancer risks for all chemicals in a specific exposure pathway

At a given exposure scenario location, receptors may be exposed through a number of exposure pathways. Risks from multiple exposure pathways that apply to the same individual should be summed within each exposure scenario. That is, risks should be summed across the scenario-exposure pathway combinations which have been identified for the study. The total risk posed to a receptor is the sum of total risks from each individual exposure pathway expressed as follows:

$$Total\ Cancer\ Risk = \Sigma Cancer\ Risk_T$$

Where:

Total Cancer Risk = Total cancer risk from multiple exposure pathways
$\Sigma Cancer\ Risk_T$ = Sum of cancer risks for individual exposure pathways

The oral and inhalation CSF values used for this analysis are discussed in Section 6.1 and are incorporated into the IRAP software model.

6.4.3 Methodology for Estimating Noncancer Health Risk - Hazard Quotient

A hazard quotient (HQ) is an expression of the potential for developing a noncarcinogenic health effect as a result of exposure to potentially toxic chemicals, averaged over an appropriate

[19] Cancer risk estimates can be presented in a number of notations or short-hand forms. For example, a cancer risk of 2 in 100,000 could be abbreviated as 2×10^{-5} or as 2E-05; each notation has the same quantitative meaning.

exposure period. A HQ is not a probability but actually an indicator (calculated as a ratio) that the exposure level of a chemical would be expected to cause adverse health effects by comparison to a standard exposure level. The standard exposure level (i.e., an RfD or RfC) is a chemical dose or chemical concentration that is projected to pose no significant likelihood of adverse health effects even with long-term exposure.

Standard risk assessment models assume that noncarcinogenic effects exhibit a threshold; that is, there is a level of exposure below which no adverse effects will be observed. The potential for noncarcinogenic health effects resulting from exposure to a chemical is generally assessed by: (1) comparing an exposure estimate to an RfD for oral exposures, or (2) comparing an estimated chemical-specific air concentration to an RfC for direct inhalation exposures. An RfD is a daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a specific exposure duration. Similarly, an RfC is an estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations.

The comparisons of exposure estimates or chemical-specific air concentrations to RfD and RfC values are known as HQs, which are calculated as follows:

$$HQ = \frac{ADD}{RfD} \quad or \quad HQ = \frac{Ca}{RfC}$$

Where:

HQ	=	Hazard quotient (unitless)
ADD	=	Average daily dose (mg/kg-day) of the chemical
Ca	=	Chemical air concentration (mg/m^3)
RfD	=	Reference dose (mg/kg-day)
RfC	=	Reference concentration (mg/m^3)

The general interpretation of HQ is that a value of less than or equal to 1 is considered health-protective. Because RfDs and RfCs do not have equal accuracy or precision, and are not based on the same severity of effect, the level of concern does not increase linearly as an HQ approaches and exceeds 1. This means that adverse health effects are not predicted to occur as soon as an HQ exceeds a value of 1. But in general, the more that the HQ value exceeds 1, the greater the level of concern becomes.

As with carcinogenic chemicals in a specific exposure pathway, a receptor may be exposed to multiple chemicals associated with noncarcinogenic health effects. The total noncarcinogenic hazard for each exposure pathway is calculated by following the procedures outlined in USEPA (1986). Specifically, the total noncarcinogenic hazard attributable to exposure to all COCs through a single exposure pathway is known as a hazard index (HI). Consistent with the procedure for addressing carcinogenic risks, the noncarcinogenic hazards are summed for each chemical constituent in a given exposure pathway. The HI is calculated as follows:

$$HI = \Sigma HQ_i$$

Where:

HI = HI; total hazard for a specific exposure pathway
ΣHQ_i = Sum of HQs for all chemicals in a specific exposure pathway

This summation methodology assumes that the health effects of the various chemicals to which an individual is exposed are additive. Specifically, this methodology is a simplification of the HQ concept because it does not directly consider the portal of entry (i.e., oral or inhalation) associated with each exposure pathway or the differences in toxic endpoints, target organs, and toxicity mechanisms of the various chemicals.

The potential uncertainty created by the use of the simple summation methodology has been discussed in the *Risk Assessment Guidance for Superfund* (USEPA 1989). As stated in the guidance, the assumption of dose additivity most properly applies to chemical contaminants which induce the same toxicological effect by the same mechanism of action. Consequently, application of the HI additivity concept to a number of chemicals that are not expected to induce the same type of effects or that do not act by the same mechanism could overestimate the potential for effects. However, the additivity approach is appropriate to apply for a screening level evaluation of the HI. The uncertainty associated with the approach should not be a concern if only one or two chemical substances are responsible for driving the HI above unity (i.e., above 1). If the HI is greater than unity as a consequence of summing several HQs of similar value, it would be more appropriate to segregate the chemicals by target effect and by mechanism of action. Then chemicals which share a similar target effect or mechanism of action should be placed into separate groups for deriving HIs.

For this SRA, the screening level approach presented in the *Risk Assessment Guidance for Superfund* (USEPA 1989) was applied with the understanding that the resulting HI values may cause an overestimate of the actual potential for adverse health effects.

As discussed earlier for carcinogenic risks, a receptor may be exposed to chemicals associated with noncarcinogenic health effects through more than one exposure pathway. For the purposes of the risk assessment, it is reasonable to estimate a receptor's total hazard as the sum of the HIs for each of the identified exposure pathways. Specifically, a receptor's total hazard is the sum of hazards from each individual exposure pathway, expressed as follows:

$$Total\ HI = \Sigma\ HI$$

Where:

$Total\ HI$ = Total HI from multiple exposure pathways
HI = Sum of HI values for individual exposure pathways

The reference toxicity factors (RfDs or RfCs) used for this analysis are discussed in Section 6.1 and are incorporated into the IRAP software model.

6.4.4 Estimates of Cancer Risk and Hazard Index using IRAP-h

Because the evaluation of multiple chemicals, multiple exposure pathways, and multiple fate and transport processes can be a very challenging computational exercise, the SRA utilized a computer software program to accomplish running the risk assessment model for the SRA. For this project, the software system called *Industrial Risk Assessment Protocol - Human Health*

(IRAP-h View™) was used. This software package (abbreviated "IRAP") was developed by Lakes Environmental Software (Waterloo, Ontario, Canada). IRAP was expressly designed to closely follow the recommendations, chemical-specific parameters, and fate and transport algorithms given in USEPA's 2005 combustion risk guidance. IRAP is a Microsoft Windows application that can be run in the Windows 3.1, Windows 3.11, Windows 95, Windows 98, and Windows NT operating systems. (The USEPA-Region 5 copy of IRAP is a 32-bit version that runs in the Windows NT operating system).

The major features of the IRAP system are its ability to:

- Import ISCST3 plot files containing the output from the ISCST3 air dispersion/deposition model runs
- Provide a graphical display of the ISCST3 receptor grid node locations
- Directly import GIS-generated land use/land cover data (e.g., residential, farming, and water body locations)
- Define the perimeter of water bodies and water sheds using a polygon drawing tool
- Define an area of concern by selecting the receptor grid nodes that cover important land use characteristics such as residential zones, recreational zones, and agricultural land
- Guide the user through the process recommended in the HHRAP combustion guidance for defining exposure scenarios and selecting exposure pathways
- Eliminate the need to perform hand calculations and write multiple interconnected computation spreadsheets
- Simultaneously calculate risk values (cancer risks and HQs) for multiple chemicals emitted from a single source or from multiple sources at multiple locations.

The IRAP system incorporated information on the geographic location of the CDF and local land use and demographic information in order to construct a Study Area of interest and define specific residential neighborhoods/zones and activity areas (e.g., schools) for which human health risk will be evaluated. (The development of the Study Area was described in Section 4 and Section 6.3.1).

Due to its computation power, the IRAP software is capable of generating a very large number of potential health risk estimates within the Study Area. The risk estimates (cancer risk and HQ) could vary significantly by geographic location based on the air dispersion/deposition model results combined with the exposure scenario which is applied at a given location. In order to generate a consistent and understandable representation of risk estimates within the Study Area, the IRAP risk estimation procedure was utilized in the following way for the this SRA:

1) IRAP recognizes the geographical boundary placed on each residential neighborhood/ zone and the school zone. Within each boundary zone, IRAP recognizes all receptor grid node points associated with the ISCST3 air model output files.

2) From the collection of ISCST3 grid points, IRAP selects the grid points associated with the "highest-parameter points" from the ISCST3 output. In each boundary area, there can be up to sixteen highest-parameter points. These "highest-parameter points" are determined from the air dispersion modeling results and are based on the emissions phase (i.e., vapor, particle and particle-bound, air concentration, dry deposition rate, wet deposition rate, and total deposition rate). In practice, there are usually between one and six distinct highest-parameter point locations in the air modeling output files, with all other highest-parameter points overlaying these point locations (i.e., co-located).

3) For each distinct highest-parameter point location, IRAP calculates total cancer risk and HI using the exposure pathways and exposure factors that apply within each zone.

4) Based on the calculated risk and hazard results, the IRAP user can identify the parameter point location corresponding to the highest calculated risk and hazard for the combination of exposure pathways that apply within a boundary. The results for this location are referred to as "highest-combined exposure pathway risk" estimate for the boundary zone.

The procedure described above was adopted to derive cancer risk and HQ estimates for the SRA. In addition to the total cancer risk and HI, IRAP also estimates individual contaminant cancer risks and contaminant HQs so that chemicals which contribute the most to cancer risk and HI can be identified.

Using the methodology described in Section 6.4.2 and Section 6.4.3, cancer risk estimates and noncancer hazard estimates were derived for each exposure scenario selected in the SRA:

6.4.4.1 Local Resident Scenario

The applicable exposure pathways are:

- Inhalation of volatile contaminants and particulate contaminants
- Incidental ingestion of soil containing deposited contaminants
- Dermal contact with soil containing deposited contaminants
- Ingestion of contaminants incorporated into produce from a typical home garden.

Table 6-8 lists the estimated cancer risks for an adult resident individual assumed to reside in one of the five residential neighborhood zones selected for evaluation in the SRA. The estimated cancer risks are displayed by exposure pathway and by additive risk across the combination of pathways.

TABLE 6-8: Local Adult Resident Cancer Risk – Exposure Pathway Estimates

Residential Zone	Inhalation	Soil Ingestion	Dermal Absorption	Home Garden Consumption	Sum of Pathway Risks
Calumet	3.3E-07	4.9E-08	1.2E-08	2.3E-07	6.2E-07
Hammond	1.6E-06	3.8E-08	9.3E-09	1.8E-07	1.8E-06
Marktown	4.6E-06	6.9E-08	1.7E-08	3.2E-07	5.0E-06
North Harbor/East Harbor	8.0E-07	2.6E-08	6.3E-09	1.2E-07	9.5E-07
Northside/Southside	4.2E-06	8.4E-07	2.1E-07	4.0E-06	9.2E-06
Robertsdale	7.8E-06	< 1.0E-10	3.2E-10	< 1.0E-10	7.8E-06

Table 6-9 lists the estimated cancer risks for a child resident individual assumed to reside in one of the five residential neighborhood zones selected for evaluation in the SRA. The estimated cancer risks are displayed by exposure pathway and by additive risk across the combination of pathways.

TABLE 6-9: Local Child Resident Cancer Risk – Exposure Pathway Estimates

Residential Zone	Inhalation	Soil Ingestion	Dermal Absorption	Home Garden Consumption	Sum of Pathway Risks
Calumet	6.6E-08	9.2E-08	1.6E-08	6.5E-08	2.4E-07
Hammond	3.2E-07	7.1E-08	1.2E-08	5.0E-08	4.5E-07
Marktown	9.2E-07	1.3E-07	2.2E-08	9.1E-08	1.1E-06
North Harbor/East Harbor	1.6E-07	4.8E-08	8.3E-09	3.4E-08	2.5E-07
Northside/Southside	8.4E-07	1.6E-06	2.7E-07	1.1E-06	3.8E-06
Robertsdale	1.6E-06	< 1.0E-10	4.1E-10	< 1.0E-10	1.6E-06

Table 6-10 lists the estimated noncancer HI values for an adult resident individual assumed to reside in one of the five residential neighborhood zones selected for evaluation in the SRA. The estimated HI values are displayed by exposure pathway and as a sum across the combination of pathways.

TABLE 6-10: Local Adult Resident Hazard Index – Exposure Pathway Estimates

Residential Zone	Inhalation	Soil Ingestion	Dermal Absorption	Home Garden Consumption	Sum of Pathway Index
Calumet	0.0079	0.00015	0.00018	0.0024	0.011
Hammond	0.0390	0.00012	0.00014	0.0019	0.041
Marktown	0.1120	0.00022	0.00026	0.0035	0.116
North Harbor/East Harbor	0.0196	0.00008	0.00009	0.0013	0.021
Northside/Southside	0.0990	0.0026	0.00301	0.0399	0.144
Robertsdale	0.1907	0.00001	0.00002	0.00033	0.191

Table 6-11 lists the estimated noncancer HI values for a child resident individual assumed to reside in one of the five residential neighborhood zones selected for evaluation in the SRA. The estimated HI values are displayed by exposure pathway and as a sum across the combination of pathways.

TABLE 6-11: Local Child Resident Hazard Index – Exposure Pathway Estimates					
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Sum of Pathway Index**
Calumet	0.0079	0.0014	0.00115	0.0033	0.013
Hammond	0.0390	0.0011	0.00092	0.0026	0.044
Marktown	0.1120	0.0021	0.00167	0.0049	0.121
North Harbor/East Harbor	0.0196	0.00076	0.00062	0.0018	0.023
Northside/Southside	0.099	0.0245	0.0197	0.0558	0.199
Robertsdale	0.1907	0.00012	0.00012	0.00046	0.191

6.4.4.2 Local Fisher Scenario

A local fisher is an individual (adult or child) who is a resident of the local area in the vicinity of the CDF and who also obtains a significant portion of the diet from consuming fish harvested from a local water body. Consequently, the applicable exposure pathways are:

- Consumption of fish fillets harvested from a local water body
- Exposure pathways expected for a Local Area Resident (as described above).

A water body located within the Study Area is determined to be candidate water body for evaluation if: (1) significant fishing is known to occur, and/or (2) information is available to indicate that access for fishing is possible and fishing is recommended by a State natural resource agency or other organization which promotes recreational fishing.

The individual who harvests fish from one of these lakes is assumed to be a high-end fish consumer in keeping with the concept that the SRA will evaluate RME levels within the selected scenarios. The two water bodies selected for evaluation were Lake George and Powderhorn Lake (Section 6.3). Because of similarities in the size of the two lakes and their distance and direction from the CDF, it was not possible, *a priori*, to determine which lake might receive higher impacts from CDF contaminants due a combination of direct deposition and overland runoff of contaminants. Consequently, it was necessary to evaluate contaminant impacts to both lakes. The following estimates of cancer risk and noncancer hazard were obtained based on the assumption that each water body could support the intake level of a high-end consumer.

Table 6-12: Estimated Cancer Risk and Hazard Index from Local Fish Consumption				
Water body	**Cancer Risk**		**Hazard Index**	
	Adult Fisher	Child Fisher	Adult Fisher	Child Fisher
Lake George	< 1.0E-08	< 1.0E-08	0.0738	0.0479
Powderhorn Lake	5.3E-06	6.8E-07	0.0655	0.0425

As shown in Table 6-12, the evaluation indicated that fishing from Powderhorn Lake gave significantly higher estimates of cancer risk than fishing from Lake George. Fishing from Lake George gave slightly higher estimates of HI than fishing from Powderhorn Lake. The values shown in Table 6-12 were used to complete the following tables which summarize the estimated cancer risks and HI values for a Local Fisher who is also assumed to be a local resident.

Table 6-13: Adult Resident Fisher Cancer Risk – Exposure Pathway Estimates (Fish Obtained from Lake George)

Residential Zone	Inhalation	Soil Ingestion	Dermal Absorption	Home Garden Consumption	Fish Consumption	Sum of Pathway Risks
Calumet	3.3E-07	4.9E-08	1.2E-08	2.3E-07	< 1.0E-08	6.3E-07
Hammond	1.6E-06	3.8E-08	9.3E-09	1.8E-07	< 1.0E-08	1.8E-06
Marktown	4.6E-06	6.9E-08	1.7E-08	3.2E-07	< 1.0E-08	5.0E-06
North Harbor/ East Harbor	8.0E-07	2.6E-08	6.3E-09	1.2E-07	< 1.0E-08	9.6E-07
Northside/ Southside	4.2E-06	8.4E-07	2.1E-07	4.0E-06	< 1.0E-08	9.3E-06
Robertsdale	7.8E-06	< 1.0E-10	3.2E-10	< 1.0E-10	< 1.0E-08	7.8E-06

Table 6-14: Child Resident Fisher Cancer Risk – Exposure Pathway Estimates (Fish Obtained from Lake George)

Residential Zone	Inhalation	Soil Ingestion	Dermal Absorption	Home Garden Consumption	Fish Consumption	Sum of Pathway Risks
Calumet	6.6E-08	9.2E-08	1.6E-08	6.5E-08	< 1.0E-08	2.5E-07
Hammond	3.2E-07	7.1E-08	1.2E-08	5.0E-08	< 1.0E-08	4.6E-07
Marktown	9.2E-07	1.3E-07	2.2E-08	9.1E-08	< 1.0E-08	1.2E-06
North Harbor/ East Harbor	1.6E-07	4.8E-08	8.3E-09	3.4E-08	< 1.0E-08	2.6E-07
Northside/ Southside	8.4E-07	1.6E-06	2.7E-07	1.1E-06	< 1.0E-08	3.8E-06
Robertsdale	1.6E-06	< 1.0E-10	4.1E-10	< 1.0E-10	< 1.0E-08	1.6E-06

Table 6-15: Adult Resident Fisher Hazard Index – Exposure Pathway Estimates (Fish Obtained from Lake George)						
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Fish Consumption**	**Sum of Pathway Index**
Calumet	0.0079	0.00015	0.00018	0.0024	0.0738	0.084
Hammond	0.0390	0.00012	0.00014	0.0019	0.0738	0.115
Marktown	0.1120	0.00022	0.00026	0.0035	0.0738	0.190
North Harbor/ East Harbor	0.0196	0.00008	0.00009	0.0013	0.0738	0.095
Northside/ Southside	0.0990	0.0026	0.00301	0.0399	0.0738	0.218
Robertsdale	0.1907	0.00001	0.00002	0.00033	0.0738	0.265

Table 6-16: Child Resident Fisher Hazard Index – Exposure Pathway Estimates (Fish Obtained from Lake George)						
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Fish Consumption**	**Sum of Pathway Index**
Calumet	0.0079	0.0014	0.00115	0.0033	0.0479	0.062
Hammond	0.0390	0.0011	0.00092	0.0026	0.0479	0.092
Marktown	0.1120	0.0021	0.00167	0.0049	0.0479	0.168
North Harbor/ East Harbor	0.0196	0.00076	0.00062	0.0018	0.0479	0.071
Northside/ Southside	0.099	0.0245	0.0197	0.0558	0.0479	0.247
Robertsdale	0.1907	0.00012	0.00012	0.00046	0.0479	0.239

Table 6-17: Adult Resident Fisher Cancer Risk – Exposure Pathway Estimates (Fish Obtained from Powderhorn Lake)						
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Fish Consumption**	**Sum of Pathway Risks**
Calumet	3.3E-07	4.9E-08	1.2E-08	2.3E-07	5.3E-06	5.9E-06
Hammond	1.6E-06	3.8E-08	9.3E-09	1.8E-07	5.3E-06	7.1E-06
Marktown	4.6E-06	6.9E-08	1.7E-08	3.2E-07	5.3E-06	1.0E-05
North Harbor/ East Harbor	8.0E-07	2.6E-08	6.3E-09	1.2E-07	5.3E-06	6.2E-06
Northside/ Southside	4.2E-06	8.4E-07	2.1E-07	4.0E-06	5.3E-06	1.4E-05
Robertsdale	7.8E-06	< 1.0E-10	3.2E-10	< 1.0E-10	5.3E-06	1.3E-05

Table 6-18: Child Resident Fisher Cancer Risk – Exposure Pathway Estimates (Fish Obtained from Powderhorn Lake)						
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Fish Consumption**	**Sum of Pathway Risks**
Calumet	6.6E-08	9.2E-08	1.6E-08	6.5E-08	6.8E-07	9.2E-07
Hammond	3.2E-07	7.1E-08	1.2E-08	5.0E-08	6.8E-07	1.1E-06
Marktown	9.2E-07	1.3E-07	2.2E-08	9.1E-08	6.8E-07	1.8E-06
North Harbor/ East Harbor	1.6E-07	4.8E-08	8.3E-09	3.4E-08	6.8E-07	9.3E-07
Northside/ Southside	8.4E-07	1.6E-06	2.7E-07	1.1E-06	6.8E-07	4.5E-06
Robertsdale	1.6E-06	< 1.0E-10	4.1E-10	< 1.0E-10	6.8E-07	2.3E-06

Table 6-19: Adult Resident Fisher Hazard Index – Exposure Pathway Estimates (Fish Obtained from Powderhorn Lake)						
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Fish Consumption**	**Sum of Pathway Index**
Calumet	0.0079	0.00015	0.00018	0.0024	0.0655	0.076
Hammond	0.0390	0.00012	0.00014	0.0019	0.0655	0.106
Marktown	0.1120	0.00022	0.00026	0.0035	0.0655	0.182
North Harbor/ East Harbor	0.0196	0.00008	0.00009	0.0013	0.0655	0.086
Northside/ Southside	0.0990	0.0026	0.00301	0.0399	0.0655	0.210
Robertsdale	0.1907	0.00001	0.00002	0.00033	0.0655	0.256

Table 6-20: Child Resident Fisher Hazard Index – Exposure Pathway Estimates (Fish Obtained from Powderhorn Lake)						
Residential Zone	**Inhalation**	**Soil Ingestion**	**Dermal Absorption**	**Home Garden Consumption**	**Fish Consumption**	**Sum of Pathway Index**
Calumet	0.0079	0.0014	0.00115	0.0033	0.0425	0.056
Hammond	0.0390	0.0011	0.00092	0.0026	0.0425	0.086
Marktown	0.1120	0.0021	0.00167	0.0049	0.0425	0.164
North Harbor/ East Harbor	0.0196	0.00076	0.00062	0.0018	0.0425	0.066
Northside/ Southside	0.099	0.0245	0.0197	0.0558	0.0425	0.242
Robertsdale	0.1907	0.00012	0.00012	0.00046	0.0425	0.234

6.4.4.3 Local Area Student Scenario

For a local student who attends school in the zone corresponding to the location of Central High School and West Side Junior High School, the applicable exposure pathways are the following:

- Inhalation of volatile contaminants and particulate contaminants during school attendance
- Incidental ingestion of soil contaminants during school attendance or as a "student-athlete"
- Dermal contact with soil contaminants during activities typical of a "student-athlete" during school attendance.

Table 6-21 lists the estimated cancer risks and noncancer HI values.

Table 6-21: Local Area Student Cancer Risk and Hazard Index – Exposure Pathway Estimates			
Exposure Pathway	**Cancer Risk**	**Exposure Pathway**	**Hazard Index**
Inhalation	1.3E-06	**Inhalation**	0.1579
Soil Ingestion	2.3E-07	**Soil Ingestion**	0.0036
Dermal Absorption	1.0E-07	**Dermal Absorption**	0.0077
Sum of Pathway Risk	1.6E-06	**Sum of Pathway Hazard Index**	0.169

6.4.5 Intake of Lead: Uptake-Biokinetic Model for Lead

The CDF is an example of an area source from which lead emissions are possible due to particulate emissions to ambient air followed by air transport and deposition to soil. Consequently, operation of the CDF could cause an incremental addition of lead to air and soil in the vicinity of the CDF. The results of the air emissions modeling and the air dispersion/deposition modeling were combined with the fate and transport characteristics of lead in soil after deposition. The IRAP Model calculated estimates of the incremental contribution of lead to ambient air and soil within the Study Area. The results of the lead transport and deposition analysis are summarized in Table 6-22.

Table 6-22: Estimated Incremental Increases in Soil and Air Lead Concentrations Due to Particulate Lead Emissions from the Confined Disposal Facility

Residential Zone or Neighborhood	Emission Source at the CDF	Estimated Soil Lead Concentration		Estimated Lead Ambient Air Concentration ($\mu g/m^3$)
		Average[3] (mg/kg)	Maximum[4] (mg/kg)	
Calumet	East Cell:	0.74	1.39	1.16E-05
	West Cell:	0.10	0.18	1.83E-06
	Total:	0.84	1.57	1.34E-05
Hammond	East Cell:	0.11	0.21	1.81E-06
	West Cell:	0.55	1.02	1.15E-05
	Total:	0.66	1.23	1.33E-05
Marktown	East Cell:	0.50	0.93	9.87E-06
	West Cell:	0.70	1.30	1.32E-05
	Total:	1.20	2.23	2.31E-05
North Harbor / East Harbor	East Cell:	0.17	0.33	3.03E-06
	West Cell:	0.27	0.50	4.95E-06
	Total:	0.44	0.83	7.98E-06
Northside / Southside	East Cell:	6.3	11.8	9.60E-05
	West Cell:	8.2	15.3	1.63E-04
	Total:	14.5	27.1	2.59E-04
Robertsdale	East Cell:	< 0.01	< 0.01	< 1.00E-06
	West Cell:	< 0.01	< 0.01	< 1.00E-06
	Total:	< 0.01	< 0.01	< 1.00E-06
School Location	East Cell:	5.8	10.9	9.21E-05
	West Cell:	25.6	47.8	5.16E-04
	Total:	31.4	58.7	6.08E-04

[1] Predicted increase in soil lead concentration compared to existing background level.
[2] Predicted increase in ambient air lead concentration compared to existing background level.
[3] Average increase in soil lead concentration during time period of deposition (30 years).
[4] Maximum increase in soil lead concentration at end of deposition period (30 years).

Table 6-22 presents the average and maximum predicted increases in soil lead concentration due to CDF emissions within each residential zone and at the school zone.[20] The average soil lead concentration corresponds to the approximate average or mean increase in lead concentration which is predicted during the time period (i.e., zero to 30 years) in which the CDF is in active operation. The maximum soil lead concentration corresponds to the highest increase in lead concentration which is predicted at the completion of the active CDF operation (i.e., at completion of 30 years).

USEPA's IEUBK Model was used to evaluate whether lead emissions from the CDF could have a significant impact on the predicted blood lead level of children assumed to reside in the vicinity of the CDF. The IEUBK Model is used to evaluate children assumed to live in each local neighborhood selected for evaluation within the risk assessment study area. The required

[20] The predicted incremental increase in soil lead and ambient air lead at the School Zone is shown for comparison purposes. Since young children (infant to seven years) will not reside at or frequent the School Zone in the foreseeable future, the School Zone is not evaluated in the IEUBK Model for child lead exposure.

inputs to the IEUBK Model were described in Section 6.3.5.9 and listed in Table 6.7. The site-specific inputs to the IEUBK Model include the following quantitative values:

1) A background ambient air concentration of lead that applies before the CDF begins operating. Ambient air monitoring data reported at a monitoring station located at East Chicago Central High School were evaluated in order to obtain a site-specific value. From these data, the highest quarterly ambient air lead concentration was determined for the time period from April 2002 - December 2005. This value was determined to be 0.035 $\mu g/m^3$.

2) A background soil lead concentration that applies before the CDF begins operating. Site-specific data on lead (or other contaminants of interest) within East Chicago soils that could be used for determining a valid site-specific background level of lead in soil were not available at the time the SRA was developed. Consequently, the IEUBK Model-recommended default background level of 200 mg/kg (200 ppm) was used for the analysis. This is considered a reasonably conservative (i.e., reasonably high) value to apply in the absence of site-specific data (USEPA 2001a).

3) The incremental increase in soil lead due to CDF operation. For each residential zone, the IEUBK Model was run using the maximum incremental increase in soil lead as given in Table 6-22. (The maximum incremental increase corresponds to the assumption that a child's exposure to soil begins after deposition of lead from the CDF is complete).

4) The incremental increase in the ambient air concentration of lead during the period of deposition from operation of the CDF. IRAP calculates an annual average increase in the ambient air concentration of lead during the CDF operating period.

The IEUBK Model generates two primary output predictions of interest to the general user:

1) A probability distribution curve that determines the geometric mean blood lead concentration for a population of children each exposed to lead under a specific exposure scenario (e.g., a fixed set of lead environmental levels and intake exposure factors).

2) The probability of exceeding the specified blood lead level of concern. In this case, the blood lead level of concern is the CDC reference exposure level of 10 µg/dL. This probability may be interpreted as the percentage of children at the same specific exposure scenario who are expected to exceed the level of concern.

The IEUBK Model was run for each residential neighborhood scenario using the corresponding input values described above. The results are summarized in Table 6-23. The corresponding probability distribution curves and text output files are presented in Appendix 6-5.

Table 6-23: Summary of IEUBK Model Results for Child Exposure to Lead			
Residential Zone or Neighborhood	**Soil Lead Input to Model**	**Predicted Geometric Mean Blood Lead Level (µg/dL)**	**Predicted Percent of Children Above 10 µg/dL**
All Zones – CDF Not in Operation	Background soil lead (200 mg/kg)	3.36	0.97
Calumet	Background soil lead (200 mg/kg) + Incremental soil lead (1.6 mg/kg)	3.37	0.97
Hammond	Background soil lead (200 mg/kg) + Incremental soil lead (1.2 mg/kg)	3.37	0.97
Marktown	Background soil lead (200 mg/kg) + Incremental soil lead (2.2 mg/kg)	3.38	0.97
North Harbor/ East Harbor	Background soil lead (200 mg/kg) + Incremental soil lead (0.8 mg/kg)	3.36	0.97
Northside/ Southside	Background soil lead (200 mg/kg) + Incremental soil lead (27.1 mg/kg)	3.60	1.39
Robertsdale	Background soil lead (200 mg/kg) + Incremental soil lead (0.01 mg/kg)	3.36	0.97

6.4.6 Margin of Exposure Evaluation for Dioxin Intake

Most chemical compounds for which RfDs are derived are not widely distributed and/or persistent in the environment. Therefore, background exposures are generally very low and not taken into account in a risk assessment. USEPA's draft "Dioxin Reassessment" documents (USEPA 2000, 2003a) concluded that it would be inappropriate to develop a reference dose for