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Revised Risk Assessment for the Air Characteristic Study Volume II Technical Background Document

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**Revised Risk Assessment for the Air
Characteristic Study
Volume II
Technical Background Document**

Office of Solid Waste
U.S. Environmental Protection Agency
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1.0 Introduction

The U.S. Environmental Protection Agency (EPA), Office of Solid Waste (OSW), has analyzed the potential risks to human health posed by the inhalation of vapor (gaseous) and particulate (nongaseous) air emissions from a set of chemicals and metals when managed in certain waste management units. An analysis of these risks was initially performed in 1998 as part of the *Air Characteristic Study* (U.S. EPA, 1998a). In accordance with Agency policy, the risk assessment conducted for the 1998 Air Characteristic Study was peer reviewed to ensure that science was used credibly and appropriately in the work performed. Based on comments made by the peer reviewers, EPA has revised the original risk assessment.

This report presents the revised risk assessment in three volumes. This document is Volume II, the Technical Background Document. This volume provides a detailed description of the methodologies, data, and supporting analyses used for the risk assessment. A discussion of the changes made from the 1998 Air Characteristic Study, a general overview of the risk assessment, and the integration of the revised risk assessment results with the May 1998 regulatory gaps and occurrence analyses can be found in Volume I, *Revised Risk Analysis for the Air Characteristic Study: Overview*. The complete results of the analysis are presented in Volume III, *Revised Risk Analysis for the Air Characteristic Study: Results* (on CD-ROM).

1.1 Purpose and Requirements of the Air Characteristic Study

This report and the 1998 Air Characteristic Study are among the initial steps for EPA in fulfilling a long-standing goal to review the adequacy and appropriateness of the hazardous waste characteristics. The first step in achieving this goal was the Hazardous Waste Characteristic Scoping Study (U.S. EPA, 1996) that the Agency completed November 15, 1996, under a deadline negotiated with the Environmental Defense Fund. This study was conducted to identify potential gaps in the current hazardous waste characteristics, as well as other modifications and updates that are necessary to ensure that the definition of characteristics is complete, up-to-date, and based on state-of-the-art methodologies. Based on the initial bounding analysis of potential risks due to air emissions done as part of the Scoping Study, as well as follow-up analysis on potential gaps in regulatory coverage under the Clean Air Act and Subpart CC of the Resource Conservation and Recovery Act (RCRA), OSW identified air emissions from waste management units as one of the areas meriting further analysis.

The Air Characteristic Study addresses this area by examining the potential direct inhalation risks due to emissions from certain waste management units. On May 15, 1998, in accordance with a consent decree, EPA completed the first portion of the study. According to the consent decree with EDF, a second part of the Air Characteristic Study, covering surface impoundments receiving wastewaters that never exhibited a characteristic, will be completed

March 26, 2001. The purpose of the 1998 Air Characteristic Study, as outlined by the consent decree, was to investigate gaps in the current hazardous waste characteristics and CAA programs. In addition, resulting potential risks to human health posed by the inhalation of air emissions from wastes managed in certain waste management units were to be investigated.

The 1998 Air Characteristic Study has three components: an evaluation of the coverage and potential regulatory gaps in RCRA Subtitle C and the CAA, a risk analysis of air emissions from waste management units, and an evaluation of the occurrence of these constituents in nonhazardous industrial waste. The risk assessment component has undergone a peer review, and EPA has made a number of changes to the risk assessment based on peer reviewer comments. In addition, other revisions have been made based on public comments and improvements initiated by the Agency. Since the other components of the May 1998 Air Characteristic Study have not been revised, those analyses are not covered in this document. The original results of these analyses are used in Volume I of this report to present the significant findings from the integration of the revised risk assessment results with the regulatory gaps and occurrence analyses.

1.2 Overview of Risk Assessment

The risk assessment described in this document is a national analysis designed to assess the potential human health risk attributable to inhalation exposures when certain chemicals and metals are managed as waste in certain types of waste management units. The purpose of the analysis is to determine which chemicals and waste management units are of potential national concern purely from a risk perspective; it is not intended to draw conclusions concerning regulatory coverage. This information, combined with preliminary information on regulatory coverage and on the presence of these chemicals in nonhazardous waste, will be useful in determining the need for expanded regulatory coverage. Specifically, the purpose of this study is to provide technical information on the potential risk from WMU emissions to assist EPA in determining the need to expand regulatory coverage in the future.

The analysis presented in this report addresses specific chemicals that when managed as a waste may pose a risk through direct inhalation exposures. Tables 1-1 and 1-2 list the chemicals and metals included in this analysis. The analysis is structured so that the results of the risk assessment are the concentrations of each constituent that can be present in each type of WMU and still be protective of human health. The protective concentrations in waste were developed for three types of receptors: adult residents, child residents, and workers. Three risk endpoints—chronic (1 year), subchronic (1 month), and acute (1 day)—were evaluated.

The protective waste concentrations were estimated by modeling the emissions from a waste management unit, the transport through the ambient environment, and the exposure to a receptor to backcalculate a threshold concentration in a waste below which the risk to human health would fall below a pre-established threshold. The waste management scenario modeled in this analysis is storage, disposal, or treatment of industrial waste streams in RCRA subtitle D waste management units.

Table 1-1. Constituents Modeled for All WMUs

Constituent	CAS No.
Acetaldehyde [ethanal]	75-07-0
Acetone [2-propanone]	67-64-1
Acetonitrile [methyl cyanide]	75-05-8
Acrolein	107-02-8
Acrylonitrile	107-13-1
Allyl chloride	107-05-1
Arsenic	7440-38-2
Barium	7440-39-3
Benzene	71-43-2
Beryllium	7440-41-7
Bromodichloromethane [dichlorobromomethane]	75-27-4
Bromoform [tribromomethane]	75-25-2
Bromomethane [methyl bromide]	74-83-9
1,3-Butadiene	106-99-0
Cadmium	7440-43-9
Carbon disulfide	75-15-0
Carbon tetrachloride	56-23-5
Chlorobenzene	108-90-7
Chlorodibromomethane [dibromochloromethane]	124-48-1
Chloroform	67-66-3
Chloromethane [methyl chloride]	74-87-3
Chloroprene [2-chloro-1,3-butadiene]	126-99-8
Chromium VI	7440-47-3
Cobalt	7440-48-4
Cumene [isopropyl benzene]	98-82-8
Cyclohexanol	108-93-0
1,2-Dibromo-3-chloropropane	96-12-8
1,2-Dichlorobenzene [<i>o</i> -dichlorobenzene]	95-50-1
1,4-Dichlorobenzene [<i>p</i> -dichlorobenzene]	106-46-7
Dichlorodifluoromethane [CFC-12]	75-71-8
1,2-Dichloroethane [ethylene dichloride]	107-06-2
1,1-Dichloroethylene [vinylidene chloride]	75-35-4
1,2-Dichloropropane [propylene dichloride]	78-87-5
<i>cis</i> -1,3-Dichloropropylene	10061-01-5
<i>trans</i> -1,3-Dichloropropylene	10061-02-6
1,4-Dioxane [1,4-diethyleneoxide]	123-91-1
Epichlorohydrin [1-chloro-2,3-epoxypropane]	106-89-8
1,2-Epoxybutane	106-88-7
2-Ethoxyethanol [ethylene glycol monoethyl ether]	110-80-5
2-Ethoxyethanol acetate [2-EEA]	111-15-9
Ethylbenzene	100-41-4
Ethylene dibromide [1,2-dibromoethane]	106-93-4

(continued)

Table 1-1. (continued)

Constituent	CAS No.
Ethylene oxide	75-21-8
Formaldehyde	50-00-0
Furfural	98-01-1
Hexachloroethane	67-72-1
<i>n</i> -Hexane	110-54-3
Lead	7439-92-1
Manganese	7439-96-5
Mercury	7439-97-6
Methanol	67-56-1
2-Methoxyethanol	109-86-4
2-Methoxyethanol acetate [2-MEA]	110-49-6
Methyl tert-butyl ether	1634-04-4
Methylene chloride [dichloromethane]	75-09-2
Methyl ethyl ketone [2-butanone][MEK]	78-93-3
Methyl isobutyl ketone [hexone] [4-methyl-2-pentanone]	108-10-1
Methyl methacrylate	80-62-6
Naphthalene	91-20-3
Nickel	7440-02-0
2-Nitropropane	79-46-9
N-Nitrosodi- <i>n</i> -butylamine	924-16-3
N-Nitrosodiethylamine	55-18-5
N-Nitrosopyrrolidine	930-55-2
Propylene oxide	75-56-9
Pyridine	110-86-1
Styrene	100-42-5
1,1,1,2-Tetrachloroethane	630-20-6
1,1,2,2-Tetrachloroethane	79-34-5
Tetrachloroethylene [perchloroethylene]	127-18-4
Toluene	108-88-3
1,1,1-Trichloroethane [methyl chloroform]	71-55-6
1,1,2-Trichloroethane [vinyl trichloride]	79-00-5
Trichloroethylene	79-01-6
Trichlorofluoromethane [trichloromonofluoromethane]	75-69-4
1,1,2-Trichloro-1,2,2-trifluoroethane [freon 113]	76-13-1
Triethylamine	121-44-8
Vanadium	7440-62-2
Vinyl acetate	108-05-4
Vinyl chloride	75-01-4
Xylenes, mixed isomers [xylenes, total]	1330-20-7

Table 1-2. Constituents Modeled for Tanks Only

Constituent	CAS No.
Acrylamide	79-06-1
Acrylic acid	79-10-7
Aniline	62-53-3
Benzidine	92-87-5
Benzo(<i>a</i>)pyrene	50-32-8
2-Chlorophenol [<i>o</i> -chlorophenol]	95-57-8
Cresols, total	1319-77-3
7,12-Dimethylbenz[<i>a</i>]anthracene	57-97-6
N,N-Dimethyl formamide	68-12-2
3,4-Dimethylphenol	95-65-8
2,4-Dinitrotoluene	121-14-2
1,2-Diphenylhydrazine	122-66-7
Ethylene glycol	107-21-1
Hexachlorobenzene	118-74-1
Hexachloro-1,3-butadiene [hexachlorobutadiene]	87-68-3
Hexachlorocyclopentadiene	77-47-4
Isophorone	78-59-1
3-Methylcholanthrene	56-49-5
Nitrobenzene	98-95-3
Phenol	108-95-2
Phthalic anhydride	85-44-9
2,3,7,8-TCDD [2,3,7,8-tetrachlorodibenzo- <i>p</i> -dioxin]	1746-01-6
<i>o</i> -Toluidine	95-53-4
1,2,4-Trichlorobenzene	120-82-1

Emissions, transport, and exposure were modeled somewhat differently for the three risk endpoints (chronic, subchronic, and acute). For emissions and transport, different averaging times were used for each endpoint (one year for chronic, one month for subchronic, and one day for acute) to generate emission rates and dispersion factors. For exposure, subchronic and acute exposures were modeled deterministically, using the point of maximum exposure at a specific distance. Chronic exposures were modeled probabilistically using a Monte Carlo approach to capture variation in receptor location and exposure factors. The waste management units assessed are aerated treatment tanks, nonaerated treatment tanks, storage tanks, landfills, waste piles, and land application units. The risk assessment was structured to capture national variations in environmental settings. In addition, Monte Carlo analysis was used in the modeling to include the variations in receptor characteristics such as exposure parameters and location around the facility. The following section, Section 2, provides a more detailed overview of the risk analysis framework.

1.3 Organization of Report

The remainder of this report is organized as follows. Section 2 presents a brief overview of the modeling approach. Section 3 discusses waste management unit data and how they were used in the analysis. Section 4 describes the development of emissions for each unit type. Development of dispersion coefficients and health benchmarks are discussed in Sections 5 and 6, respectively. Section 7 describes the development of risk-specific waste concentration distributions. Section 8 discusses how uncertainty and variability are addressed in this analysis. References are provided in Section 9, and supporting analyses are included in Appendixes A through D.

1.4 Companion Documents

Volume I of this report, the *Overview*, contains a general discussion of the risk assessment and provides detailed information on the changes that were made to the risk assessment performed in 1998. In addition, Volume I presents significant findings from the integration of the revised risk assessment results with the May 1998 regulatory gaps and occurrence analyses.

Volume III of this report, *Results* (provided on CD-ROM), presents the detailed results of the risk analysis.

2.0 Modeling Approach and Data Sources

This section provides a general overview of the modeling approach and primary sources of data and describes how the risk analysis was conducted. Further detail on the methodology, models used, and a complete set of inputs and associated references are provided in Sections 3 through 8 of this document.

2.1 Overview of Modeling Approach

The overall goal of this risk analysis is to estimate the concentrations of constituents that can be present in a waste management unit (WMU) and remain protective of human health. These protective waste concentrations were calculated for 104 constituents¹ including volatiles, semi-volatiles, and metals. These constituents were selected for their potential to result in risk from inhalation exposure. Workers, adults, and children were evaluated for three different types of exposures or risk endpoints: chronic (over 1 year), subchronic (1 month), and acute (1 day). Estimating protective concentrations required a multistep modeling process that could relate the concentrations in ambient air at a receptor point that could create a health effect to a concentration in the waste management unit. To achieve this, the analytical approach for this analysis is based on three primary components:

- # Emissions modeling—characterizing emissions from a WMU
- # Dispersion modeling—describing the transport of these emissions through the ambient environment

The Air Characteristic Study addresses:

- # 105 constituents
- # 4 WMU types
 - landfill
 - land application unit (LAU)
 - wastepile (WP)
 - tank
- # 5 receptors
 - adult resident, exposure starting age 19 years
 - child resident, exposure starting age 0-3 years
 - child resident, exposure starting age 4-10 years
 - child resident, exposure starting age 11-18 years
 - off-site worker
- # direct inhalation only
- # volatiles and particulates
- # 6 distances from the site
- # 3 risk endpoints or averaging times
 - chronic (over 1 year)
 - subchronic (1 month) - LAU, WP
 - acute (1 day) - LAU, WP

¹ 105 were addressed but one constituent, 3,4 dimethylphenol, did not have an inhalation benchmark.

- # Exposure modeling/risk estimation—estimating exposure to a receptor and then backcalculating to arrive at a waste concentration (C_w) that presents a risk equal to a prespecified risk level (e.g., 1 in 1 million, or 1E-6).

To illustrate the scenario that was modeled for this study, Figure 2-1 is a conceptual diagram of a waste site. Constituents managed in the WMU can be released as gases if they volatilize and as particulates if the constituent attaches to solid particles in the waste. Once the constituent is released from the site, the ambient air provides a medium for the transport of the airborne constituent. The direction the constituent travels and its concentration in the air are determined by meteorological conditions in the surrounding area such as wind direction, air temperature, and atmospheric stability at the time it is released. Because meteorological patterns are dynamic, the concentration of the constituents in the air varies over time and people who live and work at various locations around the WMU have different inhalation risks. The risk to an individual from the release of a constituent also depends upon characteristics of that individual such as body weight, inhalation rate, and the length of time that individual remains in the area around the WMU. These last characteristics are the reason that this assessment considers the exposure to multiple types of receptors: adult residents, child residents of various ages, and workers.

In order to model the scenario described above, the preliminary requirements for the analysis included:

- # Emissions models for the various WMUs to provide estimates of gas and particle releases from the unit
- # A dispersion model capable of modeling area sources for chronic (over 1 year), subchronic (1 month), and acute (1 day) releases

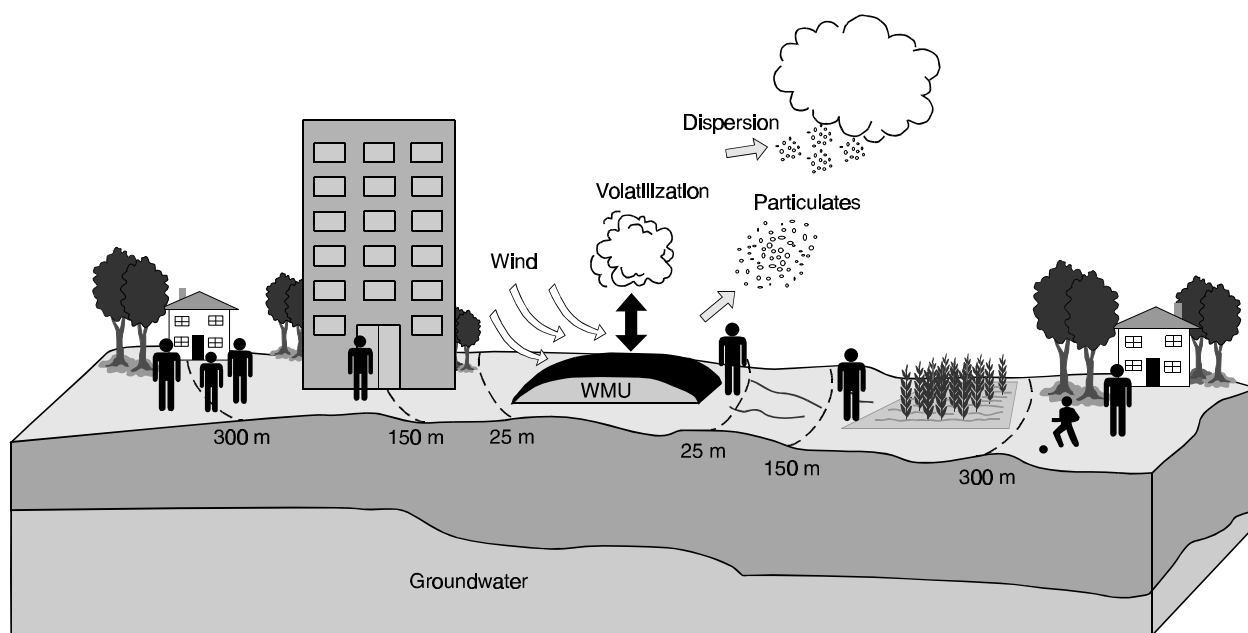


Figure 2-1. Conceptual diagram of a waste site.

- # An exposure model for locating receptors proximate to the WMUs and estimating their exposure
- # A risk model that combines the exposure characteristics of different types of receptors with constituent-specific toxicity benchmarks.
- # The ability to backcalculate C_w from a prespecified risk level (e.g., 1E-6).

For each constituent and each WMU type, EPA wanted to be able to specify a C_w that would not exceed a target risk level (e.g., 1 in 100,000, 1E-5) in more than a specified percentage (e.g., 10 percent) of the cases being modeled. Therefore, a probabilistic modeling approach, which would produce a distribution of C_w 's, was needed, as opposed to a deterministic approach, which would only produce a point estimate. A deterministic analysis produces a point estimate because it uses a single value for each parameter in the analysis. A probabilistic approach considers the variability in the inputs required to estimate the concentration nationally. This type of approach produces a distribution of results because the method iterates through the analysis more than once, allowing the input parameters in the analysis to take on different values for each iteration from a distribution of values. For this analysis, EPA used a Monte Carlo simulation. This is a type of probabilistic analysis that can be used when the distribution of some or all input variables is known or can be estimated. A large number of iterations of the calculations are performed (i.e., 1,000), with a value for each input variable selected at random from the variable's distribution and the result (in this case, C_w) calculated for each iteration. The results of each iteration are combined into a distribution of C_w . It was assumed that the modeled cases represent the national distribution of risk-specific concentrations.

The probabilistic approach described above was used to model chronic exposures. A deterministic approach designed to produce a more high-end point estimate was used to model acute and subchronic exposures. The acute/subchronic approach uses the maximum exposure point at any given distance, so no variability in receptor location is accounted for. It also uses the meteorological conditions that produce the maximum air concentration for a 24-hour or 30-day time period over 5 years of meteorological data. The results from the acute/subchronic analysis are comparable to the 100th percentile of the distribution generated for the chronic analysis. It should be noted that acute/subchronic exposures were only assessed for land application units and wastepiles, which may have episodic loading events. There are a variety of other differences in the acute/subchronic approach in how the emission rates and dispersion factors were calculated; these are described in more detail in Sections 4 and 5.

To estimate volatile emissions from each type of WMU, EPA's CHEMDAT8 model was used. For the landfill, LAU, and wastepile, the concentration of hazardous constituent in the surface layer of the soil (hereafter referred to as soil concentration) was estimated using a mass balance approach (i.e., competing pathways such as volatilization, adsorption, and biodegradation are accounted for). Particulate emissions due to wind erosion were modeled for land-based units (landfills, LAUs, and wastepiles). Landfills and LAUs were modeled as ground-level sources using the Cowherd model (U.S. EPA, 1985b, 1988). Wastepiles were modeled as elevated sources using the AP-42 model for wind erosion from aggregate storage piles (U.S. EPA, 1985a). To obtain the emission rate of constituent sorbed to particulate matter,

the emission rate of particulate matter was multiplied by the soil concentration calculated by CHEMDAT8. This was done to account for the portion of the original waste concentration that would remain in the waste after volatilization and biodegradation losses, and so would realistically be available for emission in the particulate phase.

The modeling assumes waste is continuously added to landfills and tanks, while LAUs and wastepiles have noncontinuous, episodic waste loadings. To capture potential peaks in emissions immediately after episodic loading events, acute and subchronic exposures were evaluated for LAUs and wastepiles.

Dispersion modeling was performed for each WMU using EPA's Industrial Source Complex Model Short-Term (ISCST3) to develop unitized air concentrations (UACs) for vapors and particulates. UACs are dispersion coefficients based on a unit emission (i.e., $1 \mu\text{g}/\text{m}^2\cdot\text{s}$) for use in a backcalculation. UACs varied depending on the averaging time (i.e., chronic, subchronic, or acute), the size of the WMU, the distance and direction of the receptor from the WMU, and the associated meteorological station. Dispersion modeling for vapors did not account for depletion, as sensitivity analysis showed that depletion of vapors has a negligible impact on air concentration of vapors. Dispersion modeling for particulates accounted for dry depletion of particles, since a sensitivity analysis showed that dry depletion has a potentially significant impact on air concentrations of particulates. Wet depletion of particulates was not accounted for in the dispersion modeling, as sensitivity analysis showed that wet depletion has little impact on air concentration.

The air concentration at any specific receptor is the product of the emission rate (in $\mu\text{g}/\text{m}^2\cdot\text{s}$) and appropriate UAC (in $[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\cdot\text{s}]$). Air concentrations were estimated for chronic, subchronic, and acute exposures (using averaging times of 1 year, 1 month, or 1 day), based on a combination of volatile and particulate emissions.

Many previous risk analyses have used the maximum point of exposure at some prespecified distance from the WMU as the point for analysis. Such an approach is usually criticized as being overly conservative because it does not consider the possibility of no one living at that exact point. Because individuals may potentially be located in any direction and at various distances from a facility, this analysis developed an explicit way to incorporate this consideration. First, a sensitivity analysis was conducted to determine a reasonable distance at which to bound the analysis. This sensitivity analysis showed that, beyond 1,000 m, most air concentrations are a small percentage (less than 10 percent) of the concentration at the point of maximum exposure. Therefore, 1,000 m was used as the outer bound on the distance of receptors included in this analysis. A receptor grid was set up to allow individuals to reside in any of 16 directions and at distances of 25, 50, 75, 150, 500, and 1,000 m from the edge of the unit.

For this analysis, five receptors were included: an adult resident, a child resident with exposure starting between 0 and 3 years old, a child resident with exposure starting between 4 and 10 years old, a child resident with exposure starting between 11 and 18 years old, and an off-site worker. These receptors could be located in any of 16 directions and at distances of 25, 50, 75, 150, 500, and 1,000 m from the edge of the unit. Each distance was evaluated separately and the location of a receptor was allowed to vary among any of the 16 directions. The 16 directions

were equally weighted, so there is equal probability of a receptor being located anywhere around the WMU. The 16 directions were equally weighted, so there is equal probability of a receptor being located anywhere around the WMU. For acute and subchronic exposures, receptors were modeled at 25, 50, and 75 m because it was assumed that the greatest possibility of acute exposure would be closest to the site.

2.2 Conducting the Analysis

As discussed earlier, the analysis consists of three main parts: emissions modeling, dispersion modeling, and exposure modeling/risk estimation. Figure 2-2 shows the model framework. Emissions and dispersion modeling was performed first and the results used as inputs to the exposure modeling/risk estimation. In addition, a database containing characterizations of WMUs was used.

The goal of the analysis is to backcalculate a waste concentration that will result in a specified risk. Because risk is assumed to be linear with waste concentration under most circumstances, a waste concentration was generated by forward-calculating a risk associated with a unit concentration in the waste (i.e., 1 mg/kg for land-based units and 1 mg/L for tanks), then scaling the unit concentration using the ratio of target risk to calculated risk. The assumption of linearity is accurate for the dispersion modeling and the exposure and risk modeling. The emissions model is linear for land-based units and tanks without biodegradation. The emissions model for tanks with biodegradation is nonlinear at the concentration where biodegradation shifts from first order to zero order. The results for tanks with biodegradation were backcalculated using first order emission rates; however, if this result exceeded the concentration at which biodegradation becomes zero order, the result was adjusted to be based on zero order emission rates. Even when the emissions model is linear, it is possible, using this approach, to backcalculate waste concentrations that exceed the solubility or soil saturation concentration for the chemical. Results that exceed the solubility or soil saturation concentration under neutral conditions are footnoted in the result tables. Soil saturation concentration and solubility can vary according to site-specific temperature and pH conditions.

Emissions modeling was performed for all WMUs and all chemicals, assuming a unit concentration of the chemical in the waste (1 mg/kg for land-based units or 1 mg/L for tanks). These emissions were used as inputs to Step 2 of the exposure modeling/risk estimation portion of the model.

Dispersion modeling was performed for 76 representative WMU areas and height combinations and 29 meteorological locations, assuming a unit emission rate of $1 \mu\text{g}/\text{m}^2\text{-s}$. This produced vapor and particle-phase UACs for each area/height combination, meteorological station, and receptor location, which were used as the basis from which to interpolate in Step 4 of the exposure modeling/risk estimation portion of the model.

The analytical framework shown in Figure 2-2 consists of a series of steps and loops. In Step 1, a chemical and WMU type (e.g., landfills) were selected (thus, all landfills were analyzed as a group for each chemical, and so on).

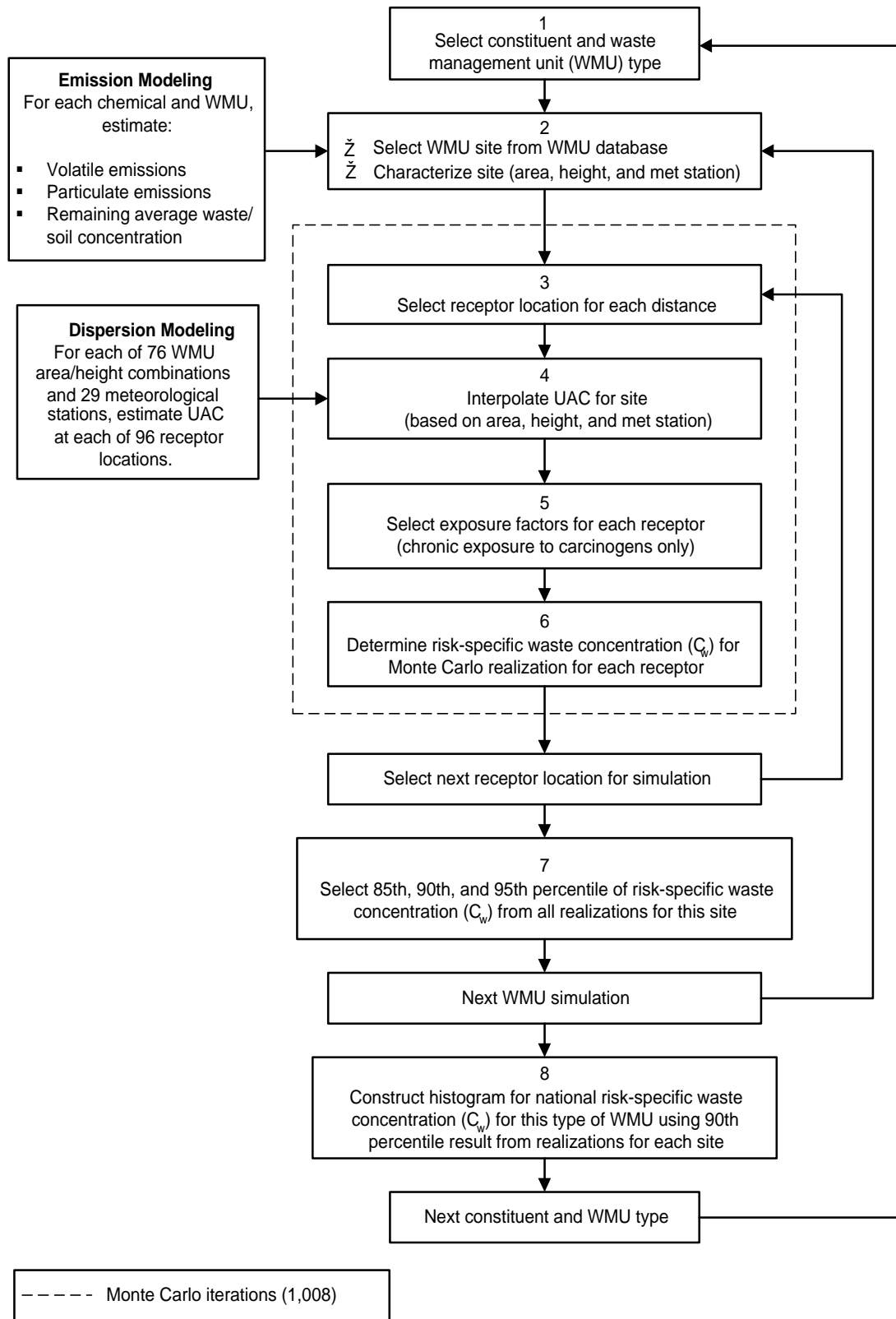


Figure 2-2. Model framework.

In Step 2, a WMU was selected from the data file for that unit type. For example, for landfills, the database has a data record containing the facility identification and WMU characteristics such as surface area, depth, and waste quantity managed per year for each of 801 landfill units. The database also has a sampling weight for each facility that defines how many facilities nationally were represented by that facility. An assigned meteorological station was added to the database based on locational information for each WMU. The model simulation starts with the first record and moves to each successive record. For each WMU record, the associated emission rate for that WMU and chemical was obtained from the emission modeling results.

In Step 3, receptor locations were selected by choosing at random one of the 16 directions modeled in the dispersion modeling. Receptors were modeled in that direction at each of six distances from the site.

In Step 4, a UAC was interpolated for the WMU. Due to the long run time of ISCST3 for area sources, UACs were modeled for only 76 selected WMU area/height combinations for each meteorological station and receptor location. To calculate a UAC corresponding to the WMU's actual area and height, EPA first chose the modeled height closest to the actual unit height, then interpolated between the UACs for the two closest of the areas modeled. For example, the first three areas modeled for wastepiles were 20, 162, and 486 m². These were modeled at heights of 1, 2, 4, 6, and 8 m. For a WMU with an actual area of 100 m² and an actual height of 3.5 m, the UAC was interpolated from the UACs for 20 m²/4 m high and 162 m²/4 m high. For a WMU with an actual area of 200 m² and an actual height of 6.9 m, the UAC was interpolated from the UACs for 162 m²/6 m high and 486 m²/6 m high.

In Step 5, for chronic exposures to carcinogens, values of exposure factors such as body weight, inhalation rate, and exposure duration were chosen at random from distributions of these parameters (developed from data in the *Exposure Factors Handbook*, U.S. EPA, 1997c and 1997d) to capture the variability in exposure factors for a given receptor. These exposure factors differ for different receptor types (such as adults, children, and workers). Noncarcinogens were not assessed in this manner because the health benchmarks, such as EPA's reference concentration (RfC), are expressed in terms of ambient concentration and cannot be adjusted for variations in these exposure factors. Similarly, acute and subchronic health benchmarks are expressed as ambient exposure concentrations and cannot be adjusted for variability in exposure factors.

In Step 6, the emission rate, UACs, and, if applicable, the exposure factors, were combined with the health benchmark for the chemical to estimate risk (for chronic exposure to carcinogens) or hazard quotient (for acute and subchronic exposures, and chronic exposures to noncarcinogens) associated with the unit concentration modeled. This risk was then compared to the target risk of either 1 in 1 million, 1 in 100,000, or 1 in 10,000 (i.e., 1E-6, 1E-5, or 1E-4) for carcinogens, and the ratio was used to scale the unit concentration to a concentration in the waste (C_w) that would result in the target risk at that receptor. A similar technique was used for scaling the hazard quotient for noncarcinogens.

Steps 3 through 6, which form the core of the Monte Carlo simulation, were then repeated 1,008 times for each WMU, resulting in a distribution of C_w for that WMU for each receptor (adult, child, or worker) at each distance from the site (25, 50, 75, 150, 500, and 1,000 m) for a specific risk criteria (i.e., 1E-4, 1E-5, or 1E-6 for carcinogens and 10, 1, or 0.25 for noncarcinogens). Once 1,008 iterations had been performed for a WMU, various percentiles were selected from the distribution to characterize it. These percentiles represent the percentage of receptors protected at the WMU.

Steps 2 through 6 were then repeated to obtain distributions of C_w for each WMU in the database. These distributions are somewhat different for carcinogens and noncarcinogens and for chronic, subchronic, and acute exposures. For chronic exposure to carcinogens, they represent both the potential variability in location around a WMU, as well as the variability in exposure duration, inhalation rate, and body weight for each receptor type. For noncarcinogens and for subchronic and acute exposures, variability in these exposure factors is not considered because the measure of risk is a ratio of air concentrations. For chronic exposures to noncarcinogens, the distributions represent the variability in location around the WMU at a specific distance. For subchronic and acute exposures, only point estimates were made at various distances using the receptor located at the point of maximum air concentration for that distance.

The cumulative distribution of C_w for each WMU is presented as the percentage of receptors that are at or below the risk criteria for any C_w (see Figure 2-3, left side). For example, 90 percent of all adult residents at a distance of 150 meters have a predicted risk at or below 1 in 100,000 (1E-5) if the concentration of the chemical (e.g., cumene) in the landfill is 1 mg/kg (see point a). A second landfill may have a 90 percent protection level for all adult residents at 150 meters at a concentration of 10 mg/kg (point b), and a third landfill at a concentration of 100 mg/kg (point c). Thus, in Step 7, for each WMU, the distribution shows the percent of potential receptors at or below a specified risk level for each concentration of constituent in the WMU (C_w) for each distance and each receptor.

In Step 8, once all WMUs of a certain type had been modeled, the distributions of C_w for all individual WMUs of the same type (e.g., landfills) were combined to produce a cumulative distribution that presents the variability in C_w across all units of a certain type. For a given percentage of protected receptors (e.g., 90 percent) as described above, the C_w was combined across all WMUs of a specified type (e.g., landfills) to provide a distribution of the percentage of sites considered protective at that level, as shown in Figure 2-3 (right side). Figure 2-3, for example, shows the cumulative distribution of C_w at a 90 percent protection level across all landfills. From this distribution, the 90th percentile C_w value for all 90 percent protection levels across all landfills could be estimated. As described above, three landfills that give a 90 percent protection level (i.e., at 1E-5) for a resident at 150 meters from the unit boundary have corresponding C_w values of 1 mg/kg, 10 mg/kg, and 100 mg/kg (see points labeled a, b, and c).

These values plus similar values from all other landfills constitute the cumulative distribution. The C_w value that is protective of 90 percent of receptors across 90 percent of the sites is referred to in this study as the 90/90 protection level. These distributions were developed for each unit type, each receptor type, each risk criteria, and each distance from the WMU.

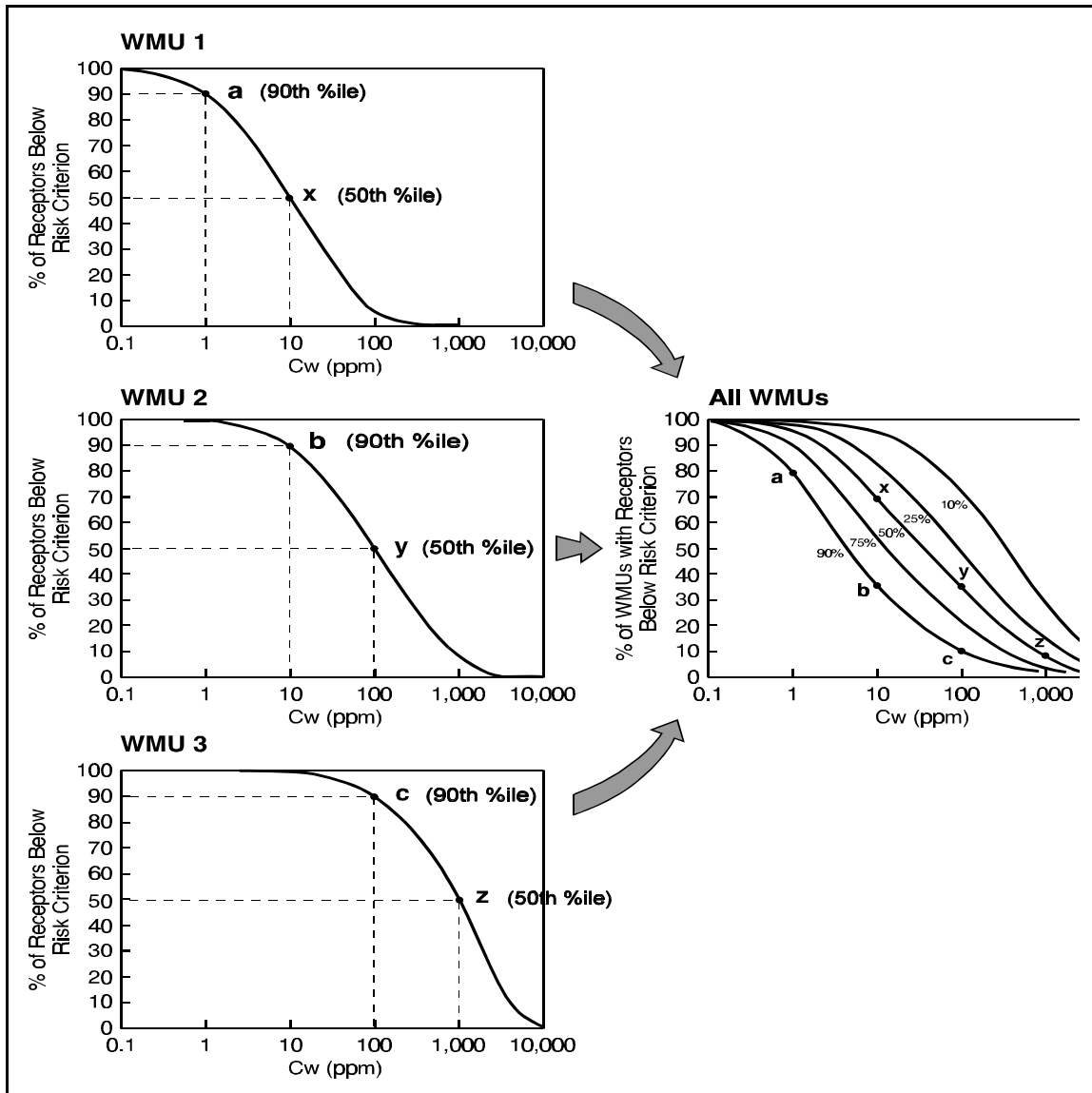


Figure 2-3. Combination of results for individual WMUs into a distribution across all WMUs.

These cumulative distributions are intended to encompass the variability across WMUs. Thus, the variability in WMU characteristics and in meteorological settings is included in these distributions.

This process was repeated from Step 1 for each chemical and WMU type analyzed in this study.

2.3 Data Sources

The Industrial D Survey database (Shroeder et al., 1987) was the primary source of data on WMUs used in this analysis. This database provides information on each of the WMUs assessed, with the exception of tanks. Tank data are from EPA's National Survey of Hazardous Waste Treatment, Storage, Disposal and Recycling Facilities (TSDR Survey, U.S. EPA, 1987). The Industrial D Survey database contains information on the size and capacity of a statistical sample of each WMU type, general location information, and statistical weights for each facility in the sample. The statistical sample was designed to represent all industrial waste management units not regulated under the RCRA hazardous waste program at the time the survey was conducted in 1987. The weights in the database indicate the number of facilities represented by each facility in the sample. For this assessment, it is assumed that the data contained in this database provide an appropriate representation of the characteristics of each WMU type and of the general location of these types of facilities with respect to climate regions of the country.

Meteorological stations provided temperature and windspeed data as inputs to the emissions model and a large set of inputs for the dispersion model. Although meteorological data are available at over 200 meteorological stations in the United States (see, for example, *Support Center for Regulatory Air Models (SCRAM) Bulletin Board* at <http://www.epa.gov/scram001>), various resource constraints prevented the use of all available data sets in this analysis. Therefore, a set of 29 stations was used that had been selected as representative of the nine general climate regions in the contiguous United States in an assessment for EPA's Superfund Soil Screening Level (SSL) program (EQM, 1993).

In EPA's Superfund study, it was determined that 29 meteorological stations would be a sufficient sample to represent the population of 200 meteorological stations and predict mean dispersion values with a high (95 percent) degree of confidence. The 29 meteorological stations were distributed among the nine climate regions based on meteorological representativeness and variability across each region. Large-scale regional average conditions were used to select the actual stations.

The 29 meteorological stations are listed in Section 5. To assign each Industrial D or TSDR facility to a meteorological station, EPA used a geographic information system (GIS) to construct areas around each station that encompass the areas closest to each station. The boundaries of these areas were then adjusted to ensure that each boundary encloses an area that is most similar in meteorological conditions to those measured at the meteorological station. First, the boundaries were adjusted to correspond to Bailey's ecological divisions (Bailey et al., 1994), which are defined primarily on physiography and climate. The boundaries were further adjusted for coastal (including Great Lakes) areas and the central valley of California to ensure that these stations were used only in regions with similar meteorology. Based on zip codes in the Industrial D Survey database and EPA IDs in the TSDR database, the sites were then overlaid on this GIS coverage, and meteorological station assignments were then exported for use in the modeling exercise. Several sites in Alaska, Hawaii, and Puerto Rico were deleted from the analysis at this point because the 29 meteorological stations are limited to the continental United States.

3.0 Waste Source Characteristics

Waste sources modeled in the air characteristic risk analysis are landfills, land application units, wastepiles, aerated treatment tanks, nonaerated treatment tanks, and storage tanks. The dimensions and operating characteristics of these waste management units (WMUs) are important determinants of the modeled emission rates and dispersion factors used to estimate inhalation risks to receptors. This section describes how data specific to these unit types were collected and processed for use as emission and air model inputs. Source parameters that are critical to emissions but not to dispersion are discussed in Section 4.0, *Source Emission Estimates*.

The primary source of data used to characterize waste sources is the Industrial D Screening Survey (Schroeder et al., 1987). These survey data provide information (for all WMUs except tanks) on the number of each type of WMU, annual quantity of waste managed in the WMU (in 1985), and some information on the dimensions of the WMU (area and capacity). Along with waste concentration, these parameters are the primary waste source characteristics that impact the emission model estimates. The dispersion model also requires WMU area for area sources (landfills, wastepiles, and land application units) as well as the height of the source for wastepiles and tanks. The primary source of data for tanks is EPA's National Survey of Hazardous Waste Treatment, Storage, Disposal and Recycling Facilities (TSDR survey, U.S. EPA, 1987).

Facility locations also were required so that facilities could be assigned to the 29 meteorological stations used to provide meteorological data for the air model. These were derived by matching Industrial D and TSDR facilities, using the Dun and Bradstreet DUNS[®] number, name, street address, and zip code, and EPA ID to EPA's Envirofacts system to obtain the "best" location for each facility as defined by EPA's Locational Data Improvement Project (LDIP) and represented by data in the Envirofacts Locational Reference Table (LRTs) (http://www.epa.gov/enviro/html/lrt/lrt_over.html). For sites that could not be matched to the LRT tables, the latitude/longitude of the facility's zip code centroid was determined and used as the facility location.

3.1 Estimation of Missing or Replacement Data for WPs, LAUs, and LFs

The Industrial D Screening Survey provided the data needed to characterize land-based WMUs except for wastepile height and landfill depth, which had to be estimated. In developing these characterizations, the data set was reviewed to ensure that facilities reporting insufficient information or outlier data were not included. In some cases, replacement values were required for waste quantity and landfill capacity due to screened unrealistic values or missing data. Units were culled for any of the following reasons:

- # Surface area was not reported
- # Address information was not provided
- # Sites were located outside the contiguous United States
- # Reported data were identified as outliers
- # Site was identified as a Bevill facility as discussed in Section 3.3.

Table 3-1 shows the total number of facilities reporting each WMU in response to the survey, those culled from the data set, and those remaining in the database whose data were used in developing the source characterizations.

3.1.1 Wastepile Height

Reported data on wastepile height were not available in the Industrial D database. In general, the height of a wastepile is a function of the area of the unit and the annual waste quantity managed in the unit. In the absence of reported data, the relationships between pile height and the annual waste quantity per unit area shown in Table 3-2 were developed. The height of a wastepile depends on the annual waste quantity, the area of the pile, the waste density, and the retention time. Given a waste density and retention time, a pile with a greater ratio of waste quantity to area must be higher. A low height of 1 m was set, as differences in height below 1 m make little difference to the dispersion modeling. The high end height of 10 m was chosen as a reasonable maximum, given that waste would have to be added to the top of a pile, so equipment adding waste would have to be able to reach the highest pile height. A height of 10 m is about the equivalent of a 3-story building. A discrete set of heights was selected to simplify the meteorological component of the data required for conducting dispersion modeling. (Specifically, a calculation is required to adjust the windspeed from the height of the measuring device to the height of the wastepile.) The annual waste quantity and area were known from the Industrial D survey data for the wastepiles modeled. The ratio of waste quantity to area ranged from 0.08 to 1,900 Mg/yr-m², with 77 percent of piles falling below 10 Mg/yr-m² and less than 3 percent falling above 80 Mg/yr-m². This information was combined with the range of pile height selected to establish the relationship shown in Table 3-2. Given a reasonable range of waste bulk density from 1 to 1.5 g/cm³, these heights imply retention times on the order of months to a year.

Using this approach, wastepile height assignments were made for each wastepile in the Industrial D data set. Approximately 77 percent of the total number of wastepiles included in the air characteristics study data set were assigned a wastepile height of 1 meter, and approximately 95 percent of the wastepiles were assigned heights of 4 meters or less.

3.1.2 Landfill Depth and Capacity

Landfill depth was calculated consistent with previous EPA modeling efforts using the Industrial D data (U.S. EPA, 1996a). The following equation was used, assuming the same waste bulk densities (fixed by unit type) used in the previous modeling efforts:

$$\text{depth} = \text{total waste capacity} / (\text{surface area} \times \text{bulk density}) .$$

Table 3-1. Summary of Land-Based WMUs Removed from Industrial D Survey Database

Description	Wastepiles	Land Application Units	Landfills
Number of Sites Reporting WMU Type in Industrial D Screening Survey	853	354	827
Culled Sites: Surface area was not reported	6	2	3
Culled Sites: Address information was not provided	15	5	20
Culled Sites: Outside contiguous United States	3	1	2
Culled Sites: Surface area identified as outlier	0	1 (area = 40,000 acres)	1 (area =120,000 acres)
Culled Sites: Bevill facilities	84	NA	NA
Total Number of Sites included in Air Characteristics Study Data Set	745	345	801

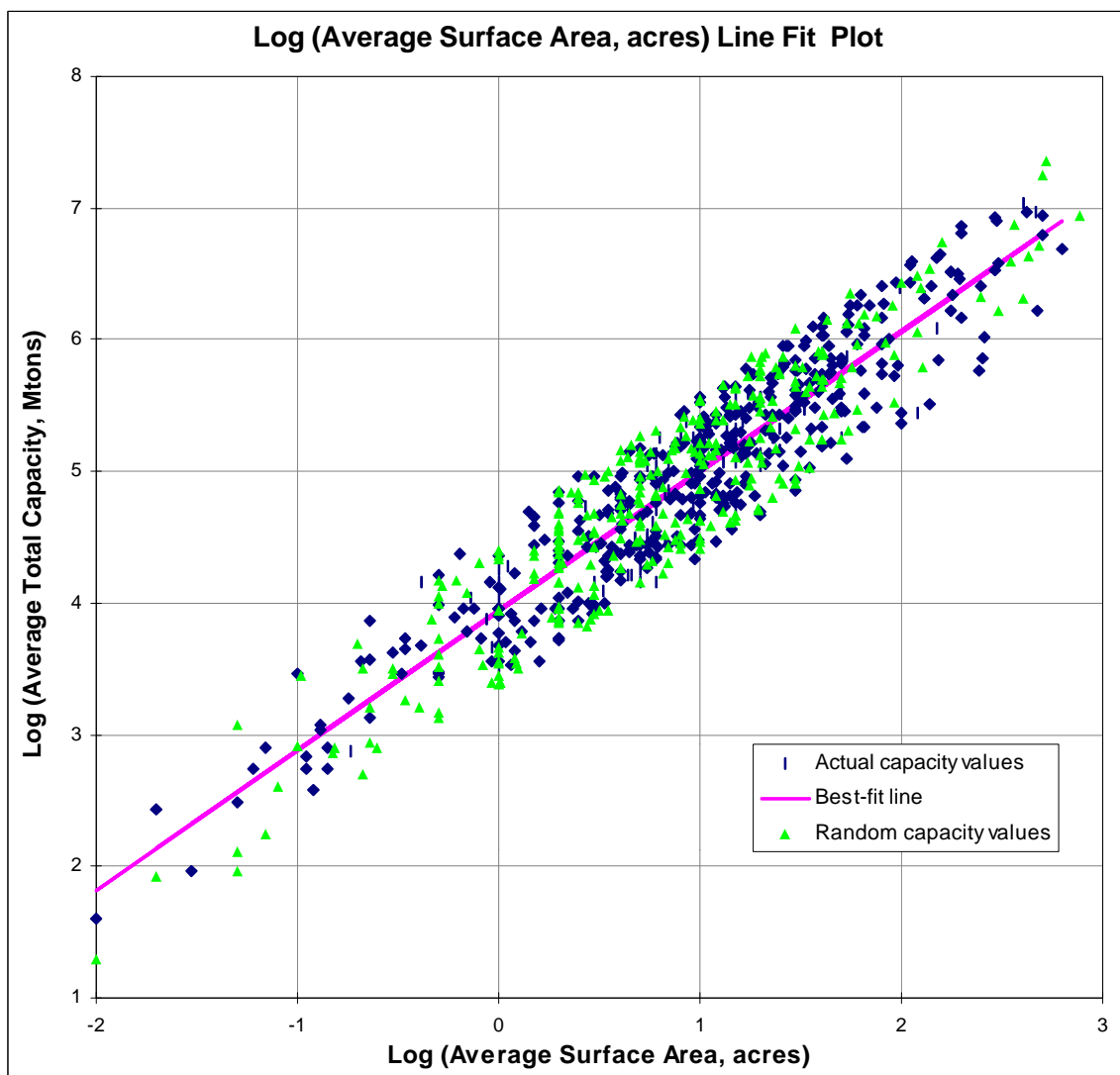
Table 3-2. Assignment of Wastepile Height

Annual Waste Quantity/Surface Area (Mg/m ² /y)	Pile Height (m)
<10	1
≥10, <20	2
≥20, <40	4
≥40, <60	6
≥60, <80	8
≥80	10

The bulk density used for landfills in previous efforts was 1.1 g/cm³; this was also used in this study for consistency.

Also in accordance with previous EPA modeling efforts using the Industrial D Screening Survey (U.S. EPA, 1996a), landfill capacities were removed from the Industrial D data when depth or capacity constraints were violated. These constraints were imposed to eliminate unrealistic values for depth and capacity. The constraints were: landfill depth had to be greater than or equal to 2 feet and less than or equal to 33 feet, and, within the Industrial D database, landfill total capacity had to be greater than the remaining capacity (U.S. EPA, 1996a, p. 3-6). Of the 801 landfills modeled, 103 had a depth less than 2 feet, 87 had a depth greater than 33 feet, and 21 had remaining capacity that exceeded the total capacity. In addition, 92 facilities were missing data on total capacity, remaining capacity, or both. Thus landfill capacity was missing or screened for 303 landfills.

Landfill capacities to replace the 303 missing or removed values were estimated based on the correlation between surface area and capacity of the waste management unit in the Industrial D data. First, a statistical regression of log (average total capacity) versus log (average surface area) was done on the facilities with known capacities. The regression yielded an equation for a best fit line through the known values. This equation gave the capacity as a function of area, so the missing or screened capacities could be estimated based on the known areas. To provide a more probabilistic sampling of average capacities, and since the known capacities seemed to be in a limited range above and below the best fit line, a positive or negative random number was generated within that range and added to the calculated log (average total capacity) to replace each missing capacity with a random value that was reasonable with respect to landfill area. This value was then used to calculate landfill depth as described above. Figure 3-1 shows the regression plots, including the replaced (“random capacity”) values, for landfills.



Landfill Regression Plot

Figure 3-1. Landfill characteristics from Industrial D Screening Survey.

3.1.3 Waste Quantity Replacement Values

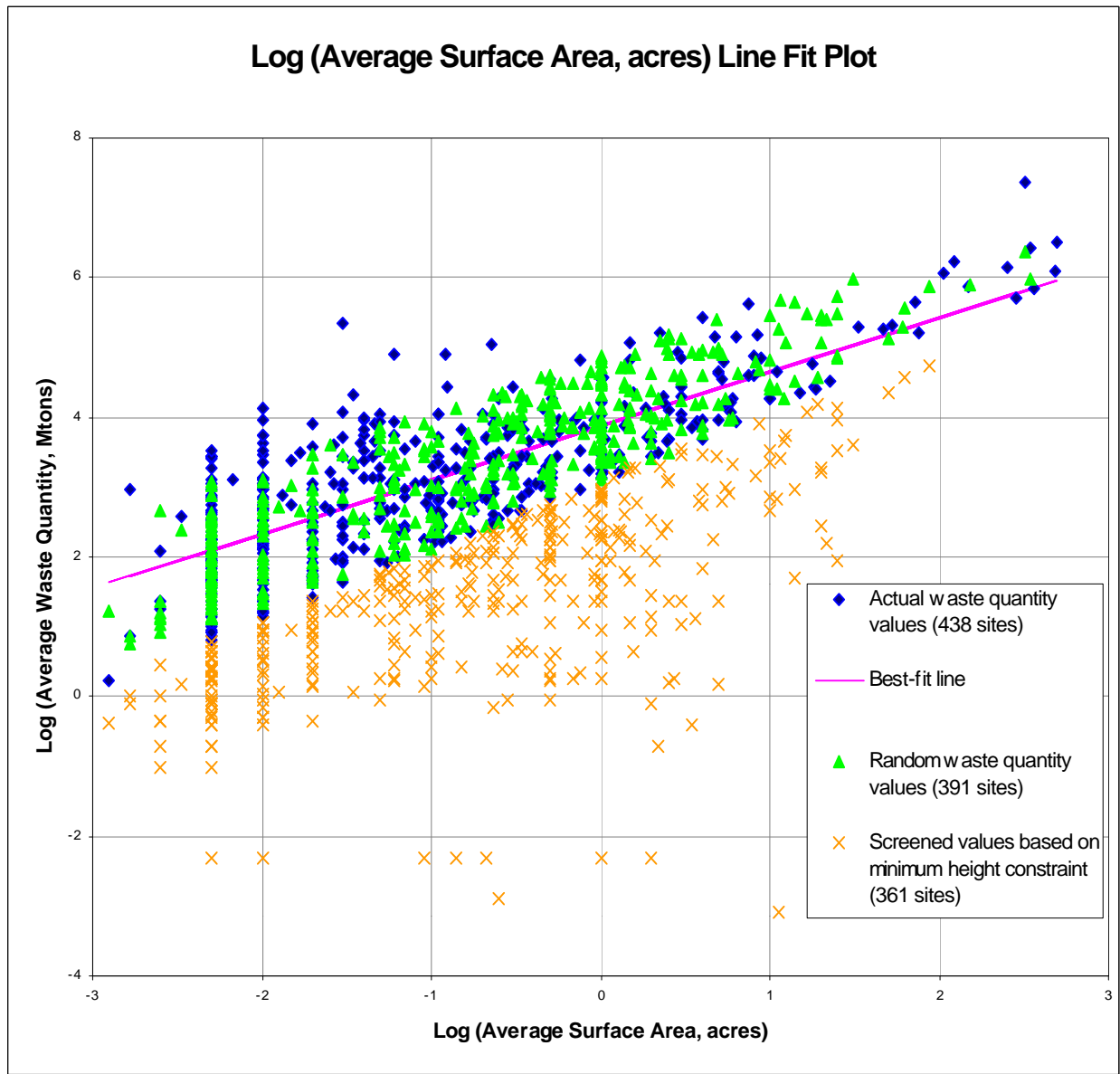
Replacement values were calculated for waste quantity due to screened unrealistic values or missing data. Waste quantity replacement values were calculated under three conditions: (1) if surface area data were reported for a given unit but the waste quantity was not provided, (2) if the minimum wastepile height constraint of 1 meter was violated, and (3) if the application rate for land application units exceeded 10 Mg/m²-yr. The minimum 1-meter wastepile height constraint was imposed to avoid a situation in which the wastepile replacement frequency is less than once every 5 years. The maximum application rate for LAUs was established as a cut-off in previous EPA modeling efforts using the Industrial D data (U. S. EPA, 1996a) and was retained in this study for consistency. The first condition applies to all unit types, while the second and third apply only to wastepiles and land application units, respectively.

Waste quantity replacement values were calculated for 391 wastepiles and 20 LAUs, based on the three conditions described above. Thirty wastepiles and 8 LAUs reported no waste quantity, 361 wastepiles violated the minimum height constraint, and 12 LAUs violated the maximum application rate constraint.

Similar to the approach applied in estimating landfill capacity, replacement values were developed for the screened and missing waste quantities. These values were estimated based on the correlation between surface area and waste quantity of the waste management units in the Industrial D data. A statistical regression of log (waste quantity) versus log (average surface area) was done on the facilities with known quantities. The regression yielded an equation for a best fit line through the known values. This equation gave the waste quantity as a function of area, so the missing or screened capacities could be estimated based on the known areas. To provide a more probabilistic sampling of average waste quantities, and since the known quantities seemed to be in a limited range above and below the best fit line (with some outliers), a positive or negative random number was generated within that range and added to the calculated log (average waste quantity) to replace each missing waste quantity with a random value that was reasonable with respect to wastepile area. Figures 3-2 and 3-3 show the regression plots, including the replaced (“random waste quantity”) values for wastepiles and land application units, respectively.

3.2 Waste Management Unit Data and Size Categories Used in Dispersion Model

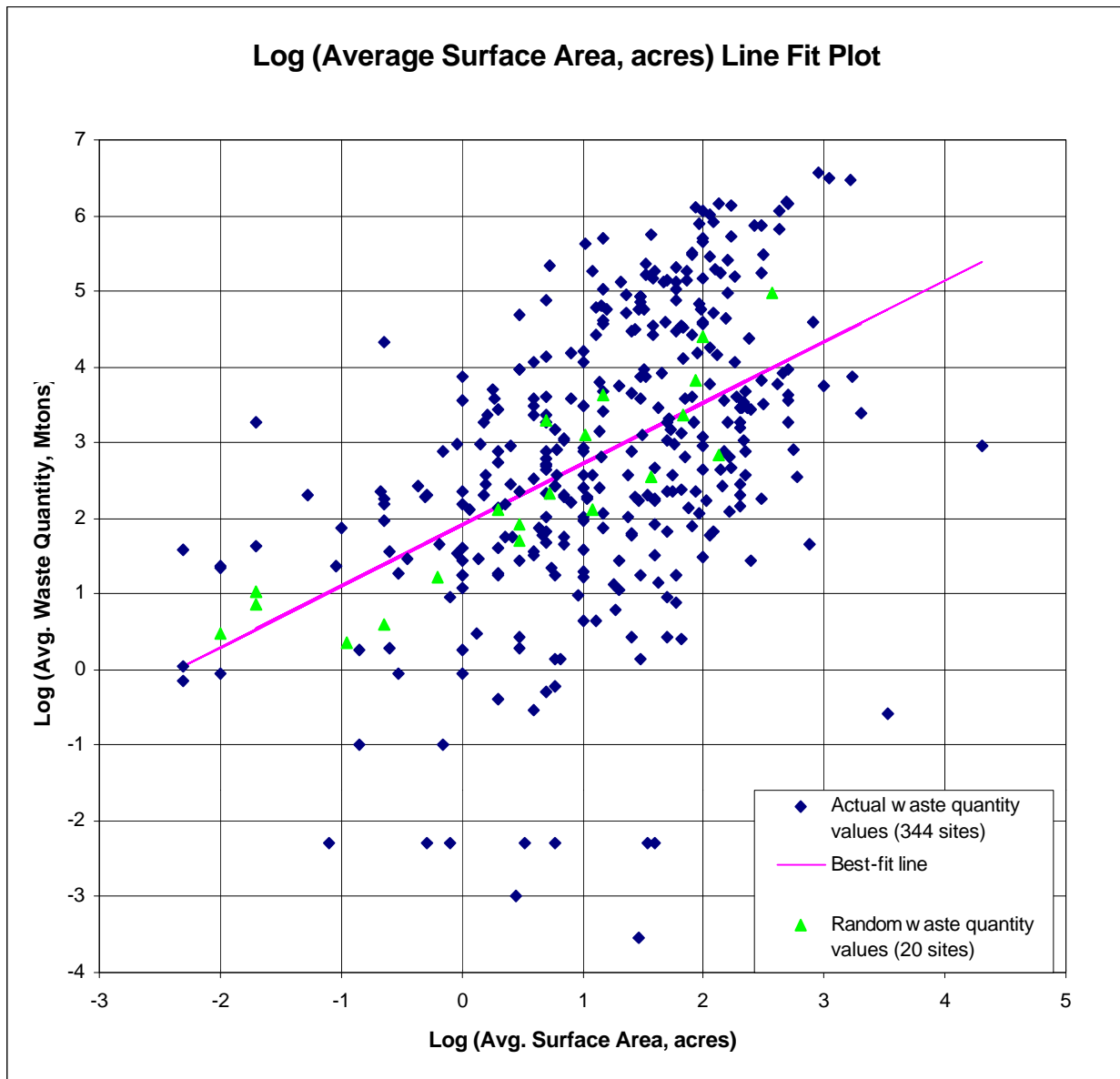
The area of a WMU is a very sensitive parameter in the dispersion model. However, because of run-time limitations associated with the air dispersion model, it was necessary to develop area strata for the land-based WMU types. Dispersion modeling was conducted for the median area of each of these strata and the resulting unitized air concentrations (UACs) were used as inputs to an interpolation routine used to determine UACs for each WMU (see Section 5).



Waste Pile Regression Plot

Figure 3-2. Wastepile characteristics from Industrial D Screening Survey.

US EPA ARCHIVE DOCUMENT



Land Application Unit Plot

Figure 3-3. Land application unit characteristics from Industrial D Screening Survey.

US EPA ARCHIVE DOCUMENT

Initially, a regular distribution of strata cutpoints (5th, 25th, 50th, 75th, and 95th percentiles) was considered to provide the representative WMU surface areas. However, examination of the distribution of areas for each WMU type showed that the distributions were all extremely skewed to the right, with a large number of facilities at the smaller end of the distribution and a few facilities with very large areas. This raised the concern that the regular cutpoints would not adequately cover the tail of the distribution (i.e., WMUs with large areas) for the interpolation process.

To improve the coverage of the cutpoints, a statistical methodology called the Dalenius-Hodges procedure was used as a starting point. This technique is designed to break the distribution of the known variable X (in this case, facility area), which is assumed to be highly correlated with the characteristic Y (in this case, the UACs), into L strata in an optimal way. From the L strata, an equal number of observations, n, are picked at random (assuming each observation has an equal weight). The process was actually employed in this case using the facility weights in the Industrial D data. To develop the initial WMU strata, L was set at 5; that is, 5 "optimal" strata were initially formed using this approach. This process produces a set of percentiles with the following property: if the distribution is skewed to the right, there will be more emphasis on characterizing the right tail. Appendix A describes the steps of the Dalenius-Hodges procedure in detail.

Based on ISCST3 model run-time estimates, the target number of strata for all WMU types was set at 20. Because land application units and landfills were modeled in the same fashion using ISCST3, it was possible to combine the areas for these WMU types to cover the distribution with as many strata as possible. Fifteen strata were initially formed for this distribution using the Dalenius-Hodges procedure. Wastepiles could not be combined with the other waste management units because they are modeled in ISCST3 as elevated area sources, not as ground-level sources like the others. Five Dalenius-Hodges strata were initially developed for this unit type.

In each case, the Dalenius-Hodges procedure yielded a good characterization of the right tail of the distribution but a poor characterization of the lower portion of the distribution. Because the UACs are most sensitive to area in this region of the distribution, the procedure was modified by collapsing two or more of the highest strata into one and successively splitting the lowest stratum into four strata. The splitting was performed by choosing stratum breakpoints as described in Appendix A.

For the combined landfill and land application distribution, the four highest strata (12, 13, 14, and 15) were collapsed into one and the facility nearest the median of the combined stratum was used to represent that stratum. The next two highest strata (10 and 11) were also collapsed into one and, again, the facility nearest the median of the combined stratum was used to represent that stratum. The first stratum (1) was then split, similar to the wastepiles, into four strata (1A, 1B, 1C, and 1D). The result was a total of 14 strata for the combined waste management units and 21 strata overall. Table 3-3 shows the final area strata for landfills and land application units, the medians used for the air dispersion modeling runs and the cumulative percentile of the distribution each stratum represents. Air dispersion modeling was conducted using the median surface area for each stratum.

Table 3-3. Area Strata Modeled for Landfills and Land Application Units

Strata	Average area (m ²)			Cumulative percentile (Ind D data)
	Low	Median	High	
1A	14	81	293	17
1B	310	567	789	24
1C	809	1,551	2,293	39
1D	2,307	4,047	7,487	54
2	7,588	12,546	26,980	74
3	27,115	40,500	59,653	81
4	60,300	78,957	119,000	87
5	120,763	161,880	210,000	92
6	210,444	243,000	295,000	94
7	303,525	376,776	546,345	96
8	554,439	607,000	728,460	97
9	753,754	906,528	999,609	98
10 & 11	1,007,703	1,408,356	2,430,000	99
12 to 15	2,521,281	8,090,000	13,500,000	100

For wastepiles, the Dalenius-Hodges stratification resulted in most (90 percent) of the Industrial D facilities falling into the lowest stratum where more interpolation points are necessary to accurately estimate the UACs. To correct this, the two highest strata (4 and 5) were collapsed into one. The first stratum (1) was then split into two (1a and 1b). This still resulted in a large number of facilities in the lowest strata, so it was split again into two strata (1a' and 1a''). The new lowest strata (1a') was further split into two strata (1a'A and 1a'B). The result was a total of seven strata for wastepiles.

All wastepiles in the database were assigned one of the six heights as discussed in Section 3.2. Using wastepile surface area data from the Industrial D data set, it was possible to identify which area strata were associated with each assigned pile height. Review of height/area strata data revealed that not all seven strata were associated with all heights. Based on these data, ISCST3 modeling was conducted for the 29 area/height combinations shown in Table 3-4. These combinations represent the height/strata combinations identified based on the Industrial D data set and a few additional combinations identified to facilitate interpolation between UACs (see Section 5).

Tanks were modeled in the same manner as wastepiles, using the area/height combinations shown in Table 3-5. The heights that were modeled represent the range of heights (1 to 6 meters) imputed from depth as described in Section 3.4.2. The 10 areas modeled were

Table 3-4. Areas and Source Heights Modeled for Wastepiles

Assigned Height (m)	Surface Area (m ²)						
	20	162	486	2,100	10,100	101,000	1,300,000
1	x	x	x	x	x	x	x
2	x	x	x	x	x	x	x
4	x	x	x	x	x		
6	x	x	x	x			
8	x	x					
10	x	x	x	x			

x = Combination modeled.

Table 3-5. Areas and Source Heights Modeled for Tanks

Assigned Height (m)	Surface Area (m ²)									
	2	3	5	10	25	50	100	400	1,500	5,000
1	x	x	x	x	x	x	x	x	x	x
2	x	x	x	x	x	x	x	x	x	x
4		x	x	x	x	x	x	x	x	x
6							x	x	x	x

x = Combination modeled.

selected based on a sensitivity analysis conducted with ISCST3. These areas were identified to minimize error (not to exceed 10 percent) when interpolating between UACs.

3.3 Removal of Wastepiles Containing Bevill-Excluded Wastes

RCRA excludes from hazardous waste regulation certain wastes related to mining and mineral processing. These wastes are commonly known as Bevill wastes. Enacted on October 21, 1980, Public Law 96-482 amended RCRA Section 8002 to include subsection (p), which requires the Administrator to study the adverse effects on human health and the environment, if any, of the disposal and utilization of solid waste from the extraction, beneficiation, and processing of ores and minerals, including phosphate rock and overburden from the mining of uranium ore. Section 7 of these amendments (the Bevill Amendment) amended Section 3001 of RCRA to exclude these wastes from regulation under Subtitle C of RCRA pending completion of the studies called for in Sections 8002(f) and (p). On November 19, 1980, EPA published an interim final amendment to its hazardous waste regulations to reflect the mining waste exclusion (40 CFR 261.4(b)(7)). Since that time, 261.4(b)(7) has been amended to clarify which solid wastes fall under the exclusion as wastes from processing of ores and minerals.

Because an air characteristic would not apply to WMUs containing Bevill Amendment wastes, an analysis of the presence of such facilities in the Industrial D data was conducted to determine whether such facilities would bias the WMU area distributions used in the risk analysis. Identification of facilities that may be handling Bevill wastes was based upon Standard Industrial Classification (SIC) codes assigned to the Bevill waste streams. SIC codes assigned to industries producing Bevill wastes are as follows:

<u>SIC Code</u>	<u>Description</u>	<u>SIC Code</u>	<u>Description</u>
2816	Inorganic Pigments	10 - 1099	Metal Mining
2819	Ind. Inorganic Chem., Nec.	12 - 1299	Coal Mining
2874	Phosphatic Fertilizers	13 - 1399	Oil & Gas Extraction
3312	Blast Furnaces & Steel Mills	14 - 1499	Mining & Quarrying
3331	Primary Copper		of Nonmetallic
3339	Primary Nonferrous Metals, Nec.		Minerals Except Fuels

Electric power facilities (SIC code 4911, electric services) were also included in the analysis because their high-volume waste streams are also exempt from RCRA Subtitle C regulation.

3.3.1 Identification of Bevill Industrial D Facilities

Two approaches were used to identify Bevill facilities from the Industrial D database. The first removed facilities using a relationship between the Bevill-related SIC codes and the industry groups in the Industrial D database:

<u>Industry Group Removed</u>	<u>Group Description</u>	<u>SIC Codes</u>
6	Inorganic Chemicals	2812 - 2819
3	Fertilizer & Agr. Chem.	2873 - 2879
2	Primary Iron & Steel	3312 - 3321
9	Primary Nonferrous Metals	3331 - 3399
4	Electric Power Generation	4911

To provide additional resolution to the analysis (i.e., to reduce the number of facilities inappropriately culled because of the breadth of the Industrial D industry groups), 1,722 Industrial D facilities were matched to EPA IDs and SIC codes in EPA's Facility Index System (FINDS) database. The FINDS SIC codes are multiple per facility and were originally obtained from Dun & Bradstreet. The 1,722 facilities with EPA IDs were then culled by the SIC codes listed above for Bevill and electric power wastes; unlike the "industry group" approach, this included the mining waste SIC codes. Because of the multiple SIC codes for many facilities, a facility-by-facility review of 4911, 2819, and some mining SIC codes was conducted using Resource Conservation and Recovery Information System (RCRIS) data to identify facilities that should not be culled (i.e., facilities that could be producing and managing hazardous wastes not covered under Bevill, typically large-quantity generators as indicated in RCRIS). Thus, some facilities that would have been culled based on SIC code alone were not removed from the analysis.

This approach is not a perfect screen. It may leave in some facilities that manage primarily Bevill wastes, and it may exclude some facilities that manage some non-Bevill wastes. However, there are insufficient data available to refine the screening approach further.

3.3.2 Bevill Facilities Culled from Industrial D Data

To decide whether or not to remove the Bevill WMUs, the area percentiles for each WMU type in the entire Industrial D database were compared with those from the Industrial D datasets with Bevill facilities removed. For landfills and land application units, the area percentiles for the entire Industrial D universe were very similar to those with the Bevill WMUs removed, and the Bevill facilities were not removed for these WMU types. However, removing the Bevill wastepiles resulted in significantly lower wastepile areas for the upper percentile ranges. Based on the SIC codes and facility names, the largest 20 wastepiles could be classified as follows:

- # 9 phosphate mines
- # 5 other mines
- # 6 facilities that do not appear to be mining-related.

Only 3 of the 17 sites with average wastepile area greater than 250,000 square meters were not connected to mining activities.

Based on this assessment, the wastepiles identified as likely to be receiving Bevill waste were identified and removed from the Industrial D WMU data used in this analysis. In total, 84 wastepiles were removed because they contained Bevill wastes, leaving 745 facilities with wastepiles for the risk analysis.

3.4 Characterization of Tanks

The Industrial Subtitle D Survey (Schroder et al., 1987) did not include tanks. Therefore, a tanks database was developed for this analysis that compiled flow rates and tank volumes. The primary source for these data was the EPA's 1986 National Survey of Hazardous Waste Treatment, Storage, Disposal, and Recycling Facilities (TSDR) Database (U.S. EPA, 1987). This database is the result of a comprehensive survey of 2,626 hazardous waste treatment, storage, disposal, and recycle (TSDR) facilities, requested information concerning 1986 waste management practices and quantities. Responses were received from 2,322 facilities. The TSDR survey included a specific questionnaire concerning tanks used at each facility. Responses to this questionnaire provided tank information for about 18,773 tanks at 1,700 facilities.

A subset of the TSDR survey responses was available for facilities that received any quantity of waste from off-site. This subset of data contained information on 8,510 tanks located at 710 facilities (approximately 45 percent of all of the tanks contained in the TSDR Survey). This reduced data set was used to characterize tanks for this analysis. Although it would have been preferable to use the complete data set, including tanks at facilities that treat only wastes generated on-site, these data are unfortunately no longer available. However, it is likely that the subset of data used represents the range of tank volumes reported for all tanks. The subset data

include a broad range of tank volumes ranging from less than 55 gallons to over 5 million gallons.

Table 3-6 shows the total number of tanks in the database, those culled from the dataset, and those remaining in the database that was used in this study. The totals add to somewhat less than 8,510 because the database included 1,270 units that, based on process codes, were not actually tanks. The remaining 7,240 were categorized as treatment or storage based on process codes, as described in Section 3.4.1; there were 472 tanks that specified both treatment and storage codes and that were therefore included in both treatment and storage datasets.

Several criteria were used in guiding the development of the tanks database. These criteria were applied to the TSDR survey data to determine which tanks should be excluded from the data set.

- # **Flow rate.** Only those tanks reporting nonzero flow rates were included in the analysis.
- # **Open versus covered tanks.** Only open storage and treatment tanks were considered in the analysis; closed or covered tanks were dropped because this study is only concerned with Industrial D scenarios, and RCRA has no requirement to cover nonhazardous tanks.
- # **Tank volume.** All tanks with a volume of 55 gallons or less were excluded from the analysis. These smaller-volume containers should be classified as drums and not tanks due to their size.

Additionally, two very large tanks (approximately 30 million gallons), one aerated treatment and one nonaerated treatment, were reviewed, because these tanks were many times larger than the next largest tanks and appeared to be nonrepresentative. The facility that owns both tanks was contacted and it was determined that both actually have volumes of 3 million gallons, a value within the range

Table 3-6. Summary of Tanks Removed from TSDR Survey Database

Description	Treatment Tanks	Storage Tanks
Number of Sites Reporting WMU Type in TSDR Database	2,346	5,366
Culled Sites: Zero size or flow	464	793
Culled Sites: Tank covered or cover not specified	979	3,885
Culled Sites: Size \leq 55 gallons	6	48
Culled Sites: Outside contiguous United States	4	2
Total Number of Sites included in Air Characteristics Study Data Set	893	638

represented by the other tanks in the database. Both values were corrected to 3 million gallons for this analysis.

- # **Location.** Tanks located outside the continental United States were excluded from the database, because suitable meteorological stations were not available to model these sites.

3.4.1 Tank Classification

Industrial tanks can be used for either treatment or storage of wastes. Treatment tanks can be either quiescent or aerated/agitated. Examples of quiescent treatment tanks are clarifiers and filters (such as sand or mixed-media filters). Storage tanks are, by definition, quiescent because they do not include aeration processes. In the absence of aeration, both treatment and storage tanks are still subject to small amounts of agitation during filling and emptying operations if they have above-surface intakes. Aeration or agitation in a wastewater treatment system transfers air to the liquid to improve mixing or increase biodegradation. The turbulence caused by aeration/agitation also enhances mass transfer to the air, thus increasing emissions. Therefore, for a given treatment volume, a facility with aerated tanks will have higher emissions than a facility with quiescent tanks.

To reflect emission characteristics associated with treatment versus storage tanks and differences within the treatment tank category related to aeration intensity, three different tank categories were identified and modeled:

- # Storage tanks
- # Aerated treatment tanks
- # Nonaerated treatment tanks.

Sorting of the tanks in the database into these three categories was done using the WMU code reported for each unit. Within the TSDR survey, the respondents were requested to provide a WMU code to describe the type of process for which each tank was used. Those tanks with WMU codes of either 2A (accumulation in tanks) or 2ST (storage in tanks) were classified as storage tanks. The TSDR Survey used a broad range of WMU treatment codes (including codes for incinerators and belt filter presses). For this reason, classification of treatment tanks was limited to those processes listed in Table 3-7.

The treatment tank WMU codes were evaluated further to determine the level of aeration used. HI aeration was assigned to tanks reporting processes that actively mix the liquid surface for the purpose of aeration or that add diffused air. LO aeration was assigned to tanks reporting processes that are likely to require mixing devices due to the addition of chemicals or other purposes. NO aeration was used for tanks that are purposefully operated to minimize mixing or agitation (e.g., a clarifier). The aeration level assignments for each WMU code are shown in Table 3-7. The high- versus low-aeration classification was based on the nature of the process description associated with the various process codes.

Table 3-7. TSDR Survey Wastewater Treatment Codes Used in Identifying Treatment Tanks

Process Code/Process	Aeration Level	Process Code/Process	Aeration Level
Equalization		Filtration	
1WT Equalization	LO	34WT Diatomaceous earth	NO
Cyanide oxidation		35WT Sand	NO
2WT Alkaline chlorination	LO	36WT Multimedia	NO
3WT Ozone	LO	37WT Other filtration	NO
4WT Electrochemical	LO	Sludge dewatering	
5WT Other cyanide oxidation	LO	38WT Gravity thickening	NO
General oxidation (including disinfection)		Air flotation	
6WT Chlorination	LO	43WT Dissolved air flotation	HI
7WT Ozonation	LO	44WT Partial aeration	HI
8WT UV radiation	LO	45WT Air dispersion	HI
9WT Other general oxidation	LO	46WT Other air flotation	HI
Chemical precipitation		Oil skimming	
10WT Lime	LO	47WT Gravity separation	NO
11WT Sodium hydroxide	LO	48WT Coalescing plate separation	NO
12WT Soda ash	LO	49WT Other oil skimming	NO
13WT Sulfide	LO	Other liquid phase separation	
14WT Other chemical precipitation	LO	50WT Decanting	NO
Chromium reduction		51WT Other liquid phase separation	NO
15WT Sodium bisulfite	LO	Biological treatment	
16WT Sulfur dioxide	LO	52WT Activated sludge	HI
17WT Ferrous sulfate	LO	54WT Fixed film--rotating contactor	LO
18WT Other chromium reduction	LO	57WT Anaerobic	NO
19WT Complexed metals treatment	LO	58WT Other biological treatment	HI
Emulsion breaking		Other wastewater treatment	
20WT Thermal	NO	60WT Neutralization	LO
21WT Chemical	LO	61WT Nitrification	LO
22WT Other emulsion breaking	LO	62WT Denitrification	LO
Evaporation		63WT Flocculation and/or coagulation	NO
31WT Solar	NO	64WT Settling (clarification)	NO
Fuel blending		66WT Other wastewater treatment	LO
1FB Fuel blending	LO	Other Processes	
		1TR Other treatment	LO

- # Equalization, cyanide oxidation, general oxidation, chemical precipitation, and chromium reduction all involve adding and mixing a chemical into the wastewater followed by a quiescent period. Therefore, these tanks were classified as LO aeration because the chemical addition and mixing involve more agitation than a storage tank but involve no processes with intense agitation or forced air.
- # Emulsion breaking included two different processes. Thermal heating simply involves heating and letting the wastewater stand, whereas chemical emulsion breaking involves chemical addition and mixing followed by a quiescent period. Therefore, thermal emulsion breaking was classified as NO and chemical emulsion breaking was classified as LO. The category “other emulsion breaking” was classified as LO because the other processes in the emulsion breaking category ranged from NO to LO, so this represented a conservative default classification in the absence of more specific process data.
- # Filtration processes are quiet and generally covered; therefore, these were classified as NO aeration. Many of these, in fact, were eliminated from the database because covered tanks, as a class, were removed.
- # Air flotation processes all involve high-energy forced air operations and are therefore all classified as HI aeration.
- # Oil skimming involves liquid phase separation, which requires quiescent conditions; therefore NO aeration was assumed for these processes. Similarly, liquid phase separation processes were classified as NO aeration.
- # Biological treatment processes are quite diverse and include HI aeration activated sludge processes and LO aeration film processes. The “other biological treatment” processes were classified as HI because the other processes in the biological treatment category ranged from NO to HI and HI represents a conservative default classification in the absence of more specific process data.
- # Finally, the “other wastewater treatment” process in the “other wastewater treatment” category and the “other processes” category were classified as LO aeration as a default since no process information can be inferred from the description.

The numbers of tanks included in each classification are summarized in Table 3-8. Some tanks reported multiple process codes that included both a storage code (2A or 2ST) and a treatment code (one of the 50 in Table 3-7). These 472 tanks were included in both the storage tank database and one of the treatment tanks databases. However, there is no overlap between the two treatment tank databases.

The tank database does appear to underrepresent highly aerated tanks. This may be due to the age of the survey data, reflecting that highly aerated biological processes were in less widespread use at that time than now. This underrepresentation introduces some uncertainty into the analysis, the result of which is that risks from aerated tanks may be underestimated.

Table 3-8. Numbers of Tanks by Classification

Tank Classification	Number
Storage tanks	638
Aerated treatment tanks	620
High aeration	29
Low aeration	591
Nonaerated treatment tanks	273
Total	1,531

3.4.2 Additional Tank Data Used for Imputation

To address tank-specific data gaps in the tanks database, additional data sources were identified. These data included information collected in 1985 and 1986 during EPA site visits to aerated treatment systems in support of the development of RCRA Air Emission Standards. The systems visited were selected to represent a range of aeration processes and reflect a variety of industries and waste types. At that time, candidate facilities were identified through numerous phone contacts with state and local environmental agencies. From those conversations, information on wastewater treatment systems at 54 facilities was collected, and then site visits to these facilities were conducted in 1985 and 1986. Data on the individual tanks (both aerated and nonaerated) were provided by the facilities during the site visits, including data on tank dimensions. The data on aerated tanks are summarized in Coburn et al. (1988) and Eichinger (1985) (these documents are available in EPA Docket No. F-90-CESP, Hazardous Waste Treatment, Storage, and Disposal Facilities. RCRA Air Emission Standards for Proposal: Studies, Reports, and Background Information. Document numbers are F-90-CESP-S00251 and F-90-CESP-S00130, respectively). The data on nonaerated tanks were collected at the same site visits and are unpublished. Added to these data were five tanks from the TSDf BID (U.S. EPA, 1991). This resulted in a supplemental database of 49 tanks (13 with high aeration, 9 with low aeration, and 27 with no aeration), presented in Table 3-9.

In addition to these data, several tank vendors were contacted to establish a reasonable high end for tank capacity and depth based on design principles. As a result, a reasonable maximum capacity for an open, partially or completely aboveground tank was defined to be approximately 3 million gallons and the depth of such a tank would not be expected to exceed 10 m (about 32 ft) (Kendall Smith, personal communication, AO Smith Industrial, March 16, 1999). These site visit tanks and hypothetical tanks were used only as a basis for imputing values and were not modeled in the analysis in order to maintain the integrity of the source database.

3.4.3 Estimation of Missing Data

The TSDR Survey provided flow rate and tank volume data for use in characterizing tanks for this analysis. However, other key parameters, including depth, surface area, and height, also needed to be defined. In the absence of reported TSDR Survey data, these parameters were calculated as described below. Other operating parameters (aeration parameters), which impact emission estimates but not dispersion, are discussed in Section 4.0.

Table 3-9. Summary of Tank Size Information Collected in EPA Site Visits for RCRA Air Emission Standards

Type of unit	Aeration	Type of Aerator	Volume (m3)	Area (m2)	Depth (m)
Aerated Trtmt Tank	HI	Mechanical	108	27	4.0
Aeration Tank	HI	Mechanical	112	34	3.4
Bubbling Pit	HI	Diffused	453	74	6.1
Aerated Trtmt Tank	HI	Mechanical	1,600	430	3.7
Aeration Tank	HI	Diffused	1,666	159	10.5
Aeration Tank	HI	Mechanical	3,367	910	3.7
Aeration Tank	HI	Diffused	3,785	730	5.2
Aeration Tank	HI	Diffused	4,542	618	7.4
Aeration Tank	HI	Mechanical	5,678	931	6.1
Aeration Tank	HI	Diffused	5,764	1,051	5.5
Aux. Aer. Tank	HI	Mechanical	21,804	4,459	4.9
Aeration Tank	HI	Mechanical	26,546	5,806	4.6
Aeration Tank	HI	Mechanical	41,261	11,241	3.7
Treatment Tank	LO		30	13	2.4
Mixing Tank	LO	Mechanical	68	9.3	7.3
Treatment Tank	LO		76	26	2.7
Mixing Tank	LO	Mechanical	112	34	3.4
No Eq. Basin	LO	Mechanical	191	84	2.3
So Eq. Basin	LO	Mechanical	240	109	2.2
Eq. Basin	LO	Mechanical	681	200	3.4
Treatment Tank	LO		800	65	12.0
Eq. Basin	LO	Mechanical	41,261	11,241	3.7
Gravitator	NO		5.6	1.8	3.1
Pre-Filter	NO		132	39	3.4
Final Filter	NO		154	42	3.7
Backwash Clarifier	NO		207	71	2.9
Clarifier	NO		283	46	6.1
Bio-sludge Holding Tanks	NO		300	66	4.6
Bio-sludge Holding Tanks	NO		300	66	4.6
Primary Clarifier	NO		641	263	2.4
Digestor	NO		819	117	7.0
API Separator	NO		836	457	1.8
Primary Clarifier	NO		1,803	591	3.1
Biosludge Thickener	NO		1,803	591	3.1
Clarifier/Thickener	NO		2,504	410	6.1
Final Clarifier	NO		2,513	687	3.7
Final Clarifier	NO		2,513	687	3.7
Final Clarifier	NO		2,513	687	3.7
Clarifier	NO		2,670	730	3.7
Clarifier	NO		2,670	730	3.7
Clarifier	NO		2,670	730	3.7
Clarifier	NO		3,065	804	3.8
Clarifier	NO		3,065	804	3.8
Clarifier	NO		3,065	804	3.8
Clarifier	NO		3,065	804	3.8
Ship's Ballast Water	NO		3,394	1,271	2.7
Final Clarifier	NO		3,918	1,430	2.7
Ship's Ballast Water	NO		10,244	1,051	9.8
Solid Waste Disposal Basin	NO		386,464	60,385	6.4

The depth of the waste was imputed from the reported tank volume (or capacity). This was accomplished by developing a regression of log (depth) versus log (capacity) using data in the supplemental data set discussed in Section 3.4.2 (49 tanks from the site visit/TSDf BID database plus TSDf BID tanks). Because the site visit data did not include any very small tanks or many very large tanks, a cube-shaped 55-gallon tank and a 3-million gallon/32-ft-deep tank (based on the vendor information) was included in the regression derivation. Regression lines were derived for aerated tanks (Equation 3-1) and nonaerated tanks (Equation 3-2), on the assumption that these might have different volume-to-depth relationships since aerated tanks may be shallower to facilitate aeration.

$$D = 10^{[0.1358 \times \log(V) + 0.2236]} \quad (3-1)$$

$$D = 10^{[0.1334 \times \log(V) + 0.1657]} \quad (3-2)$$

where

$$\begin{aligned} D &= \text{depth (m)} \\ V &= \text{volume (m}^3\text{)} \end{aligned}$$

However, as can be seen in Figure 3-4, the two regressions were nearly identical. Therefore, a single regression was developed using all 49 tanks from the site visit/TSDf BID database plus the hypothetical tanks, as follows:

$$D = 10^{[0.1057 \times \log(V) + 0.2804]} \quad (3-3)$$

Comparisons of this regression with regressions done without one or both hypothetical tanks indicate that the hypothetical tanks do not unduly dominate the regression.

This equation was then examined for the reasonableness of the depths predicted. Using Equation 3-3, 60-gallon tanks (the smallest tanks in the database) are approximately 1.9 m (6.2 ft) deep and about 39 cm (15.4 inches) in diameter. This seemed unrealistically tall and narrow; consequently, for very small tanks, a second equation was derived from the assumption of a cube-shaped tank:

$$D = V^{0.333} \quad (3-4)$$

At tanks of approximately 10 m³, Equation 3-3 predicts approximately cube-shaped tanks; therefore, Equation 3-4 is used for tanks smaller than 10 m³.

The largest tank in the TSDR database in the NO and LO aeration categories is 25,000 m³, and the projected depth for this tank using Equation 3-3 is 5.6 m (18 ft), which is acceptable for mixing tanks.

The largest tank in the TSDR database in the HI aeration category is 23,000 m³, and the projected depth for this tank is 5.5 m (18 ft). In evaluating the predicted depth of HI aeration, the

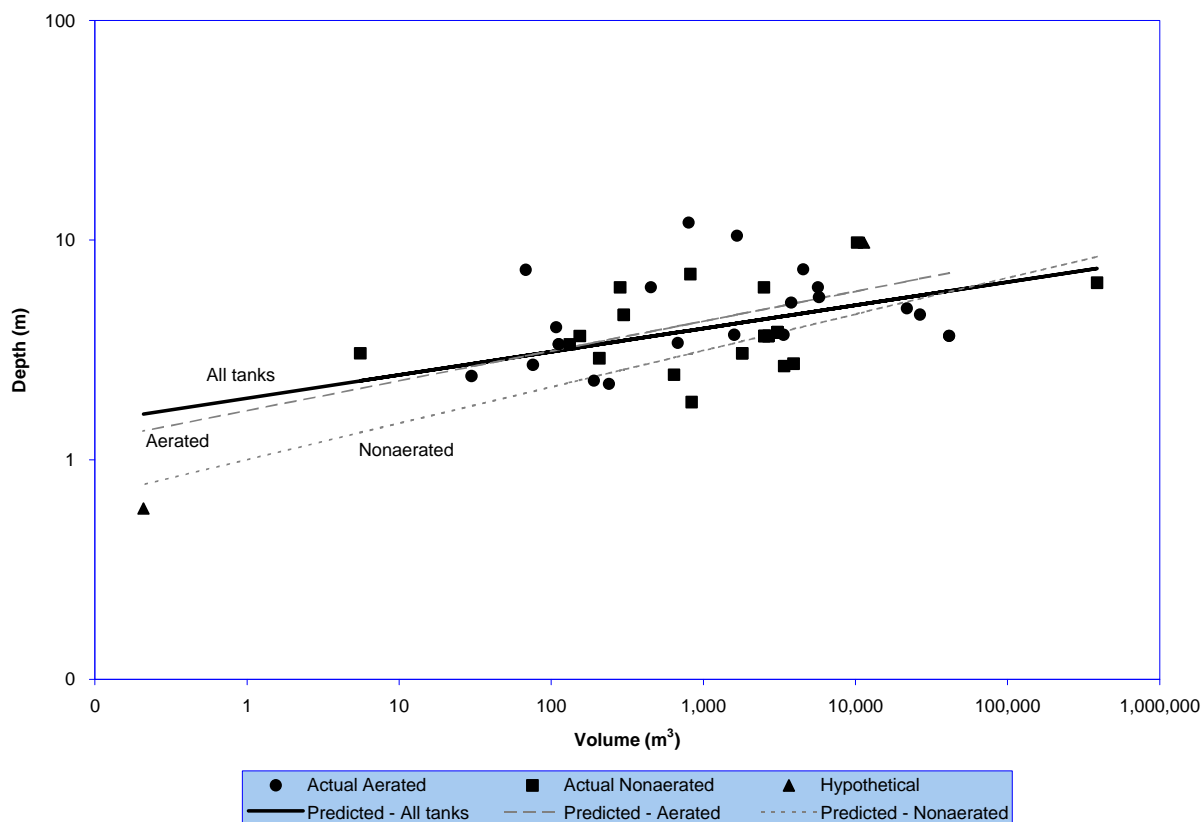


Figure 3-4. Comparison of tank depth regression lines.

eight mechanically aerated tanks from the site visits were considered. The maximum depth from these data was 6.1 m (20 ft) and even this appeared to be an outlier compared to the other HI aeration, mechanically aerated tanks. Data for the other seven site visit tanks all have depths ranging from 3.35 m (11 ft) to 4.88 m (16 ft). The mid-range of the latter depths is approximately equivalent to a 1,000-m³ tank as evaluated using Equation 3-3. Therefore, for HI aeration tanks greater than 1,000 m³, a random depth was assigned using a uniform distribution with endpoints of 3.5 m and 4.8 m.

Table 3-10 summarizes the methods used to make an initial estimate of tank depth for each type of tank. While these methods were intended to represent the actual relationship between volume and depth as closely as possible, they imply a certain precision that is unrealistic. In fact, there will be variation in the dimensions of tanks of the same volume. To address that variation, a random variation was applied to these initial estimates using a normal distribution with a mean of 1 and 90 percent of the values between 0.8 and 1.2. The initial depth estimate was multiplied by this random factor to obtain a final depth estimate used in this analysis.

Surface area data were not provided in the TSDR Survey. In the absence of these data, surface area for each of the TSDR tanks was calculated by dividing tank volume by depth.

Table 3-10. Summary of Depth Imputation Techniques

Tank Type	Volume Range (m ³)	Imputation Technique
HI Aeration	<10	Equation 3-4
	10-1,000	Equation 3-3
	>1,000	Uniform distribution from 3.5-4.8 m
LO Aeration	<10	Equation 3-4
	≥10	Equation 3-3
NO Aeration	<10	Equation 3-4
	≥10	Equation 3-3

The height of the top of the tank above the ground is needed for dispersion modeling. Height is related to depth, but not necessarily equal to depth, as tanks may be partially in the ground. In the absence of height data being reported in the TSDR Survey, height was imputed from depth using a two-step process:

1. A number was selected at random from 0 to 20 (uniform distribution).
2. If this number was less than the depth in meters + 0.5, it was used as the height. If it was greater than the depth in meters + 0.5, set height = depth + 0.5 m.

None of the tank depths imputed were greater than 9.5 m, therefore, none of the heights above ground were more than 10 m (9.5 + 0.5) using this method; 10 m above the ground is the realistic maximum height from a structural point of view, according to tank vendor contacts.

This approach establishes percentages of tanks of certain depths that will be all above-ground vs. partially or completely in ground:

- # For a 10-m tank, about half are partly or all in ground (when the random selection is between 0 and 10; as the random number increases from 0 to 10, more and more of the tank depth is aboveground, until at 10 all of it is), and about half are all aboveground (when the random pick is between 10 and 20).
- # For a 1-m tank, about 5 percent are partly or all in ground (random numbers from 0 to 1) and 95 percent all aboveground (random numbers from 1 to 20).
- # For a 5-m tank, 25 percent are partly or all in ground (random numbers from 0 to 5) and 75 percent all aboveground (random numbers from 5 to 20).

4.0 Source Emission Estimates

This chapter describes the source-specific emission model and assumptions used to develop the emission estimates for each waste management unit type. Section 4.1 discusses the selection of a general volatilization model and a particulate emission model to use for the emission estimates. Section 4.2 describes some of the critical model input parameters required to run the volatilization and particulate emission models. Section 4.3 describes modifications made to CHEMDAT8 for land-based units. Subsequent sections describe unit-specific modeling scenarios and assumptions used for the volatilization model effort. The final section of this chapter describes the particulate emission model estimates. Both volatile and particulate emissions were estimated for landfills, land application units, and wastepiles (referred to as land-based WMUs), while only volatile emissions were estimated for tanks.

4.1 Model Selection

4.1.1 Volatile Emission Model Selection

Several factors were considered in selecting emission models for assessing the potential for contaminant exposure through inhalation. In developing acceptable contaminant limits for wastes, the ideal emission model would provide emission estimates that are as accurate as possible without underestimating the contaminant emissions. Because both volatile emissions (for all WMU types) and particulate emissions due to wind erosion (for land-based WMUs) were required in the risk analysis, the volatile emission model had to estimate both volatile emission rates and long-term average soil concentration in the unit (for land-based WMUs). Ideally, the model would provide a relatively consistent modeling approach (in terms of model complexity and accuracy) for each of the different emission sources under consideration. Additionally, the emission model would have to be reviewed both internally by EPA and externally by both state and local agencies and industry representatives. Finally, the model would have to be publicly available for use in more site-specific evaluations.

Based on these considerations, EPA's CHEMDAT8 model was selected as the model to estimate volatile emission rates and long-term average soil concentrations in the WMU. The CHEMDAT8 model was originally developed in projects funded by EPA's Office of Research and Development (ORD) and Office of Air Quality Planning and Standards (OAQPS) to support National Emission Standards for Hazardous Air Pollutants (NESHAPs) from sources such as tanks, surface impoundments, landfills, wastepiles, and land application units for a variety of industry categories including chemical manufacturers, pulp and paper manufacturing, and petroleum refining. It also has been used to support the emissions standards for hazardous waste treatment, storage, and disposal facilities (U.S. EPA, 1991) regulated under Subpart CC rules of RCRA, as amended in 1984. The CHEMDAT8 model is publicly available and has undergone

extensive review by both EPA and stakeholder representatives. The CHEMDAT8 spreadsheet model and model documentation may be downloaded at no charge from EPA's web page (<http://www.epa.gov/ttn/chief/software.html>).

The CHEMDAT8 model considers most of the competing removal pathways that might limit air emissions, including adsorption, hydrolysis (for tanks only), and biodegradation. Adsorption/absorption is the tendency of a chemical or liquid media to attach or bind to the surface or fill the pores of particles in the soil or waste and therefore not volatilize into the air. This tendency to adsorb to or absorb in particles is an important process for estimating the concentration of the chemical on particles emitted to the air due to wind erosion. CHEMDAT8 in its original form models adsorption for land-based units by presuming that the entered waste concentration is in liquid phase. Because waste concentrations are more typically measured as total concentration (liquid plus solid phase), CHEMDAT8 was modified to model adsorption explicitly for an entered total waste concentration for land-based units. Biodegradation is the tendency of a chemical to be broken down or decomposed into less complex chemicals by organisms in the waste or soil. Similarly, hydrolysis is the tendency of a chemical to be broken down or decomposed into less complex chemicals by reaction with water. Chemicals that decompose due to either biodegradation or hydrolysis have lower potential for emission to the air as gases or particles than those that do not. Loss of contaminant by leaching or runoff is not included in the CHEMDAT8 model. Both leaching and runoff are a function of a chemical's tendency to become soluble in water and follow the flow of water (e.g., due to rainfall) down through the soil to groundwater (leaching) or downhill to surface water (runoff). These two mechanisms would also result in less chemical being available for emission to the air as gases or particles. As such, CHEMDAT8 is considered to provide reasonable to slightly high (environmentally protective) estimates of air emissions from the land-based units.

The CHEMDAT8 model was used to estimate the emissions for all WMUs with some modifications. CHEMDAT8 calculates a fraction of chemical emitted. Some additional equations, which are described in Sections 4.4 through 4.7, were added to calculate emission rates in $\text{g}/\text{m}^2\text{-sec}$ and remaining concentration in mg/kg from the fraction emitted and other inputs. This document does not present the equations used by CHEMDAT8 to calculate fraction emitted other than to show modifications made to model adsorption for total waste concentration instead of just liquid-phase waste concentration. The reader interested in the CHEMDAT8 algorithms is referred to the CHEMDAT8 documentation (U.S. EPA, 1994e). Additionally, certain equations were modified to prevent division by zero when certain volatilization parameters (Henry's law constant or vapor pressure) were zero (e.g., for metals).¹

¹ Specifically, the CHEMDAT8 model was modified to prevent division by zero as follows:

- “If-statements” were added to set the biodegradation rate to a negligible level or zero and prevented division by zero when no biodegradation rate constants were available.
- “If-statements” were added to prevent division by zero for chemicals that did not have vapor pressure, Henry's law constant, or diffusivity inputs (e.g., metals).

4.1.2 Particulate Emission Model Selection

The model selection criteria for the particulate emission models were similar to those for the volatilization model. Specifically, the particulate emission model would provide as accurate emission estimates as possible without underestimating the contaminant emissions. The model would provide a relatively consistent modeling approach (in terms of model complexity and accuracy) for each of the different emission sources under consideration, and the emission model would have to be both reviewed and publicly available for use for more site-specific evaluations. Two different models were selected to model wind erosion: one for wastepiles (elevated sources) and one for landfills and land application units (ground-level sources). Based on the considerations above, the Cowherd model (U.S. EPA 1985b, 1988) was selected for modeling wind erosion emissions from ground-level sources, and the AP-42 model for wind erosion from aggregate storage piles (U.S. EPA, 1985a) was selected for modeling wind erosion emissions from wastepiles. Newer versions of both of these models are available; however, the newer versions are event-based algorithms that require extensive site-specific data that were not available for the sites modeled in this analysis. The versions used probably result in somewhat higher particulate emission estimates than the event-based algorithms would. This overestimation of particulate emissions is not significant for volatile chemicals, as particulate emissions were found to be a negligible fraction (less than 2 percent in most cases) of total emissions for the volatile chemicals modeled in land-based units. The protective waste concentrations (C_w 's) for metals other than mercury (which do not volatilize and are therefore based solely on particulate emissions) may be somewhat lower as a result of this overestimation of emissions.

4.2 Emission Model Input Parameters

This section discusses the various parameters that impact the estimated volatilization and particulate emission rates. Inputs that influence these rates include input parameters specific to the physical and chemical properties of the constituent being modeled, the physical and chemical characteristics of the waste material being managed, input parameters specific to the process and operating conditions of the WMU being modeled, and meteorological parameters.

A general discussion of the physical and chemical properties of the constituents is provided in Section 4.2.1. Critical input parameters for the remaining sets of inputs are discussed first for land-based WMUs and then for tanks. A sensitivity analysis was performed for the 1998 Air Characteristic Study to better understand the impact of certain modeling assumptions on the model results. While the models and data have changed somewhat, those changes would not alter the conclusions drawn; therefore, these sensitivity analyses are not included here. The interested reader is referred to Appendix C of the May 1998 Air Characteristic Study.

4.2.1 Chemical-Specific Input Parameters

Key chemical-specific input parameters include: air-liquid equilibrium partitioning coefficient (vapor pressure or Henry's law constant), liquid-solid equilibrium partitioning coefficient (log octanol-water partition coefficient for organics), biodegradation rate constants, and liquid and air diffusivities. The Hazardous Waste Identification Rule (HWIR) chemical properties database (RTI, 1995; U.S. EPA, 1995b) was used as the primary data source for the

physical and chemical properties for the constituents being modeled. This chemical properties database provided the following chemical-specific input parameters: molecular weight, vapor pressure, Henry's law constant, solubility, liquid and air diffusivities, and log octanol-water partition coefficient. Soil biodegradation rate constants were obtained from Howard et al. (1991). The CHEMDAT8 chemical properties database (U.S. EPA, 1994b) was used as a secondary data source for the physical and chemical properties for the constituents being modeled. This chemical properties database provided the following chemical-specific input parameters: density, boiling point, Antoine's coefficients (to adjust vapor pressure for different temperatures), and biodegradation rate constants for tanks. The biodegradation rate constants in the downloaded CHEMDAT8 database file were compared with the values reported in the summary report that provided the basis for the CHEMDAT8 tank biodegradation rate values (Coburn et al., 1988). Tank biodegradation rate constants for compounds with no data were assigned biodegradation rates equal to the most similar compound in the biodegradation rate database (or set to zero for metals). The specific chemical properties input database used for emission modeling is provided in Appendix B.

4.2.2 Critical Input Parameters for Land-Based WMU Emission Models

4.2.2.1 Volatile Emissions and Waste Concentration. The input parameters used for the CHEMDAT8 land-based unit emissions model are presented in Table 4-1. (Note: The data entry form in the CHEMDAT8 model refers to oil rather than waste; the term waste is used here for clarity.) Of these parameters, two are actually flags to determine which model equations to apply (Input ID Nos. L7 and L9). The most important flag for emission estimates is probably the aqueous waste flag (Input ID No. L7). This flag tells the CHEMDAT8 model which equilibrium partitioning model to use between the liquid and gas phases. For organic wastes, the model uses Raoult's law and the liquid-to-air partition coefficient becomes proportional to the contaminant's partial vapor pressure. For aqueous wastes, the model uses Henry's law and the liquid-to-air partition coefficient becomes proportional to the contaminant's Henry's law coefficient. All land-based WMUs were run twice; once assuming unit concentration (concentration set to 1 mg/kg, assuming Henry's law applies) and once assuming pure component (concentration set to 1E+6 mg/kg, assuming Raoult's law applies). The results presented in Volumes I and III are based on the aqueous phase emission rates (unit concentration and Henry's law). The pure component emission rates were used only to identify chemicals for which greater emissions occur from the organic phase than from the aqueous phase (which is rare) or to identify chemicals for which the aqueous-based results exceeded soil saturation concentrations, and note for these whether the target risk or hazard quotient would be exceeded modeling pure component.

The annual waste quantity is a critical source (site-specific input) parameter. This parameter along with assumptions concerning the frequency of contaminant addition and the dimensions of the unit combine to influence a number of model input parameters (Input ID Nos. L1, L2, L3, L8, and L12).

The CHEMDAT8 model is insensitive to windspeeds for long-term emission estimates from land-based units. Temperature affects the air diffusivity, which affects the volatilization rate and potentially affects the biodegradation rate (biodegradation rates were independent of temperature above 5°C and were set to zero below 5°C). Consequently, temperature is the only

Table 4-1. CHEMDAT8 Land-Based Unit Model Input Requirements

Input ID No.	Input Parameter	Data Source/Assumption
L1	Loading (g waste/cm ³ soil)	Waste quantity and/or density from Ind D Survey
L2	Concentration in waste (ppmw)	1 for unit concentration run; 1E+6 for pure component run
L3	Depth of tilling (or unit) (cm)	Assumed or set by capacity
L4	Total porosity	Assumed default value of 0.5
L5	Air porosity (0 if unknown)	Assumed default value of 0.25
L6	Molecular weight of waste	18 for unit concentration run; 147 for pure component run
L7	For aqueous waste, enter 1	1 for unit concentration run; 0 for pure component run
L8	Time of calculation (d)	Dependent on type of WMU
L9	For biodegradation, enter 1	Dependent on type of WMU
L10	Temperature (°C)	Set by location of WMU
L11	Windspeed (m/s)	Set by location of WMU
L12	Area (m ²)	Input from Ind D Survey
L13	Fraction organic carbon	Assigned randomly from distribution

meteorological data input that potentially impacts the emission results for the CHEMDAT8 model for the land-based WMU.

The total porosity and air porosity values that were used in the emission assessments were the default CHEMDAT8 model values for these parameters. These assumed porosity values appear to be reasonable for a waste and waste/soil matrices that have a density of 1.1 g/cm³.

For aqueous wastes, the molecular weight of the waste (Input ID No. L6) does not impact the calculations.

The molecular weight of the waste for the "pure component" runs using Raoult's law was set to 147 g/mol, which is the CHEMDAT8 default value for this input parameter. If the waste were truly pure constituent, then the appropriate molecular weight input for the waste would be the specific constituent's molecular weight. However, the pure component run is used to backcalculate an appropriate waste concentration limit that is often considerably less than pure component. Therefore, the scenario modeled is not actually pure constituent, and modeling the waste at the molecular weight of the constituent is not appropriate. If the actual molecular weight of the waste is higher than 147 g/mol, the default molecular weight used may underestimate volatile emissions. Conversely, if the actual molecular weight of the waste is lower than 147, the default value may overestimate volatile emissions. The magnitude of this under- or over estimation is expected to be small over the range of likely waste molecular weights.

The process of biodegradation is an important one because it lowers both the emission rate and the average soil concentration. Consequently, biodegradation is an important input parameter, and the biodegradation rate constants used in the model are critical parameters.

Biodegradation was treated differently for the various WMUs. Landfills are not designed for biodegradation, and waste in wastepiles managed over short periods will not be affected substantially. Therefore, both the landfill emission runs and the short-term wastepile emission runs did not include biodegradation losses. First-order biodegradation was included in the LAU emission runs and long-term wastepile emission runs. Note that the default CHEMDAT8 model method of calculating biodegradation rates was not used. CHEMDAT8 biodegradation rates were derived primarily from wastewater studies and applied to model biodegradation in soils using an assumed, low-biomass concentration. Because the first-order biodegradation rate constants obtained from Howard et al. (1991) were either based on soil studies or explicitly evaluated for applicability to soil, they were used instead. These biodegradation rate constants provide a more direct link to soil-based biodegradation and are considered more appropriate for modeling biodegradation in land-based WMUs.

The fraction of organic carbon, f_{oc} , affects adsorption. The f_{oc} of interest is the f_{oc} of the waste (assuming the waste contains sludge/solids) and not of the soil. Little data exist concerning the f_{oc} of the waste itself; therefore, default values for this parameter were defined and applied in the absence of data. A distribution was developed to represent f_{oc} at all sites. Because this parameter is a fraction, it must range from 0 to 1. A beta distribution was selected for the distributional form because the beta distribution also varies between 0 and 1. This distribution is defined by two parameters called alpha and beta. Fraction organic carbon is waste- and site-specific but is most often less than 0.1. Therefore, the distribution was fitted with the criteria that half the values generated should be less than 0.1, and that 90 percent of the values should be less than 0.5. The fitted distribution has an alpha value of 0.455 and a beta value of 2.05. Individual f_{oc} values for each WMU were then selected randomly from the distribution.

4.2.2.2 Particulate Emissions. Particulate emissions due to wind erosion were modeled for land-based units (landfills, land application units, and wastepiles). Landfills and LAUs were modeled differently than wastepiles because they are ground-level sources and wastepiles are elevated sources. Wind erosion emissions from landfills and LAUs were modeled using the Cowherd model (U.S. EPA, 1985b, 1988). This model estimates the emission of respirable particles (i.e., PM_{10}) due to wind erosion from a ground-level surface with an unlimited reservoir of erodible particles. Surfaces are defined as having a limited or unlimited reservoir based on threshold friction velocity (U^*); surfaces with a U^* greater than 0.5 m/s are considered limited; those with U^* less than 0.5 m/s are considered unlimited (U.S. EPA, 1988). Threshold friction velocity is a measure of the windspeed at the ground surface that would be required to remove particles from the surface. Examples of limited reservoirs include nonhomogeneous surfaces with stones, clumps of vegetation, or other nonerodible elements or crusted surfaces. Further, wind erosion is considered unlikely to occur from surfaces with full vegetative cover.

Wind erosion emissions from wastepiles were modeled using an equation from AP-42 (U.S. EPA, 1985a) for estimating emissions from wind erosion from active storage piles. The equation gives emissions of total suspended particulates (TSP). Typically, an equation-specific particle size multiplier would be applied to reduce the emissions to a desired size category, in this case, PM_{10} . No particle size multipliers (PSM) are given for this equation in AP-42; however, Cowherd (U.S. EPA, 1988) gives a PM_{10} particle size multiplier of 0.5 for use with this equation.

Important input parameters for particulate emissions include silt content of waste (i.e., percent with small particle size), number of days with greater than 0.01 inches of rainfall, and percent of time that windspeed exceeds 5.4 m/s. Data on the silt content of the wastes being modeled were not available. A median silt content for miscellaneous fill material of 12 percent (U.S. EPA, 1988) was used. The number of precipitation days and the frequency of windspeed greater than 5.4 m/s were location-specific; values were obtained from the National Oceanic and Atmospheric Administration (NOAA, 1992) and are summarized in Section 4.7.2.

4.2.3 Critical Parameters for Tank Emissions Model

Table 4-2 presents the required CHEMDAT8 input parameters² along with units and comments on the source of the parameter values. As shown in Table 4-2, only one parameter (flow rate) has values that are taken directly from the TSDR survey data as discussed in Section 3.4 (that is, the data were provided by facility owner/operators at the time the survey was conducted). Volume data, which were used to impute a number of model input parameter values for tanks, were also taken directly from the TSDR survey data. The imputation procedures for the aeration and waste characteristics parameter values are discussed in this section. The procedures applied to estimate unit design parameters, which are also critical to air dispersion modeling, are discussed in Section 3.4.

Factors that affect the relative surface area of turbulence and the intensity of that turbulence are important in determining the fate of chemicals in tanks. The tank model has several input parameters that impact the degree and intensity of the turbulence created by the aeration (or mixing). Note that many of these parameters are determined for both aerated and some nonaerated (both treatment and storage) tanks. For nonaerated tanks with an above-surface intake (based on information available from the survey data), a small degree of aeration is modeled to account for the agitation of splash loading. Although there is not actually an aerator in these tanks, one is characterized to simulate the effects of splash loading. For nonaerated tanks with below-surface intakes, no agitation is modeled, and the aeration parameters are all set to zero. The values of most of the aeration parameters were estimated based on data collected in 1985 and 1986 during EPA site visits to aerated treatment systems (Coburn et al., 1988; Eichinger, 1985) and hypothetical tanks from the TSDF BID (U.S. EPA, 1991) (see Section 3.4.2 for further details). Aerator parameters for 16 tanks and surface impoundments (which are expected to have similar aerator properties to tanks) from these site visits and the TSDF BID are shown in Table 4-3. These data were used to provide a sense of the range and typical values of the aeration parameters.

The tank model is most sensitive to the fraction aerated; the total power, number of aerators, and impeller diameter have some impact on the emission results; and the other parameters have little to no impact on the estimated emissions.

² Note that this table also includes one parameter, height, that is not used in CHEMDAT8 but is used in dispersion modeling. For clarity, it is presented here as it is derived from depth.

Table 4-2. CHEMDAT8 Tank Model Input Requirements

Input Parameter	Source	Imputation Method
Unit Design		
Flow rate (m ³ /s)	Survey	None
Depth (m)	Imputed	Based on volume
Average surface area (m ²)	Imputed	Based on volume and depth
Height above ground (m)	Imputed	Based on depth
Aeration Parameters		
Fraction agitated	Estimated distribution	
Total power (hp)	Imputed	Based on volume
Number of impellers	Imputed	Based on total power
Impeller diameter (cm)	Estimated constant = 61	
Impeller speed (rad/s)	Estimated constant = 130	
Power efficiency (unitless)	Estimated constant = 0.83	
O ₂ transfer rate (lbO ₂ /h-HP)	Estimated constant = 3	
Submerged air flow (m ³ /s)	Estimated constant = 0	
Waste Characteristics		
Active biomass conc. (kg/m ³)	Estimated distribution	Depends on treatment code
Total solids in (kg/m ³)	Estimated distribution	
Total organics (COD) In (g/m ³)	Estimated distribution	
Total biorate (mg/g-h)	Estimated constant = 19	
Meteorological Data		
Temp (°C)	Imputed	Based on meteorological station
Windspeed (m/s)	Imputed	Based on meteorological station

The fraction aerated depends on the level of aeration. Distributions for this parameter were developed for each aeration level. Highly aerated tanks should have a higher fraction aerated than less-aerated tanks. No tank can have a fraction aerated greater than 1 or less than zero, and realistically, the fraction aerated for an aerated tank should not be close to zero. A non aerated tank may have a small fraction aerated to simulate the agitation from splash loading from an above-surface intake. For HI aeration tanks, fraction aerated is randomly assigned from a normal distribution with a mean of 0.75 and a standard deviation of 0.1. Values greater than 1 are truncated to 1. For LO aeration tanks, fraction aerated is randomly assigned from a uniform distribution with endpoints of 0.2 and 0.8. For NO aeration and storage tanks with above-surface intakes, fraction aerated was randomly assigned from a normal distribution with a mean of 0.08 and a standard deviation of 0.03. Values less than zero were truncated at zero, and values that implied an area agitated more than 10 m² were truncated so that the agitated area was 10 m². The rationale for this is that splash loading would not affect an area greater than 10 m². For NO aeration tanks with below-surface intakes, the fraction aerated was set to zero.

Table 4-3. Summary of Mechanical Aerator Information Collected in EPA Site Visits for RCRA Air Emission Standards

Type of unit	Aeration	Total Power (hp)	# Aerators	Impeller diameter (cm)	Impeller speed (rad/s)	Oxygen transfer rate (lb/hp-h)	Power/volume (hp/m ³)	Aerator Power for Total Power >100 hp (hp)
Aeration tank	HI	300	3				0.053	100
Aeration tank	HI	7.5	1				0.067	
Aeration tank	HI	900	9	259	188	3.0	0.022	100
Aux. aer. tank	HI	450	6	259	126	3.0	0.021	75
Aeration tank	HI	900	6	274			0.034	150
Aeration tank	HI	150	2	50	124	3.0	0.045	75
Aerated trtmnt tank	HI	7.5					0.069	
Aerated trtmnt tank	HI	120					0.075	
So eq. basin	LO	30	2	107	7.1		0.125	
No eq. basin	LO	20	1	152	5.9		0.105	
Mixing tank	LO	3	1				0.027	
Eq. basin	LO	150	5	122	7.1		0.004	30
Mixing tank	LO	1.5	1	183	1.0		0.022	
Aerated lagoon	SI	30	2	14	367		0.009	
Aerated lagoon	SI	270	6	42	123		0.011	45
Aerated lagoon	SI	1800	28				0.040	64

Total aerator power depends on the volume of the tank and the level of aeration. For HI aeration tanks, total power per million gallons of volume was randomly assigned using a normal distribution with a mean of 115 hp/million gallons and 90 percent of the values between 80 and 150 hp/million gallons (Metcalf and Eddy, 1979). For LO aeration tanks, total power per million gallons of volume was randomly assigned using a normal distribution with a mean of 30 hp/million gallons and 90 percent of the values between 15 and 45 hp/million gallons. This was based on industry comments on the 1998 ACS that indicated that surface aerators used for mixing typically have power levels between 15 and 20 hp/million gallons, and aerators used for activated sludge have a minimum power of 20 to 30 hp/million gallons. The upper value of 30 was adjusted upward by a factor of 1.5 to provide values above the minimum, and the resulting range of 15 to 45 hp/million gallons was presumed to encompass 90 percent of all values. For both HI and LO aerated tanks, the power per million gallons was multiplied by the volume in millions of gallons to determine total power. For NO aeration and storage tanks, the power per million gallons was determined in the same way as for LO aeration tanks. However, the total power was then calculated by multiplying the power per million gallons by fraction aerated and volume (in million gallons) to estimate total power. A minimum value of 0.25 hp was set. Fraction aerated is included in this calculation to account for the fact that the whole tank volume is not affected by the agitation caused by splash loading.

The number of aerators (or impellers) for aerated tanks was derived from the total power and the power per aerator. Typical values of power per aerator for different total power levels were based on the data in Table 4-3 and the number of aerators set as follows:

- # For Total Power \leq 25 HP, one aerator
- # For 25 HP < Total Power < 80 HP, randomly pick one or two aerators with equal probability
- # For Total Power \geq 80 HP, randomly pick a power per aerator using a uniform distribution with endpoints of 60 and 100, then divide the Total Power by the random power per aerator and round up to the next integer. While some of the power per aerator values associated with the data in Table 4-3 fell outside the range of 60 to 100, this range represented most of the values.

For nonaerated tanks, the number of impellers was set to 1 if the tank had an above-surface intake and to zero if it had a below-surface intake.

Impeller diameter and rotational speeds appeared to be related parameters. Generally, the longer (or high-diameter) impellers found in the site visit data (see Table 4-3) had lower rotational speeds and the shorter impellers had faster rotational speeds. Consequently, independent random assignments of these variables was determined to be inappropriate. Rather than attempting to develop a correlation between the two parameters based on limited data, the fixed values used for the model tanks developed for the Hazardous Waste TSDF air rules (U.S. EPA, 1991) were selected. These values are reasonable central tendency values based on the limited available data presented in Table 4-3:

- # Impeller diameter fixed at 61 cm (2 ft), based on data reported by Watkins (1990)
- # Impeller rotational speed fixed at 130 rad/s, based on data reported by Watkins (1990)
- # Oxygen transfer rating and power efficiency do not have much impact on the emission, and vary over a very small range. Therefore, these values were fixed, as follows:
 - # Oxygen transfer rating fixed at 3.0 lb O₂/HP-h; U.S. EPA (1991) reports a range of 2.9 to 3 lb O₂/hp-h
 - # Power efficiency fixed at 0.83; U.S. EPA (1991) reports a range of 0.8 to 0.85.

Submerged air flow was set to zero for all tanks because aeration is modeled as mechanical aeration, not diffused air.

Waste characteristics that influence the rate of biodegradation are important in determining emissions from both aerated and storage tanks. As shown in Table 4-2, these parameters include active biomass concentration, total solids in, total organics in, and total biorate. Limited data were available on waste characteristics for tanks; most facilities only need to measure these parameters for the final wastewater discharge and do not measure them in the influent. From a review of the site visit reports described previously, the data estimations

discussed below were derived and used for aerated tanks reporting biological treatment. Aerated tanks reporting other types of treatment, nonaerated treatment tanks, and storage tanks were modeled with no biodegradation.

Unlike the biodegradation rate model that was used for the land-based units, the biodegradation rate model used in CHEMDAT8 for tanks is dependent on the amount of active biomass in the WMU. Therefore, the active biomass concentration is a critical parameter for aerated tanks. Because this parameter can vary widely for different types of tanks, biomass concentrations were set on a tank-by-tank basis for aerated tanks using process code information (WMU codes) from the TSDR Survey. For biological treatment aerated tanks, the active biomass concentration measured as mixed liquor volatile suspended solids (MLVSS) from the site visit reports range up to 4 g/L, and one source test measured MLVSS up to 6 g/L. However, the typically observed MLVSS concentration fell in the 1.5- to 3.0-g/L range. Many of the biodegradation rate constants developed for CHEMDAT8 used 2 g/L as a default MLVSS concentration to normalize the constituent disappearance rates. Therefore, 2 g/L was considered the most appropriate central tendency value for the active biomass concentration. Consequently, the following algorithm was used to select the active biomass concentration.

- # For biological treatment units (WMU codes 52WT and 58WT), this value was randomly assigned for each tank, using a uniform distribution with endpoints of 1 and 3 g/L
- # For all other aerated tanks (as well as storage and nonaerated treatment tanks), active biomass concentration was set to 0 g/L.

The "total biomass(solids) in," "total organics in," and "total biorate" (Input ID Nos. T6, T8, and T9) impact the rate of biomass production and subsequently the amount of contaminant that is absorbed onto the solids. (Note: The "biomass solids in" does not affect the biodegradation rate and is more appropriately labeled simply "solids in."). These inputs, however, have little or no impact on the estimated emission rates for most of the contaminants modeled in this analysis. The value for "total solids in" was randomly assigned for each tank using a uniform distribution with endpoints of 0.1 and 1 g/L, based on best professional judgment. CHEMDAT8 (U.S. EPA, 1994e) suggests a range of 0.1 to 0.4 for surface impoundments designed for biodegradation. The "total organics in" value was randomly assigned for each tank, using a uniform distribution with endpoints of 100 and 1,000 mg/L, based on best professional judgment. The CHEMDAT8 default value is 250 mg/L (U.S. EPA, 1994e).

The input parameter listed as "total biorate, $\text{mg/g}_{\text{bio-h}}$ " is used in conjunction with the active biomass concentration to estimate the growth or replacement rate of biomass, e.g., how much of the "old" biomass is consumed to grow "new" biomass that is now available for contaminant adsorption. This biomass replacement rate is used in conjunction with the influent total solids and total organic concentrations to determine the total rate at which total suspended solids (TSS) are removed (or "wasted") from the system. Because this input parameter impacts only tanks that have active biomass greater than zero (a small fraction of the total number of tanks in the data set) and this biomass replacement factor will generally have only a small, if any, contribution to the adsorptive losses, no additional research was performed for this parameter.

Instead, the total biorate is fixed at 19 mg/g biomass-h, which is the default value recommended for CHEMDAT8 (U.S. EPA, 1994e).

Due to the nonlinearity of the biodegradation rate model used in the tank emission estimates, direct backcalculation of an acceptable waste concentration may not be appropriate for some compounds. Unlike the emission results from the land-based units, the contaminant concentration used in the analysis may impact the predicted "normalized" emission rate (i.e., the emission rate in $\text{g/m}^2\text{-s}$ per mg/L of contaminant). Therefore, the tanks with biodegradation were run at a low concentration (i.e., 0.001 mg/L) and at a high concentration (i.e., the constituent's solubility). The most appropriate backcalculated emission value was then selected based on the concentration range of the backcalculated values and the constituent's biodegradation characteristics (see Section 7.9 for further details).

Meteorological inputs are also important for the tank emission model. For nonaerated treatment tanks and storage tanks, the emission estimates are impacted by both temperature and windspeed. Because the emissions for aerated tanks are predominantly driven by the turbulent area and associated mass transfer coefficients, the emissions from the aerated tanks are not strongly impacted by the windspeed. Aerated tank emissions are impacted by temperature. Annual average temperatures were used as input to the model based on tank locations. (Note that, dependent on the residence time of the waste in the tank, the temperature of the waste in the tank was not expected to vary significantly with changing atmospheric temperatures, and annual average temperatures were used to estimate the average waste temperature in the tanks). The location of each tank is available from the TSDR survey data. Based on this information, each tank was assigned to one of the 29 meteorological stations used in the dispersion modeling. As discussed in Section 5, these assignments were made based on both proximity and similarity of the climatological characteristics that affect meteorological data. The windspeed and temperature used in emissions modeling are the annual averages for the assigned meteorological station, taken from NOAA (1992).

4.3 Modifications to CHEMDAT8 for Land-Based Units

The CHEMDAT8 model estimates emissions from land-based WMUs (such as land application units, open landfills, and wastepiles) using a simple emissions model that accounts for contaminant partitioning between a liquid waste matrix and the air, diffusion of vapors through a porous media, and contaminant loss through biodegradation. The CHEMDAT8 model, however, does not accommodate entered total waste concentrations (i.e., liquid and solid phase). The assumption of an entered waste concentration in liquid phase was based on the petroleum wastes for which CHEMDAT8 was originally developed and may not apply to the chemicals considered in this analysis. Therefore, a method for including adsorptive partitioning for total waste concentrations was developed and is presented below.

Assuming three-phase partitioning (adsorbed, dissolved, and volatile), the total concentration of a contaminant can be expressed as the sum of the masses of the contaminant adsorbed on the soil, dissolved in the liquid, and in the air spaces divided by the total mass of contaminated soil as follows:

$$C_T = C_s \rho_b + \theta_w C_w + \theta_a C_a \quad (4-1)$$

where

- C_T = total contaminant concentration ($\mu\text{g}/\text{cm}^3_{\text{soil}} = \text{g}/\text{m}^3_{\text{soil}}$)
 C_s = concentration of contaminant adsorbed on soil ($\mu\text{g}/\text{g}_{\text{soil}} = \text{g}/\text{Mg}_{\text{soil}}$)
 ρ_b = soil dry bulk density ($\text{g}_{\text{soil}}/\text{cm}^3_{\text{soil}} = \text{Mg}_{\text{soil}}/\text{m}^3_{\text{soil}}$)
 θ_w = water-filled soil porosity ($\text{m}^3_{\text{water}}/\text{m}^3_{\text{soil}}$)
 C_w = concentration of contaminant dissolved in liquid ($\mu\text{g}/\text{cm}^3_{\text{water}} = \text{g}/\text{m}^3_{\text{water}}$)
 θ_a = air-filled soil porosity ($\text{m}^3_{\text{air}}/\text{m}^3_{\text{soil}}$)
 C_a = concentration of contaminant in air ($\mu\text{g}/\text{cm}^3_{\text{air}} = \text{g}/\text{m}^3_{\text{air}}$).

The adsorbed contaminant concentration is assumed to be linearly related to the liquid phase concentration as follows:

$$C_s = K_d C_w \quad (4-2)$$

where

- C_s = concentration of contaminant adsorbed on soil ($\mu\text{g}/\text{g}_{\text{soil}} = \text{g}/\text{Mg}_{\text{soil}}$)
 K_d = soil-water partition coefficient ($\text{cm}^3/\text{g} = \text{m}^3/\text{Mg}$)
 C_w = concentration of contaminant dissolved in liquid ($\mu\text{g}/\text{cm}^3_{\text{water}} = \text{g}/\text{m}^3_{\text{water}}$)

For organic constituents:

$$K_d = K_{oc} f_{oc} \quad (4-3)$$

where

- K_{oc} = organic-carbon partition coefficient ($\text{cm}^3/\text{g} = \text{m}^3/\text{Mg}$)
 f_{oc} = weight fraction organic carbon content of the solid matrix ($\text{g}/\text{g} = \text{Mg}/\text{Mg}$).

The contaminant concentration in the vapor phase is assumed to be linearly related to the liquid phase concentration as follows:

$$C_a = H' C_w \quad \text{or} \quad C_w = \frac{C_a}{H'} \quad (4-4)$$

where

- H' = dimensionless Henry's law constant = $H/RT = 41 \times H$ at 25°C where
 H = Henry's law constant at 25°C ($\text{atm}\cdot\text{m}^3/\text{mol}$).

Combining Equations 4-1, 4-2, and 4-4 by replacing C_s in Equation 4-1 with the term in Equation 4-2 and C_w in Equation 4-1 with the term in Equation 4-4 yields the following expression in terms of the gas phase concentration C_a :

$$C_T = C_a \left[\frac{\rho_b K_d}{H'} + \frac{\theta_w}{H'} + \theta_a \right]. \quad (4-5)$$

The total contaminant concentration, C_T , represents the measured soil concentration. Equation 4-5 can be rearranged to calculate the gas phase concentration given the total contaminant concentration as follows:

$$C_a = \frac{C_T H'}{K_d \rho_b + \theta_w + \theta_a H'} \quad (4-6)$$

This partitioning theory, as represented by the above equations, was used to include adsorption in CHEMDAT8, as described below.

The CHEMDAT8 land treatment emission model is based on the diffusion of a gas from a semi-infinite slab that initially has a uniform concentration of diffusing material throughout and that has equal concentrations of diffusing material at each surface (U.S. EPA, 1994e). The emission equations presented in CHEMDAT8 are in terms of the mass (as opposed to concentration) of contaminant in the gas phase.

CHEMDAT8 uses an equilibrium partitioning factor, K_{eq} , as a multiplier to correct the effective diffusion coefficient. The partitioning factor, K_{eq} , represents the ratio of the mass of organics in the vapor phase to the mass of organics in the soil/waste mixture and, therefore, is used to estimate the amount of material that partitions into the vapor phase based on equilibrium conditions within the soil/waste mixture. The CHEMDAT8 solution, as described in the CHEMDAT8 documentation, requires a ratio of the total mass of contaminant in the gas phase to the total mass of contaminant in the soil/waste matrix. The mass ratio for the partitioning correction factor (including adsorption) that was used in the CHEMDAT8 model, $K_{eq, ads}$, therefore, was defined as follows:

$$K_{eq, ads} = \frac{M_a}{M_T} \quad (4-7)$$

where

$$\begin{aligned} M_a &= \text{mass of constituent in the air-filled soil porosity (g)} \\ M_T &= \text{total mass of constituent in the soil/waste mixture (g)}. \end{aligned}$$

The masses of constituent in the air-filled soil porosity and in the soil/waste mixture are equal to their respective concentrations times volumes:

$$\frac{M_a}{M_T} = \frac{C_a V_a}{C_T V_T} \quad (4-8)$$

where

$$\begin{aligned} V_a &= \text{total volume in the air-filled soil porosity (m}^3\text{)} \\ V_T &= \text{total volume of the soil/waste mixture (m}^3\text{)}. \end{aligned}$$

Using the relationship between C_a and C_T presented in Equation 4-6, and the definition of porosity shown in the following equation,

$$\theta_a = \frac{V_a}{V_T}, \quad (4-9)$$

the terms in Equation 4-8 can be substituted for, and $K_{eq, ads}$ can be rewritten as follows:

$$K_{eq, ads} = \frac{\theta_a H'}{(\rho_b K_d + \theta_w + \theta_a H')} \quad (4-10)$$

The CHEMDAT8 model was modified to include Equation 4-10. This equation presents an expression for $K_{eq, ads}$ that achieves the goal of including adsorptive partitioning of total waste concentration in the model. This equation always yields a partitioning value of 1 or less. At high Henry's law values, $K_{eq, ads}$ is necessarily equal to 1, providing the same emission rate predictions as if the total initial mass of contaminant was in the vapor phase with no partitioning.

4.4 Development of Volatile Emissions and Waste Concentrations for Landfills

The basic assumptions used for modeling landfills are as follows:

- # The landfill operates for 20 years filling 20 cells of equal size sequentially.
- # The active cell is modeled as being instantaneously filled at time $t=0$ and remains open for 1 year.
- # Emissions are calculated only for one cell for 1 year (after 1 year, the cells are either depleted of the constituent or capped).
- # The waste is homogeneous with an initial concentration of 1 mg/kg.

- # The waste matrix may be aqueous (Henry's law partitioning applies) or organic (Raoult's law partitioning applies).
- # Annual average temperature is used (determined by assigned meteorological station).
- # Acute and subchronic exposures were not modeled.

The results presented in Volumes I and III are based on the aqueous-phase emission rates (i.e., assuming a concentration of 1 mg/kg and Henry's law partitioning). Most of the waste streams managed in land-based units are expected to contain constituents in the aqueous, rather than the organic, phase; therefore, this is the most realistic scenario. However, results based on organic phase emissions are of interest in two circumstances:

- # Most chemicals are better able to volatilize from an aqueous medium than from an organic medium; therefore, for most chemicals, the aqueous-phase emission rates are considerably higher than organic-phase emission rates. However, for a few chemicals (most notably formaldehyde), the organic-phase emissions are higher than the aqueous-phase emissions. When this is the case, the results based on aqueous-phase emissions are footnoted to indicate that organic-phase emissions would be higher, so a concentration based on organic-phase emissions would be lower. This does not invalidate the aqueous-phase results, as that is still the most likely waste matrix.
- # Some of the backcalculated waste concentrations based on aqueous-phase emissions exceed the soil saturation concentration at a neutral pH and temperature of 20 to 25°C. This is the maximum possible aqueous-phase concentration in soil; once this is exceeded, free (organic-phase) product will occur in the soil. This soil saturation concentration has been estimated for each chemical in the analysis, but this is a somewhat site- and waste-specific value because it depends on solubility, soil properties such as bulk density and porosity, and temperature (see Section 7.10.2 for the equation used to calculate the soil saturation concentration). Therefore, a backcalculated concentration may exceed it in some situations but not in others. When the backcalculated concentration based on aqueous-phase emissions exceeded the calculated soil saturation concentration at a neutral pH and temperature of 20 to 25°C, the result was footnoted to indicate whether pure component (i.e., a concentration of 10⁶ mg/kg) would result in a risk exceeding the target risk when modeled using organic phase assumptions and Raoult's law.

Table 4-1 provides the CHEMDAT8 model input requirements for land-based units with some commentary about each input parameter. The inputs that were calculated from the Industrial D Screening Survey data were calculated as follows:

- # All total quantities, capacities, and areas in the Industrial D Screening Survey were divided by the number of landfills at the facility to get landfill-specific estimates.
- # Loading = bulk density = 1.1 g/cm³.
- # Tilling depth (cm) = landfill depth, l, calculated as follows:

$$D_{till} = \frac{100 \times C}{A \times BD} \quad (4-11)$$

where

- D_{till} = landfill depth (cm)
- 100 = unit conversion (cm/m)
- C = capacity (Mg)
- A = landfill area (m²)
- BD = bulk density (g/cm³ = Mg/m³)

If the calculated depth was less than 2 feet or more than 33 feet, then the method described in Section 3.1 was used.

- # Total landfill surface area was divided by 20 to get surface area of landfill cell.
- # The total landfill capacity was divided by 20 to get the average annual quantity of waste, Q_{annual} .

The landfill cell areas and depth were entered into the CHEMDAT8 input table (along with average ambient temperature), and the emission fraction for the "intermediate time" (365.25 days) was calculated. This emission fraction was then multiplied by the annual waste quantity and waste concentration and divided by the area of the cell to calculate the output emission rate as follows:

$$E = \frac{Q_{annual} \times C_{waste} \times f_{emit}}{A_{cell} \times 31,557,600} \quad (4-12)$$

where

- E = emission rate (g/m²-s)
- Q_{annual} = annual waste quantity (Mg/yr)
- C_{waste} = waste concentration (mg/kg = g/Mg)
- f_{emit} = fraction emitted (unitless)
- A_{cell} = cell area (m²)
- 31,557,600 = unit conversion factor (s/yr).

The average concentration of the waste in the landfill cell was estimated from the emission fraction and the biodegradation fraction (although the biodegradation fraction was zero – no biodegradation – for the landfill) by assuming first-order contaminant (concentration) disappearance. Assuming first-order kinetics with respect to the contaminant concentration in the landfill cell, an exponential decay can be written in terms of the apparent overall first-order decay rate. The concentration at a given time is equal to the initial concentration as follows:

$$C_{\text{waste},t} = C_{\text{waste},0} \times e^{(-K_{1,\text{all}} t)} \quad (4-13)$$

where

$$\begin{aligned} C_{\text{waste},t} &= \text{waste concentration at time } t \text{ (mg/kg)} \\ C_{\text{waste},0} &= \text{waste concentration at time } 0 \text{ (mg/kg)} \\ K_{1,\text{all}} &= \text{apparent first-order decay rate (yr}^{-1}\text{)} \\ t &= \text{time period of calculation (yr).} \end{aligned}$$

At the end of 1 year, $C_{\text{waste},t}/C_{\text{waste},0} = 1 - \text{emission fraction} - \text{biodegraded fraction}$. Therefore, the $K_{1,\text{all}} t$ term, at the time period for which the fraction loss terms were calculated, is simply:

$$K_{1,\text{all}} t = -\ln(1 - f_{\text{emit}} - f_{\text{bio}}) \quad (4-14)$$

where

$$\begin{aligned} K_{1,\text{all}} &= \text{apparent first-order decay rate (yr}^{-1}\text{)} \\ t &= \text{time period of calculation (yr)} \\ f_{\text{emit}} &= \text{fraction emitted (unitless)} \\ f_{\text{bio}} &= \text{fraction biodegraded (unitless).} \end{aligned}$$

The concentration versus time profile (Equation 4-13) can then be integrated to calculate the average waste concentration, $C_{\text{waste,ave}}$, over the time period of the calculation:

$$C_{\text{waste,ave}} = C_{\text{waste},0} \times \frac{1 - e^{-K_{1,\text{all}} t}}{K_{1,\text{all}} t} \quad (4-15)$$

where

$$\begin{aligned} C_{\text{waste,ave}} &= \text{average waste concentration (mg/kg)} \\ C_{\text{waste},0} &= \text{waste concentration at time } 0 \text{ (mg/kg)} \\ K_{1,\text{all}} &= \text{apparent first-order decay rate (yr}^{-1}\text{)} \\ t &= \text{time period of calculation (yr).} \end{aligned}$$

The input parameters required for the landfill are presented in Table 4-1. The annual waste quantity and unit dimensions are the critical source parameters. For landfills, the loading

rate is pure waste material so that loading (Input ID No. L1) is basically the waste density. A waste density of 1.1 g/cm³ was used for the landfill to be consistent with the waste densities used in the analysis of the Industrial D Screening survey data. The annual waste quantity is also combined with the area of the landfill to calculate the depth of the landfill.

Temperature and porosities have some impact on predicted emissions. The biodegradation flag was set to zero (no biodegradation) for landfills. Therefore, temperature variations should have less of an impact on the annual emission rates from landfills than from land application units. The model is insensitive to molecular weight of the waste (for aqueous wastes) and windspeed (for long-term emission estimates).

4.5 Development of Volatile Emissions and Waste Concentrations for Land Application Units

4.5.1 Chronic Exposure Analysis

Because the same basic CHEMDAT8 model was used for landfills and land application units, the emission estimates for land application units have some similarities to the landfill emission estimates, but there are also a number of differences. The basic modeling assumptions used for modeling land application units are as follows:

- # The land application unit emissions are modeled as pseudo-steady-state. Emissions are actually time-dependent (depending on how recently waste has been added) but are modeled as a series of steady-state emissions for short time intervals, which are then averaged to produce a long-term emission rate.
- # Emissions in year 40 are used to estimate long-term emissions. This does not reflect an assumed operating life of the unit but is simply a sufficiently long period to ensure that steady state has been reached, if it is ever going to be, (typically, steady state is reached in 1 or 2 years) and to exceed most of the exposure durations used in the modeling.
- # Waste application occurs twice monthly (i.e., 24 times per year).
- # The waste is homogeneous with an initial concentration of 1 mg/kg.
- # The waste matrix may be aqueous (Henry's law partitioning applies) or organic (Raoult's law partitioning applies).
- # Temperature is determined by assigned meteorological station; monthly average temperature was used.
- # Biodegradation occurs at temperatures greater than 5 °C.

The results presented in Volumes I and III are based on the aqueous-phase emission rates (i.e., assuming a concentration of 1 mg/kg and Henry's law partitioning). Most of the waste

streams managed in land-based units are expected to contain constituents in the aqueous, rather than the organic, phase; therefore, this is the most realistic scenario. However, results based on organic phase emissions are of interest in two circumstances:

- # Most chemicals are better able to volatilize from an aqueous medium than from an organic medium; therefore, for most chemicals, the aqueous-phase emission rates are considerably higher than organic-phase emission rates. However, for a few chemicals (most notably formaldehyde), the organic-phase emissions are higher than the aqueous-phase emissions. When this is the case, the results based on aqueous-phase emissions are footnoted to indicate that organic-phase emissions would be higher, so a concentration based on organic-phase emissions would be lower. This does not invalidate the aqueous-phase results, as that is still the most likely waste matrix.
- # Some of the backcalculated waste concentrations based on aqueous-phase emissions exceed the soil saturation concentration at a neutral pH and temperature of 20 to 25°C. This is the maximum possible aqueous-phase concentration in soil; once this is exceeded, free (organic-phase) product will occur in the soil. This soil saturation concentration has been estimated for each chemical in the analysis, but this is a somewhat site- and waste-specific value because it depends on solubility, soil properties such as bulk density and porosity, and temperature (see Section 7.10.2 for the equation used to calculate the soil saturation concentration). Therefore, a backcalculated concentration may exceed it in some situations but not in others. When the backcalculated concentration based on aqueous-phase emissions exceeded the calculated soil saturation concentration at a neutral pH and temperature of 20 to 25°C, the result was footnoted to indicate whether pure component (i.e., a concentration of 10⁶ mg/kg) would result in a risk exceeding the target risk when modeled using organic phase assumptions and Raoult's law.

The inputs that were calculated for the land application units were calculated as follows:

- # The total annual waste quantities and surface areas for each facility, as reported in the Industrial D Screening Survey, were divided by the number of LAUs at the facility to get LAU-specific estimates.
- # Tilling depth (cm) = 20 cm if $Q_{\text{annual}}(\text{Mg/yr})/\text{Area}(\text{m}^2) \leq 0.2$. If $Q_{\text{annual}}/\text{Area} > 0.2$, then depth (cm) = $100 \times Q_{\text{annual}}/\text{Area}$.
- # Loading rate, L, is calculated as follows:

$$L = \frac{Q_{\text{annual}} \times t}{A \times D_{\text{till}}} \quad (4-16)$$

where

L = loading rate ($\text{Mg}/\text{m}^3 = \text{g}/\text{cm}^3$)
 Q_{annual} = annual waste quantity (Mg/yr)
 t = time period of calculation (yr)
 A = LAU area (m^2)
 D_{till} = tilling depth (m).

- # Biodegradation is assumed to occur if temperature is greater than 5°C . If the temperature is 5°C or lower, biodegradation is turned off.
- # Time of calculation = $365.25/24$ applications per year = 15.2 days.
- # Monthly temperature and windspeeds were calculated by averaging the hourly temperature and windspeeds.

A sensitivity analysis was conducted that investigated the impact that application frequency (monthly versus quarterly) and the averaging period for meteorological data (monthly versus yearly) had on emission estimates. Results from this analysis indicated that both application frequency and averaging period do impact emissions. Therefore, modeling was conducted using monthly average meteorological data. In the absence of reported unit-specific data on application frequency in the Industrial D database, it was assumed that waste was applied to the unit two times during each month of the year (i.e., 24 times per year).

Land treatment generally involves the application of wastes to the land in either a liquid or a semi-solid form with treatment occurring through the biological degradation of the hazardous constituents. Waste is assumed to be delivered by tank trucks and is applied uniformly across the entire unit area. Based on the literature reviewed, it appears that the frequency at which waste is to be applied is dependent on a number of variables, including waste characteristics (e.g., constituent concentrations and oil content), soil type, vegetation, and climatic conditions. The application frequencies found in literature range from yearly to an extreme of 260 times per year. U.S. EPA (1989), which cites data presented in *Land Treatment Practices in the Petroleum Industry (Environmental Research & Technology, 1983)*, reports a typical range for refineries of 2 to 52 applications per year. Martin et al. (1986) presents frequencies for 13 operating petroleum refineries that represent the geographical distribution of refineries. The reported frequencies ranged from 1 to 260 times per year, with the majority of the sites reporting monthly or yearly frequencies. The *Handbook of Land Treatment Systems for Industrial and Municipal Wastes* (Reed and Crites, 1984) reports that an application of once per week is commonly used but suggests that determination of application frequency should consider site-specific conditions. One of the site-specific variables that can impact the frequency of application is the number of months out of the year the unit is active. Martin et al. (1986) reported that the number of months facilities were actively used varied from 6 months (colder climates) to 12 months (warmer climates).

Based on the information from the literature review described above, the relationship between number of applications per year and waste quantity shown in Table 4-4 was developed.

The relationships shown in Table 4-4 were applied to the Industrial D data set for land application units. Table 4-5 summarizes the frequency of waste application estimated using the Industrial D data. As shown, approximately 86 percent of the land application units were predicted to have application frequencies 52 times or less per year and only 26 percent were estimated to have waste applied 4 times or less per year. The median number of applications per year is 16.

It was not feasible from a modeling perspective to assign different frequencies of application to each unit. Also, the use of monthly meteorological data suggested an application frequency that could be expressed as an integer on an applications per month basis. Therefore, an application frequency of 24 applications per year (2 applications per month) was selected as best representing the data available within these modeling constraints.

Table 4-4. Relationship Between Frequency of Application and Waste Quantity

Annual Waste Quantity at Unit (Mg/yr)	Number of applications/year
$\leq 1,500$	Annual waste quantity at unit/15
$\geq 1,500, < 15,000$	Annual waste quantity at unit/150
$\geq 15,000, < 150,000$	Annual waste quantity at unit/1,500
$\geq 150,000$	Annual waste quantity at unit/15,000

Table 4-5. Estimated Frequency of Application Using Industrial D Data

Frequency of Waste Application	Total Number of Land Application Units	Percentage of Total Land Application Units
$\leq 1/\text{yr}$	49	14
$\leq 4/\text{yr}$	90	26
$\leq 12/\text{yr}$	133	39
$\leq 16/\text{yr}$	176	51
$\leq 24/\text{yr}$	217	62
$\leq 52/\text{yr}$	295	86
$\leq 100/\text{yr}$	341	99
$\leq 250/\text{yr}$	345	100

The CHEMDAT8 model was run for each monthly meteorological condition using half-month (15.2 days) “time of calculation.”

The fraction emitted and the fraction biodegraded for each of the monthly meteorological conditions, as calculated by CHEMDAT8, were stored and used to project the disappearance and accumulation of contaminant due to sequential applications. Given the initial concentration just after a waste application, the ending concentration (just prior to the next application) is calculated as follows:

$$C_{end,n} = C_{start,n} \times (1 - f_{emit} - f_{bio}) \quad (4-17)$$

where

$C_{end,n}$	=	ending soil concentration after nth application (mg/kg)
$C_{start,n}$	=	initial soil concentration after nth application (mg/kg)
f_{emit}	=	fraction emitted (unitless)
f_{bio}	=	fraction degraded (unitless).

It is assumed that the volume of the land application unit remains constant. Therefore, as more waste is applied, it is assumed that an equal volume of waste/soil mixture becomes buried or otherwise removed from the active tilling depth. Including the contaminant loss by burial/removal, the initial soil concentration for any given application number is calculated as follows:

$$C_{start,n} = C_{end, n-1} \times \left(1 - \frac{L}{BD}\right) + C_{waste} \times \frac{L}{BD} \quad (4-18)$$

where

$C_{start,n}$	=	initial soil concentration after nth application (mg/kg)
$C_{end,n-1}$	=	ending soil concentration after nth-1 application (mg/kg)
L	=	loading rate (g/cm ³)
BD	=	bulk density (g/cm ³)
C_{waste}	=	waste concentration (mg/kg).

Assuming no background contaminant concentration, $C_{end,0} = 0$ mg/kg. Equations 4-17 and 4-18 were solved sequentially using the fraction emitted and fraction degraded for Month 1 (January) twice (once for each 15.2-day interval in Month 1), then twice using the fraction emitted and fraction degraded calculated for Month 2, etc., until semimonthly concentrations had been calculated for 40 years (the assumed life of the unit). For the 40th year, the starting and ending concentrations of the month were used to calculate a log-mean average soil concentration for each month. These 12 monthly soil concentrations were then arithmetically averaged to yield an average long-term concentration, which was then output for each of the 40 years modeled.

Long-term emission rates were calculated by multiplying the initial soil concentration for each application rate by the fraction emitted, the bulk density, the tilling depth, and then by dividing by time period of the calculation as follows:

$$E_n = \frac{C_{start, n} \times f_{emit} \times BD \times D_{till}}{t \times 86,400} \quad (4-19)$$

where

E_n	=	emission rate over n^{th} application period ($\text{g}/\text{m}^2\text{-s}$)
$C_{start, n}$	=	initial soil concentration after n^{th} application (mg/kg)
f_{emit}	=	fraction emitted (unitless)
BD	=	bulk density ($\text{g}/\text{cm}^3 = \text{Mg}/\text{m}^3$)
D_{till}	=	tilling depth (m)
t	=	time period of calculation (d)
86,400	=	unit conversion factor (s/d).

The long-term emission rates were calculated for all 24 waste applications during the 40th year, then the arithmetic average of these emission rates was calculated and output.

The input parameters required for the land application unit are presented in Table 4-1. The annual waste quantity and unit dimensions are the critical source parameters. The area of the land application unit is a site-specific input parameter. The tilling depth was assumed to be 20 cm unless the annual quantity of waste added to the land application unit was greater than the volume of the land application unit with a set depth of 20 cm. If the annual waste quantity exceeded the volume of the land application unit given a depth of 20 cm, the depth of the land application unit was calculated so that the land application unit could hold the entire annual waste quantity. Twice-monthly waste applications were employed (time of calculation, Input ID No. L8, was set to 15.2 days).

A waste density of $1.1 \text{ g}/\text{cm}^3$ was used to test the unit size and to calculate the tilling depth, if needed. This waste density was used for the land application unit to be consistent with the waste densities used in the analysis of the Industrial D Screening survey data. Air porosity has the greatest influence on the predicted emissions of these parameters. Nonetheless, because these parameters typically do not vary over wide ranges, they are considered secondary parameters for the emission estimates.

Because biodegradation both lowers the emission rate and the average soil concentration, the biodegradation flag is an important input parameter, and the biodegradation rate constants used in the model are also critical parameters. The biodegradation flag was set to 1 for all LAU unit runs; however, the default CHEMDAT8 model method of calculating the biodegradation rate was not used. Instead, values for first-order biodegradation rate constants were obtained from Howard et al. (1991). These biodegradation rate constants are based, for the most part, on contaminant half-lives in soil. The biodegradation rates used in CHEMDAT8 were derived

primarily from wastewater studies, and then a low biomass concentration was used in CHEMDAT8 to model biodegradation in soils. Because the Howard et al. biodegradation rate constants were more directly linked to soil-based WMUs, they were used for all land-based WMUs that included biodegradation.

The Taylor series approximation used to estimate emissions in CHEMDAT8 is inaccurate for chemicals of very low volatility and low biodegradability. For a few chemicals with the lowest Henry's law constants, this resulted in chronic emissions greater than acute emissions (which are instantaneous rates based on an exact solution rather than the Taylor series approximation). For chemicals with very low volatility and biodegradability, the instantaneous emission rates are not expected to change much over time. Therefore, for these chemicals, the acute (instantaneous) emission rates and soil concentrations were also used for chronic exposure estimates.

4.5.2 Acute and Subchronic Exposure Analysis

Acute (1-day) and subchronic (1-month) emission estimates were made for LAUs. These emission estimates used the same general modeling approach as used for the chronic approach; however, the emission estimates were made for the first 24 hours immediately after a waste application (during the 40th year, to ensure steady-state had been reached) or averaged over 30 days. As with the chronic exposure emission estimates, biodegradation was included in the model.

The acute emission estimates were projected from the CHEMDAT8 "instantaneous" emission values by weighting the instantaneous emission rate with a corresponding time interval (see box). The "instantaneous" emission rates were then normalized by the initial concentration used for the calculation, and normalized emission rates were output for each of the 12 monthly meteorological conditions. The appropriate normalized emission rate was then multiplied by the corresponding initial soil concentration for each of the applications occurring during the 40th year. The maximum (1-day) emission rate and the maximum initial concentration occurring during the 40th year were output.

Time at Which Instantaneous Emissions were Calculated	Period of Time Represented by Instantaneous Emissions
0.25 h	0 - 0.5 h
1 h	0.5 - 2 h
4 h	2 - 6 h
10 h	6 - 14 h
18 h	14 - 24 h

The subchronic emission rates used the same emission fractions as calculated for the chronic exposure. Average monthly emissions were estimated using the paired application emission rates for January, February, and so on. The maximum monthly emission rate and the maximum average monthly concentration occurring during the 40th year were output.

Acute and subchronic emission estimates were made for both unit and pure component concentrations. The Taylor series approximation used to estimate short-term (i.e., subchronic) emissions in CHEMDAT8 is inaccurate for chemicals of very low volatility and low biodegradability. For six chemicals with the lowest Henry's law constants, this resulted in subchronic emissions greater than acute emissions (which are instantaneous rates based on an exact solution rather than the Taylor series approximation). For chemicals with very low volatility and biodegradability, the instantaneous emission rates are not expected to change much over time. Therefore, for these six chemicals, the acute (instantaneous) emission rates and soil concentrations were also used for subchronic exposure estimates.

4.6 Development of Volatile Emissions and Waste Concentrations for Wastepiles

4.6.1 Chronic Exposure Analysis

The same basic CHEMDAT8 model that was used for landfills and land application units was also used for wastepiles. Similar to landfills, waste is applied, and the basic modeling equations used for landfills apply. However, subsequent calculations of the wastepile results are required to account for monthly variations, similar to the land application units. The basic modeling assumptions used for modeling land application units are as follows:

- # The wastepile operates with a fixed volume; depths (heights) ranging from 1 to 10 meters were assigned based on waste quantity and surface area; modeling was conducted for various wastepile height/surface area strata combinations (see Section 3.2).
- # The waste is homogeneous with an initial concentration of 1 mg/kg.
- # The waste matrix may be aqueous (Henry's law partitioning applies) or organic (Raoult's law partitioning applies).
- # Monthly average temperatures are used; temperature determined by assigned meteorological station.
- # Biodegradation occurs at temperatures greater than 5 °C.

The results presented in Volumes I and III are based on the aqueous-phase emission rates (i.e., assuming a concentration of 1 mg/kg and Henry's law partitioning). Most of the waste streams managed in land-based units are expected to contain constituents in the aqueous, rather than the organic, phase; therefore, this is the most realistic scenario. However, results based on organic phase emissions are of interest in two circumstances:

- # Most chemicals are better able to volatilize from an aqueous medium than from an organic medium; therefore, for most chemicals, the aqueous-phase emission rates are considerably higher than organic-phase emission rates. However, for a few chemicals (most notably formaldehyde), the organic-phase emissions are higher

than the aqueous-phase emissions. When this is the case, the results based on aqueous-phase emissions are footnoted to indicate that organic-phase emissions would be higher, so a concentration based on organic-phase emissions would be lower. This does not invalidate the aqueous-phase results, as that is still the most likely waste matrix.

- # Some of the backcalculated waste concentrations based on aqueous-phase emissions exceed the soil saturation concentration at a neutral pH and temperature of 20 to 25 °C. This is the maximum possible aqueous-phase concentration in soil; once this is exceeded, free (organic-phase) product will occur in the soil. This soil saturation concentration has been estimated for each chemical in the analysis, but this is a somewhat site- and waste-specific value because it depends on solubility, soil properties such as bulk density and porosity, and temperature (see Section 7.10.2 for the equation used to calculate the soil saturation concentration). Therefore, a backcalculated concentration may exceed it in some situations but not in others. When the backcalculated concentration based on aqueous-phase emissions exceeded the calculated soil saturation concentration at a neutral pH and temperature of 20 to 25 °C, the result was footnoted to indicate whether pure component (i.e., a concentration of 10⁶ mg/kg) would result in a risk exceeding the target risk when modeled using organic phase assumptions and Raoult's law.

Inputs that were calculated for the wastepile were calculated as follows:

- # The total annual waste quantities and surface areas for all wastepiles at each facility, as reported in the Industrial D Screening Survey, were divided by the number of wastepiles at the facility to get wastepile-unit-specific estimates.
- # Loading = waste bulk density = 1.1 g/cm³.
- # Biodegradation is assumed to occur if temperature is greater than 5 °C. If the temperature is 5 °C or lower, biodegradation is turned off.
- # Time of calculation = average residence time of waste in the wastepile, t_{ave} , as follows:

$$t_{ave} = \frac{A \times D_{till} \times BD \times 365.25}{Q_{annual}} \quad (4-20)$$

where

t_{ave}	=	average residence time of waste in the wastepile (d)
A	=	wastepile areas (m ²)
D_{till}	=	tilling depth (m)
BD	=	bulk density (g/cm ³ = Mg/m ³)
365.25	=	unit conversion factor (d/yr)
Q_{annual}	=	annual waste quantity (Mg/yr).

- # The same monthly temperature and windspeeds calculated for land application units were used for wastepiles.

The average waste concentration and emission rate for wastepiles can be calculated using the equations presented for the landfill model (Equations 4-12 and 4-15).

The same input parameters required for land application units and landfills were used for wastepiles (see Table 4-1). The annual waste quantity and unit dimensions are still the critical source parameters. As with landfills, the loading for wastepiles is pure waste material so that the loading is basically the waste density. A waste density of 1.1 g/cm³ was used for the wastepile to be consistent with the waste densities used in the analysis of the Industrial D Screening survey data. Wastepile emission (and concentration) estimates were made for wastepiles representing various height/surface area combinations (see Section 3.2). The annual waste quantity combined with the capacity (dimensions) of the wastepile were used to calculate residence time for the waste in the wastepile (i.e., time of calculation - Input ID No. L8).

Temperature and porosities have some impact on emissions. Biodegradation was assumed to occur for wastepiles. The model is insensitive to molecular weight of the waste (for aqueous wastes) and windspeed (for long-term emission estimates).

4.6.2 Acute and Subchronic Exposure Analysis

Acute (1-day) and subchronic (1-month) emission estimates were made for wastepiles following the same approach used for LAUs as described in Section 4.5.2, with the following differences:

- # The wastepile was assumed to be completely filled with new waste at time $t = 0$.
- # The time of calculation used for subchronic exposure was 1 month (365/12 days).
- # Biodegradation was **not** included (a lag period is generally associated with biodegradation, especially for the assumed "all new waste" scenario. This is because wastepiles are not designed for biodegradation, so it takes time for enough acclimated biomass to accumulate for biodegradation to occur. This time is typically several months, longer than the 1-day or 30-day periods modeled for acute and subchronic).
- # Since there was no biodegradation, only maximum-temperature runs were needed, which maximize volatile emissions.

4.7 Development of Volatile Emissions for Tanks

The basic modeling assumptions used (or inherent in CHEMDAT8) for the tank model emission estimates include:

- # The WMU operates at steady state.

- # The WMU is well mixed.
- # Emission estimates were performed for three different influent concentrations: an influent concentration of either 0.001 (for aerated tanks) or 1 mg/L (for nonaerated and storage tanks; the low concentration modeled was set to a different, lower, value for aerated tanks to ensure first-order biodegradation), an influent concentration equal to the constituent's solubility (to estimate aqueous-phase emissions with zero-order biodegradation), and an influent concentration of 1E+6 mg/L (pure component).
- # The waste matrix may be aqueous (Henry's law partitioning applies) or organic (Raoult's law partitioning applies).
- # Annual average temperatures are used; temperature is determined by assigned meteorological stations.
- # Biodegradation rate is first order with respect to biomass concentrations. (Only some aerated tanks were modeled with biodegradation.)
- # Biodegradation rate follows Monod kinetics with respect to contaminant concentrations.
- # Hydrolysis rate is first order with respect to contaminant concentrations.
- # Acute and subchronic exposures were not modeled.

For tanks with biodegradation (some aerated tanks), biodegradation shifts from first order at low concentrations to zero order at high concentrations. To account for this, two runs were done using aqueous phase waste: one at 0.001 mg/L (a concentration low enough to ensure first-order biodegradation for all chemicals) and one at the chemical's solubility (the maximum possible for aqueous phase, which will produce zero-order biodegradation for all chemicals that shift to zero-order at concentrations below solubility). Backcalculated concentrations were initially calculated using the first-order biodegradation emissions. If this concentration exceeded the half-saturation concentration for the chemical, suggesting that zero-order biodegradation would be more appropriate, it was replaced with a backcalculated concentration based on the zero-order emission rate.

For tanks, the volume and flow were taken directly from the TSDR Survey. Other unit design parameters, such as surface area and depth, were imputed from the survey data. Modeling was conducted for both treatment (aerated and nonaerated) and storage tanks.

The CHEMDAT8 model is used to calculate the emission fractions for each tank. The emission rate, in $\text{g}/\text{m}^2\text{-s}$, is calculated from the fraction emitted, the flow rate, waste concentration, and the surface area as follows:

$$E = \frac{Q_{flow} \times C_{infl} \times F_{emit}}{A} \quad (4-21)$$

where

E	=	emission rate (g/m ² -s)
Q _{flow}	=	flow rate (m ³ /s)
C _{infl}	=	waste concentration in influent (mg/L = g/m ³)
F _{emit}	=	fraction emitted (unitless)
A	=	tank surface area (m ²).

4.8 Development of Particulate Emissions

Two different models were used to model wind erosion – one for wastepiles (elevated sources) and one for landfills and land application units (ground-level sources). The Cowherd model (U.S. EPA, 1985b, 1988) was selected for modeling wind erosion emissions from ground-level sources, and the AP-42 model for wind erosion from aggregate storage piles (U.S. EPA, 1985a) was selected for modeling wind erosion emissions from wastepiles.

For both types of WMU, the models described in this section predict the emission rate of particulate matter released from a site due to wind erosion. To obtain the emission rate of constituent sorbed to particulate matter, the emission rate of particulate matter must be multiplied by the soil or waste concentration. That calculation is described in Section 7.4.

4.8.1 Landfills and Land Application Units

Wind erosion emissions from landfills and LAUs were modeled using the Cowherd model (U.S. EPA, 1985b). A newer version of Cowherd's model is available in U.S. EPA (1988). However, the newer version is an event-based model that requires detailed site-specific information unavailable for this analysis. Therefore, it was not used. The older Cowherd model tends to slightly overestimate emissions relative to the event-based version. Although the degree to which it overestimates is not known, it is expected to be relatively small. Because particulate emissions are negligible compared to volatile emissions for the volatile chemicals modeled, this is only of concern for the metals (other than mercury), which are based only on particulate emissions.

The Cowherd model estimates the emission of respirable particles (i.e., PM₁₀) due to wind erosion from a ground-level surface with an unlimited reservoir of erodible particles. Surfaces are defined as having a limited or unlimited reservoir based on threshold friction velocity (U*); surfaces with a U* greater than 0.5 m/s are considered limited, while those with U* less than 0.5 m/s are considered unlimited (U.S. EPA, 1988). Threshold friction velocity is a measure of the windspeed at the ground surface that would be required to remove particles from the surface. Examples of limited reservoirs would include nonhomogeneous surfaces with stones, clumps of

vegetation, or other nonerodible elements or crusted surfaces. Further, wind erosion is considered unlikely to occur from surfaces with full vegetative cover.

Wind erosion emissions were calculated as follows (U.S. EPA, 1985b):

$$E_{10} = 0.036 \times (1-V) \times \left(\frac{[u]}{U_t} \right)^3 \times F(x) \quad (4-22)$$

where

- E_{10} = emission rate of PM_{10} (g/m^2-h)
- V = vegetative cover (fraction)
- $[u]$ = average annual windspeed (m/s)
- U_t = threshold windspeed (m/s).

$F(x)$ is a complex function of x , which is a ratio of threshold windspeed and average annual windspeed. The following is a linear approximation of $F(x)$ from a graph of $F(x)$ in U.S. EPA (1985b, Figure 4-3), except for the equation for values of x greater than 2, which is given in Appendix B of U.S. EPA (1985b).

$$F(x) = \begin{cases} 1.91 & x \leq 0.5 \\ 2.06 - 0.33x & 0.5 < x < 0.8 \\ 2.6 - x & 0.8 < x < 1 \\ 2.9 - 1.3x & 1 < x < 2 \\ 0.18(8x^3 + 12x)e^{-x^2} & x \geq 2 \end{cases} \quad (4-23)$$

$$x = 0.886 \times \frac{U_t}{[u]} \quad (4-24)$$

The threshold windspeed, U_t , is a function of threshold friction velocity and roughness height, as follows:

$$U_t = \frac{1}{0.4} \times \ln \left(\frac{Z}{z_0} \right) \times U^* \quad (4-25)$$

where

- U_t = threshold windspeed (m/s)
- Z = anemometer height (cm)
- z_0 = roughness height (cm)
- U^* = threshold friction velocity (m/s).

The inputs used for these equations (other than average annual windspeed and anemometer height, which are location-specific determined by the meteorological station) are summarized in Table 4-6.

The vegetative cover, V , is an important modifying factor that can range from 0 (bare ground) to 1 (100 percent vegetative cover). Because this analysis focuses on active industrial landfills and land application units, the assumption of no vegetative cover was applied (i.e., vegetative cover was set to 0). For active industrial landfills, it was assumed that the constant filling and dumping taking place would not allow for vegetative growth to occur. Active land application units were assumed to be tilled and thus were also assumed to have no vegetative cover.

The roughness height, z_0 , accounts for the size and spacing of surface roughness elements (which may include vegetation, or even buildings). U.S. EPA (1985b) provides a range of roughness heights for various surfaces ranging from 0.1 cm (natural snow) to 1,000 cm (high-rise buildings). A value of 1 cm, corresponding to a plowed field, was selected as most representative of the conditions expected to occur in an active landfill or LAU.

The threshold friction velocity, U^* , is a function of the physical condition of the soil and would normally be considered a site-specific parameter. In the absence of data, a value of 0.5 m/s was selected based on the assumption that the WMU is an unlimited reservoir.

The following inputs and intermediate values are location-specific: anemometer height, threshold windspeed, average annual windspeed, x , and $F(x)$. These are shown in Table 4-7 along with the calculated particulate emissions rate for each meteorological location used in the analysis. The anemometer heights and average annual windspeed values are from NOAA (1992). Values for U_t , x , $F(x)$, and E_{10} have been rounded to two significant figures.

4.8.2 Wastepiles

Wind erosion emissions from wastepiles were modeled using an equation from AP-42 (U.S. EPA, 1985a) for estimating emissions from wind erosion from active storage piles. The equation gives emissions of total suspended particulates. Typically, an equation-specific particle size multiplier would be applied to reduce the emissions to a desired size category, in this case, PM_{10} . No particle size multipliers are given for this equation in AP-42; however, Cowherd (U.S. EPA, 1988) gives a PM_{10} particle size multiplier of 0.5, which was used with this equation.

Table 4-6. Inputs and Intermediate Values Used for Wind Erosion from Landfills and LAUs

Symbol	Parameter	Units	Value	Source
V	Vegetative cover	fraction	0	Assumption
z_0	Roughness height	cm	1	U.S. EPA (1985b)
U^*	Threshold friction velocity	m/s	0.5	Assumption

Table 4-7. Calculated Particulate Emission Rates for Landfills and LAUs

Location	Z (cm)	U _t (m/s)	[u] (m/s)	x (unitless)	F(x) (unitless)	E ₁₀ (g/m ² -h)
Albuquerque	700	8.2	4.1	1.8	0.06	0.0027
Atlanta	610	8.0	4.6	1.5	0.89	0.0061
Bismarck	610	8.0	6.2	1.1	1.4	0.023
Boise	610	8.0	4.6	1.5	0.89	0.0061
Casper	610	8.6	7.2	1.1	1.5	0.032
Charleston	610	8.0	4.1	1.7	0.65	0.0031
Chicago	610	8.0	4.6	1.5	0.89	0.0061
Cleveland	1010	8.6	5.1	1.5	0.95	0.0070
Denver	1010	8.6	4.1	1.9	0.47	0.0018
Fresno	610	8.0	3.6	2.0	0.34	0.0011
Harrisburg	610	8.1	4.6	1.6	0.86	0.0056
Hartford	1010	8.6	4.1	1.9	0.47	0.0018
Houston	610	8.0	4.1	1.7	0.65	0.0031
Huntington	610	8.0	3.6	2.0	0.34	0.0011
Las Vegas	610	8.0	5.1	1.4	1.1	0.010
Lincoln	610	8.0	5.1	1.4	1.1	0.010
Little Rock	610	8.0	3.6	2.0	0.34	0.0011
Los Angeles	910	8.5	4.1	1.8	0.51	0.0020
Miami	700	8.2	4.6	1.6	0.85	0.0054
Minneapolis	1010	8.6	5.7	1.3	1.2	0.012
Philadelphia	610	8.0	4.6	1.5	0.89	0.0061
Phoenix	1010	8.6	3.1	2.5	0.060	0.00010
Portland, ME	610	8.0	4.6	1.5	0.89	0.0061
Raleigh-Durham	610	8.0	4.1	1.7	0.65	0.0031
Salem, OR	610	8.0	4.6	1.5	0.89	0.0061
Salt Lake City	610	8.0	4.6	1.5	0.89	0.0061
San Francisco	1010	8.6	6.2	1.2	1.3	0.017
Seattle	610	8.0	5.1	1.4	1.1	0.010
Winnemucca	1010	8.6	4.1	1.9	0.47	0.0018

Emissions were calculated as follows:

$$E_{10} = 1.9 \times PSM \times \frac{S}{1.5} \times \frac{365-p}{235} \times \frac{f}{15} \quad (4-26)$$

where

- E₁₀ = emission rate of PM₁₀ (kg/ha-d)
- PSM = particle size multiplier for PM₁₀ (unitless) = 0.5
- S = silt content of waste (%)
- p = number of days with ≥0.25 mm (0.01 in) of precipitation per year (d/yr)
- f = percentage of time that the unobstructed windspeed exceeds 5.4 m/s (12 mph) (%).

The emission rate was converted to g/m²-h as follows:

$$E' = \frac{E \times 10^3}{10^4 \times 24} \quad (4-27)$$

where

E'	=	emission rate (g/m ² -s)
E	=	emission rate (kg/ha-d)
10 ³	=	unit conversion factor (g/kg)
10 ⁴	=	unit conversion factor (m ² /ha)
24	=	unit conversion factor (h/d).

Silt content varies depending on the waste stored in the wastepile. AP-42 contains a table of typical silt and moisture contents of materials at various industries. Based on these data, a default value of 12 percent was used. This value was identified in the AP-42 table as the mean silt content for miscellaneous fill materials.

The number of precipitation days and the frequency of windspeed greater than 5.4 m/s were location specific. These data were collected at the meteorological stations and not at the waste management unit; values were obtained from NOAA (1992) and are summarized in Table 4-8 for each of the 29 meteorological locations modeled. Also included in Table 4-8 are the particulate emission rates in both kg/ha-d and g/m²-h that were calculated using Equations 4-26 and 4-27; these values have been rounded to two significant figures. The particle size multiplier value of 0.5 used in calculating these estimates was chosen based on 10- μ m particles and was taken directly from U.S. EPA (1988).

Table 4-8. Calculated Particulate Emission Rates for Wastepiles

Location	p (d/yr)	f (%)	E (kg/ha-d)	E (g/m ² -h)
Albuquerque	58	22.0	15	0.061
Atlanta	116	21.0	11	0.047
Bismarck	96	33.2	19	0.080
Boise	91	21.0	12	0.052
Casper	95	47.3	28	0.11
Charleston	113	19.1	10	0.043
Chicago	125	31.7	16	0.068
Cleveland	157	33.6	15	0.063
Denver	89	20.9	12	0.052
Fresno	89	7.4	4.4	0.018
Harrisburg	125	16.6	8.6	0.036
Hartford	126	21.9	11	0.047
Houston	101	16.3	9.3	0.039

(continued)

Table 4-8. (continued)

Location	p (d/yr)	f (%)	E (kg/ha-d)	E (g/m²-h)
Huntington	142	8.2	3.9	0.016
Las Vegas	27	25.9	19	0.079
Lincoln	91	31.1	18	0.077
Little Rock	104	14.4	8.1	0.034
Los Angeles	33	14.7	11	0.044
Miami	128	25.5	13	0.054
Minneapolis	113	35.2	19	0.080
Philadelphia	117	25.6	14	0.057
Phoenix	37	6.8	4.8	0.020
Portland, ME	129	23.0	12	0.049
Raleigh-Durham	110	14.5	8.0	0.033
Salem, OR	146	15.2	7.2	0.030
Salt Lake City	92	20.1	12	0.049
San Francisco	63	37.4	24	0.10
Seattle	157	22.1	9.9	0.041
Winnemucca	67	17.2	11	0.046

5.0 Dispersion Modeling

Dispersion describes the transport of chemical emissions through the air to a receptor. In this risk analysis, dispersion modeling was used to estimate air concentrations associated with a unit emission ($1 \mu\text{g}/\text{m}^2\text{-s}$) (unitized air concentrations, or UACs) at a variety of potential receptor locations. The following sections discuss overview of the approach, model selection, the critical parameters of the model, and the model results or UACs.

5.1 Overview of Approach

A dispersion model (ISCST3) was run to calculate air concentrations associated with a standardized unit emission rate ($1 \mu\text{g}/\text{m}^2\text{-s}$) to obtain a unitized air concentration (UAC), also called a dispersion factor, which is measured in $\mu\text{g}/\text{m}^3$ per $\mu\text{g}/\text{m}^2\text{-s}$. Total air concentration estimates are then developed by multiplying the constituent-specific emission rates derived from CHEMDAT8 with this dispersion factor.

Running ISCST3 to develop a dispersion factor for each of the approximately 3,400 individual WMUs modeled in this study would have been very time consuming due to the run time of the area source algorithm in ISCST3. In addition, modeling for many different locations requires extensive preprocessing to generate the detailed meteorological data needed for each location modeled. Therefore, a database of dispersion factors was developed by running ISCST3 for many separate scenarios designed to cover a broad range of unit characteristics, including:

- # both ground-level and elevated sources;
- # 14 surface area sizes for landfills and land application units, 29 surface area-height combinations for waste piles, and 33 surface area height combinations for tanks;
- # 6 receptor distances from the unit (25, 50, 75, 150, 500, and 1,000 meters) placed in 16 directions in relation to the edge of the unit.

Based on the size and location of a specific unit, an appropriate dispersion factor was interpolated from the database of dispersion factors using the closest location and the two closest unit sizes.

In addition, WMUs were assigned to and dispersion modeling was performed for 29 meteorological stations. These were chosen from the more than 200 available to represent the nine general climate regions of the continental U.S.

Each UAC in the database is specific to one meteorological station, one area-height combination, one distance from the unit, and one direction from the unit.

5.2 Model Selection

A number of dispersion models are available on the EPA Support Center for Regulatory Air Models (SCRAM) Bulletin Board (<http://www.epa.gov/scram001/>). These dispersion models were developed for a variety of applications and each has its own strengths and weaknesses. This analysis required a model with the capability to model the dispersion of vapors and particulates from landfills, land application units, wastepiles, and tanks to off-site receptors for chronic, subchronic, and acute averaging times. Therefore, a dispersion model was needed that could model (1) area sources; (2) ground-level and elevated sources; (3) off-site impacts; (4) vapors and particulates; and (5) annual, monthly, and daily averaging times.

Five models were considered for this analysis:

- # Industrial Source Complex - Short Term v.3 (ISCST3) - U.S. EPA, 1995a
- # Industrial Source Complex - Long Term v.3 (ISCLT3) - U.S. EPA, 1995a
- # Toxic Modeling System - Short Term (TOXST) - U.S. EPA, 1994c
- # Fugitive Dust Model (FDM) - U.S. EPA, 1992
- # COMPDEP - U.S. EPA, 1990.

Table 5-1 summarizes the capabilities of these commonly used air dispersion models with respect to the requirements of this analysis. The ISCST3 (U.S. EPA, 1995a) was selected for all aspects of this analysis because it met all the criteria. This model, however, requires considerable run time, which limited the number of meteorological stations included in this analysis.

5.3 Critical Parameters

This section discusses the critical parameters of the selected model, ISCST3, and the results of sensitivity analyses performed to investigate several of the model parameters. Results of the sensitivity analyses are presented in Appendix C.

Table 5-1. Air Dispersion Model Capabilities

Model	Source Geometry		Receptor Location		Chemical Phase		Averaging Period		
	Area Source	Source Height Elevated Ground	Off-site Air Conc.	Vapor	Particulate	Annual	Monthly	Daily	
ISCST3	✓	✓	✓	✓	✓	✓	✓	✓	✓
ISCLT3	✓	✓	✓	✓	✓	✓	✓		
TOXST	✓	✓	✓ ^a	✓	✓	✓		✓	✓
FDM	✓	✓		✓		✓			✓
COMPDEP		✓		✓	✓	✓			✓

^aMinimum height of source for modeling is 0.5 meters.

5.3.1 General Assumptions

This section discusses depletion, rural vs. urban, and terrain assumptions.

5.3.1.1 Depletion. Air concentrations can be calculated in ISCST3 with or without wet depletion for vapors and particulates, and with or without dry depletion for particulates only (ISCST3 cannot model dry depletion of vapors.) Modeled concentrations without depletions are higher than those with depletions. The run time for calculating concentrations using the ISCST3 model with depletion options, however, is 15 to 30 times longer than the run time without depletions. Even using the database approach described in Section 5.1 rather than modeling all 3,400 WMUs separately, the additional run time needed to model depletion was considerable. To be as efficient as possible, a sensitivity analysis was conducted to determine the sensitivity of model results to depletion processes that could be modeled for vapors and particulates.

For particulates, the sensitivity analysis, which is presented in Appendix C, shows that the differences in the maximum concentrations with and without wet depletion are small at close-to-source receptors, increasing only slightly as the distance from the source increases. The sensitivity analysis, however, also shows that very large differences in concentration can occur when results with and without dry depletion are compared for particulates. (The difference is greater for larger sources; see sensitivity analysis in Appendix C for details.) Therefore, concentrations for particulates were calculated with dry depletion but without wet depletion in this analysis.

A similar sensitivity analysis for vapors showed that wet depletion of vapors is negligible (differences in concentration with and without depletion were mostly less than 1 percent). Therefore, wet depletion of vapors was not modeled. ISC cannot model dry depletion of vapors, so this was also not modeled; however, dry depletion of vapors is also expected to be negligible.

5.3.1.2 Rural vs. Urban. ISCST3 may be run in rural or urban mode, depending on land use within a 3-km radius from the source. These modes differ with respect to wind profile exponent and potential temperature gradients. Unless the site is located in a metropolitan area, the rural option is generally more appropriate. Because the types of WMUs being assessed are typically in nonurban areas, the rural option was used in this analysis.

Assumptions Made for Dispersion Modeling

- # Dry depletion was activated in the dispersion modeling for particulates. Depletion was not considered for vapors.
- # The rural option was used in the dispersion modeling since the types of WMUs being assessed are typically in nonurban areas.
- # Flat terrain was assumed.
- # An area source was modeled for all WMUs.
- # To minimize error due to site orientation, a square area source with sides parallel to X- and Y- axes was modeled.
- # Receptor points were placed on 25, 50, 75, 150, 500, and 1,000 m receptor squares starting from the edge of the source with 16 receptor points on each square.
- # Modeling was conducted using a unit emission rate of $1 \mu\text{g/s}\cdot\text{m}^2$.

5.3.1.3 Terrain. Flat terrain for both the source and the surrounding area was assumed in the modeling analysis for two reasons: (1) ISCST3 models all area sources as flat, and (2) complex terrain simulations in the surrounding area result in air concentrations that are highly dependent upon site-specific topography. A specific WMU's location in relation to a hill or valley produces results that would not be applicable to other locations. Because complex terrain applications are extremely site-specific, model calculations from one particular complex terrain location cannot be applied to another. Conversely, simulations from flat terrain produce values that are more universally applicable.

5.3.2 Meteorological Stations and Data

Meteorological data at over 200 meteorological stations in the United States are available on the SCRAM Bulletin Board (<http://www.epa.gov/scram001/>) and from a number of other sources. To develop the dispersion factor database described in Section 5.1, a set of 29 meteorological stations selected in an assessment for EPA's Superfund program Soil Screening Levels (SSLs) (EQM, 1993) as being representative of the nine general climate regions of the continental United States was used.

In EPA's SSL study, it was determined that 29 meteorological stations would be a sufficient sample to represent the population of 200 meteorological stations and predict mean dispersion values with a high (95 percent) degree of confidence. The 29 meteorological stations were distributed among nine climate regions based on meteorological representativeness and variability across each region. These climate regions were:

# North Pacific Coastal	# Northwest Mountains	# Midwest
# South Pacific Coastal	# Central Plains	# Northern Atlantic
# Southwest	# Southeast	# South Florida.

Large-scale regional average conditions were used to select the actual stations (EQM, 1993).

The 29 meteorological stations are listed in Table 5-2. To assign each Industrial D facility to a meteorological station, EPA used a geographic information system (GIS) to construct Thiessen polygons around each station that enclose the areas closest to each station. The boundaries of these areas were then adjusted to ensure that each boundary encloses an area that is most similar in meteorological conditions to those measured at the meteorological station. To assist in this process, a GIS coverage of Bailey's ecoregion divisions and provinces (Bailey et al., 1994) was used to conflate the boundaries to correspond to physiographic features likely to influence climate or boundaries corresponding to changes in temperature or precipitation. General wind regimes were also considered in the conflation process.

Key factors considered in the conflation process include: defining coastal regimes as narrow polygons, which generally stretched about 25 to 50 miles inland, to capture regions dominated by coastal climate effects; maintaining tropical/subtropical and arid/semiarid divisions in the southwestern United States; and using the ecoregion boundaries in Washington, Oregon, and California to separate the more humid marine/redwood or Mediterranean mountain regimes

Table 5-2. Meteorological Stations Used in the Air Characteristic Study

City	Met Station		Latitude		Longitude	
	State	#	Degree	Minute	Degree	Minute
Albuquerque	NM	23050	35	3	106	37
Atlanta	GA	13874	33	39	84	25
Bismarck	ND	24011	46	46	100	45
Boise	ID	24131	43	34	116	13
Casper	WY	24089	42	55	106	28
Charleston	SC	13880	32	54	80	2
Chicago	IL	94846	41	59	87	54
Cleveland	OH	14820	41	25	81	52
Denver	CO	23062	39	46	104	52
Fresno	CA	93193	36	46	119	43
Harrisburg	PA	14751	40	13	76	51
Hartford	CT	14740	41	56	72	41
Houston	TX	12960	29	58	95	21
Huntington	WV	03860	38	22	82	33
Las Vegas	NV	23169	36	5	115	10
Lincoln	NE	14939	40	51	96	45
Little Rock	AR	13963	34	44	92	14
Los Angeles	CA	23174	33	56	118	24
Miami	FL	12839	25	49	80	17
Minneapolis	MN	14922	44	53	93	13
Philadelphia	PA	13739	39	53	75	15
Phoenix	AZ	23183	33	26	112	1
Portland	ME	14764	43	39	70	19
Raleigh-Durham	NC	13722	35	52	78	47
Salem	OR	24232	44	55	123	0
Salt Lake City	UT	24127	40	47	111	57
San Francisco	CA	23234	37	37	122	23
Seattle	WA	24233	47	27	122	18
Winnemucca	NV	24128	40	54	117	48

Source; EQM (1993).

from the deserts to the east. In general, Thiessen polygons were used to define the meteorological station areas for the remainder of the country.

Based on facility locations derived from Industrial D or TSDR survey data, the WMU sites were then overlaid on the GIS coverage of the conflated meteorological boundaries and meteorological station assignments were then exported for use in the modeling exercise. Several sites in Alaska, Hawaii, and Puerto Rico were deleted from the analysis because the 29 meteorological stations are limited to the continental United States. Figures 5-1 to 5-4 show the final meteorological station boundaries used for the study along with the locations of the Industrial D or TSDR facility sites for landfills, LAUs, wastepiles, and tanks, respectively.

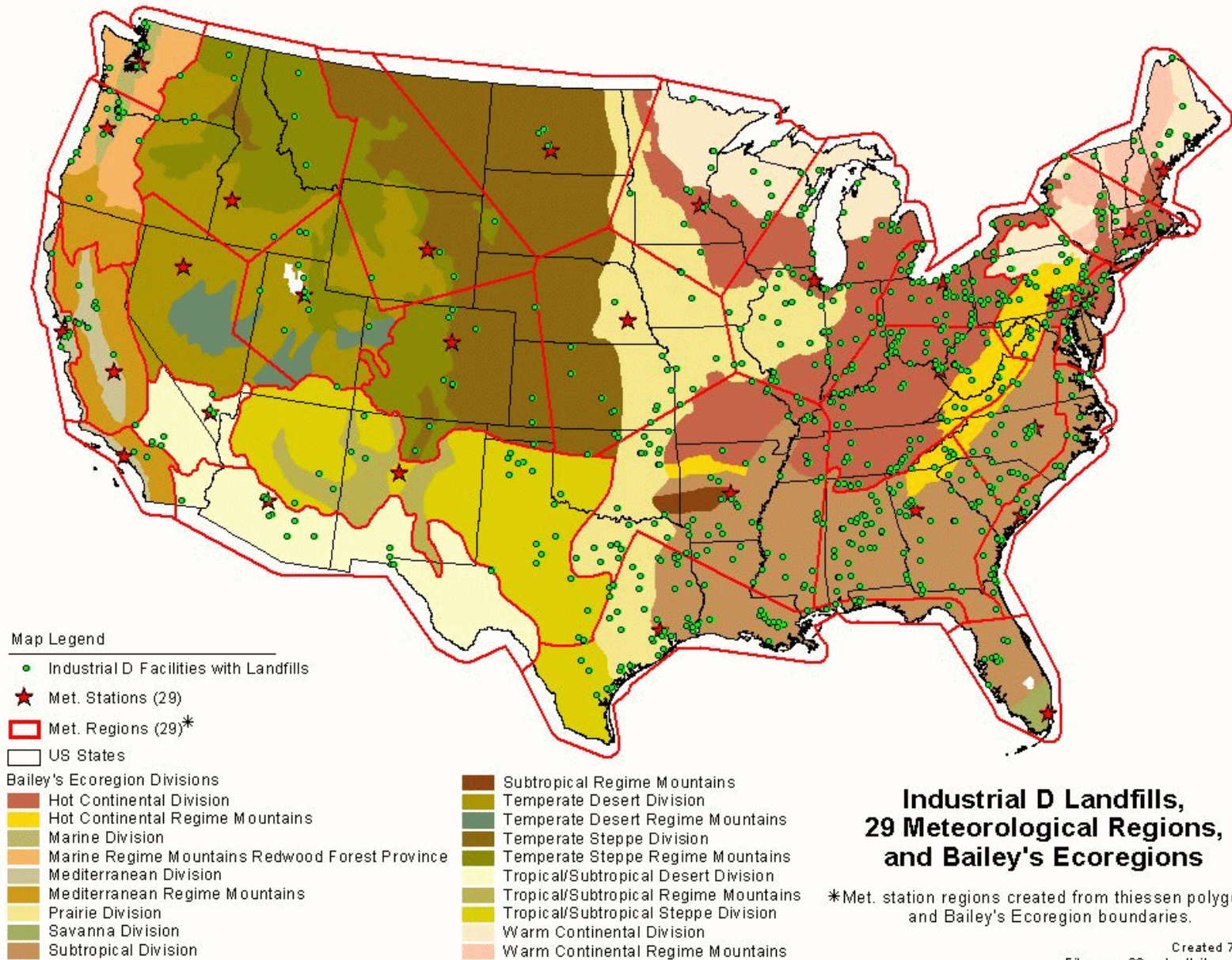


Figure 5-1. Meteorological station regions and landfill locations.

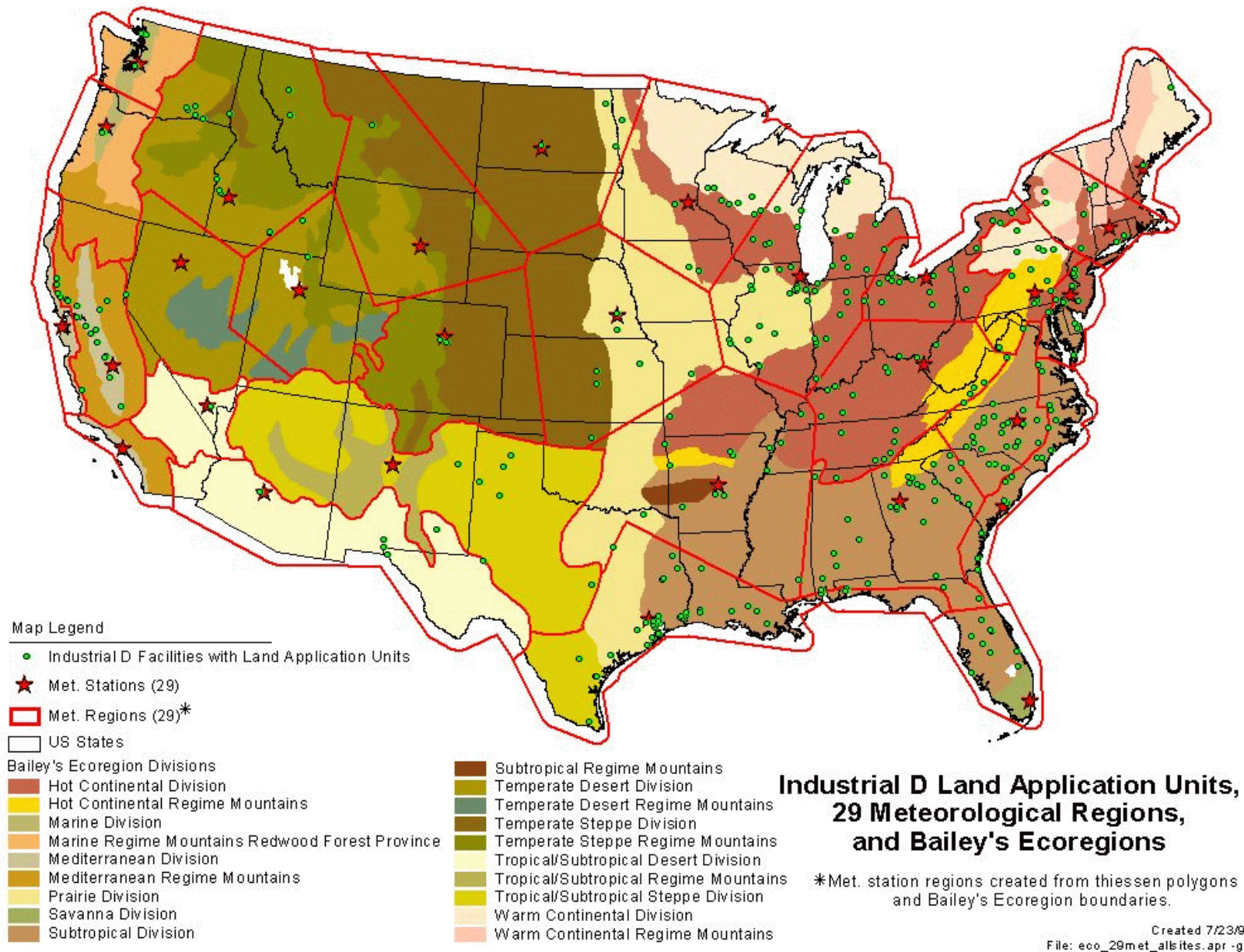


Figure 5-2. Meteorological station regions and LAU locations.

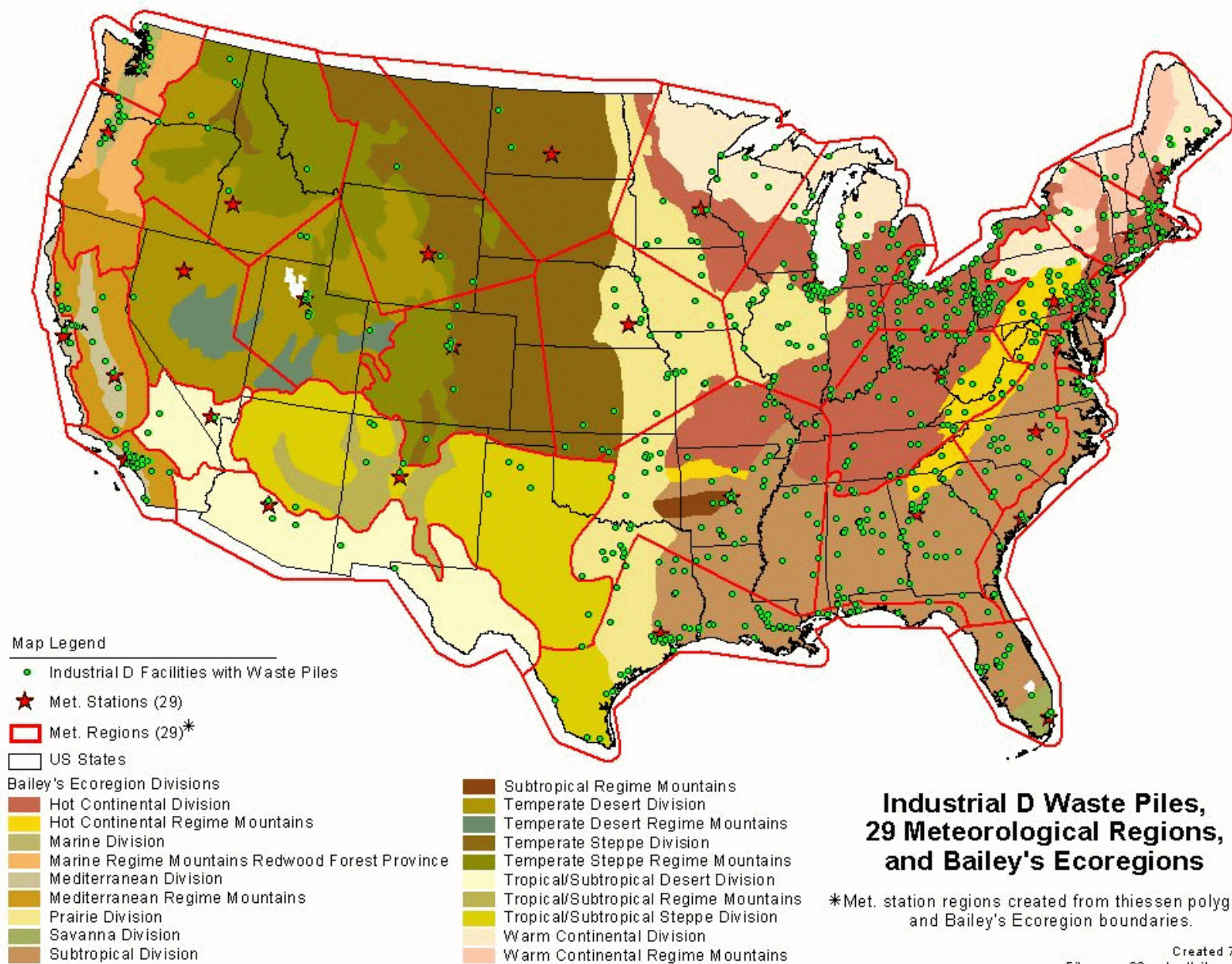


Figure 5-3. Meteorological station regions and wastepile locations.

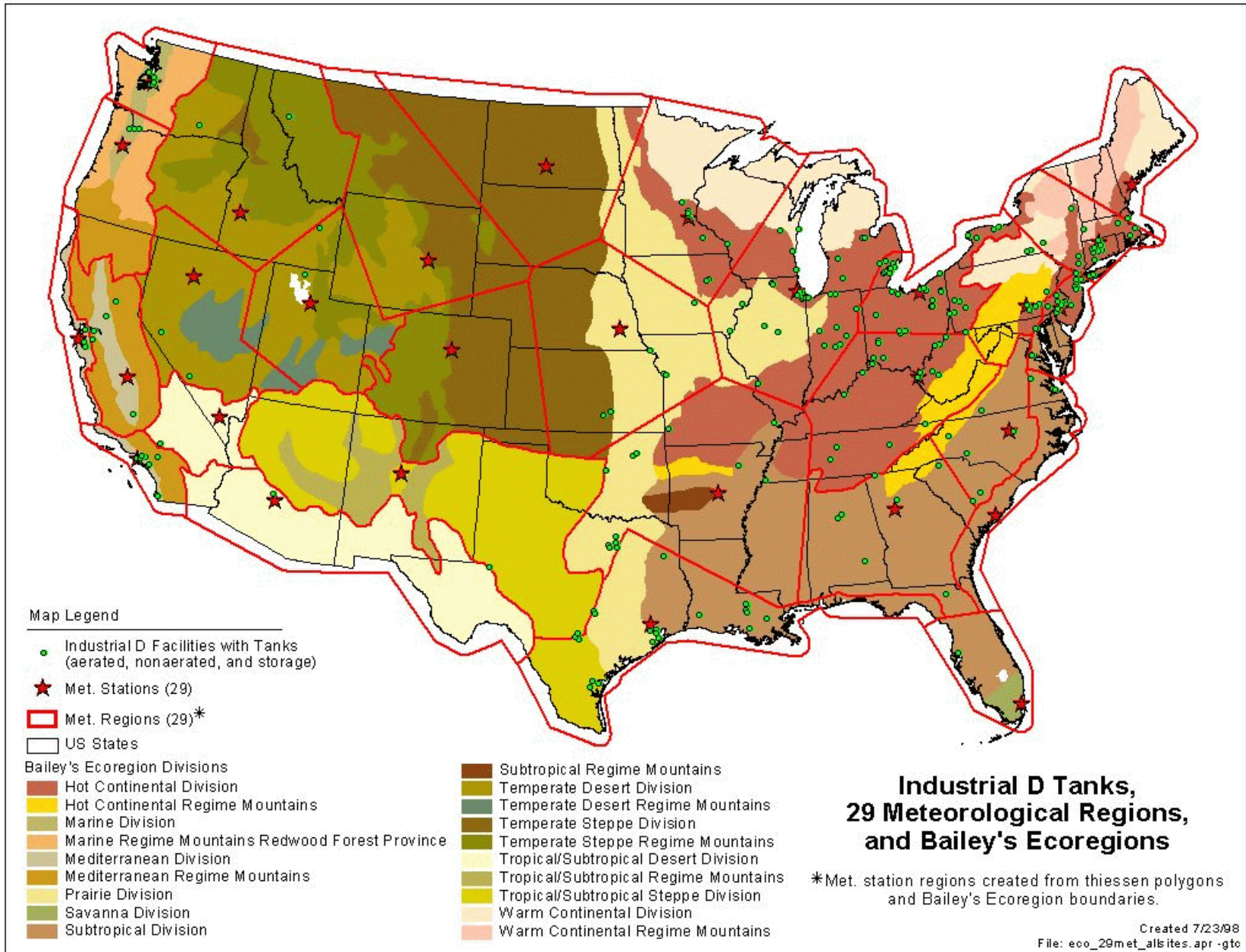


Figure 5-4. Meteorological station regions and tank locations.

The modeling analysis was conducted using 5 years of representative meteorological data from each of the 29 meteorological stations. Five-year wind roses representing the frequency of wind directions and windspeeds for the 29 meteorological stations were analyzed. These show that the 29 meteorological stations represent a variety of wind patterns and are presented in Appendix C.

Shape of Wind Rose for 29 Meteorological Stations

Shape of Wind Rose	No. of Stations
Narrowly distributed	10
Moderately distributed	4
Evenly distributed	6
Bimodally distributed	9

Meteorological Data for the ISCST3 Model without Depletion

Wind Direction (or Flow Vector)
Windspeed
Ambient Temperature
Stability Class
Mixing Height

Wind direction and windspeed are typically the most important meteorological inputs for dispersion modeling analysis. Wind direction determines the direction of the greatest impacts. Windspeed is inversely proportional to ground-level air concentrations, so that the lower the windspeed, the higher the air concentration.

Mixing height determines the heights to which pollutants can be diffused vertically. Stability class is also an important factor in determining the rate of lateral and vertical diffusion. The more unstable the air, the greater the diffusion. This increase would lower concentration at the centerline of the plume. Note that air concentration is highest at the centerline.

5.3.3 Source Release Parameters

This section describes the source parameters and assumptions used in the dispersion modeling, including source type and elevation, source shape and orientation, and source areas.

5.3.3.1 Source Type and Elevation. All WMU types modeled in this analysis were modeled as area sources. Landfills and land application units were modeled as ground-level sources, and wastepiles and tanks were modeled as elevated sources.

5.3.3.2 Source Shape and Orientation. The ISCST3 models an area source as a rectangle or combination of rectangles. The user may also specify an angle of rotation relative to a north-south orientation. A sensitivity analysis was conducted to compare the air concentrations from a square area source, a rectangular area source oriented east to west, and a rectangular area source oriented north to south to determine what role source shape and orientation play in determining dispersion coefficients of air pollutants. The results show that the differences in unitized air concentration between the square area source and the two rectangular area sources are less than the differences between the two rectangular sources. In addition, a square area source has the least amount of impact on orientation. Because information on source shapes or orientations is not available, a square source was chosen to minimize the errors caused by source shapes and orientations. (See sensitivity analysis in Appendix C for details.)

5.3.3.3 Source Areas Modeled. In the modeling analysis, four types of WMUs were considered (i.e., landfill, land application unit, wastepile, and tank). Because the ISCST3 model is sensitive to the size of the area source, the relationship between air concentrations and size of the area source was analyzed. As illustrated in Figure 5-5, the results show that, for relatively small area sources, air concentrations increase significantly as the size of the area source increases. For large area sources, this increase in air concentrations is not as significant.

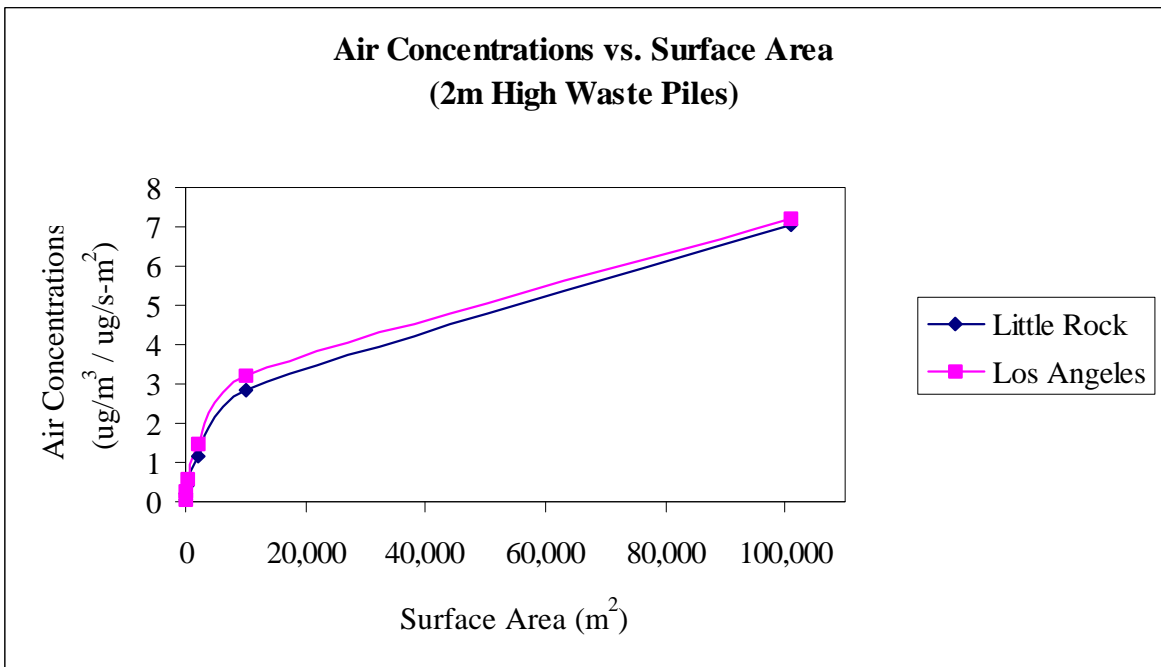
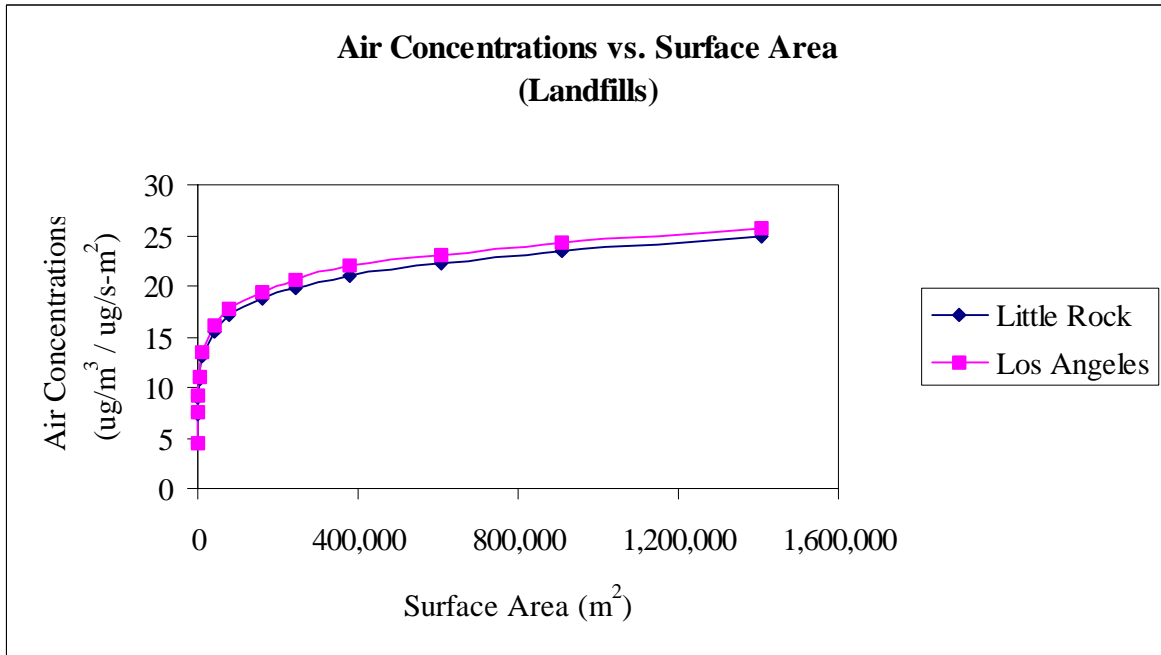
To address this model sensitivity yet avoid modeling approximately 3,400 separate WMUs, EPA developed area strata that represented the distribution of the surface area for each of the WMU types. Landfills and land application units were modeled as ground-level area sources, while wastepiles and tanks were treated as elevated area sources. Separate area strata were developed for ground-level and elevated sources. Tables 5-3, 5-4, and 5-5 present the source areas and heights used in the modeling analysis. Fourteen area strata were selected for landfills and land application units; all 14 were modeled at zero height, since landfills and LAUs are not elevated.

For wastepiles, seven areas and six heights were modeled. All wastepiles in the database were assigned one of the seven heights as described in Section 3. Each wastepile has a different area and UACs were extrapolated between areas modeled; therefore, for any given wastepile, a UAC is needed for each of the two areas bracketing the actual area, at the assigned height. An analysis of the wastepile database revealed that only 29 of the possible 42 combinations of the seven areas and six heights were needed; therefore, UACs were modeled only for those 29 area-height combinations. Tanks were modeled in the same manner as wastepiles, using 10 areas and 4 heights. Analysis of the tank databases revealed that only 33 of the possible 40 combinations of 10 areas and 4 heights were needed.

These areas or area-height combinations were modeled for each of the 29 meteorological locations. This provided a set of UACs for use in the analysis. For any specific WMU, a UAC was then estimated using an interpolation routine that used the UACs associated with modeled areas immediately above and below the actual area of the unit. The interpolation routine provides a technique for minimizing the number of ISCST3 runs required for a WMU while also minimizing the error associated with the difference between the UACs for preselected areas and the UAC for the actual area of the WMU. The interpolation is described in more detail in Section 7.

5.3.4 Receptors

The ISCST3 model allows the user to specify receptors with Cartesian receptor grid and/or polar receptor grid. In general, Cartesian receptors are used for near-source receptors and polar grid receptors for more distant receptors. The number of receptors modeled greatly impacts run time. However, if too few receptors are modeled, the location of peak concentration may be missed. A sensitivity analysis was conducted to determine receptor locations and spacings that would provide adequate resolution without modeling an excessive number of receptors. (See Appendix C for details.) The results of the sensitivity analysis show that the maximum concentrations are generally higher for a dense receptor grid (i.e., 64 or 32 receptors on each



Note: Largest areas modeled for each WMU type have been omitted from the chart to improve clarity.

Figure 5-5. Air concentration vs. size of area source.

Table 5-3. Areas Modeled for Landfills and Land Application Units

Source Area (m ²)	Source Height (m)
81	0
567	0
1,551	0
4,047	0
12,546	0
40,500	0
78,957	0
161,880	0
243,000	0
376,776	0
607,000	0
906,528	0
1,408,356	0
8,090,000	0

Table 5-4. Areas and Source Heights Modeled for Wastepiles

Assigned Height (m)	Surface Area (m ²)						
	20	162	486	2,100	10,100	101,000	1,300,000
1	x	x	x	x	x	x	x
2	x	x	x	x	x	x	x
4	x	x	x	x	x		
6	x	x	x	x			
8	x	x					
10	x	x	x	x			

x = Combination modeled.

Table 5-5. Areas and Source Heights Modeled for Tanks

Assigned Height (m)	Surface Area (m ²)									
	2	3	5	10	25	50	100	400	1,500	5,000
1	x	x	x	x	x	x	x	x	x	x
2	x	x	x	x	x	x	x	x	x	x
4		x	x	x	x	x	x	x	x	x
6							x	x	x	x

x = Combination modeled.

square) than for a scattered receptor grid (i.e., 16 receptors on each square). However, the differences of the maximum receptor concentrations are not significant between a dense and a scattered receptor grid. Therefore, 16 evenly spaced receptor points on each square were used in the modeling. The sensitivity analysis also shows that the maximum downwind concentrations decrease sharply from the edge of the area source to about 1,000 meters from the source. After the first 1,000 meters from the edge of the area source, concentrations decrease very slowly as the downwind distance increases. Therefore, for annual average concentrations, the receptor points were placed on 25, 50, 75, 150, 500, and 1,000 meter receptor squares starting from the edge of the source, with 16 receptor points on each square.

5.4 Unitized Air Concentrations

Unitized air concentrations were calculated by running ISCST3 with a unit emission rate (i.e., 1 $\mu\text{g}/\text{m}^2\text{-s}$). The selected areas for each type of WMU were modeled with 29 representative meteorological locations in the continental United States to estimate UACs. The 5-year average UACs at all receptor points were calculated for the long-term or chronic exposure scenario. They were used as input to the Monte Carlo analysis and as input to the interpolation routine discussed above.

A similar methodology and assumptions were used to model dispersion for acute and sub-chronic exposures. Since the ISCST3 model uses hourly meteorological data, the outputs from the model can be used to develop any averaging times equal to or greater than 1 hour. One set of ISCST3 runs (for the 76 area-height combinations and 29 meteorological stations) was done for both acute and subchronic, resulting in 5 years of hourly average concentrations at each receptor. For each area, meteorological location, and receptor location, the maximum air concentration for any 24-hour period over the 5 years was selected. Then, for each area and meteorological station, the maximum 24-hour air concentration among all receptor locations at each distance modeled was selected, and this value was used as the UAC for that area and meteorological station for acute exposure. The same method was used to determine the subchronic UAC, except that the maximum 30-day period over the 5 years was used instead of the maximum 24-hour period.

Typically, the location of maximum impacts with respect to the source are determined by the prevailing wind direction. For ground-level area sources (i.e., landfills and land application units), maximum annual average UACs are always located on the first receptor square (closest to source). For elevated area sources, the maximum annual average UACs are usually located on the first receptor square and occasionally located on the second or third receptor square. Annual average UACs increase with the increasing area size of the sources.

Maximum UACs vary with meteorological location. For landfills and land application units, the maximum UACs at some meteorological locations can be twice as much as those at other locations. For wastepiles and tanks, the maximum UACs at some meteorological locations are more than twice those at other meteorological locations.

6.0 Development of Inhalation Health Benchmarks

Inhalation health benchmarks for chronic, subchronic, and acute exposure durations were needed for the risk characterization model. This section describes the noncancer and cancer benchmarks used in this study.

6.1 Chronic Inhalation Health Benchmarks Used in This Study

Chronic inhalation health benchmarks used in the Air Characteristic study include inhalation reference concentrations (RfCs) for noncarcinogens and inhalation unit risk factors (URFs) and inhalation cancer slope factors (CSFs) for carcinogens. URFs and CSFs were used in the analysis for carcinogenic constituents, regardless of the availability of an RfC. Inhalation health benchmarks were identified in the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997a, 1999). IRIS and HEAST are maintained by the Agency, and values from IRIS and HEAST were used in the analysis whenever available. Provisional EPA benchmarks and Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels (MRLs) were used to fill in data gaps (see Section 6.1.1). Additional chronic inhalation health benchmarks were derived for use in this analysis for constituents lacking EPA or ATSDR values (see Section 6.1.2).

Figure 6-1 describes the approach used to identify or develop the chronic inhalation health benchmarks used in this analysis. The benchmarks are summarized in Table 6-1.

6.1.1 Alternate Chronic Inhalation Health Benchmarks Identified

If IRIS or HEAST chronic inhalation health benchmarks were not available, benchmarks from alternative sources were sought. Provisional EPA benchmarks, ATSDR inhalation MRLs, and California EPA noncancer chronic reference exposure levels (CalEPA, 1997a) were included whenever available. Alternate RfCs were identified for

- # Acetone
- # Cyclohexanol
- # Isophorone
- # Phenol
- # Pyridine
- # Tetrachloroethylene
- # 1,1,1-Trichloroethane
- # Xylenes.

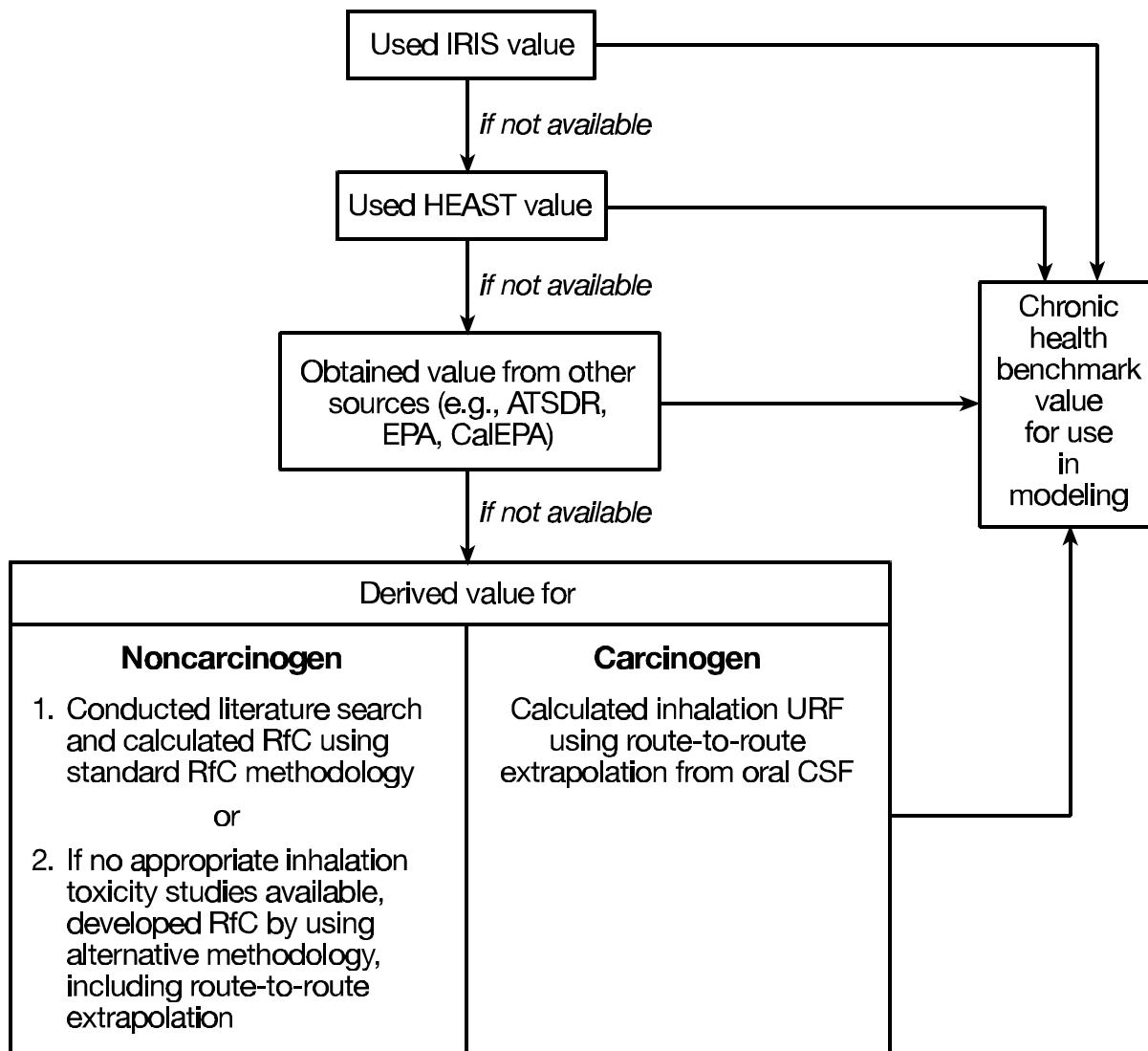


Figure 6-1. Approach used to select chronic inhalation health benchmark values.

Table 6-1. Chronic Inhalation Health Benchmarks Used in the Air Characteristic Analysis

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
75-07-0	Acetaldehyde	9.0E-03	Respiratory	I	2.2E-06	7.7E-03	I
67-64-1	Acetone	3.1E+01	Neurological	A	NA	NA	
75-05-8	Acetonitrile	6.0E-02	Mortality	I	NA	NA	
107-02-8	Acrolein	2.0E-05	Respiratory	I	NA	NA	
79-06-1	Acrylamide	NA			1.3E-03	4.6E+00	I
79-10-7	Acrylic acid	1.0E-03	Respiratory	I	NA	NA	
107-13-1	Acrylonitrile	2.0E-03	Respiratory	I	6.8E-05	2.4E-01	I
107-05-1	Allyl chloride	1.0E-03	Neurological	I	NA	NA	
62-53-3	Aniline	1.0E-03	Spleen	I	NA	NA	
7440-38-2	Arsenic	NA			4.3E-03	1.5E+01	I
7440-39-3	Barium	5.0E-04	Reproductive	H	NA	NA	
71-43-2	Benzene	NA			8.3E-06	2.9E-02	I
92-87-5	Benzidine	NA			6.7E-02	2.3E+02	I
50-32-8	Benzo(a)pyrene	NA			8.8E-04	3.1E+00	N
7440-41-7	Beryllium	2.0E-05	Respiratory	I	2.4E-03	8.4E+00	I
75-27-4	Bromodichloromethane	NA			1.8E-05	6.2E-02	D
75-25-2	Bromoform (Tribromomethane)	NA			1.1E-06	3.9E-03	I
106-99-0	Butadiene, 1,3-	NA			2.8E-04	1.8E+00	I
7440-43-9	Cadmium	NA			1.8E-03	6.3E+00	I
75-15-0	Carbon disulfide	7.0E-01	Neurological	I	NA	NA	
56-23-5	Carbon tetrachloride	NA			1.5E-05	5.3E-02	I
126-99-8	Chloro-1,3-butadiene, 2- (Chloroprene)	7.0E-03	Respiratory	H	NA	NA	
108-90-7	Chlorobenzene	2.0E-02	Kidney and liver	H	NA	NA	
124-48-1	Chlorodibromomethane	NA			2.4E-05	8.4E-02	D
67-66-3	Chloroform	NA			2.3E-05	8.1E-02	I
95-57-8	Chlorophenol, 2-	1.4E-03	Repro/developmental	D	NA	NA	
7440-47-3	Chromium VI	1.0E-04	Respiratory	I	1.2E-02	4.2E+01	I
7440-48-4	Cobalt	1.0E-05	Respiratory	D	NA	NA	
1319-77-3	Cresols (total)	4.0E-04	Hematological	D	NA	NA	
98-82-8	Cumene	4.0E-01	Kidney and adrenal	I	NA	NA	
108-93-0	Cyclohexanol	2.0E-05	Muscle	FR	NA	NA	

(continued)

Table 6-1. (continued)

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
96-12-8	Dibromo-3-chloropropane, 1,2-	2.0E-04	Reproductive	I	6.9E-07	2.4E-03	H
95-50-1	Dichlorobenzene, 1,2-	2.0E-01	Body weight	H	NA	NA	
106-46-7	Dichlorobenzene, 1,4-	8.0E-01	Liver	I	NA	NA	
75-71-8	Dichlorodifluoromethane	2.0E-01	Liver	H	NA	NA	
107-06-2	Dichloroethane, 1,2-	NA			2.6E-05	9.1E-02	I
75-35-4	Dichloroethylene, 1,1-	NA			5.0E-05	1.8E-01	I
78-87-5	Dichloropropane, 1,2-	4.0E-03	Respiratory	I	NA	NA	
10061-01-5	Dichloropropene, cis-1,3-	2.0E-02	Respiratory	I	3.7E-05	1.3E-01	H
10061-02-6	Dichloropropene, trans-1,3-	2.0E-02	Respiratory	I	3.7E-05	1.3E-01	H
57-97-6	Dimethylbenz(<i>a</i>)anthracene, 7,12-	NA			2.4E-02	8.4E+01	D
68-12-2	Dimethylformamide, <i>N,N</i> -	3.0E-02	Liver	I	NA	NA	
95-65-8	Dimethylphenol, 3,4-	NA	NA		NA	NA	
121-14-2	Dinitrotoluene, 2,4-	NA			1.9E-04	6.8E-01	D
123-91-1	Dioxane, 1,4-	8.0E-01	No liver, kidney, or hemato effects	D	NA	NA	
122-66-7	Diphenylhydrazine, 1,2-	NA			2.2E-04	7.7E-01	I
106-89-8	Epichlorohydrin	1.0E-03	Respiratory	I	1.2E-06	4.2E-03	I
106-88-7	Epoxybutane, 1,2-	2.0E-02	Respiratory	I	NA	NA	
111-15-9	Ethoxyethanol acetate, 2-	3.0E-01	Reproductive (male), hemato	D	NA	NA	
110-80-5	Ethoxyethanol, 2-	2.0E-01	Reproductive (male), hemato	I	NA	NA	
100-41-4	Ethylbenzene	1.0E+00	Developmental	I	NA	NA	
106-93-4	Ethylene dibromide	2.0E-04	Reproductive (male)	H	2.2E-04	7.7E-01	I
107-21-1	Ethylene glycol	6.0E-01	Respiratory	D	NA	NA	
75-21-8	Ethylene oxide	NA			1.0E-04	3.5E-01	H
50-00-0	Formaldehyde	NA			1.3E-05	4.6E-02	I
98-01-1	Furfural	5.0E-02	Respiratory	H	NA	NA	
87-68-3	Hexachloro-1,3-butadiene	NA			2.2E-05	7.7E-02	I
118-74-1	Hexachlorobenzene	NA			4.6E-04	1.6E+00	I
77-47-4	Hexachlorocyclopentadiene	7.0E-05	Respiratory	H	NA	NA	
67-72-1	Hexachloroethane	NA			4.0E-06	1.4E-02	I
110-54-3	Hexane, <i>n</i> -	2.0E-01	Respiratory and neurological	I	NA	NA	

(continued)

Table 6-1. (continued)

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
78-59-1	Isophorone	1.2E-02	Body weight	FR	NA	NA	
7439-96-5	Manganese	5.0E-05	Neurological	I	NA	NA	
7439-97-6	Mercury	3.0E-04	Neurological	I	NA	NA	
67-56-1	Methanol	1.3E+01	Developmental	D	NA	NA	
110-49-6	Methoxyethanol acetate, 2-	3.0E-02	Reproductive (male)	D	NA	NA	
109-86-4	Methoxyethanol, 2-	2.0E-02	Reproductive (male)	I	NA	NA	
74-83-9	Methyl bromide (bromomethane)	5.0E-03	Respiratory	I	NA	NA	
74-87-3	Methyl chloride (chloromethane)	NA			1.8E-06	6.3E-03	H
78-93-3	Methyl ethyl ketone	1.0E+00	Developmental	I	NA	NA	
108-10-1	Methyl isobutyl ketone	8.0E-02	Kidney and liver	H	NA	NA	
80-62-6	Methyl methacrylate	7.0E-01	Respiratory	I	NA	NA	
1634-04-4	Methyl <i>tert</i> -butyl ether	3.0E+00	Kidney and liver	I	NA	NA	
56-49-5	Methylcholanthrene, 3-	NA			2.1E-03	7.4E+00	D
75-09-2	Methylene chloride	3.0E+00	Liver	H	4.7E-07	1.6E-03	I
91-20-3	Naphthalene	3.0E-03	Respiratory	I	NA	NA	
7440-02-0	Nickel	NA			2.4E-04	8.4E-01	I
98-95-3	Nitrobenzene	2.0E-03	Kidney, liver, hematological, adrenal	H	NA	NA	
79-46-9	Nitropropane, 2-	2.0E-02	Liver	I	2.7E-03	9.4E+00	H
55-18-5	Nitrosodiethylamine	NA			4.3E-02	1.5E+02	I
924-16-3	Nitrosodi- <i>n</i> -butylamine	NA			1.6E-03	5.6E+00	I
930-55-2	<i>n</i> -Nitrosopyrrolidine	NA			6.1E-04	2.1E+00	I
108-95-2	Phenol	6.0E-03	No effects	FR	NA	NA	
85-44-9	Phthalic anhydride	1.2E-01	Respiratory	H	NA	NA	
75-56-9	Propylene oxide	3.0E-02	Respiratory	I	3.7E-06	1.3E-02	I
110-86-1	Pyridine	7.0E-03	Liver	O	NA	NA	
100-42-5	Styrene	1.0E+00	Neurological	I	NA	NA	
1746-01-6	TCDD, 2,3,7,8-	NA			3.3E+01	1.6E+05	H
630-20-6	Tetrachloroethane, 1,1,1,2-	NA			7.4E-06	2.6E-02	I
127-18-4	Tetrachloroethylene	3.0E-01	Neurological	A	5.8E-07	2.0E-03	SF
79-34-5	Tetrachloroethane, 1,1,2,2-	NA			5.8E-05	2.0E-01	I

(continued)

Table 6-1. (continued)

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
108-88-3	Toluene	4.0E-01	Respiratory and neurological	I	NA	NA	
95-53-4	Toluidine, o-	NA			6.9E-05	2.4E-01	D
76-13-1	Trichloro-1,2,2-trifluoroethane, 1,1,2-	3.0E+01	Body weight	H	NA	NA	
120-82-1	Trichlorobenzene, 1,2,4-	2.0E-01	Liver	H	NA	NA	
71-55-6	Trichloroethane, 1,1,1-	1.0E+00	Neurological	SF	NA	NA	
79-00-5	Trichloroethane, 1,1,2-	NA			1.6E-05	5.6E-02	I
79-01-6	Trichloroethylene	NA			1.7E-06	6.0E-03	SF
75-69-4	Trichlorofluoromethane	7.0E-01	Kidney and respiratory	H	NA	NA	
121-44-8	Triethylamine	7.0E-03	Respiratory	I	NA	NA	
7440-62-2	Vanadium	7.0E-05	Respiratory	D	NA	NA	
108-05-4	Vinyl acetate	2.0E-1	Respiratory	I	NA	NA	
75-01-4	Vinyl chloride	NA			8.4E-05	3.0E-01	H
1330-20-7	Xylenes (total)	4.0E-01	Neurological	A	NA	NA	

CAS = Chemical Abstract Service.

CSF = Cancer slope factor.

NA = Not available.

RfC = Reference concentration.

URF = Unit risk factor.

^a Sources:

I = IRIS (U.S. EPA, 1999)

H = HEAST (U.S. EPA, 1997a).

A = Agency for Toxic Substances Disease Registry (ATSDR) minimal risk levels (MRLs).

SF = Superfund Risk Issue Paper (U.S. EPA, 1996b; U.S. EPA, n.d.).

FR = 63 FR 64371-0402 (U.S. EPA, 1998b)

N = NCEA Risk Assessment Issue Paper (U.S. EPA, 1994f).

D = Developed for this study.

O = Other source (see Sections 6.1.1 and 6.1.2).

For acetone, tetrachloroethylene, and total xylenes, ATSDR's chronic inhalation MRLs were used. Provisional RfCs were identified for cyclohexanol, isophorone, and phenol in a *Federal Register* notice (63 FR 64371) of the final listing rules for solvents (U.S. EPA, 1998b). An acceptable daily intake (ADI) was identified for pyridine (U.S. EPA, 1986). An RfC for 1,1,1-trichloroethane was identified in a Superfund risk issue paper (U.S. EPA, 1996c). Table 6-2 summarizes the alternate RfCs identified for this analysis, as well as the target organs, sources, and critical studies.

Table 6-2. Alternate Chronic Inhalation Health Benchmarks

CAS #	Chemical Name	Inhalation Benchmark and Benchmark Value	Target Organ	Source
67-64-1	Acetone (2-propanone)	RfC = 13 ppm (31 mg/m ³)	Neurological	ATSDR chronic inhal MRL based on Stewart et al., 1975. <i>Acetone: Development of a Biological Standard for the Industrial Worker by Breath Analysis</i> , Cincinnati, OH: NIOSH. NTIS PB82-172917.
108-93-0	Cyclohexanol	Provisional RfC = 0.00002 mg/m ³	Muscle	63 FR 64376 (U.S. EPA 1998b); standard RfC methodology
78-59-1	Isophorone	Provisional RfC= 0.012 mg/m ³	Body weight	63 FR 64376 (U.S. EPA 1998b); standard RfC methodology
108-95-2	Phenol	Provisional RfC = 0.006 mg/m ³	No effects	63 FR 64376 (U.S. EPA 1998b); standard RfC methodology
110-86-1	Pyridine	Inhalation ADI= 0.002 mg/kg/d; converts to 0.007 mg/m ³	Liver	Cited in Health and Environmental Effects Profile (HEEP) for Pyridine (U.S. EPA, 1986)
127-18-4	Tetrachloroethylene	RfC = 0.04 ppm (0.3 mg/m ³)	Neurological	ATSDR chronic inhal MRL based on Ferroni et al., 1992. Neurobehavioral and neuroendocrine effects of occupational exposure to perchloroethylene. <i>Neurotoxicology</i> 12: 243-247.
71-55-6	1,1,1-Trichloroethane	RfC= 1.0 mg/m ³	Neurological	Superfund risk issue paper (U.S. EPA, 1996b)
1330-20-7	Xylenes (total)	RfC = 0.1 ppm (0.4 mg/m ³)	Neurological	ATSDR chronic inhal MRL based on Uchida et al., 1993. Symptoms and signs in workers exposed predominantly to xylenes. <i>Int Arch Occup Environ Health</i> 64:597-605.

6.1.2 Chronic Inhalation Health Benchmarks Derived for This Study

Chronic inhalation health benchmarks were developed for constituents lacking IRIS, HEAST, alternative EPA, or ATSDR values. RfCs were developed for

- # 2-Chlorophenol
- # Cobalt
- # Cresols
- # 1,4-Dioxane
- # 2-Ethoxyethanol acetate
- # Ethylene glycol
- # Methanol

- # 2-Methoxyethanol acetate
- # Vanadium.

For cobalt, cresols, 1,4-dioxane, ethylene glycol, and methanol, appropriate inhalation studies were identified and RfCs were developed using EPA's standard RfC methodology as detailed in *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994d). For vanadium, the study that the ATSDR acute inhalation MRL is based on was used but was adjusted for chronic exposure. For 2-chlorophenol, an RfC was developed using route-to-route extrapolation of the oral RfD (U.S. EPA, 1999). RfCs were derived for 2-ethoxyethanol acetate and 2-methoxyethanol acetate based on RfCs for 2-ethoxyethanol and 2-methoxyethanol, respectively.

EPA examined the toxicity data for 3,4-dimethylphenol and determined that it was not appropriate to derive an RfC. Very little toxicity or metabolism data specific to 3,4-dimethylphenol are available. Although an RfD has been developed by EPA (U.S. EPA, 1999), route-to-route extrapolation is not recommended because of the potential for respiratory tract effects following inhalation exposure (i.e., "portal-of-entry" effects) and "first-pass" effects following ingestion exposure (due to extensive metabolism in the liver which may result in differences in toxicity between oral and inhalation exposures).

Inhalation unit risk factors and inhalation cancer slope factors were developed for

- # Bromodichloromethane
- # Chlorodibromomethane
- # 7,12-Dimethylbenz[*a*]anthracene
- # 2,4-Dinitrotoluene
- # 3-Methylcholanthrene
- # o-Toluidine.

For bromodichloromethane, chlorodibromomethane, 2,4-dinitrotoluene, and o-toluidine, the oral CSFs (U.S. EPA, 1997a, 1999) were used to develop inhalation URFs for the compounds. For 7,12-dimethylbenz[*a*]anthracene and 3-methylcholanthrene, inhalation URFs developed by California's EPA (CalEPA 1997b) were used as the cancer benchmarks.

Table 6-3 summarizes the RfCs, inhalation unit risk factors, and inhalation cancer slope factors derived for use in the air characteristic analysis, the method of development and critical studies used, and the target organs identified. Details on the derivation of these inhalation benchmark values are provided in Appendix D.

6.2 Subchronic Inhalation Health Benchmarks

Information on intermediate or subchronic inhalation noncancer benchmark values for constituents considered in this study is summarized in Table 6-4. Data collected include subchronic RfCs and ATSDR intermediate inhalation MRLs.

Table 6-3. Chronic Inhalation Health Benchmarks Derived for This Study

CAS #	Chemical Name	Inhalation Benchmark and Benchmark Value	RfC Target Organ	Method of Derivation
75-27-4	Bromodichloromethane (dichlorobromomethane)	Inhal CSF = 6.2E-02 per mg/kg/d Inhal URF = 1.8E-05 per µg/m ³		Inhal CSF and URF based on IRIS oral CSF (renal)
124-48-1	Chlorodibromomethane (dibromochloromethane)	Inhal CSF = 8.4E-02 per mg/kg/d Inhal URF = 2.4E-05 per µg/m ³		Inhal CSF and URF based on IRIS oral CSF (hepatocellular adenoma/carcinoma)
95-57-8	2-Chlorophenol (o-)	RfC = 0.0014 mg/m ³	Repro/ developmental	Route-to-route extrapolation of IRIS RfD (0.005 mg/kg/d for reproductive effects)
7440-48-4	Cobalt	RfC = 0.00001 mg/m ³	Respiratory	Standard RfC derivation based on NTP TR-471 (1996a); supported by: ATSDR intermediate inhal MRL = 3E-05 mg/m ³ based on NTP TR-TOX-5 (1991) and CalEPA RfC = 5E-06 mg/m ³ based on Bucher et al., 1990. Inhalation toxicity studies of cobalt sulfate in F344/N rats and B6C3F1 mice. <i>Fundam Appl Toxicol</i> 15:357-372.
1319-77-3	Cresols, total	RfC = 0.0004 mg/m ³	Hematological	Standard RfC derivation based on: Uzhdavini ER, Astaf'yeva K, Mamayeva AA, Bakhtizina GZ. 1972. [Inhalation toxicity of o-cresol]. <i>Trudy Ufimskogo Nauchno-Issledovatel'skogo Instituto Gigiyeny Profzabolevaniya</i> , 7:115-9. (Russian)
57-97-6	7,12-Dimethylbenz[a]anthracene	Inhal CSF = 8.4E+01 per mg/kg/d Inhal URF = 2.4E-02 per µg/m ³		Inhal CSF and URF derived by CalEPA (1997b) based on TD ₅₀ approach
95-65-8	3,4-Dimethylphenol	NA - RfC derivation is inappropriate		
121-14-2	2,4-Dinitrotoluene	Inhal CSF = 6.8E-01 per mg/kg/d Inhal URF = 1.9E-04 per µg/m ³		Inhal CSF and URF based on IRIS oral CSF (liver, mammary gland)
123-91-1	1,4-Dioxane (1,4-diethyleneoxide)	RfC = 0.8 mg/m ³	Liver, kidney, hematological	Standard RfC derivation based on Torkelson et al. 1974. 1,4-Dioxane. II. Results of a 2-year inhalation study in rats. <i>Toxicol Appl Pharmacol</i> 30:287-298.

(continued)

Table 6-3. (continued)

CAS #	Chemical Name	Inhalation Benchmark and Benchmark Value	RfC Target Organ	Method of Derivation
111-15-9	2-Ethoxyethanol acetate	RfC= 0.3 mg/m ³	Repro (male)/ hematological	Derived from RfC for ethoxyethanol
107-21-1	Ethylene glycol	RfC= 0.6 mg/m ³	Respiratory	Derived using standard RfC methodology
67-56-1	Methanol	RfC = 13 mg/m ³	Developmental	Standard RfC derivation based on Rogers et al., 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose-response modeling for estimation of benchmark doses. <i>Teratology</i> 47(3):175-188.
110-49-6	2-Methoxyethanol acetate	RfC= 0.03 mg/m ³	Reproductive (male)	Derived from RfC for methoxyethanol
56-49-5	3-Methylcholanthrene	Inhal CSF = 7.4E+00 per mg/kg/d Inhal URF = 2.1E-03 per µg/m ³		Inhal CSF and URF derived by CalEPA (1997b) based on TD ₅₀ approach
95-53-4	o-Toluidine	Inhal CSF = 2.4E-01 per mg/kg/d Inhal URF = 6.9E-05 per µg/m ³		Inhal CSF and URF based on HEAST oral CSF (skin fibroma)
7440-62-2	Vanadium	RfC = 0.00007 mg/m ³	Respiratory	RfC based on ATSDR acute inhal MRL study, but adjusted for chronic exposure. Zenz and Berg, 1967. Human responses to controlled vanadium pentoxide exposure. <i>Arch Environ Health</i> 14:709-12. (acute inhal MRL = 0.0002 mg/m ³)

Table 6-4. Subchronic Inhalation Health Benchmarks

CAS #	Name	Subchronic RfC (mg/m ³)	Target Organ	Source
75-07-0	Acetaldehyde	9.0E-02	Respiratory	RfC based on subchronic study - removed UF
67-64-1	Acetone	3.1E+01	Neurological	ATSDR intermediate MRL
75-05-8	Acetonitrile	6.0E-02	Death	IRIS
107-02-8	Acrolein	2.0E-04	Respiratory	RfC based on subchronic study - removed UF
79-10-7	Acrylic acid	3.0E-03	Respiratory	HEAST
107-13-1	Acrylonitrile	2.0E-02	Respiratory	RfC based on chronic study - adjusted w/ AF
107-05-1	Allyl chloride	1.0E-02	Neurological	HEAST
62-53-3	Aniline	1.0E-02	Spleen	HEAST
7440-39-3	Barium	5.0E-03	Developmental	HEAST
71-43-2	Benzene	1.3E-02	Neurological	ATSDR intermediate MRL
75-15-0	Carbon disulfide	7.0E-01	Neurological	HEAST
56-23-5	Carbon tetrachloride	3.1E-01	Liver	ATSDR intermediate MRL
126-99-8	Chloro-1,3-butadiene, 2-(chloroprene)	7.0E-02	Respiratory	HEAST
108-90-7	Chlorobenzene	2.0E-01	Liver, kidney	Superfund Risk Assessment Issue Paper
67-66-3	Chloroform	2.4E-01	Liver	ATSDR intermediate MRL
7440-47-3	Chromium VI	5.0E-04	Respiratory	ATSDR intermediate MRL
7440-48-4	Cobalt	3.0E-05	Respiratory	ATSDR intermediate MRL
1319-77-3	Cresols (total)	1.2E-03	Hematological	RfC based on subchronic study - removed UF
98-82-8	Cumene	4.0E+00	Kidney, adrenal	RfC based on subchronic study - removed UF
108-93-0	Cyclohexanol	2.0E-04	Muscle	RfC based on subchronic study - removed UF
96-12-8	Dibromo-3-chloropropane, 1,2-	2.0E-03	Reproductive	RfC based on subchronic study - removed UF
95-50-1	Dichlorobenzene, 1,2-	2.0E+00	Body weight	HEAST
106-46-7	Dichlorobenzene, 1,4-	2.5E+00	Liver	HEAST
75-71-8	Dichlorodifluoromethane	2.0E+00	Liver	HEAST
107-06-2	Dichloroethane, 1,2-	8.1E-01	Liver, immunological	chronic & acute MRLs are equal; subchronic = chronic also
75-35-4	Dichloroethylene, 1,1-	7.9E-02	Liver	ATSDR intermediate MRL
78-87-5	Dichloropropane, 1,2-	1.3E-02	Respiratory	HEAST
10061-01-5	Dichloropropene, cis-1,3-	2.0E-02	Respiratory	HEAST

(continued)

Table 6-4. (continued)

CAS #	Name	Subchronic RfC (mg/m ³)	Target Organ	Source
10061-02-6	Dichloropropene, trans-1,3-	2.0E-02	Respiratory	HEAST
68-12-2	Dimethylformamide, <i>N,N</i> -	3.0E-02	Liver, GI	HEAST
123-91-1	Dioxane, 1,4-	8.0E+00	No liver, kidney, or hematological effects	RTI-derived RfC based on chronic study - adjusted w/ AF
106-89-8	Epichlorohydrin	1.0E-02	Respiratory	HEAST
106-88-7	Epoxybutane, 1,2-	2.0E-01	Respiratory	RfC based on chronic study - adjusted w/ AF
110-80-5	Ethoxyethanol, 2-	2.0E+00	Hematological	HEAST
100-41-4	Ethylbenzene	8.7E-01	Developmental	ATSDR intermediate MRL
106-93-4	Ethylene dibromide	2.0E-03	Reproductive	HEAST
107-21-1	Ethylene glycol	6.0E+00	Respiratory	RfC based on subchronic study - removed UF
75-21-8	Ethylene oxide	1.6E-01	Kidney	ATSDR intermediate MRL
50-00-0	Formaldehyde	1.2E-02	Respiratory	ATSDR intermediate MRL
98-01-1	Furfural	5.0E-01	Respiratory	HEAST
77-47-4	Hexachlorocyclopentadiene	7.0E-04	Respiratory	HEAST
67-72-1	Hexachloroethane	5.8E+01	Neurological	ATSDR intermediate MRL
110-54-3	Hexane, <i>n</i> -	2.0E-01	Neurological	HEAST
78-59-1	Isophorone	1.2E-01	Body weight	RfC based on subchronic study - removed UF
7439-96-5	Manganese	5.0E-04	Neurological	RfC based on chronic study - adjusted w/ AF
7439-97-6	Mercury	3.0E-04	Neurological	HEAST
109-86-4	Methoxyethanol, 2-	2.0E-01	Reproductive	HEAST
74-83-9	Methyl bromide (bromomethane)	1.9E-01	Neurological	ATSDR intermediate MRL
74-87-3	Methyl chloride (chloromethane)	4.1E-01	Liver	ATSDR intermediate MRL
78-93-3	Methyl ethyl ketone	1.0E+00	Developmental	HEAST
108-10-1	Methyl isobutyl ketone	8.0E-01	Liver, kidney	HEAST
80-62-6	Methyl methacrylate	7.0E+00	Respiratory	RfC based on chronic study - adjusted w/ AF
1634-04-4	Methyl <i>tert</i> -butyl ether	2.1E+00	Neurological	ATSDR intermediate MRL
75-09-2	Methylene chloride	3.0E+00	Liver	HEAST

(continued)

Table 6-4. (continued)

CAS #	Name	Subchronic RfC (mg/m ³)	Target Organ	Source
91-20-3	Naphthalene	3.0E-02	Respiratory	RfC based on chronic study - adjusted w/ AF
7440-02-0	Nickel	2.0E-03	Respiratory	chronic MRL avail. - adjusted w/ AF
98-95-3	Nitrobenzene	2.0E-02	Hematological, adrenal, kidney, liver	HEAST
79-46-9	Nitropropane, 2-	2.0E-02	Liver	HEAST
108-95-2	Phenol	6.0E-02	No effects	RfC based on subchronic study - removed UF
85-44-9	Phthalic anhydride	1.2E-01	Respiratory	HEAST
75-56-9	Propylene oxide	3.0E-02	Respiratory	HEAST
100-42-5	Styrene	3.0E+00	Neurological	HEAST
79-34-5	Tetrachloroethane, 1,1,2,2-	2.7E+00	Liver	ATSDR intermediate MRL
127-18-4	Tetrachloroethylene	3.0E+00	Neurological	RTI-derived RfC based on chronic study - adjusted w/ AF
108-88-3	Toluene	4.0E+00	Respiratory, neurological	RfC based on chronic study - adjusted w/ AF
76-13-1	Trichloro-1,2,2-trifluoroethane, 1,1,2-	3.0E+01	Body weight	HEAST
120-82-1	Trichlorobenzene, 1,2,4-	2.0E+00	Liver	HEAST
71-55-6	Trichloroethane, 1,1,1-	3.8E+00	Neurological	ATSDR intermediate MRL
79-01-6	Trichloroethylene	5.4E-01	Neurological	ATSDR intermediate MRL
75-69-4	Trichlorofluoromethane	7.0E+00	Kidney, respiratory	HEAST
121-44-8	Triethylamine	7.0E-02	No effects	RfC based on subchronic study - removed UF
7440-62-2	Vanadium	7.0E-05	Respiratory	RfC based on acute study (subchronic = chronic)
108-05-4	Vinyl acetate	2.0E-01	Respiratory	HEAST
75-01-4	Vinyl chloride	7.7E-02	Liver	ATSDR intermediate MRL
1330-20-7	Xylenes (total)	3.0E+00	Developmental	ATSDR intermediate MRL

Figure 6-2 describes the approach used to select the subchronic inhalation health benchmarks used in this analysis. Subchronic inhalation RfCs were identified in HEAST (U.S. EPA, 1997a) for 33 constituents. If subchronic RfCs were not available from HEAST, then they were derived from existing chronic RfCs or chronic inhalation MRLs. Ten chronic RfCs cited in

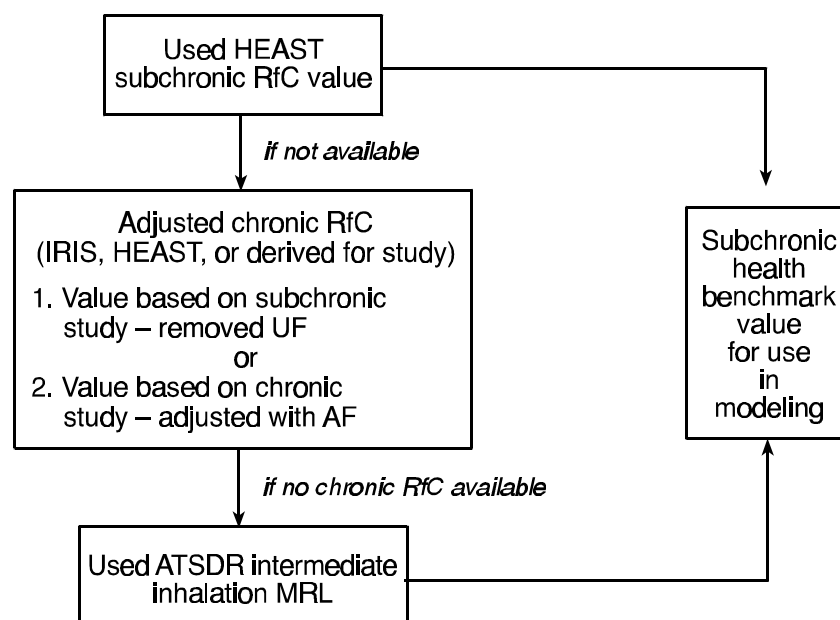


Figure 6-2. Approach used to select subchronic noncancer inhalation benchmark values.

IRIS or HEAST were based on subchronic studies; for these constituents, the uncertainty factor (UF) applied to extrapolate from subchronic to chronic duration (usually 10) for the derivation of the chronic RfC was removed, resulting in subchronic RfCs. Six RfCs cited in IRIS were based on chronic studies; for these constituents, an adjustment factor (AF) of 10 was applied to extrapolate from chronic to subchronic duration to derive subchronic RfCs.

ATSDR intermediate inhalation MRLs are for use with exposure durations of 15 to 364 days and are derived from subchronic toxicological or epidemiological studies. ATSDR intermediate inhalation MRLs were sought if subchronic RfCs from HEAST were not available or if chronic RfCs could not be adjusted to derive subchronic RfCs; intermediate MRLs were identified for 19 constituents. In addition, a chronic MRL was available for nickel ($2.0E-4$ mg/m³); an adjustment factor was used to extrapolate from chronic to subchronic duration, resulting in an interim subchronic RfC of $2.0E-3$ mg/m³. RfCs derived for this study for 1,4-dioxane and tetrachloroethylene were based on chronic inhalation studies; an adjustment factor was applied to these RfCs to extrapolate from chronic to subchronic duration.

Seventy-five subchronic RfCs or intermediate inhalation MRLs were identified for the 105 selected constituents; however, because subchronic exposures were modeled only for LAUs and wastepiles for volatile chemicals and metals, only 64 of these benchmarks were used. The remaining 11 were for semivolatiles not needed for assessing LAUs and wastepiles.

6.3 Acute Inhalation Health Benchmarks

Acute noncancer inhalation benchmark values for constituents considered in this study are summarized in Tables 6-5 and 6-6. Acute benchmarks identified include ATSDR acute

Table 6-5. ATSDR Acute Inhalation MRLs

CAS #	Name	ATSDR acute inhal MRL (mg/m ³)	Target Organ
67-64-1	Acetone	6.2E+01	Neurological
107-02-8	Acrolein	1.1E-04	Ocular
107-13-1	Acrylonitrile	2.2E-01	Neurological
71-43-2	Benzene	1.6E-01	Immunological
56-23-5	Carbon tetrachloride	1.3E+00	Liver
67-66-3	Chloroform	4.9E-01	Liver
106-46-7	Dichlorobenzene, 1,4-	4.8E+00	Developmental
107-06-2	Dichloroethane, 1,2-	8.1E-01	Immunological
78-87-5	Dichloropropane, 1,2-	2.3E-01	Respiratory
107-21-1	Ethylene glycol	1.3E+00	Kidney
50-00-0	Formaldehyde	6.1E-02	Respiratory
67-72-1	Hexachloroethane	5.8E+01	Neurological
74-83-9	Methyl bromide (bromomethane)	1.9E-01	Neurological
74-87-3	Methyl chloride (chloromethane)	1.0E+00	Neurological
1634-04-4	Methyl <i>tert</i> -butyl ether	6.1E+00	Neurological
75-09-2	Methylene chloride	1.0E+01	Neurological
127-18-4	Tetrachloroethylene	1.4E+00	Neurological
108-88-3	Toluene	1.5E+01	Neurological
71-55-6	Trichloroethane, 1,1,1-	1.1E+01	Neurological
79-01-6	Trichloroethylene	1.1E+01	Neurological
7440-62-2	Vanadium	7.0E-04	Respiratory
75-01-4	Vinyl chloride	1.3E+00	Developmental
1330-20-7	Xylenes (total)	4.3E+00	Neurological

Table 6-6. CalEPA's 1-Hour Acute Inhalation Reference Exposure Levels (RELs)

CAS #	Name	CalEPA 1-h REL (mg/m ³)	Effect Level	Effect
79-10-7	Acrylic acid	6.0E+00	I	Respiratory irritation
7440-38-2	Arsenic	4.0E-04	II	Reproductive/developmental
75-15-0	Carbon disulfide	2.0E+01	II	Reproductive/developmental
123-91-1	Dioxane, 1,4-	6.0E+00	I	Eye irritation
106-89-8	Epichlorohydrin	3.0E+00	I	Eye & respiratory irritation
111-15-9	Ethoxyethanol acetate, 2-	3.0E-01	II	Reproductive/developmental
110-80-5	Ethoxyethanol, 2-	9.0E-01	II	Reproductive/developmental
7439-97-6	Mercury	2.0E-03	II	Reproductive/developmental

(continued)

Table 6-6. (continued)

CAS #	Name	CalEPA 1-h REL (mg/m ³)	Effect Level	Effect
67-56-1	Methanol	3.0E+01	I	Mild neurological
109-86-4	Methoxyethanol, 2-	2.0E-02	I	Hematological
78-93-3	Methyl ethyl ketone	3.0E+01	I	Eye & respiratory irritation
7440-02-0	Nickel	1.0E-02	I	Respiratory irritation; immunological
108-95-2	Phenol	6.0E+00	I	Eye & respiratory irritation
75-56-9	Propylene oxide	6.0E+00	I	Eye & respiratory irritation
100-42-5	Styrene	2.0E+01	I	Eye & respiratory irritation

I = mild; II = severe

inhalation MRLs, EPA acute exposure guideline levels (AEGLs), and CalEPA (1998) 1-h acute inhalation reference exposure levels (RELs). Twenty-three acute ATSDR MRLs were identified. ATSDR acute inhalation MRLs are for use with exposure durations of 14 days or less and are derived from acute toxicological or epidemiological studies (see Table 6-5). AEGLs have been derived for aniline and ethylene oxide. CalEPA (1998) derived 1-h acute inhalation RELs for 29 of the 105 constituents. ATSDR acute MRLs were considered most appropriate for use in modeling, therefore CalEPA acute inhalation RELs were used only when MRLs were unavailable (for 15 constituents, see Table 6-6). Figure 6-3 describes the approach used to select the acute inhalation health benchmarks used in this analysis.

CalEPA's RELs can be applied in risk characterization for routine industrial emissions and planned releases as well as unplanned releases. These acute RELs are similar to EPA's AEGLs; both incorporate the recommendations of the National Academy of Science (NAS) published in *Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances* (NAS, 1993). AEGLs represent short-term threshold or ceiling exposure values intended for the protection of the general public, including susceptible or sensitive individuals. The AEGLs represent biological reference values for each of four different exposure periods of 30 minutes, 1 hour, 4 hours, and 8 hours. CalEPA's acute RELs and EPA's AEGLs have been established for three grades of effect:

- # Level I and AEGL-1: discomfort or mild effect level
- # Level II and AEGL-2: disability or serious effect level
- # Level III and AEGL-3: life-threatening effect level.

Only AEGL-1s were used in this study. The 8-h AEGL-1 for aniline is 3.8 mg/m³; no AEGL-1 levels have been derived for ethylene oxide (U.S. EPA, 1997b).

A total of 39 acute benchmarks were identified for the 105 constituents in this study; however, because acute exposures were modeled only for LAUs and wastepiles for volatile chemicals and metals, only 35 of these benchmarks were used. The remaining four were for semivolatiles not modeled in LAUs and wastepiles.

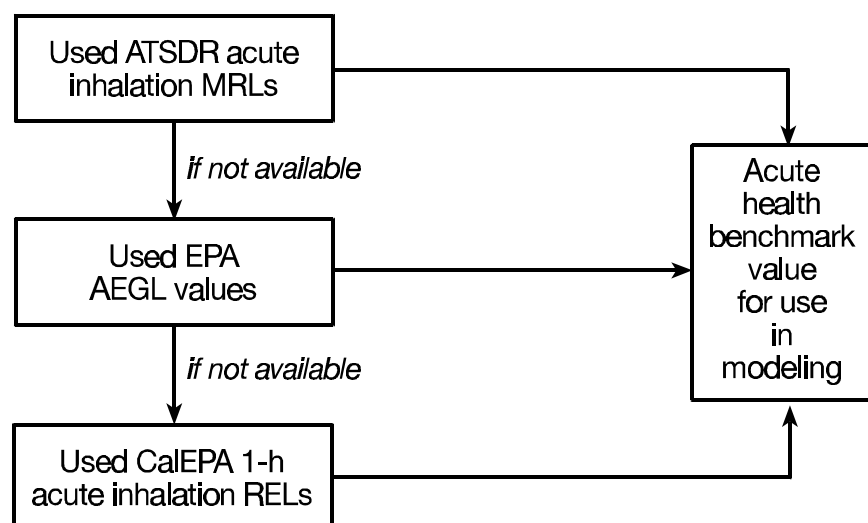


Figure 6-3. Approach used to select acute noncancer inhalation health benchmark values.

6.4 Comparison of Chronic, Subchronic, and Acute RfCs

RfCs for chronic, subchronic, and acute exposure durations were compared to verify the appropriate progression from chronic (lowest) to acute (highest). Acrolein, 1,2-dichloroethane, 1,4-dioxane, 2-ethoxyethanol, ethylbenzene, ethylene glycol, 2-methoxyethanol, methyl *tert*-butyl ether, and tetrachloroethylene have an inconsistent progression between their chronic, subchronic, and/or acute RfCs. Discrepancies in this progression are due to the fact that RfCs of different durations were derived by different agencies (i.e., U.S. EPA, ATSDR, CalEPA, Superfund). Some of these differences are due to the different dates on which values were derived (e.g., less complete data available to EPA for the derivation of an IRIS health benchmark in 1987 as compared to ATSDR for the derivation of an MRL in 1998), the use of different methodologies, or the use of different toxicity endpoints (e.g., for nickel, a respiratory effect is the endpoint used for the subchronic RfC and an immunological effect is the endpoint used for the acute RfC).

The chronic and subchronic RfCs for vanadium are both $7E-5$ mg/m³ because they were derived from an acute-duration study and had an uncertainty factor of 10 applied to account for the extrapolation from acute to subchronic and chronic exposure duration (the acute RfC is $7E-4$ mg/m³).

The chronic and subchronic RfCs for acetonitrile are both $6E-2$ mg/m³ because the chronic RfC is based on a subchronic-duration study that does not require the application of an uncertainty factor to adjust for extrapolation to longer exposures according to EPA. That is, EPA would have derived the same value for both chronic and subchronic RfCs based on this study. EPA's rationale for not applying an uncertainty factor to account for the use of a subchronic study was that "there was no mortality in the longer term mouse study. Therefore, although this endpoint is of concern based on the subchronic study, increased exposure would not be expected to increase the sensitivity to this endpoint" (U.S. EPA, 1999).

7.0 Development of Risk-Specific Waste Concentration Distribution

This section describes the models used to combine results of the emissions modeling (Section 4.0) and dispersion modeling (Section 5.0) with exposure factor distributions to calculate air concentration, risk or hazard quotient, and risk-specific waste concentration (C_w) for chronic, subchronic, and acute exposures. In addition, it describes the stability analysis performed for the Monte Carlo model used to calculate chronic risk-specific waste concentrations for carcinogens and modifications made to the methodology for lead. The section begins with an overview of the modeling, then describes the modeling in detail step by step. Following that are subsections describing the development of exposure factors, the Monte Carlo model stability analysis, and modifications for lead.

7.1 Overview

The risk model calculates risk-specific waste concentrations for carcinogens and noncarcinogens for three averaging times (chronic, subchronic, and acute) for adults and children. The model structure varies somewhat among these different categories.

7.1.1 Chronic Exposures to Carcinogens

For carcinogens, a Monte Carlo analysis was performed in which the location of the receptor at a given distance and various exposure factors (body weight, inhalation rate, and exposure duration) were varied (exposure frequency was kept fixed as it has little variability). For each constituent and waste management unit type combination, a separate Monte Carlo simulation was run for each WMU in the Industrial D Survey database (Shroeder et al., 1987) or TSDR tank database (U.S. EPA, 1987). The emission rate for the specific constituent from the specific WMU was used as an input to the Monte Carlo simulation and was not varied across iterations within a simulation.

Approximately 1,000 iterations were performed for each WMU, resulting in a distribution of waste concentrations (C_w) that would result in the specified risk criterion. This distribution captures the range in waste concentration attributable to the variability in the potential location and to the exposure factors associated with each receptor. From this distribution, the 85th, 90th, and 95th percentiles were selected to characterize the distribution. These percentiles represent the percentage of receptors that are protected at the risk criterion for a specific WMU.

When the Monte Carlo simulation had been run for all the WMUs, a cumulative distribution across all facilities for each protection level (85, 90, or 95 percent of receptors) was

obtained for each receptor at each distance. This distribution reflects the variability across facilities. In developing this distribution, the results were weighted using the facility weights from the Industrial D Survey data. These weights indicate the number of U.S. facilities represented by a particular facility in the Industrial D Survey database. The resulting cumulative distribution accounts for variability across all facilities represented, not just those actually modeled. The TSDR survey data used to characterize tanks did not include facility weights; therefore, the tank distributions are not weighted.

7.1.2 Chronic Exposures to Noncarcinogens

Hazard quotients for noncarcinogens depend only on air concentration and the health benchmark (a reference concentration). Therefore, exposure factors are not used and only the location of the receptor is relevant of the variables varied in the Monte Carlo analysis. Because the location of the receptor is such a simple distribution, a Monte Carlo analysis was unnecessary for noncarcinogens; the distribution of hazard quotient (and therefore C_w) based on the distribution of the location of the receptor can be obtained analytically by calculating hazard quotient (and C_w) for each of the 16 receptor locations (or directions around the site) and taking the desired percentiles from those 16 values.

7.1.3 Acute/Subchronic Exposures

Exposure and risk modeling for subchronic and acute exposures differed somewhat from the modeling for chronic exposures in several respects. All acute and subchronic health benchmarks are analogous to chronic noncarcinogen benchmarks, so exposure factors were not used. Neither receptor location around the site was varied for acute and subchronic exposures. Therefore, a Monte Carlo analysis was not performed for subchronic and acute exposures. Instead, a point estimate of C_w was calculated for each WMU in the Industrial D database using the single sector that resulted in the maximum air concentration. This point estimate represents the maximum, or 100th percentile, concentration, and therefore is most comparable to the 100th percentile of the distribution generated by the Monte Carlo model for chronic exposures. The point estimate can be interpreted as the level at which 100 percent of receptors are protected at a particular WMU. A distribution across all WMUs of a specific type was generated from these point estimates, and the 90th percentile of that distribution is presented in the results for subchronic and acute exposures.

7.1.4 Adult Exposure Approach

Adult receptors modeled include adult residents and off-site workers. Risk for adults is calculated using long-term average air concentration that is constant over the entire exposure duration.

The inhalation rate, exposure frequency, and exposure duration differ for residents and workers. Body weight is the same for all adults, whether resident or worker. All exposure factors for adults are held constant over the entire exposure duration. The actual point estimates or distributions of the exposure factors used are described in Section 7.11.

7.1.5 Child Exposure Approach

Three child age groups, or cohorts, were used to model child exposures: 0 to 3, 4 to 10 and 11 to 18 years of age. These cohorts reflect the age cohorts for which inhalation rate data are available. Results were calculated and saved for receptors falling into each of these three age cohorts at the start of exposure and presented as child 1 (0-3), child 2 (4-10), and child 3 (11-18). An exposure duration was selected randomly for each of the three starting age cohorts from a distribution specific to each starting age cohort. For each age cohort, exposure begins at a starting age selected at random within the cohort and then continues through succeeding age cohorts and into adulthood as necessary until the exposure duration selected for that starting age cohort is reached.

Annual risk for each year of exposure (from starting age to starting age plus exposure duration) was calculated and summed over the exposure duration for each child receptor. If the child reached age 19 before the exposure duration ended, adult exposure factors were used for the remainder of the exposure duration. This approach requires both body weight and inhalation rate distributions by year of age; however, only body weight is available by year. Inhalation rate is available only for the age groups used to define the cohorts (0-3, 4-10, and 11-18 years). Because inhalation rate data could not be disaggregated to individual years of age, we retained year-by-year body weights and used the inhalation rate for the cohort associated with each year of age for that year. Thus, the inhalation is a constant for all ages within an age cohort and changes only when the receptor ages from one cohort to the next. Both EPA and a statistician experienced in working with EFH exposure factor data (L. Myers, RTI, personal communication with Anne Lutes, RTI, March 16, 1998) preferred this approach over the alternative of pooling body weights to the age cohort age ranges as retaining the most detail from the available data without sacrificing statistical rigor.

7.1.6 Specific Steps Required in the Risk Analysis

Most of the following steps are common to the risk model structure for carcinogens and noncarcinogens, all three averaging times, and both adults and children:

- # Select a receptor location
- # Obtain the appropriate unitized air concentration (UAC)
- # Calculate air concentration
- # Select exposure factors from distributions
- # Obtain health benchmarks
- # Calculate risk or hazard quotient
- # Backcalculate risk-specific waste concentration (C_w)
- # Adjust tank results for nonlinearity in biodegradation
- # Flag results exceeding soil saturation concentration or solubility.

For chronic exposure to carcinogens, these steps were repeated for each iteration in the Monte Carlo simulation. For chronic exposure to noncarcinogens, these steps were performed once for each of the 16 receptor locations. For acute and subchronic exposures, these steps were performed once. Sections 7.2 through 7.10 describe these above steps in more detail.

7.2 Select Receptor Location

7.2.1 Chronic Exposure

Dispersion coefficients were modeled for each of 6 distances and 16 directions as described in Section 5.0. The 6 distances were 25, 50, 75, 150, 500, and 1,000 meters. The 16 directions were equally spaced around the WMU. Calculations were performed for each distance, and direction was allowed to vary.

Five receptors were modeled: an adult resident, a child resident ages 0 to 3 years at the start of exposure, a child resident ages 4 to 10 years at the start of exposure, a child resident ages 11 to 18 years at the start of exposure, and an off-site worker. These receptors were assessed at all six distances. All receptors could be located in any of the 16 directions.

Direction was modeled as a uniform distribution so that a receptor had a probability of 1 in 16 (or 0.0625) of being located in any one of the 16 directions. The same direction was used for all receptors for a particular iteration.

For carcinogens, Crystal Ball[®] was used to randomly sample the distributions and generate values of the exposure factors for each iteration. However, receptor locations (i.e., direction from the site) were not generated using Crystal Ball[®]. Preliminary analysis suggested that random sampling from this distribution would not achieve stability unless considerably more than 1,000 iterations were used. However, it is not necessary to sample from this distribution, because the distribution is discrete and is completely described by the uniform probability (1/16th) of a receptor being located in any particular direction. Thus, this distribution is computationally most efficiently represented in the Monte Carlo simulation, not by random sampling but rather by simply “visiting” each of the 16 directions an equal number of times during the course of the Monte Carlo simulation. That is, if one visits each of the 16 directions (deterministically) an equal number of times, there is no remaining direction-related variability from which to “sample,” because the entire population has been visited. Visiting the directions an equal number of times for each Monte Carlo simulation required a number of iterations that was a multiple of 16 (e.g., 1,008). We then associated each direction with one set of exposure factors generated by the Monte Carlo simulation. In this way, we ensured that an equal selection of the directions was made and that the directions were associated randomly with exposure factors.

For noncarcinogens, exposure factors are not used, so results were calculated once for each of the 16 directions.

7.2.2 Acute/Subchronic Exposures

Dispersion coefficients were modeled for each of 3 distances and 16 directions as described in Section 5.0. The 3 distances were 25, 50, and 75 meters. The 16 directions were equally spaced around the WMU. Calculations were performed for each distance, and the direction that gave the maximum air concentration was used. The direction of maximum air concentration varied from WMU to WMU depending on prevailing wind directions.

7.3 Obtain Unitized Air Concentrations

After a receptor location had been selected, vapor-phase and particulate-phase UACs for that location were calculated. The UACs were estimated by interpolating between the UACs developed for areas immediately above and below the actual area of the unit, as follows:

$$UAC = \left(\frac{A - A_i}{A_j - A_i} \right) \times (UAC_j - UAC_i) + UAC_i \quad (7-1)$$

where

UAC = unitized air concentration for specific WMU ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$)

A = area of specific WMU (m^2)

A_i = area modeled in dispersion modeling immediately below area of specific WMU (m^2)

A_j = area modeled in dispersion modeling immediately above area of specific WMU (m^2)

UAC_j = unitized air concentration developed for area j ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$)

UAC_i = unitized air concentration developed for area i ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$).

A few WMUs had areas that fell either below the smallest area or above the largest area modeled in the dispersion modeling. If the area was less than the smallest area modeled, A_j and UAC_j were set to the values for the smallest area modeled, and A_i and UAC_i were set to zero. If the area was greater than the largest area modeled, the A_i , UAC_i , A_j , and UAC_j were set to correspond to the two largest areas modeled, based on the assumption that the UAC continues to increase with the same slope above the largest area modeled.

For WMUs that were elevated (i.e., wastepiles and tanks), UACs were developed for several different heights for each modeled area. The UACs for the modeled height closest to each unit's actual height were used in the interpolation.

7.4 Calculate Air Concentration

Air concentration was calculated from the WMU emission rate (which remains constant through all iterations of the Monte Carlo simulation for a particular WMU) and the interpolated UACs. Recall that the emission rates are emissions associated with a unit waste concentration of 1 mg/kg (or 1 mg/L for tanks), so the resulting air concentration is also associated with a unit waste concentration. Air concentration was calculated as follows:

$$C_{air} = (E_{vapor} \times 10^6 \mu\text{g/g} \times UAC_{vapor} + C_{soil} \times \frac{E_{partic}}{3600 \text{ s/h}} \times UAC_{partic}) \times 0.001 \text{ mg}/\mu\text{g} \quad (7-2)$$

where

C_{air} = air concentration associated with a unit waste concentration
([mg/m³]/[mg/kg] or [mg/m³]/[mg/L])

E_{vapor} = emission rate of constituent in vapor phase ([g/m²-s]/[mg/kg] or [g/m²-s]/mg/L)

C_{soil} = average annual soil concentration in unit ([mg/kg]/[mg/kg] = [μg/g]/[mg/kg] or [μg/g]/[mg/L])

E_{partic} = emission rate of particulates (g/m²-h)

UAC_{vapor} = vapor-phase unitized air concentrations ([μg/m³]/[μg/m²-s]).

UAC_{partic} = particulate-phase unitized air concentrations ([μg/m³]/[μg/m²-s]).

Note that C_{soil} and E_{partic} are both zero for tanks. Vapor emissions (E_{vapor}) are zero for nonvolatile constituents (all metals except mercury).

7.5 Select Exposure Factors

The development of exposure factor distributions is described in Section 7.11. Values for each exposure factor (inhalation rate, body weight, and exposure duration) were selected independently for each receptor. Specifically, for child receptors, exposure factors were selected independently for a specific age or cohort for each starting age cohort. For example, one set of exposure factors for a 9-year-old child was selected for use with the child 1 receptor (a child whose exposure starts between 0 and 3 years of age) and a second set was selected independently for use with the child 2 receptor (a child whose exposure starts between 4 and 10 years of age).

Risk results are not calculated separately by gender. Therefore, we considered pooling distributions for males and females. However, pooling these distributions across genders risks losing the slightly bimodal character of the overall distribution created by differences in exposure factors for males vs. females. Therefore, it was considered preferable and more statistically rigorous to retain the separate distributions for males and females. We then used the male distribution for half the Monte Carlo iterations and the female distribution for the other half.

The health benchmarks used for chronic exposure to noncarcinogens and subchronic and acute exposure to all chemicals are expressed as ambient air concentrations, and are compared directly to the modeled air concentration, without the use of exposure factors. Therefore, this step is not needed for noncarcinogens or acute and subchronic exposures.

7.5.1 Inhalation Rate

To assess chronic exposures, an average daily inhalation rate is needed. Such a rate is based on inhalation values for a variety of activities averaged together.

The inhalation rate is linearly related to exposure and risk. A twofold change in the inhalation rate results in a twofold change in the risk estimate for a carcinogen. Thus, the sensitivity of this parameter in the analysis is a function of its variability. An examination of data presented in the draft EFH on daily inhalation rates for adults, workers, and children provides a general idea of the variability in this factor. In general, the range for both adults and workers would be around a factor of 2 or 3. Children's rates are more variable.

For children, inhalation rates were held constant over each age cohort because there were insufficient data to disaggregate them to specific years of age.

7.5.2 Body Weight

Distributions for body weight were needed that were consistent with the distributions used for inhalation rate. Therefore, body weight distributions for adult males and females (to be used for both adult residents and workers) and male and female children were needed. For children, body weights specific to each year of age were used.

7.5.3 Exposure Duration

A starting age within each child cohort was selected at random, assuming equal distributions of ages within the cohort. The assumption of equal distribution by age within each cohort is supported by Census data showing that the population is roughly equally divided among each year of age (see <http://www.census.gov/cdrom/lookup/922975351>; 100% count, national totals, Table P11-Age).

When an exposure duration extended beyond the end of the age cohort, exposure was continued in the next age cohort (or adulthood) until the exposure duration was reached. For example, if exposure began at 0 years, and exposure duration was 13 years, then 3 years were modeled as age 0 to 3, 7 years were modeled as age 4 to 10, and 3 years were modeled as age 11 to 18. An exposure duration was picked at random for each starting age cohort.

7.5.4 Exposure Frequency

Exposure frequency is held constant throughout the analysis.

7.6 Obtain Health Benchmarks

Standard health benchmarks (cancer slope factors for carcinogens and reference concentrations for noncarcinogens) were obtained for each constituent (see Section 6).

7.7 Calculate Risk or Hazard Quotient

The risk or hazard quotient associated with a unit waste concentration was calculated based on the calculated air concentration and, if relevant, the exposure factors selected.

Risk for carcinogens for adults was calculated as follows:

$$Risk_{calc'd} = \frac{C_{air} \times CSF \times IR \times ED \times EF}{BW \times AT \times 365 \text{ d/yr}} \quad (7-3)$$

where

$Risk_{calc'd}$ = individual risk associated with unit waste concentration (per mg/kg)

C_{air} = air concentration associated with a unit waste concentration ([mg/m³]/[mg/kg])

CSF = cancer slope factor (per mg/kg-d)

IR = inhalation rate (m³/d)

ED = exposure duration (yr)

EF = exposure frequency (d/yr)

BW = body weight (kg)

AT = averaging time (yr) = 70.

The air concentration is a long-term average and is constant over the entire exposure duration. Averaging time is a fixed input to this equation (with a value of 70) because it must be consistent with the averaging time used to develop standard cancer slope factors.

For children, annual risk for each year of exposure (from starting age to starting age plus exposure duration) was calculated and summed over the exposure duration for each child receptor. Ideally, we would have body weight and inhalation rate data specific to each single year of age, and risk would be calculated as follows for each starting age cohort:

$$Risk_j = \sum_{i=s}^{s+ED_j} \frac{C_{air} \times CSF \times IR_i \times ED_i \times EF}{BW_i \times AT \times 365 \text{ d/yr}} \quad (7-4)$$

where

$Risk_j$ = individual risk associated with unit waste concentration for child starting exposure in cohort j (unitless)

i = age (yr)

s = age at start of exposure (yr)

ED_j = total exposure duration for starting age cohort j (yr)

C_{air} = long-term average air concentration associated with a unit waste concentration ([mg/m³] [mg/kg])

IR_i = inhalation rate for age i (m³/d)

CSF = cancer slope factor (per mg/kg-d)

ED_i = exposure duration for age i (yr). This is usually 1, but for the last year of exposure, may be less than 1 to accommodate an ED that is not an integer number of years, or greater than 1 if the child has aged into adulthood. The sum of all $ED_i = ED_j$.

EF = exposure frequency (d/yr)

BW_i = body weight for age i (kg)

AT = averaging time (yr) = 70.

As for adults, the air concentration is a long-term average and is constant over the entire exposure duration.

As described in Section 7.1.5, inhalation rate data could not be disaggregated to individual years of age; therefore, the inhalation rate for the cohort associated with age i is substituted in the above equation for IR_i . Thus, IR_i becomes a constant for all ages within an age cohort and changes only when the receptor ages from one cohort to the next.

The hazard quotient for noncarcinogens (including all chemicals for acute and subchronic exposures) was calculated as follows:

$$HQ_{calc'd} = \frac{C_{air}}{RfC} \quad (7-5)$$

where

- $HQ_{calc'd}$ = hazard quotient associated with unit waste concentration (per mg/kg)
- C_{air} = air concentration associated with a unit waste concentration
([mg/m³]/[mg/kg])
- RfC = chronic reference concentration or acute or subchronic benchmark (mg/m³).

Because the hazard quotient equation does not consider exposure factors, there is no difference in results for different receptors at the same location (e.g., adult resident, child resident, and offsite worker). Therefore, only an adult resident was modeled for noncarcinogens and for acute and subchronic exposures.

7.8 Backcalculate Risk-Specific Waste Concentration

The next step was to backcalculate the risk-specific waste concentration from the risk or hazard quotient corresponding to a unit waste concentration. Because risk is linear with respect to waste concentration,¹ in the models used in this analysis, this may be done by a simple ratio technique:

$$C_w = \frac{Risk_{crit}}{Risk_{calc'd}} \quad or \quad \frac{HQ_{crit}}{HQ_{calc'd}} \quad (7-6)$$

where

- C_w = risk-specific waste concentration (mg/kg)
- $Risk_{crit}$ = risk criterion (unitless)
- $Risk_{calc'd}$ = risk associated with unit waste concentration (per mg/kg)
- HQ_{crit} = hazard quotient criterion (unitless)
- $HQ_{calc'd}$ = hazard quotient associated with unit waste concentration (per mg/kg).

When a particular constituent had both carcinogenic and noncarcinogenic effects, the carcinogenic risk was used to continue the calculations, because it generally results in a more protective concentration.

7.9 Adjust Tank Results For Non-Linearity of Biodegradation

As mentioned, risk is assumed to be linear with waste concentration. The assumption of linearity is accurate for the dispersion modeling and the exposure and risk modeling. However, the emissions model is linear only for land-based units and tanks with no biodegradation; for tanks with biodegradation, the emissions model is nonlinear with respect to biodegradation. At

¹ The volatile emission model for tanks with biodegradation can be an exception to this assumption of linearity. Section 7.9 describes how those situations were identified and resolved.

low concentrations, biodegradation in tanks is first order. However, at concentrations in excess of the half-saturation level, biodegradation becomes zero order. In order to address this, emissions were modeled in the aqueous phase at 0.001 mg/L to capture first-order biodegradation and at the solubility to capture zero-order biodegradation. These emission rates then were normalized to a unit concentration by dividing by 0.001 or the solubility. When the backcalculated waste concentration based on first order biodegradation exceeded the half-saturation constant, suggesting that biodegradation would be zero order, it was recalculated based on the normalized solubility limit emission rate. Figure 7-1 summarizes this approach for tanks with biodegradation (i.e., some aerated tanks).

7.10 Flag Results That Exceed Soil Saturation Concentration or Solubility

The results for all WMU types presented in Volumes I and III were calculated as described above using aqueous-phase emission rates. Most of the waste streams managed in the types of units modeled are expected to contain constituents in the aqueous, rather than the organic, phase; therefore, this is the most realistic scenario. However, results based on organic-phase emissions are of interest in two circumstances: when organic-phase emissions are higher than aqueous-phase emissions, and when backcalculated results based on aqueous-phase emissions exceed physical limitations on the aqueous phase such as the soil saturation concentration or solubility. These are discussed below.

7.10.1 Organic-Phase Emissions Higher than Aqueous-Phase Emissions

Most chemicals are better able to volatilize from an aqueous medium than from an organic medium; therefore, for most chemicals, the aqueous-phase emission rates are

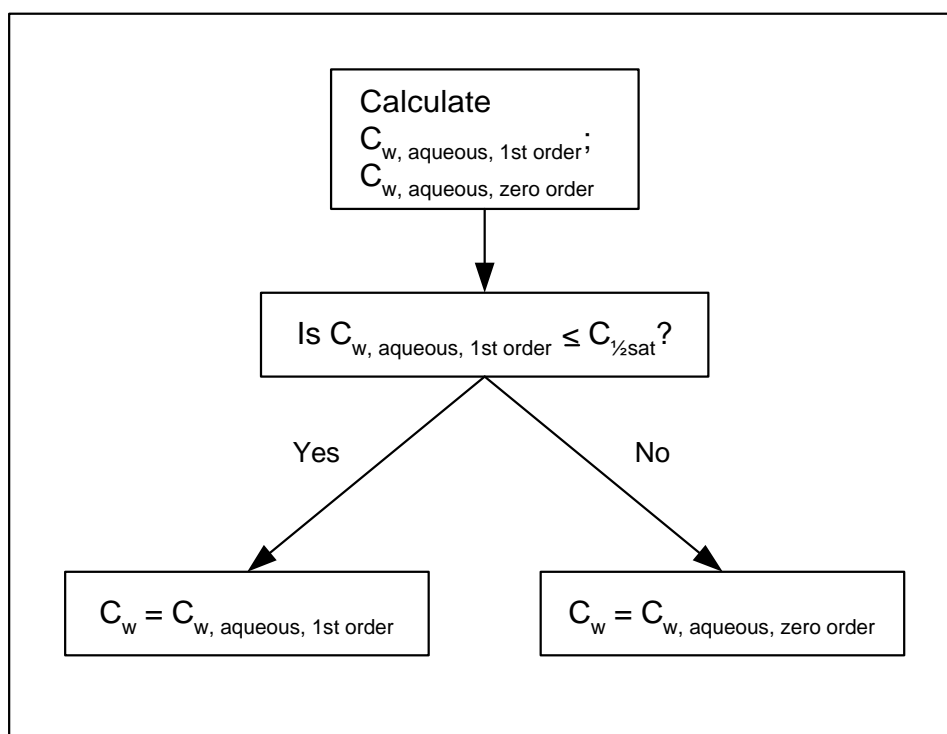


Figure 7-1. Linearity adjustments for tanks with biodegradation.

considerably higher than organic-phase emission rates. However, for a few chemicals (most notably formaldehyde), the organic-phase emissions are higher than the aqueous-phase emissions and waste concentrations based on organic-phase emissions would be lower than waste concentrations based on aqueous-phase emissions. When this is the case, the results based on aqueous-phase emissions are footnoted to indicate this. This does not invalidate the aqueous-phase results, as that is still the most likely waste matrix.

7.10.2 Backcalculated Concentrations Exceed Physical Limitations on Aqueous Phase

For land-based units, some of the backcalculated waste concentrations based on aqueous-phase emissions exceed the soil saturation concentration. This is the maximum possible aqueous-phase concentration in soil; once this is exceeded, free (organic-phase) product will occur in the soil. This soil saturation concentration has been estimated for each chemical in the analysis, using the following equation:

$$C_{sat} = \frac{S}{\rho_b} (K_d \times \rho_b + \theta_w + H' \times \theta_a) \quad (7-7)$$

where

- C_{sat} = soil saturation limit (mg/kg)
- S = solubility limit (mg/L)
- ρ_b = bulk density of soil / waste matrix (kg/L)
- K_d = soil-water partition coefficient (L/kg)
- θ_w = water-filled soil porosity (unitless)
- H' = dimensionless Henry's law constant (unitless, = H/RT)
- θ_a = air-filled soil porosity (unitless).

The soil saturation concentration is a somewhat site- and waste-specific value. Therefore, a backcalculated concentration may exceed it in some situations but not in others. When the backcalculated concentration based on aqueous-phase emissions exceeded the typical soil saturation concentration calculated for this analysis as shown in Equation 7-7, the result was footnoted to indicate whether pure component (i.e., a concentration of 10^6 mg/kg) would result in a risk exceeding the target risk when modeled using organic-phase emission rates.

Similarly, for tanks, some of the backcalculated waste concentrations based on aqueous-phase emissions exceed the solubility. This is the maximum possible aqueous-phase concentration; once this is exceeded, free (organic-phase) product will occur in the tank and either sink, yielding aqueous-phase emissions from a concentration equal to the solubility, or float on the surface, yielding emissions from the organic phase at a concentration of pure component. The solubility under standard temperature and pH conditions (20-25 C and neutral pH) has been estimated for each chemical in the analysis, but this is a somewhat site- and waste-specific value. Therefore, a backcalculated concentration may exceed it in some situations but not in others. When the backcalculated concentration based on aqueous-phase emissions exceeded the typical solubility calculated for this analysis, the result was footnoted to indicate

whether pure component (i.e., a concentration of 10^6 mg/kg) would result in a risk exceeding the target risk when modeled using organic-phase emission rates.

7.11 Derivation of Exposure Factors

This section describes the derivation of exposure factor distributions used in the model. All data in this section are from the *Exposure Factors Handbook* (U.S. EPA, 1997c and 1997d; hereafter, the EFH).

All exposure factor distributions and point estimates were developed for the following subpopulations:

- # Adult residents
- # Children ages 0-3 years
- # Children ages 4-10 years
- # Children ages 11-18 years
- # Workers.

The age ranges for children were chosen for consistency with the data on inhalation rate in the EFH.

7.11.1 Inhalation Rate

Single values for inhalation rates are presented as recommended values for long-term dose assessments in the EFH. Values of $11.3 \text{ m}^3/\text{d}$ for women and $15.2 \text{ m}^3/\text{d}$ for men are representative of average inhalation rates for adults. Upper percentile values are not recommended. These values differ significantly from the $20 \text{ m}^3/\text{d}$ commonly assumed in past EPA risk assessments. Additional data are presented for other activity patterns that can be used for special subpopulations (athletes, outdoor workers), as well as to more accurately reflect the actual exposed population. The EFH does not make any recommendation on the use of distributional data for this parameter for any receptor.

Although the EFH provides no guidance for developing a distribution of inhalation rates, EPA is continuing to work on the development of distributions for exposure factors from the data in the EFH. As part of this effort, Myers et al. (1998) present two fitted distributions, one lognormal and one gamma, for inhalation rate for male and female residents in six age ranges (0-3 years, 4-10 years, 11-18 years, 19-30 years, 31-60 years, and >60 years) based on data from the EFH. Myers et al. found that the difference between the two distributions is negligible and recommend using the lognormal distribution (L. Myers, RTI, personal communication with Anne Lutes, RTI, March 16, 1998).

The parameters of the lognormal distribution (from Table 5.2 in Myers et al.) are presented in Table 7-1. For adult residents, we used ages 30 to 60 years (there is little difference between this and other adult age cohorts, which are 18 to 30 and over 60). It is simpler to use one representative age group than to pool the data and develop an overall distribution. For children, we used the age cohorts 0 to 3, 4 to 10, and 11 to 18 years of age as presented. The

Table 7-1. Estimated Parameters for Inhalation Rate for Residents Assuming Lognormal Distribution

Age (yr)	Gender	Mean (m ³ /d)	CV (%)	Std Dev (m ³ /d)	50%ile (m ³ /d)	90%ile (m ³ /d)	95%ile (m ³ /d)	99%ile (m ³ /d)
0-3	Male	7.52	73	5.49	6.1	14.1	17.8	27.9
0-3	Female	5.75	71	4.08	4.7	10.7	13.4	20.8
4-10	Male	9.30	30	2.79	8.9	13.0	14.5	17.7
4-10	Female	8.65	31	2.68	8.3	12.1	13.5	16.6
11-18	Male	14.58	36	5.25	13.7	21.4	24.2	30.6
11-18	Female	10.76	31	3.34	10.3	15.1	16.8	20.7
19-30	Male	16.75	31	5.19	16.0	23.7	26.5	32.6
19-30	Female	11.14	30	3.34	10.7	15.6	17.3	21.2
31-60	Male	16.32	32	5.22	15.6	23.2	25.9	32.0
31-60	Female	10.95	29	3.18	10.5	15.1	16.7	20.3
>60	Male	12.69	34	4.31	12.0	18.4	20.7	25.9
>60	Female	10.44	29	3.03	10.0	14.5	16.0	19.5

Std Dev = Mean × CV.

Note: Bolded values were used in this analysis.

Source: Myers et al., 1998, Table 5.2.

specific values that were used to define the distribution for the Monte Carlo analysis are shown in bold.

The data in the EFH are not adequate to develop a distribution of inhalation rates for workers. Therefore, a point estimate was used for the inhalation rate for workers. Table 7-2 summarizes the values for inhalation rate for workers presented in the EFH. The recommended hourly average of 1.3 m³/h was used. To convert this to a daily value, an 8-h workday was assumed, yielding a daily inhalation rate for workers of 10.4 m³/d.

To calculate risk for children on a year-by-year basis, inhalation rates must be generated in each Monte Carlo iteration for each age cohort through which the child might age. Inhalation rate in one cohort is strongly correlated with inhalation rate in the previous and subsequent cohorts: a child in the 80th percentile for inhalation rate from 0 to 3 years of age is not likely to be in the 20th percentile for inhalation rate from 4 to 10 years. Selecting the inhalation rates for each cohort independently presumes a correlation coefficient of 0 between cohorts. It would be preferable to account for the correlation. Unfortunately, no data documenting such a correlation quantitatively were readily available. The true correlation coefficient should be between 0 (meaning no correlation) and +1 (meaning a perfect correlation, in which the value of one parameter increases as the other increases) and is probably closer to +1 than 0; therefore, in the absence of data to support a more precise estimate of the correlation coefficient, we set it to +1. This ensures that inhalation rates remain roughly the same percentile from cohort to cohort. A correlation of less than +1 would result in more variation between the percentiles of the two correlated values.

Table 7-2. Recommended Inhalation Rates for Workers

Activity Type	Mean (m³/h)	Upper Percentile (m³/h)
Slow activities	1.1	NA
Moderate activities	1.5	NA
Heavy activities	2.5	NA
Hourly average	1.3	3.3

NA = Not available.

Note: Bolded values were used in this analysis.

Source: U.S. EPA, 1997c, Table 5-23.

7.11.2 Body Weight

The EFH provides data on body weight for males and females by age. For children, body weights are provided for individual years (e.g., age 5, age 6, etc.). For adults, data are provided by 10-year age groups as well as for all adults ages 18 to 75. The EFH presents mean and standard deviation for each gender and age group. In addition, means (but no standard deviations) are presented by age for both genders pooled.

To develop distribution statistics from the EFH, we used EFH Tables 7-2 (adults, including workers) and 7-3 (children). These data are summarized here in Tables 7-3 and 7-4. A lognormal distribution was found to best fit these data and was used.

For adults (residents and workers), we used the mean and standard deviation for adults ages 18 to 75 years from Table 7-2. For children, we used year-by-year means and standard deviations from Table 7-3; for age 0, we used the statistics for 6- to 11-month-olds. These two parameters are sufficient to define a lognormal distribution.

To calculate risk for children on a year-by-year basis, body weights must be generated in each Monte Carlo iteration for each age from 0 to 18. Body weight in one year is strongly correlated with body weight in the previous and subsequent years: a child in the 80th percentile for weight one year is not likely to be in the 20th percentile for weight the next year. Selecting these annual body weights independently presumes a correlation coefficient of 0 between years. It would be preferable to account for the correlation. Unfortunately, no data documenting such a correlation quantitatively were readily available. The true correlation coefficient should be between 0 (meaning no correlation) and +1 (meaning a perfect correlation in which the values of one parameter increases as the other increases) and is probably closer to +1 than 0; therefore, in the absence of data to support a more precise estimate of the correlation coefficient, we set it to +1. This ensures that body weights remain roughly the same percentile from year to year. A correlation of less than +1 would result in more variation between the percentiles of the two correlated values.

Table 7-3. Body Weights for Adults, Ages 18-74 Years (kg)

Age (years)	Males		Females	
	Mean	Std Dev	Mean	Std Dev
18-74	78.1	13.5	65.4	14.6
18-24	73.8	12.7	60.6	11.9
25-34	78.7	13.7	64.2	15.0
35-44	80.9	13.4	67.1	15.2
45-54	80.9	13.6	68.0	15.3
55-64	78.8	12.8	67.9	14.7
65-74	74.8	12.8	66.6	13.8

Note: Bolded values were used in this analysis.

Source: U.S. EPA, 1997c, Table 7-2.

Table 7-4. Body Weights for Children, Ages 6 Months to 18 Years (kg)

Age (years)	Males		Females	
	Mean	Std Dev	Mean	Std Dev
6-11 months	9.4	1.3	8.8	1.2
1	11.8	1.9	10.8	1.4
2	13.6	1.7	13.0	1.5
3	15.7	2.0	14.9	2.1
4	17.8	2.5	17.0	2.4
5	19.8	3.0	19.6	3.3
6	23.0	4.0	22.1	4.0
7	25.1	3.9	24.7	5.0
8	28.2	6.2	27.9	5.7
9	31.1	6.3	31.9	8.4
10	36.4	7.7	36.1	8.0
11	40.3	10.1	41.8	10.9
12	44.2	10.1	46.4	10.1
13	49.9	12.3	50.9	11.8
14	57.1	11.0	54.8	11.1
15	61.0	11.0	55.1	9.8
16	67.1	12.4	58.1	10.1
17	66.7	11.5	59.6	11.4
18	71.1	12.7	59.0	11.1

Source: U.S. EPA, 1997c, Table 7-3.

Body weights and inhalation rates were not correlated with each other. While there may be some correlation between these variables, there are no data to provide a quantitative estimate. Therefore, these variables were not correlated.

7.11.3 Exposure Duration

Exposure duration was determined using data on residential occupancy from the EFH, Table 15-168. These data are presented here in Table 7-5. These data represent the total time a person is expected to live at a single location, based on age. This table presents males and females combined, and there are not sufficient data in the EFH to develop separate distributions for males and females. Therefore, the pooled distribution was used for all iterations of the Monte Carlo analysis.

For adult residents, we pooled age groups from 21 to 90. For children, we used the 3-yr-old age group for the 0 to 3 cohort. We pooled the 6- and 9-yr-old age groups for the 4 to 10 cohort. We pooled the 12-, 15-, and 18-yr-old age groups for the 11 to 18 cohort.

Myers et al. (1998) found that the residential occupancy data were best fit by a Weibull distribution. The Weibull distribution as implemented in Crystal Ball[®] is characterized by three parameters: location, shape, and scale. Location is the minimum value, and in this case was presumed to be 0. Shape and scale were determined by fitting a Weibull distribution to the pooled data, as follows.

To pool residential occupancy data for each of the four desired age cohorts, we formed an arithmetic mean of data means for each of the age groups that we desired to pool (i.e., for children ages 4-10, we took the mean of the data means for the 6-yr-old age group and the 9 yr-old age group). Then, assuming a Weibull distribution, we calculated the variance within each age group (e.g., 6-yr-olds) in the age cohort. These variances in turn were pooled over the age cohort using equal weights. This is not the usual type of pooled variance, which would exclude the variation in the group means. However, we wanted the overall variance to reflect the variance of means within the age groups (e.g., within the 6-yr-old age group). The standard deviation was estimated as the square root of the variance. The coefficient of variation (CV) was calculated as the ratio of the standard deviation divided by the Weibull mean. For each cohort, we then took the Weibull distribution that agreed with the given calculated data mean for the age cohort and the CV as calculated above. The Weibull parameters used are presented in Table 7-6.

Exposure duration for adults was capped at 51 years. This is the maximum exposure duration, starting at age 19, that will not exceed the 70-year lifetime assumption implicit in the averaging time used. For landfills, exposure duration was further limited to 20 years, the assumed operating life of landfills in this analysis.

For workers, the typical default exposure values used by EPA in the past were an 8-h shift, 240 d/wk, for 40 years. The EFH presents data on occupational mobility that are in stark contrast to the assumed value of 40 years at a single place of employment. As presented in the EFH, the median occupational tenure of the working population (109.1 million people) ages 16 years of age and older in January 1987 was 6.6 years. Since the occupational tenure varies significantly according to age, the EFH recommends using age-dependent values. When age cannot be determined, use of the median tenure value of 6.6 years for working men and women 16 years and older is recommended.

Table 7-5. Descriptive Statistics for Residential Occupancy Period by Age (years)

Current Age (years)	Percentile					
	25	50	75	90	95	99
3	3	5	8	13	17	22
6	4	7	10	15	18	22
9	5	8	12	16	18	22
12	5	9	13	16	18	23
15	5	8	12	16	18	23
18	4	7	11	16	19	23
21	2	4	8	13	17	23
24	2	4	6	11	15	25
27	3	5	8	12	16	27
30	3	6	9	14	19	32
33	4	7	11	17	23	39
36	5	8	13	21	28	47
39	5	9	15	24	31	48
42	6	11	18	27	35	49
45	7	13	20	31	38	52
48	8	14	22	32	39	52
51	9	15	24	33	39	50
54	9	16	25	34	40	50
57	10	17	26	35	41	51
60	11	18	27	35	40	51
63	11	19	27	36	41	51
66	12	20	28	36	41	50
69	12	20	29	37	42	50
72	13	20	29	37	43	53
75	13	20	29	38	43	53
78	12	19	29	38	44	53
81	11	20	29	39	45	55
84	11	19	28	37	44	56
87	10	18	29	39	46	57
90	8	15	27	40	47	56

Source: U.S. EPA, 1997d, Table 15-168.

Table 7-6. Estimated Weibull Parameters for Exposure Duration

Age (years)	Location	Scale (Alpha)	Shape (Beta)
0-3	0	7.059	1.32
4-10	0	9.467	1.69
12-18	0	9.949	1.73
Adult	0	17.38	1.34

For this study, it was not possible to find age distribution data for people employed in the waste management sector. Nor was there sufficient information in the EFH to develop a distribution for exposure duration. Since full-time exposure was assumed (8 hr/day, 250

day/year) a point estimate for all full-time workers (male and female) of 7.2 years was used (EFH Table 15-160).

7.11.4 Exposure Frequency

Exposure frequency is the number of days per year that a receptor is exposed. It is not expected to vary much, so no distributions were developed for exposure frequency. A value of 350 d/yr was used for adult and child residents, and a value of 250 d/yr was used for workers. These are based, respectively, on 7 d/wk and 5 d/wk for 50 wk/yr to account for the receptor being elsewhere on vacation for 2 wk/yr.

7.12 Stability Analysis of the Monte Carlo Model

A systematic evaluation of the stability of the chronic risk Monte Carlo model was undertaken. The model was found to be stable at 1,000 iterations.

The chronic risk model varies exposure factors and receptor location in the Monte Carlo iterations. Exposure factors varied include body weight, inhalation rate, and exposure duration. The receptor location is varied among 16 directions from the site, each of which may occur with equal probability. Different receptor distances from the site are assessed in separate model runs, and the results maintained separately, so distance is not varied in the Monte Carlo analysis.

It was not feasible to run the stability analysis for all chemicals, WMUs, and sites due to the long runtime of the risk model. Therefore, a selection of chemicals, WMUs, and sites were chosen that represented all of the variables that could influence the stability. These included different receptors (because they have different exposure factors), and different meteorological locations, source areas, and source heights (because these could influence the variability of air concentration with sector). In all, we ran the stability analysis for three carcinogens (acrylonitrile, methylene chloride, and nitrosodi-*n*-butylamine), all six distances from the source, all five receptors, and a selection of sites (25 LAUs and 28 wastepiles). The sites were selected to represent all meteorological stations at which there were wastepiles or LAUs, a range of source areas, and a range of source heights for wastepiles. Source area, height, and meteorological location all potentially affect how air concentration varies by sector.

To assess the stability of the Monte Carlo model, we ran the model for the scenarios described above and extracted the 90th percentile results after 1,008, 2,000, 3,008, 4,000, 5,008, 6,000, 7,008, and 8,000 iterations. We then compared the 90th percentile results across number of iterations. In all cases, the number of iterations was chosen to be a multiple of 16 because of the approach used to select the sector values.

In all, we ran 4,764 chemical/site/distance/receptor combinations through the stability test. We then compared the 90th percentile results for each run, rounded to one significant figure, across the different numbers of iterations. Figure 7-2 shows some typical results for which the result varied slightly over 8,000 iterations. The 3 lower lines all round to a value of 40 at 1,008 iterations; the 3 upper lines all round to a value of 60 at 1,008 iterations. For all lines shown, the

rounded value at 8,000 iterations is 50; note that any value falling between 45 and 55 will round to 50.

Table 7-7 summarizes the results. In 88 percent of the runs, the result did not change at all from 1,008 iterations to 8,000 iterations. For another 12 percent of the runs, we saw minor fluctuations in the value (e.g., between 2.48 and 2.52) that result in fluctuations in the result when rounded to one significant figure. These fluctuations never resulted in a change of more than 1 digit in the single significant figure. This was not considered significant.

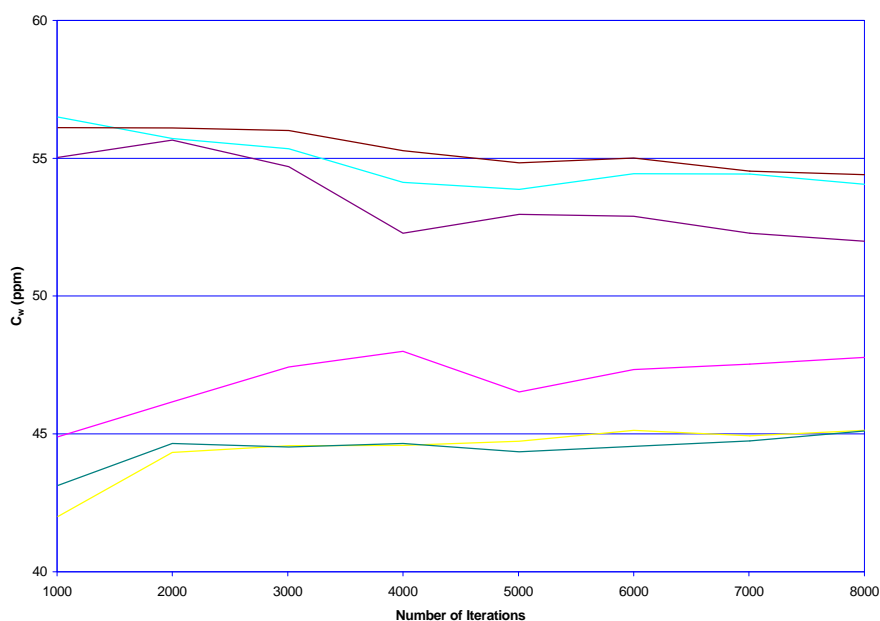


Figure 7-2. Stability analysis results showing typical variations resulting in change in results at one significant figure.

Table 7-7. Summary of Stability Analysis

Amount of change from 1008 to 8000 iterations	Number of runs	Percent of runs
None	4,173	88
1 digit	591	12
Total	4,764	

7.13 Modifications to Methodology for Lead

Human health risk assessment for lead is unique. Instead of developing an RfC in the traditional manner, all identified sources of lead exposure (including background) are used to predict blood lead (PbB) levels in the exposed individuals. The predicted PbB levels are compared to a target PbB. PbB levels have long been used as an index of body lead burdens and as an indicator of potential health effects.

The Integrated Exposure Uptake Biokinetic Model (IEUBK) (U.S. EPA, 1994a) was developed to predict PbB levels for an individual child or a population of children. The model was specifically designed to evaluate lead exposure in young children (birth to 7 years of age) because this age group is known to be highly sensitive to lead exposure.

The IEUBK is a versatile risk assessment tool that allows the user to make rapid calculations from a complex array of intake, absorption, distribution, and elimination equations. Default values representing urban background exposures to lead from soil and dust ingestion, air, food, and water are built into the model.

The IEUBK has four primary components: exposure, uptake, biokinetic, and probability distribution. The exposure component integrates 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. Thus, the exposure component determines how much lead enters the child's body over the exposure period. The uptake component calculates how much of the lead that is ingested or inhaled is actually absorbed into the blood and the biokinetic component models the distribution of lead from the blood to other body tissues and/or elimination from the body. The final component calculates a plausible probability distribution of PbB for a hypothetical child. The geometric mean PbB is calculated and the probability of exceeding a target PbB is determined.

For this analysis, the IEUBK model was used to identify air concentrations that would result in a probability of less than 5 percent of having a PbB level higher than the target PbB and that could be used in place of an RfC in the calculations. Because the IEUBK model cannot be run in a backcalculation mode, different air concentrations were modeled until one was found that satisfied the 95 percent protection level desired.

Only two receptors were modeled for lead: children ages 0 to 3 years and children ages 3 to 7 years. Adults (including workers) and older children were excluded from the analysis for lead because those age groups are considered less sensitive to lead than 0- to 7-year-olds (and, in fact, the pharmacokinetic relationships in the IEUBK model are only valid for 0- to 7-year-olds).

The IEUBK model inputs are summarized in Table 7-8. These include inhalation rate, body weight, media concentrations (including soil, indoor dust, water, and food), and indoor air concentration as a percentage of outdoor air concentration. The IEUBK model does not support Monte Carlo analysis, so these inputs must be entered as point estimates. To be as consistent as possible with the distributions developed for exposure factors, the median body weight and inhalation rates from the distributions for 0- to 3- and 3- to 10-year-olds were used for 0- to 3- and 3- to 7-year-olds, respectively. Ingestion rate for water was left at the default age-specific values recommended in the IEUBK guidance document (U.S. EPA, 1994a).

Media concentrations were left at the standard background levels. Two background soil concentrations were modeled: 75 mg/kg and 200 mg/kg. These values correspond to the range of typical soil lead levels in urban soil (U.S. EPA, 1994a). Indoor dust concentrations were set to equal soil concentrations and indoor air lead concentrations were equal to outdoor concentrations (the normal default is indoor air = 30 percent of outdoor air). Therefore, it was assumed that children would be exposed 24 h/d to constant air lead concentrations. This was done for consistency with other constituents for which exposure was assumed to occur 24 h/d with no distinction between indoor and outdoor air made.

Table 7-8. Summary of Inputs for IEUBK Model

Input	Age (yr)	Value	Source
Exposure Factors			
Inhalation rate (m ³ /d)	0-3	6.1	Median of distribution for 0- to 3-yr-olds
	3-7	8.9	Median of distribution for 3- to 10-yr-olds
Body weight (kg)	0-3	12.63	Median of distribution for 0- to 3-yr-olds
	3-7	25.9	Median of distribution for 3- to 10-yr-olds
Media Concentrations			
Soil (µg/g)		75 to 200	U.S.EPA (1994a)
Indoor dust (µg/g)		75 to 200	U.S.EPA (1994a)
Water (µg/L)		4	U.S.EPA (1994a)
Food (µg/d)		Varies by age	U.S.EPA (1994a)
Indoor Air Concentration (% of outdoor air conc.)		100%	Assumption/consistency with other constituents

Model outputs were produced for three target blood lead levels (10, 8, and 5 µg/dL) and two assumed background soil concentrations (75 and 200 µg/g); these are shown in Table 7-9. Depending on the target PbB and the background soil concentration, acceptable air lead concentrations ranged from background to 3.3 µg/m³. For comparison, the default background air lead concentration used by the IEUBK is 0.1 µg/m³.

For some combinations of target blood lead level, background soil concentration, and age, the background exposures alone led to a probability greater than 5 percent that the blood lead target would be exceeded. This would mean that no exposure to lead in air could be allowed.

A single air concentration for each age range was needed for use in the Monte Carlo analysis as a surrogate for the RfC. The blood lead level of 10 µg/dL was selected because that level has been identified as a level of concern by the Centers for Disease Control (CDC) (U.S. EPA, 1994a). The IEUBK guidance recommends a background soil concentration of 200 µg/g; therefore, that concentration was used. This resulted in air concentrations of 0.5 and 1.8 µg/m³ for the 0- to 3- and 3- to 7-year-old, respectively. These air concentrations were used in the Monte Carlo analysis described above in lieu of a health benchmark.

Table 7-9. Results of IEUBK Modeling

Target Blood Level ($\mu\text{g/dL}$)	Assumed Soil/Dust Background Concentration (mg/kg)	Air Concentration ^a ($\mu\text{g/m}^3$)	
		0- to 3-yr-old	3- to 7-yr-old
10	200	0.5	1.8
8	200	0 ^b	0.6
5	200	0 ^b	0 ^b
10	75	2.4	3.3
8	75	1.25	2.1
5	75	0 ^b	0.35

^a Air concentration at which there is a 5% probability of exceeding the target blood lead level.

^b Background exposures alone result in >5% probability of blood lead exceeding target level.

8.0 Analysis of Variability and Uncertainty

The purpose of this section is to discuss the methods that are used in this study to capture variability and uncertainty. Variability and uncertainty are discussed separately because they are fundamentally different. Variability represents true heterogeneity in characteristics such as body weight differences within a population or differences in contaminant levels in the environment. It accounts for the distribution of risk within the exposed population. Uncertainty, on the other hand, represents lack of knowledge about factors such as adverse effects from contaminant exposure, which may be reduced with additional research to improve data or models.

Variability arises from true heterogeneity in characteristics such as body weight differences within a population or differences in contaminant levels in the environment.

Uncertainty represents lack of knowledge about factors, such as the nature of adverse effects from exposure to constituents, which may be reduced with additional research.

This discussion describes the treatment of variability in some parameters used to describe human receptors and their behavior. Treatment of variability using a Monte Carlo simulation forms the basis for the risk distributions. Uncertainty necessitated the use of assumptions, default values, and imputation techniques in this study. These are discussed so that decision makers can understand the limitations of the analysis. Taken as a whole, this discussion focuses on how this treatment of variability and uncertainty affects the representativeness and accuracy of the results.

8.1 Variability

In conducting a national risk assessment, numerous parameters will vary across the nation. Variability is often used interchangeably with the term “uncertainty,” but this is not strictly correct. Variability is tied to variations in physical, chemical, and biological processes and cannot be reduced with additional research or information. Although variability may be known with great certainty (e.g., age distribution of a population may be known and represented by the mean age and its standard deviation), it cannot be eliminated and needs to be treated explicitly in the analysis. Spatial and temporal variability in parameter values used to model exposure and risk account for the distribution of risk in the exposed population.

An example is meteorological parameters used in dispersion modeling, such as wind speed and wind direction. These parameters are measured hourly by the National Weather Service at many locations throughout the United States, and statistics about these parameters are well documented. While the distributions of these parameters may be known, their actual values

vary spatially and temporally and cannot be predicted exactly. Thus, the concentration calculated by a dispersion model for a particular receptor for a particular time period will provide information on average conditions that may over- or underpredict actual concentrations. Use of models such as ISCST3 that calculate concentrations hourly and sum these hourly values to provide annual concentrations estimates account for much of the temporal variation. Additionally, using meteorological data from multiple monitoring stations located throughout the U.S. accounts for some but not all spatial variability.

In planning this analysis, it was important to specifically address as much of the variability as possible, either directly in the Monte Carlo analysis or through disaggregation of discrete parts of the analysis. For example, use of a refined receptor grid accounts for spatial variability in concentrations around an WMU. Variability in WMU characteristics is accounted for using a large database of individual WMUs that represent the range of possible WMU characteristics. Previous sections describe how distributions and point value estimates were made for input parameters and how these were combined through design of the analysis to provide facility-level and national level distributions of C_w . Variability is discussed in this section to provide an overview of the modeling techniques used to address variability. The risk assessment components discussed included:

- # Source characterization and emissions modeling
- # Fate and transport modeling
- # Exposure modeling.

8.1.1 Source Characterization and Emissions Modeling

Biodegradation in WMUs affects emissions by reducing the amount of waste constituent that could be released to the atmosphere. Biodegradation rates are affected by site-specific parameters. Of the many site specific-values that could affect biodegradation rates, only temperature was considered in their analysis. Biodegradation was assumed to occur at temperatures greater than 5°C. In order to turn biodegradation on and off, seasonal temperatures were calculated for each site based on region-specific meteorological data. The use of seasonal temperature data was intended to reduce the error in both the biodegradation rate and the temperature-dependent volatilization rate in land-based WMUs. The landfill scenario assumed that no biodegradation occurred, so temperature was a less important parameter for this WMU than for the land application unit or wastepile. In the land application units, temperature was varied monthly, as were other meteorological parameters. Monthly variation was used to capture the interaction between meteorology, waste application, and tilling.

A single biodegradation rate was used for each constituent. This biodegradation rate was applied to all WMUs in all locations. Site-specific conditions may cause the biodegradation rate to be greater or less than the default value used, but this variability was not captured by this analysis.

The effect of incorporating seasonal temperature variations in the analysis was chemical-dependent. For most chemicals in the analysis, the difference between using seasonal temperatures or an annual average temperature resulted in little or no change in the overall annual emissions. For a few chemicals, however, there was a very large impact for certain

locations in the United States. These chemicals had rapid biodegradation rates, and turning the biodegradation off for the winter months resulted in significantly higher annual emissions.

8.1.2 Fate and Transport Modeling

To capture geographic variation, dispersion modeling was conducted using meteorological datasets from 29 different meteorological stations around the continental United States. This provided regional representation of the variability in meteorological data. For landfills and land treatment units, these datasets were combined with 14 surface areas representing the distribution of WMU size. Combined, this provides 406 different sets of unit air concentrations (UACs) to use with emissions data to estimate air concentrations. As discussed below, a set of UACs consists of all UACs at 96 combinations of distances and directions at one meteorological location. Similarly, wastepiles used 812 different sets of UACs based on 29 surface area/wastepile height combinations and 29 meteorological stations. Tanks used the same 29 meteorological stations and 33 surface area/tank height combinations, providing 957 sets of UACs.

The location of receptors was an important source of variability addressed in the exposure modeling. The data used to identify and characterize WMUs contained no information on the location and types of receptors relative to the facility. Many previous risk analyses have used the maximum point of exposure at some prespecified distance from the WMU as the point for analysis. Such an approach is usually criticized as being overly conservative because it does not consider the possibility of no one's living at that exact point. Because individuals may potentially be located in any direction and at various distances from a facility, this analysis developed an explicit way to incorporate this consideration. First, a sensitivity analysis was conducted to determine a reasonable distance at which to bound the analysis. This sensitivity analysis showed that, beyond 1,000 meters, most air concentrations are a small percentage (less than 10 percent) of the concentration at the point of maximum exposure. Therefore, 1,000 meters was used as the outer bound on the distance of receptors included in this analysis. Next, a receptor grid was set up to allow individuals to reside in any of 16 directions and at distances of 25, 50, 75, 150, 500, and 1,000 meters from the edge of the unit. The Monte Carlo analysis uses a uniform distribution that gives equal probability to a receptor's being located in any of 16 directions at each distance (96 combinations).

Obviously, 29 meteorological stations do not represent every site-specific condition that could exist in the continental United States. Based on EQM (1993), however, it is believed that these stations provide a reasonable representation of the variability in meteorological conditions for the U.S. climate regions. Quantifying the error associated with the use of this limited number of meteorological datasets would require comparison of results with more site-specific data, which has not been done. A detailed analysis of the wind rose for the 29 meteorological stations is presented in Appendix C.

8.1.3 Exposure Modeling

The exposure factors used in the analysis, including inhalation rate, body weight, and duration of exposure, are quite variable. To include this variability explicitly in the analysis, distributions for these variables were used for each receptor in the analysis: workers, adult

residents, and child residents. For adults, male and female exposure factors were developed separately. For child exposures, three age groups (0 to 3, 4 to 10, and 11 to 18), representing age at the start of exposure, were considered. Body weight was developed for each age and inhalation rate for each child cohort group based on the data available. Thus, for each Monte Carlo realization for each receptor type, values for each of these variables were selected from the specified distribution.

At each WMU modeled, a distribution of C_w was developed that incorporated the variability in the location of the receptor and the variability in the exposure factors used to characterize the receptor. Note that the Monte Carlo analysis for noncarcinogenic effects incorporates variability only about receptor location since exposure factors are not used in the calculation.

The overall output of the analysis includes specific consideration of the variability in site-specific WMU information, regional-specific meteorological conditions, location of receptors, and exposure factors for each receptor to provide national distributions of a risk-specific waste concentration across all facilities of a specified type. These distributions are presented in Volume III.

8.2 Uncertainty

Uncertainty is a description of the imperfection in knowledge of the true value of a particular parameter. In contrast to variability, uncertainty is reducible by additional information gathering or analysis activities (better data, better models). EPA typically classifies the major areas of uncertainty in risk assessments as scenario uncertainty, model uncertainty, and parameter uncertainty. Scenario uncertainty refers to missing or incomplete information needed to fully define exposure and dose. Model uncertainty is a measure of how well the model simulates reality. Finally, parameter uncertainty is the lack of knowledge regarding the true value of a parameter that is used in the analysis.

While some aspects of uncertainty were directly addressed in the analysis, much of the uncertainty associated with this analysis could only be addressed qualitatively. Significant sources of uncertainty are presented in this section and are discussed with regard to how uncertainty was addressed. If the analysis directly addressed uncertainty, the approach used is described. If the analysis did not directly address uncertainty, a qualitative discussion of its importance is provided.

8.2.1 Scenario Uncertainty

Sources of scenario uncertainty include the assumptions and modeling decisions that are made to represent an exposure scenario. Because we lack information or resources to define and model actual exposure conditions, uncertainty is introduced into the analysis. Despite the complexity of this analysis, it was necessary to exclude or simplify actual exposure conditions. For example, this analysis only addresses inhalation exposures; indirect exposure pathways were excluded. Professional judgement, often coupled with using the results of sensitivity analysis, is used to decide which parameters to include in describing exposure conditions and behaviors.

These judgements are imperfect and uncertainty is introduced. Some of the uncertainties that are important to understand in interpreting the results of this study are discussed below.

Emissions from WMUs that were modeled for this study included emissions from the WMU itself (e.g., the surface of an open tank). Emissions from ancillary operations associated with the WMU were not considered. For land-based WMUs, one source of particulate emissions that was not included is vehicular traffic associated with trucks and other facility operations. These vehicles entrain particulate material to the air from utility roads at WMUs, which was not included in the model used in this analysis.

This risk analysis addresses only inhalation exposure to humans, a direct exposure pathway. Indirect exposures include exposure pathways such as contaminated produce or meats due to the uptake and bioaccumulation of contaminants in the food chain. Although this analysis focused only on releases of the contaminants to air, it is possible that these contaminants could accumulate in plants through air-to-plant transfer mechanisms or by direct deposition onto plants. Animals could then come into contact with contaminated plants, or even contaminated soil, and accumulate the contaminant in their bodies (bioaccumulation). Because of the short time frame of this project and the complexity associated with modeling indirect risks, the consent decree with EDF did not require the Agency to evaluate this pathway.

EPA conducted a preliminary analysis to determine if any constituents included in this analysis have the potential to be taken up by plants and accumulated to the point of presenting a potential concern for humans or domestic animals, such as beef or dairy cows that might graze in fields adjacent to a WMU. EPA looked at a waste stream in which polycyclic aromatic hydrocarbons (PAHs) were the constituents of concern from a risk perspective. Releases from an land application units (LAUs) were modeled with receptors located randomly between 75 and 1,000 m. The concentration of constituents in contaminated produce could be attributed to three mechanisms: vapor transfers, direct deposition from air, and uptake from soil, with vapor transfers being the predominant mechanism of contamination. The risk from ingestion of contaminated produce as a percent of total risk from all ingestion pathways is quite significant for the farmer scenario and less significant for the home gardener.

Two important points need to be made regarding this modeling exercise. First, the vapor transfer coefficients used for PAHs are very high and are thought to have a high level of uncertainty. Studies are currently under way to address this uncertainty, but the results of such studies are not available at this time. One criticism is that these vapor transfer coefficients greatly overestimate the concentration of PAHs in plants and thus lead to overestimates of risk. Second, the farmer scenario has much higher risk from homegrown produce than the home gardener due to differences in ingestion rates for these two groups. At this time, it is not known which scenario would be the most appropriate to use for the air characteristic analysis, nor is information available about the prevalence of farmers or home gardeners in the vicinity of these types of facilities.

Although this sensitivity analysis suggests that indirect pathways may be important to consider in the air characteristic risk analysis, this analysis is very limited in terms of constituents, WMUs, settings, and scenarios. In a sense, this sensitivity analysis should be

considered a bounding analysis because it is based on a set of inputs that are biased toward determining if the air-to-plant pathway could be a concern for any of the constituents.

8.2.2 Model Uncertainty

To reduce model uncertainty, EPA generally selected models that are considered state-of-the-art. Model uncertainty is associated with all models used in all phases of a risk assessment. These include the animal models used as surrogates for testing human carcinogenicity, dose-response models used in extrapolations, and computer models used to predict the fate and transport of chemicals in the environment. Computer models are simplifications of reality, requiring exclusion of some variables that influence predictions but cannot be included in models due either to increased complexity or to a lack of data on a particular parameter. The risk assessor needs to consider the importance of excluded variables on a case-by-case basis because a given variable may be important in some instances and not in others. A similar problem can occur when a model that is applicable under average conditions is used when conditions differ from the average. In addition, choosing the correct model form is often difficult when conflicting theories seem to explain a phenomenon equally well. Modeling uncertainty is not addressed directly in this study but is discussed qualitatively in this section.

With regard to model error, the CHEMDAT8 model was used for all volatile emissions estimates including acute and subchronic releases and as the basis for estimating soil concentrations that are used in the particulate emissions model. As discussed in Section 4.1, there are many features of this model that meet the needs of this analysis. The model, however, was developed to address only volatile emissions from the waste management units considered in this study. Competing mechanisms such as runoff and erosion and leaching are not included in the model. Inasmuch as these competing processes actually occur, the model would tend to slightly overestimate the volatile emissions and waste/soil concentrations in the WMU. On the other hand, one could interpret this situation as being representative of WMUs that have leachate controls, such as liners, or erosion and runoff controls. Such controls would tend to inhibit these processes and result in more volatile emissions. Similar lines of argument hold for the calculation of a waste/soil concentration that is the basis for estimating particulate emissions of a contaminant.

The ISCST3 model was used to calculate the dispersion of particle and gas emissions from a WMU. This model has many capabilities needed for this assessment, such as the ability to model area, volume, or point sources for chronic, subchronic, and acute averages. For dispersion modeling of this type, it is considered to be a fairly accurate model, with error within about a factor of 2. It does not include photochemical reactions or degradation of a chemical in the air, which results in additional model uncertainty for some chemicals. In addition, this analysis did not use wet depletion but did use dry plume depletion for particulates. As presented in Section 5 and discussed in the sensitivity analysis in Appendix C, wet deposition and associated depletion accounts for only about 2 percent of total depletion. Wet depletion, therefore, was not used in this analysis because of the time required to properly process the requisite precipitation data required for the wet depletion option. Dry deposition and associated plume depletion are important for particulates and are explicitly incorporated into this analysis.

Dispersion modeling is highly sensitive to meteorological data and the surface area and dimensions of the WMU. Meteorological data used in the dispersion modeling include windspeed and direction, temperature, precipitation type and amount, and stability class. The ISCST3 model uses hourly data as inputs, and this analysis used 5 years of hourly data to develop chronic (i.e., 1-year), subchronic (i.e., 30-day) and acute (i.e., 1-day) UACs, as discussed in Section 5.

8.2.3 Parameter Uncertainty

Parameter uncertainty occurs when (1) there is a lack of data about the parameters used in the equations, (2) the data that are available are not representative of the particular instance being modeled, or (3) parameter values cannot be measured precisely and/or accurately either because of equipment limitations or because the quantity being measured varies spatially or temporally. Random, or sample errors, are a common source of parameter uncertainty that is especially critical for small sample sizes. More difficult to recognize are nonrandom or systematic errors that result from bias in sampling, experimental design, or choice of assumptions.

Source characterization can introduce parameter uncertainty into the analysis through survey techniques used to characterize waste management units and associated parameter values. As discussed in Section 3.1, existing databases were used to identify WMUs and as a basis for determining important emissions and dispersion model input parameter values. The *Industrial Subtitle D Survey* (Schroder et al., 1987) was used to characterize landfills, LAUs, and wastepiles. Because the Industrial D database did not survey tanks, characterization of tanks was based on the *1986 National Survey of Hazardous Waste Treatment, Storage, Disposal, and Recycling Facilities* (U.S. EPA, 1987).

These databases were used to determine physical and operating characteristics for the WMUs modeled. The impact of the uncertainty associated with the information contained in these databases is unknown. There are several sources of this uncertainty, including age of the data, representativeness, missing data on waste volumes or capacity, multiple WMUs of the same type associated with a combined surface area and waste volume, accuracy of the reported data (i.e., measurement error), and limited information on WMU operating characteristics. Because these surveys were completed in 1987, uncertainty exists concerning changes in waste management practices since 1987. This is especially true for the tanks data, which may result in an underestimation of highly aerated biological treatment tanks. Underestimation of the number of highly aerated treatment tanks would result in lower emissions estimates and higher C_w s.

The data were reviewed to assess the representativeness of the survey data for the WMUs evaluated in this analysis. For example, the tank database contained many small “tanks,” or containers that were not representative of the tanks that are the target of this assessment. Tanks less than or equal to 55 gallons were deleted from the database and not included in this study. Similarly, closed tanks were deleted from the database because this analysis was concerned only with open tanks.

Any survey results will introduce uncertainty due to inaccuracy in the way the data were collected and compiled. Inaccuracy can result from respondents’ misinterpreting the survey questions. This uncertainty can be compounded by the imputation techniques used to develop

parameter values for parameters not directly surveyed. The imputation techniques used in this study that increase the uncertainty include:

- # The tank survey provides data on tank volume and flow rate. Tank depth and surface area have to be imputed based on surveyed volume data using supplemental data and engineering judgment about the relationship between tank volume, surface area, and depth.
- # When more than one WMU of the same type existed at a facility, the combined area of those WMUs was reported in the Industrial D database. This analysis used the average area of WMUs of a single type when more than one unit existed at the same facility. This is not a problematic assumption except for a few facilities with large total surface areas. In such cases, it is unknown whether one large WMU dominates this surface area or the area is evenly distributed across the WMUs.
- # The Industrial D survey does not report depth for landfills. Therefore, depth had to be imputed from the total capacity and total area data that were reported. To do this, an assumption about the bulk density of waste deposited in the landfill was made. To the extent that actual bulk density of waste differs from the assumed bulk density, depth would be over- or underestimated.
- # Like landfill depth, waste pile height was not reported in the Industrial D survey. Hence, waste pile height was imputed from total capacity and total area data that were reported. To do this, an assumption was made about the bulk density of waste deposited in the waste pile. To the extent that actual bulk density of waste differs from the assumed bulk density, waste pile height would be over- or underestimated.

Source characterization also requires making assumptions about the way WMUs are operated. For example, LAU surface area is reported in the Industrial Subtitle D Survey but details about operating practices are not. Operators of LAUs may spread waste over the entire surface area, or they may choose to spread waste in cells, rotating from cell to cell until the entire surface area is used and then repeating the process. Tilling practices for LAUs will also vary from operator to operator. Therefore, assumptions made for LAUs about active surface area and tilling frequency and depth introduce uncertainty into the parameter values used to model these units. For landfills, no information was reported on how much of the landfill total area was operational in a given year. Therefore, it was assumed that the landfill operated for 20 years, filling one cell per year, so that cell area was one-twentieth of the total area. In general, however, because this analysis captured wide variations in WMUs, the effect of uncertainties in these parameters should be mitigated.

There was a lack of site-specific values such as waste characteristics that influence emissions calculation. For particular emissions default values were used to characterize all waste streams. Use of default or average values instead of value more specific to the type of waste managed introduces uncertainty into the analysis. In addition, data were not available for the actual molecular weight of the waste, which is a required input to the CHEMDAT8 emissions

model that affects organic-phase emissions (but not aqueous-phase emissions). A default value of 147 g/mol was used. If the actual molecular weight of waste is greater than 147 g/mol, organic phase emissions would be underestimated. Conversely, if the actual molecular weight of waste is less than 147 g/mol, organic-phase emissions would be overestimated.

Biodegradation rates were based on data contained in Howard et al. (1991). Some of these rates have been determined for aqueous systems and adjusted for use in soil systems. While they are often used in this manner, there is some degree of uncertainty associated with making the adjustment for use in soil systems.

To minimize error associated with the use of discrete heights and surface areas in the dispersion modeling, an interpolation routine was used to estimate the UAC. To determine the UAC value used in the risk model, a two-step process was used. First, the UAC values for a height closest to the actual WMU height (for wastepiles and tanks) were used. Using the closest (and not actual) height introduces uncertainty that can over- or underpredict actual concentrations. Then a UAC value was interpolated between the surface areas that bracketed the actual WMU surface area (all WMUs). Areas were chosen to maintain an error of less than 10 percent for tanks. For all other WMUs, areas were chosen to best cover the distribution of areas; although minimizing interpolation error was not an explicit factor, that error is generally less than 25 percent.

Other uncertainties introduced into the analysis in dispersion modeling are related to WMU shape (for land-based WMUs). A sensitivity analysis was conducted (see Appendix C) to determine the sensitivity of different shapes on ambient ground-level air concentrations calculated by ISCST3. A square shape was selected because it minimized the error introduced by not knowing the orientation of the WMU shape to wind direction. The number of receptors selected was also based on sensitivity analyses (see Appendix C). A sparse grid would increase the likelihood of missing peak concentrations, whereas a dense receptor grid would be too resource-intensive for this analysis.

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

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

A key step in CSF development is high- to low-dose extrapolation. Depending on the model used to fit the data, extrapolations to the low dose range can vary by several orders of magnitude, reflecting the potential uncertainty associated with the cancer slope factor.

Reference Doses and Reference Concentrations. Uncertainty in the toxicological and epidemiological data from which reference doses (RfDs) and reference concentrations (RfCs) are derived are accounted for by applying uncertainty factors. An RfD (or RfC) is “an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime” (U.S. EPA, 1999). RfDs and RfCs are based on the no adverse effects level (NOAEL) or lowest observed adverse effects level (LOAEL) for the most sensitive effect in the most sensitive or most relevant species. A series of standard uncertainty factors are applied to the NOAEL or LOAEL to derive the RfD or RfC. The following uncertainty factors account for areas of scientific uncertainty:

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

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

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

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

While most of the uncertainty associated with health benchmarks is common to all risk assessment studies, some uncertainties are specific to this analysis. Data to support the development of subchronic and acute effects were limited. Hence, health benchmark values were drawn from a variety of sources based on a quality hierarchy. This resulted in some chronic health benchmark values' being higher than the corresponding subchronic or acute value. This counterintuitive result comes about because different studies were used that were from different years, used different methods, and were based on different studies. This result occurred for about 14 chemicals in this study.

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Appendix A

Basic Dalenius-Hodges Procedure for Constructing Strata

A. Basic Dalenius-Hodges Procedure for Constructing Strata (applies to census data)

1. Select an available variable X correlated with the variable Y of interest. (Both X and Y are assumed to be continuous.)
2. Form a relative frequency histogram of X with respect to M prespecified intervals with breakpoints $a_0, a_1, a_2, \dots, a_M$. Let f_i denote the frequency count for the i^{th} interval. (Note that M should be fairly large, e.g., at least 50 and preferably 100, since the ultimate strata will be formed as the union of a subset of contiguous intervals. The intervals do not have to be the same length but it may be convenient to use one or two standard lengths.)
3. For each interval i ($i=1,2,\dots,M$), compute $Z_i = \sqrt{L_i f_i}$ where $L_i = a_i - a_{i-1}$ is the length of the i^{th} interval.
4. Compute $CUMZ_i = \sum_{j=1}^i Z_j$.
5. Determine stratum breakpoints by breaking CUMZ into equal intervals of length $Q = CUMZ_M/H$, where H is the desired number of strata (e.g., if $H=5$, then stratum boundaries would be formed at $Q, 2Q, 3Q$, and $4Q$).
6. Sequentially cluster the intervals into strata in order to redefine breakpoints in terms of X variable.
7. Select random samples of size n within each stratum.

In practice, H is normally about 4, 5, or 6. This procedure produces a sample that is near optimal for estimating the population mean of Y if X and Y are highly correlated.

Modification of the Basic Procedure for the Current Context

At Step 2, determine f_i as the sum of the weights of the units falling into the i^{th} interval.¹ At Step 7, select a unit (or facility, depending on whether X and associated weights relate to units or facilities) near the midpoint of each stratum as the “representative” unit. Its percentile in the population of units would be estimated as (sum of weights for all units having $X_s \leq X$ of given unit)/(sum of all weights).

¹ Note that the current weights relate to the population of facilities; to get weights appropriate to units (which one should do if x is defined as a unit-specific measure rather than a facility-specific measure), one should multiply the number of units at a facility by the facility’s weight. As a check, the sum of the current weights should produce an estimate of the number of facilities in the population and the sum of the (weight x number of units) should produce an estimate of the number of units in the population.

The above procedure was applied to distributions for landfills (LFs), land application units (LAUs), surface impoundments (SIs), and wastepiles (WPs). It was also applied to the combined LF/LAU/SI distribution. In each case, the procedure yields a good characterization of the right tail of the distribution, but a poor characterization of the lower portion of the distribution. As a result, the procedure was modified by collapsing two or more of the highest strata into one and successively splitting the lowest stratum into two or more strata. The splitting was performed by choosing stratum breakpoints within the lowest initial at $CUMZ < Q$ (boundary of the initial stratum), $CUMZ < Q/2$, $CUMZ < Q/4$, $CUMZ < Q/8$, etc.

NOTE: If facility weights are used, but X =average unit area, then the distribution being characterized is the facility distribution of average unit areas. A percentile, X_p , of this distribution has the following interpretation: 100P% of the facilities have average unit size $< X_p$. On the other hand, if unit level weights are used, then the P^{th} percentile, X_p , is interpreted as: 100P% of the units have size $< X_p$. The latter is probably the most meaningful, but it can only be approximated since within-facility variation in unit sizes is not available from the survey data.

Appendix B
Chemical-Specific Data

B. Chemical-Specific Data

Key chemical-specific input parameters include: air-liquid equilibrium partitioning coefficient (vapor pressure or Henry's law constant), liquid-solid equilibrium partitioning coefficient (log octanol-water partition coefficient for organics), biodegradation rate constants, and liquid and air diffusivities. The HWIR chemical properties database (RTI, 1995) was used as the primary data source for the physical and chemical properties for the constituents being modeled. This chemical properties database provided the following chemical-specific input parameters: molecular weight, vapor pressure, Henry's law constant, solubility, liquid and air diffusivities, and log octanol-water partition coefficient. The soil biodegradation rate constants were obtained from Howard et al (1991). The CHEMDAT8 chemical properties database (U.S. EPA, 1994) was used as a secondary data source for the physical and chemical properties not included in the HWIR data base. The CHEMDAT8 chemical properties database primarily provided the following chemical-specific input parameters: density, boiling point, Antoine's coefficients (for adjusting vapor pressure to temperature), and biodegradation rate constants for tanks. Hydrolysis rates were taken from Kollig et al. (1993). The biodegradation rate constants in the downloaded CHEMDAT8 data base file were compared with the values reported in the summary report that provided the basis for the CHEMDAT8 tank biodegradation rate values (Coburn et al., 1988). Tank biodegradation rates constants for compounds with no data were assigned biodegradation rates equal to the most similar compound in the biodegradation rate data base (or set to zero for metals). The chemical specific input parameters used for the emission model estimates are presented in Table B-1.

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Table B-1. Chemical Specific Input Parameters

CAS #	COMPOUND NAME	Mol. Wt. (g/mol)	Density (g/cc)	VAP. Press. (mmHg)	H Law Const. (atm-m ³ /mol)	Diffusivity in Water (cm ² /sec)	Diffusivity in Air (cm ² /sec)	Antoinies' Vapor Pressure Coefficients			log Oct Water Part. Coeff.	Kmax mgVO/g-hr.	K1 L/g-hr.	Hydrol. Rate sec-1	Soil Biodeg. Rate sec-1	Solubility mg/L
							A	B	C							
50000	Formaldehyde	30.03	0.97	5240	3.4E-07	1.98E-05	1.78E-01	7.195	971	244	-0.05	5	0.25	0	6.08E-10	5.50E+05
50328	Benzo(a)pyrene	252.32	1.11	5.5E-09	1.1E-06	9.00E-06	4.30E-02	9.246	3724	273	6.11	0.001	0.31	0	4.61E-08	2.50E-02
55185	N-Nitrosodiethylamine	102.14		0.86	3.6E-06	8.00E-06	8.00E-02			273	0.48	4.4	0.45		1.56E-08	9.30E+04
56235	Carbon tetrachloride	153.82	1.59	115	0.0304	8.80E-06	7.80E-02	6.934	1242	230	2.73	1.5	1.50	0	3.13E-08	7.93E+02
56495	3-Methylcholanthrene	268.36	1.02	7.7E-09	9.4E-07	5.36E-06	2.09E-02	8.164	3364	273	6.42	0.001	0.31	0	1.22E-07	3.23E-03
57976	7,12-Dimethylbenz[a]anthracene	256.35	1.02	5.6E-09	3.1E-08	4.98E-06	4.61E-02	6.955	2163	171	6.62	0.001	0.31	0	2.43E-09	2.50E-02
62533	Aniline	93.13	1.02	0.49	1.9E-06	8.30E-06	7.00E-02	6.950	1467	177	0.98	7.1	21.00	0	6.95E-10	3.61E+04
67561	Methanol	32.04	0.79	126	4.6E-06	1.64E-05	1.50E-01	7.897	1474	229	-0.71	18	0.20	0	6.08E-10	1.00E+06
67641	Acetone	58.08	0.79	230	3.9E-05	1.14E-05	1.24E-01	7.117	1211	230	-0.24	1.3	1.15	0	6.08E-10	1.00E+06
67663	Chloroform	119.38	1.49	197	0.00367	1.00E-05	1.04E-01	6.493	929	196	1.92	28	0.79	0	2.43E-09	7.92E+03
67721	Hexachloroethane	236.74	2.09	0.21	0.00389	6.80E-06	2.49E-03	7.228	1348	133	4	0.001	0.03	0	1.56E-08	5.00E+01
68122	N,N-Dimethyl formamide	73.09	0.9445	4	1.9E-07	1.92E-05	9.39E-02	6.928	1401	196	-1.01	9.7	0.13	0	1.00E-20	1.00E+06
71432	Benzene	78.11	0.87	95	0.00558	9.80E-06	8.80E-02	6.905	1211	221	2.13	19	1.40	0	1.39E-09	1.75E+03
71556	1,1,1-Trichloroethane	133.4	1.33	124	0.0172	8.80E-06	7.80E-02	6.827	1147	219	2.48	3.5	0.74	2E-08	2.37E-08	1.33E+03
74839	Methyl bromide	94.94	1.41	1620	0.00624	1.21E-05	7.28E-02	7.566	1301	273	1.19	10.76	0.35	0	2.43E-09	1.52E+04
74873	Methyl chloride	50.49	0.95	4300	0.00882	6.50E-06	1.26E-01	7.093	949	249	0.91	10.76	0.72	0	2.43E-09	5.33E+03
75014	Vinyl chloride	62.5	0.91	2980	0.027	1.23E-05	1.06E-01	6.991	969	251	1.5	10.76	0.14	0	1.56E-08	2.76E+03
75058	Acetonitrile	41.05	0.78	91.1	3.5E-05	1.66E-05	1.28E-01	7.119	1314	230	-0.34	9.7	0.10	0	2.43E-09	1.00E+06
75070	Acetaldehyde	44.05	0.788	902	7.9E-05	1.41E-05	1.24E-01	8.005	1600	292	1.25	82.42	0.20	0	1.00E-20	1.00E+06
75092	Methylene chloride	84.93	1.34	433	0.00219	1.17E-05	1.01E-01	6.968	1074	223	1.25	18	0.38	0	2.43E-09	1.30E+04
75150	Carbon disulfide	76.14	1.26	359	0.03022	1.00E-05	1.04E-01	6.942	1169	242	2	15.3	0.89	0	1.00E-20	1.19E+03
75218	Ethylene oxide	44.06	0.87	1094	0.00012	1.45E-05	1.04E-01	7.128	1055	238	-0.3	4.2	0.91	0	1.00E-20	3.83E+05
75252	Tribromomethane	252.73	2.89	5.51	0.00054	1.03E-05	1.49E-02	7.988	2159	273	2.35	10.76	1.01	0	1.56E-08	3.10E+03
75274	Bromodichloromethane	163.83	1.97	50	0.0016	1.06E-05	2.98E-02	7.966	1847	273	2.1	10.76	0.70	0	1.00E-20	6.74E+03
75354	1,1-Dichloroethylene	96.94	1.213	600	0.0261	1.04E-05	9.00E-02	6.972	1099	237	2.13	10.76	0.90	0	1.56E-08	2.25E+03
75569	Propylene oxide	58.08	0.83	532.1	8.5E-05	1.00E-05	1.04E-01	7.067	1133	236	0.03	17.56	0.17	0	1.00E-20	4.76E+05
75694	Trichlorofluoromethane	137.37	1.49	803	0.097	9.70E-06	8.70E-02	6.884	1043	237	2.53	1.076	0.12	0	3.13E-08	1.10E+03
75718	Dichlorodifluoromethane	120.91	1.41	4850	0.343	8.00E-06	8.00E-02	7.590	1329	273	2.16	1.076	0.07	0	1.56E-08	2.80E+02
76131	1,1,2-Trichloro-1,2,2-trifluoroethane	187.38	1.41	332	0.4815	8.20E-06	7.80E-02	8.784	1894	273	3.16	0.001	0.03	0	1.00E-20	1.70E+02
77474	Hexachlorocyclopentadiene	272.77	1.7	0.0596	0.027	6.16E-06	5.61E-02	8.415	2835	273	5.39	0.001	0.03	0	2.43E-09	1.80E+00
78591	Isophorone	138.21	0.92	0.438	6.6E-06	6.76E-06	6.23E-02	7.963	2481	273	1.7	15.3	0.60	0	2.43E-09	1.20E+04
78875	1,2-Dichloropropane	112.99	1.156	52	0.0028	8.73E-06	7.82E-02	6.980	1380	223	1.97	17	1.40	0	1.12E-07	2.80E+03
78933	Methyl ethyl ketone	72.11	0.82	95.3	5.6E-05	9.80E-06	8.08E-02	7.112	1305	229	0.28	2	0.20	0	6.08E-10	2.23E+05
79005	1,1,2-Trichloroethane	133.4	1.435	23.3	0.00091	8.80E-06	7.80E-02	7.192	1480	229	2.05	3.5	0.74	0	3.17E-08	4.42E+03
79016	Trichloroethylene	131.39	1.4	73.5	0.0103	9.10E-06	7.90E-02	6.518	1019	193	2.71	3.9	0.88	0	3.13E-08	1.10E+03
79061	Acrylamide	71.08	0.84	0.007	1E-09	1.06E-05	9.70E-02	11.293	3940	273	-0.96	9.7	0.27	0	6.52E-11	6.40E+05
79107	Acrylic acid	72.06	1.12	4	1.2E-07	1.06E-05	9.60E-02	5.652	649	155	0.35	17.56	0.18	0	1.00E-20	1.00E+06

Table B-1. Chemical Specific Input Parameters

CAS #	COMPOUND NAME	Mol. Wt. (g/mol)	Density (g/cc)	VAP. Press. (mmHg)	H Law Const. (atm-m ³ /mol)	Diffusivity in Water (cm ² /sec)	Diffusivity in Air (cm ² /sec)	Antoinies' Vapor Pressure Coefficients			log Oct Water Part. Coeff. mgVO/g-hr.	Kmax L/g-hr.	K1 sec-1	Hydrol. Rate sec-1	Soil Biodeg. Rate sec-1	Solubility mg/L
							A	B	C							
79345	1,1,2,2-Tetrachloroethane	167.85	1.59	4.62	0.00035	7.90E-06	7.10E-02	6.894	1355	192	2.39	6.2	0.68	0	3.82E-09	2.97E+03
79469	2-Nitropropane	89.09	0.9876	18	0.00012	1.01E-05	9.23E-02	7.272	1531	229	0.87	9.7	0.42	0	1.56E-08	1.70E+04
80626	Methyl methacrylate	100.12	0.95	38.4	0.00034	8.60E-06	7.70E-02	6.517	1052	188	1.38	17.56	4.30	0	2.43E-09	1.50E+04
85449	Phthalic anhydride	148.12	1.33	0.00052	1.6E-08	9.60E-06	7.10E-02	8.022	2869	273	-0.62	17.56	0.08	0.0155	1.00E-20	6.20E+03
87683	Hexachloro-1,3-butadiene	260.76	1.67	0.221	0.00815	6.16E-06	5.61E-02	7.485	1956	215	4.81	0.001	0.03	0	1.56E-08	3.23E+00
91203	Naphthalene	128.17	1.14	0.085	0.00048	7.50E-06	5.90E-02	7.373	1968	223	3.36	42.47	1.00	0	4.17E-09	3.10E+01
92875	Benzidine	184.24	1.02	8E-09	3.9E-11	1.50E-05	8.00E-02	7.542	2626	163	1.66	31.1	0.66	0	6.95E-10	5.00E+02
95501	o-Dichlorobenzene	147	1.31	1.36	0.0019	7.90E-06	6.90E-02	6.883	1538	205	3.43	2.5	0.58	0	1.56E-08	1.56E+02
95534	o-Toluidine	107.16	0.989	0.32	2.7E-06	9.12E-06	7.14E-02	7.197	1683	191	1.34	31.1	0.86	0	6.08E-10	1.66E+04
95578	2-Chlorophenol	128.56	1.26	2.34	0.00039	9.46E-06	5.01E-02	6.877	1472	193	2.15	15	0.89	0	1.00E-20	2.20E+04
95658	3,4-Dimethylphenol	122.17	0.97	0.05836	2.3E-07	8.33E-06	6.02E-02	7.504	1940	197	2.23	5.5	1.05	0	1.00E-20	4.00E+04
96128	1,2-Dibromo-3-chloropropane	236.33	1.41	0.58	0.00015	7.02E-06	2.12E-02	8.073	2436	273	2.34	10.76	0.16	0	1.56E-08	1.23E+03
98011	Furfural	96.09	1.16	2.21	4E-06	1.04E-05	6.72E-02	6.575	1199	163	0.41	17.56	0.54	0	1.00E-20	1.10E+05
98828	Cumene	120.19	0.86	4.5	1.16	7.10E-06	8.60E-02	6.963	1461	208	3.58	31.1	2.88	0	6.95E-10	6.13E+01
98953	Nitrobenzene	123.11	1.2	0.245	2.4E-05	8.60E-06	7.60E-02	7.115	1747	202	1.84	11	2.30	0	1.71E-08	2.09E+03
100414	Ethylbenzene	106.17	0.87	9.6	0.00788	7.80E-06	7.50E-02	6.975	1424	213	3.14	6.8	2.10	0	8.69E-10	1.69E+02
100425	Styrene	104.15	0.9	6.12	0.00275	8.00E-06	7.10E-02	6.945	1437	208	2.94	31.1	0.11	0	2.43E-09	3.10E+02
106467	p-Dichlorobenzene	147	1.29	1	0.0024	7.90E-06	6.90E-02	7.199	1690	218	3.42	6.4	2.30	0	1.00E-20	7.38E+01
106887	1,2-Epoxybutane	72.11	0.826	207.912	0.00046	1.03E-05	1.35E-01	6.832	1141	228	1.441	10.76	0.48	0	1.00E-20	4.28E+04
106898	Epichlorohydrin	92.53	1.18	16.4	3E-05	9.80E-06	8.60E-02	8.229	2087	273	0.25	10.76	0.14	0	2.43E-09	6.59E+04
106934	Ethylene dibromide	187.86	2.7	13.3	0.00074	1.19E-05	2.17E-02	7.345	1675	245	1.96	10.76	0.55	0	1.56E-08	4.18E+03
106990	1,3-Butadiene	54.09	0.76	2110	0.0736	1.08E-05	2.49E-01	7.217	1145	269	1.99	15.3	0.69	0	1.00E-20	7.35E+02
107028	Acrolein	56.06	0.84	274	0.00012	1.22E-05	1.05E-01	7.213	1297	247	-0.01	7.8	0.34	0	2.43E-09	2.13E+05
107051	Allyl chloride	76.53	0.94	368	0.011	1.08E-05	1.17E-01	7.576	1494	273	1.45	10.76	0.31	0	1.21E-09	3.37E+03
107062	1,2-Dichloroethane	98.96	1.26	78.9	0.00098	9.90E-06	1.04E-01	7.068	1293	225	1.47	2.1	0.98	0	1.56E-08	8.52E+03
107131	Acrylonitrile	53.06	0.97	109	0.0001	1.34E-05	1.22E-01	7.110	1336	238	0.25	18	0.75	0	2.00E-09	7.40E+04
107211	Ethylene glycol	62.07	1.11	0.092	6E-08	1.22E-05	1.08E-01	8.091	2089	204	-1.36	17.56	0.06	0	1.00E-20	1.00E+06
108054	Vinyl acetate	86.09	0.93	90.2	0.00051	9.20E-06	8.50E-02	7.210	1296	227	0.73	17.56	0.30	0	1.00E-20	2.00E+04
108101	Methyl isobutyl ketone	100.16	0.8	19.9	0.00014	7.80E-06	7.50E-02	6.672	1168	192	1.19	0.74	0.45	0	6.08E-10	1.90E+04
108883	Toluene	92.14	0.87	28.4	0.00664	8.60E-06	8.70E-02	6.954	1345	219	2.75	6.7	2.40	0	1.91E-09	5.26E+02
108907	Chlorobenzene	112.56	1.11	12	0.0037	8.70E-06	7.30E-02	6.978	1431	218	2.86	0.39	10.00	0	1.30E-08	4.72E+02
108930	Cyclohexanol	100.2	0.95	1.22	4.5E-06	8.31E-06	2.14E-01	6.255	913	109	1.577	17.56	0.54	0	1.00E-20	3.60E+04
108952	Phenol	94.11	1.07	0.276	4E-07	9.10E-06	8.20E-02	7.133	1517	175	1.48	97	13.00	0	8.69E-10	8.28E+04
109864	2-Methoxyethanol	76.09		2.55697	2.6E-07	8.00E-06	8.00E-02			273	-0.77	19.8	1.00		1.00E-20	1.00E+06
110496	2-Methoxyethanol acetate	130.15		9.28503	1.6E-06	8.00E-06	8.00E-02			273	0	19.8	1.00		1.00E-20	1.00E+06
110543	n-Hexane	86.18	0.66	151	0.0143	7.77E-06	2.00E-01	6.876	1171	224	4	15.3	1.47	0	1.00E-20	1.24E+01
110805	2-Ethoxyethanol	90.12	0.9	5.31	3.5E-07	9.57E-06	9.47E-02	7.874	1844	234	-0.1	19.8	1.00	0	2.43E-09	1.00E+06
110861	Pyridine	79.1	0.98	20.8	8.9E-06	7.60E-06	9.10E-02	7.041	1374	215	0.67	35.03	0.24	0	6.08E-10	1.00E+06

Table B-1. Chemical Specific Input Parameters

CAS #	COMPOUND NAME	Mol. Wt. (g/mol)	Density (g/cc)	VAP. Press. (mmHg)	H Law Const. (atm-m ³ /mol)	Diffusivity in Water (cm ² /sec)	Diffusivity in Air (cm ² /sec)	Antoinies' Vapor Pressure Coefficients			log Oct Water Part. Coeff.	Kmax mgVO/g-hr.	K1 L/g-hr.	Hydrol. Rate sec-1	Soil Biodeg. Rate sec-1	Solubility mg/L
							A	B	C							
111159	2-Ethoxyethanol acetate	143.01		11.6912	2.2E-06	8.00E-06	8.00E-02			273	0	19.8	1.00		1.00E-20	1.00E+06
118741	Hexachlorobenzene	284.78	2.04	1.8E-05	0.00132	5.91E-06	5.42E-02	9.554	3249	203	5.89	0.001	0.03	0	1.82E-07	6.20E+00
120821	1,2,4-Trichlorobenzene	181.45	1.41	0.431	0.00142	8.23E-06	3.00E-02	7.706	2243	253	4.01	1.076	0.44	0	1.56E-08	3.00E+02
121142	2,4-Dinitrotoluene	182.14	1.31	0.00015	9.3E-08	7.06E-06	2.03E-01	7.981	3074	280	2.01	9.7	0.78	0	1.56E-08	2.70E+02
121448	Triethylamine	101.19	0.7326	57.07	0.00014	7.88E-06	8.81E-02	6.959	1272	223	1.45	9.7	1.06	0	1.00E-20	5.50E+04
122667	1,2-Diphenylhydrazine	184.24	1.19	0.00043	1.5E-06	7.36E-06	3.17E-02	13.836	5403	273	2.94	19	1.91	0	1.00E-20	6.80E+01
123911	1,4-Dioxane	88.11	1.03	38.1	4.8E-06	1.02E-05	2.29E-01	7.351	1518	238	-0.39	17.56	0.39	0	1.56E-08	1.00E+06
124481	Chlorodibromomethane	208.28	2.451	4.9	0.00078	1.05E-05	1.96E-02	8.220	2100	273	2.17	10.76	0.04	0	1.56E-08	2.60E+03
126998	Chloroprene	88.54	0.958	213.658	0.0143	1.00E-05	1.04E-01	6.161	783	180	2.08	10.76	0.22	0	1.56E-08	1.74E+03
127184	Tetrachloroethylene	165.83	1.624	18.6	0.0184	8.20E-06	7.20E-02	6.976	1387	218	2.67	6.2	0.68	0	3.13E-08	2.00E+02
630206	1,1,1,2-Tetrachloroethane	167.85	1.59	12.03	0.00242	7.90E-06	7.10E-02	6.894	1355	192	2.63	6.2	0.68	0	5.81E-09	1.10E+03
924163	N-Nitrosodi-n-butylamine	158.24		0.03	0.00032	8.00E-06	8.00E-02			273	2.41	0.0001	1.00		1.00E-20	1.27E+03
930552	N-Nitrosopyrrolidine	100.12		0.092	1.2E-08	1.04E-05	7.36E-02			273	-0.19	0.0001	1.00		1.56E-08	1.00E+06
1319773	Cresols (total)	108.1	1.03	0.3	1.6E-06	9.30E-06	6.94E-02	8.850	2795	273	0	23	17.00	0	1.00E-20	2.20E+04
1330207	Xylenes	106.17	0.86	8.04178	0.00604	9.34E-06	7.14E-02	7.940	2090	273	3.17	40.8	1.80	0	2.43E-09	1.86E+02
1634044	Methyl tert-butyl ether	88	0.97	185.949	0.00056	1.05E-05	1.02E-01	6.852	1104	223	1.901	17.56	0.71	0	1.00E-20	3.88E+04
1746016	2,3,7,8-TCDD	322	1.41	7.4E-10	1.6E-05	8.00E-06	4.70E-02	6.977	2377	159	6.64	0.001	0.03	0	1.00E-20	1.90E-05
7439921	Lead	207.2		0	0 NA	NA				273	5.447				1.00E-20	
7439965	Manganese	54.938		0	0					273					1.00E-20	
7439976	Mercury	200.59		0.00196	0.0071	6.30E-06	5.5E-02			273	3*				1.00E-20	5.62E-02
7440020	Nickel	58.69		0	0 NA	NA				273	1.914				1.00E-20	
7440382	Arsenic	74.92		0	0 NA	NA				273	1.462				1.00E-20	
7440393	Barium	137.33		0	0 NA	NA				273	2.724				1.00E-20	
7440417	Beryllium	9.012		0	0 NA	NA				273	1.845				1.00E-20	
7440439	Cadmium	112.41		0	0 NA	NA				273	2.21				1.00E-20	
7440473	Chromium (total)	51.996		0	0 NA	NA				273	1.255				1.00E-20	
7440484	Cobalt			0						273					1.00E-20	
7440622	Vanadium	50.94		0	0 NA	NA				273	1.699				1.00E-20	
10061015	cis-1,3-Dichloropropylene	110.97	1.2	32.8	0.00176	1.10E-05	5.85E-02	6.807	1328	230	2	10.76	0.76	0	9.81E-10	2.72E+03
10061026	trans-1,3-Dichloropropylene	110.97	1.2	23.3	0.00125	1.10E-05	5.85E-02	6.807	1328	230	2	10.76	0.76	0	9.81E-10	2.72E+03
16065831	Chromium (III)	51.996		0	0					273	1.255				1.00E-20	
18540299	Chromium (VI)	51.996		0	0	1.41E-05	2.00E-01			273	1.255				1.00E-20	

* This is the log Kd from the Mercury Report to Congress.

Appendix C

Sensitivity Analysis of ISC Air Model

C. Sensitivity Analysis of ISC Air Model

This appendix describes sensitivity analyses on depletion options, source shape and orientation, and receptor location and spacing.

C.1 Options With and Without Depletions

The Air Characteristic Study relies on the output of the air dispersion model ISCST3 to determine atmospheric concentrations of chemical constituents released by various waste management units. The ISCST3 model has several options for modeling deposition and depletion. Although the Air Characteristic Study is an inhalation-pathway-only analysis and does not require modeling deposition to estimate indirect pathway exposure, depletion is important. Depletion, or removal of chemical constituents from the plume due to deposition processes, can affect the estimate of air concentrations. Using ISCST3 with depletion, however, requires substantially more computer power and time to complete the extensive computer runs. Therefore, for the current study, depletion options are used only if the risk results are sensitive to the depletion option.

To determine ISCST3 model output sensitivity to depletion options for the Air Characteristic study, the magnitude of wet depletion of vapors and particulates and dry depletion of particulates were examined. ISCST3 cannot model dry depletion of vapors.

In this appendix, the setup of the sensitivity analysis is described, results are discussed, and recommendations are presented.

C.1.1 Setup of Sensitivity Analysis

Wastepiles and land application units (LAUs) were chosen to evaluate the effects of modeling air concentration of vapors and particulates with and without depletion. These two WMU types were selected to represent elevated and ground-level sources, respectively. Two areas were selected for each type of unit, one large and one small to capture differences in depletion due to source area. The two wastepile sizes were 20.25 m² and 1,300,056 m². The two land application unit sizes were 81 m² and 8,090,043 m². These sizes represent the smallest and largest size strata of each waste management unit type. Wastepiles were set to a source height of 5 meters and land application units were set to a source height of 0 meters. Receptors were placed at 25, 50, 75, 150, 500, and 1,000 meters from the edge of the source in concentric squares.

Meteorological data from Las Vegas, Nevada, and Miami, Florida, were selected for these analyses because these locations have the lowest and highest long-term average precipitation, respectively, of the 29 meteorological stations used in the Air Characteristic Study. Las Vegas averages 4.0 inches of precipitation per year and Miami averages 57.1 inches per year.

C.1.2 Analysis of Wet Depletion and Its Magnitude for Vapors and Particulates

The first sensitivity analysis was performed to evaluate the significance of wet depletion on calculated air concentrations of both vapors and particulates. The significance was determined by examining the magnitude of the difference between annual average air concentrations when wet depletion alone was selected vs. when both wet and dry depletion were selected. Both large and small wastepiles and land application units were run in this analysis, using the Hazardous Waste Identification Rule (HWIR 98) meteorological data and the 97363 version of ISCST3, which is the version of the model used for the May 1998 Air Characteristic Study. Because retention of the air dispersion model runs from the May 1998 study was desirable, the performance of this version of the model was important.

Because precipitation data in the SAMSON surface data set provided by the National Climatic Data Center are generally not complete, a specially processed meteorological data set was developed for the HWIR98 study based on precipitation data from the National Climatological Data Center (NCDC) Cooperative Station Summaries of the Day data set. Therefore, the new HWIR98 data set contains more complete precipitation data than were used in the May 1998 Air Characteristic Study and would yield a higher and more representative amount of wet depletion. The ISCST3 model was run for vapors and particulates with no depletion, wet depletion, and wet and dry depletion (particulates only) for both Miami and Las Vegas using those meteorological data.

Vapor Results. Vapors were evaluated for no depletion and wet depletion only because ISCST3 does not perform dry depletion calculations for vapor. In all cases, little difference in air concentration was observed when wet depletion was included versus excluded. Most differences were less than 1 percent, with lower air concentrations always resulting from the inclusion of wet depletion. Differences between air concentrations with and without wet depletion slightly exceeded 1 percent in a few cases when distances around 1 kilometer were examined. Only one receptor showed a difference of more than 2 percent. This receptor was for a small LAU in Miami at a distance of 1 kilometer. The difference was about 10 percent. The concentration values, however, are extremely small in magnitude due to the small size of the site. This difference is not considered significant.

Particulate Results. Large differences were observed between air concentrations with and without wet and dry depletion for particles for both LAUs and wastepiles. Maximum differences in air concentration ranged from 2 percent near the waste management unit to 57 percent at 1 kilometer from the waste management unit (see Tables C-1a and C-1b). The percent difference increases with increasing distance because the loss of material due to depletion is cumulative in nature. A comparison of air concentrations modeled with wet depletion only and those modeled with no depletion indicates that wet depletion plays a very small role in the annual average depletion of particles. Wet depletion alone yields less than 2 percent difference in concentration compared to no depletion.

Table C-1a. Maximum Percent Differences in Air Concentration with and without Wet Depletion Versus with and without Wet and Dry Depletion of Particles for Land Application Units

Distance (m)	Miami				Las Vegas			
	Large LAU		Small LAU		Large LAU		Small LAU	
	Wet Depletion	Wet/Dry Depletion	Wet Depletion	Wet/Dry Depletion	Wet Depletion	Wet/Dry Depletion	Wet Depletion	Wet/Dry Depletion
25	0.9	36.4	0.1	5.2	0.2	16.3	0.0	1.6
50	1.0	39.1	0.1	16.7	0.2	17.8	0.1	6.6
75	1.1	40.5	0.2	22.0	0.2	18.7	0.2	9.4
150	1.3	43.1	0.9	30.1	0.3	20.4	0.0	14.3
500	1.8	47.8	6.7	40.7	0.3	24.7	0.0	22.2
1000	2.2	51.0	20.0	50.0	0.4	29.1	0.0	33.3

Table C-1b. Maximum Percent Differences in Air Concentration with and without Wet Depletion Versus with and without Wet and Dry Depletion of Particles for Wastepiles

Distance (m)	Miami				Las Vegas			
	Large WP		Small WP		Large WP		Small WP	
	Wet Depletion	Wet/Dry Depletion	Wet Depletion	Wet/Dry Depletion	Wet Depletion	Wet/Dry Depletion	Wet Depletion	Wet/Dry Depletion
25	0.7	43.3	0.1	20.6	0.1	19.8	0.0	8.0
50	0.7	44.7	0.3	30.5	0.1	20.8	0.0	13.3
75	0.8	45.7	0.3	35.7	0.1	21.6	0.0	16.6
150	1.0	47.5	0.5	43.0	0.1	23.6	0.1	22.2
500	1.5	51.9	1.0	52.4	0.3	30.4	0.2	30.2
1000	2.0	57.0	1.6	56.3	0.4	34.8	0.3	34.0

C.1.3 Analysis of Different Versions of ISCST3 and Their Effects on Dry Depletion of Particles

A second analysis was performed to determine the difference between air concentration of particulates with dry depletion and without depletion for each of three different versions of ISCST3. The three versions were 96113 (1996), 97363 (1997), and 98356 (1998). The 1996 version of the model was used for the sensitivity analysis conducted as part of the May 1998 Air Characteristic Study. The 1997 version of the model was used for the production of the final results in May 1998 and is the model version used for the current study. The 1998 version, the latest release, was evaluated to determine if it should be adopted for the current study.

The algorithm for dry depletion differs in each of these versions of the model and the 1998 version contains a completely different area source integration routine from the other two versions. This analysis was performed using the large land application unit and the May 1998 Air Characteristic Study meteorological data. As shown in Section C.1.2., wet depletion is not significant for particles and thus is not included in this portion of the sensitivity analysis. Furthermore, there is no difference in the wet depletion algorithm among the three versions of ISCST3.

Results of Dry vs. No Depletion of Particles for Different Versions of ISCST3. As part of this analysis, differences in air concentration results produced by different versions of the ISCST3 model also were examined. When the three versions of ISCST3 were compared without depletion, no difference was found between the 1996 and 1997 versions of ISCST3. Small differences were found between these versions and the 1998 version and are not consistent in direction. These insignificant differences are due to changes in the area source integration technique¹ used by the model and modifications to some portions of the code.

There are, however, significant differences between the three versions of ISCST3 when dry depletion is included in the model run. The three versions were run for the largest land application unit strata (8,090,043 m²). Large land application units were selected because larger differences between concentrations with and without depletion are generally expected at most receptor distances for a larger source. The dry depletion sensitivity analysis was conducted using all three versions of the ISCST3 model and the same meteorological data that were used in the May 1998 Air Characteristic Study.

The difference in air concentrations of particles with and without dry depletion for the 1996 version of ISCST3 was about 6 percent at 0 meters and increased to 25 percent at 1,000 meters for Las Vegas meteorological data. For Miami, these differences were 7 and 31 percent, respectively. The 1997 version showed differences of 12 and 34 percent at Las Vegas and 15 percent and 38 percent at Miami. The difference between the 1996 and 1997 models stems from the change in the deposition reference height from 20 times the roughness height to 1 meter.

¹The integration technique arrives at a concentration value for a given receptor by performing a series of iterations, each representing the area source as consecutively increasing numbers of point emission sources, until there is little change in the result.

The difference in air concentrations of particles with and without depletion in the 1998 version of the model was larger. At Las Vegas, the difference was 22 percent at 0 meters and 46 percent at 1,000 meters. Miami showed 28 percent and 52 percent differences, respectively. These differences stem from changes to the algorithm used. These include a change in the integration technique and modifications that affected the vertical dispersion coefficient for area sources. Table C-1c shows the maximum differences with and without depletion for each version of ISCST3 at each distance from the source.

In addition to differences of the effect of depletion between the 1997 and 1998 versions of ISCST3, computer run time between these two versions of the model differed substantially. The 1998 version of the model required eight times the number of hours needed to complete a run than did the 1997 version of the model. The model developers were contacted for their recommendation about which version of the model to use. While they confirmed the increase in run time for the 1998 version, they offered no opinion as to which version is technically superior. Therefore, the 1997 version was chosen based on run time considerations and consistency with the previous Air Characteristic analysis.

C.1.4 Summary of Depletion Conclusions

Based on the results of these sensitivity analyses, the following decisions were made concerning the dispersion modeling:

- # Use meteorological data used in 1998 Air Characteristic Study since wet depletion will not be modeled for vapors or particulates.
- # Model vapors with no wet depletion because it is significant for vapors.
- # Model particulates with dry depletion only because wet depletion is not significant for particulates.
- # Use 1997 version of ISCST3 because it is technically valid, compatible with the May 1998 Air Characteristic Study, and the run time is substantially shorter than the run time for the 1998 version.

C.2 Source Shape and Orientation

A sensitivity analysis was conducted using the ISCST3 air model to determine what role source shape and orientation play in determining dispersion coefficients of air pollutants. A discussion of this analysis follows.

Three different sources were chosen for this analysis. The sources were a square (source No. 1), a rectangle oriented east to west (source No. 2), and a rectangle oriented north to south (source No. 3). All three sources had an area of 400 m² in order to ensure that equal emission rates were compared. The rectangles were selected to be exactly two times longer and half as wide as the square (see Figure C-2).

Table C-1c. Comparison of Maximum Percent Differences in Air Concentration of Particles with Versus Without Dry Depletion Between Three Versions of ISCST3

Distance (m)	Miami			Las Vegas		
	ISCST3 1996	ISCST3 1997	ISCST3 1998	ISCST3 1996	ISCST3 1997	ISCST3 1998
25	13	25	43	10	20	34
50	15	27	46	12	22	35
75	16	29	47	13	23	36
150	19	31	49	15	25	38
500	26	36	51	21	29	43
1000	31	39	53	25	34	47

Two meteorological stations at Little Rock, Arkansas, and Los Angeles, California, were selected for this modeling analysis in order to compare two different meteorological regimes. Little Rock was selected because of its evenly distributed wind directions and Los Angeles was selected because it has a predominantly southwest wind direction (see Figure C-3). Five years of meteorological data were used for this analysis.

Each area source was modeled with similar receptor grids to ensure consistency. Sixteen receptors were placed on the edge of each of the area sources and another 16 were placed 25 meters out from the edge. Each of these two receptor groups were modeled as a Cartesian receptor grid. Two receptor rings were also placed at 50 and 100 meters out from the center of the source. This polar receptor grid consisted of 16 receptors with a 22.5 degree interval between receptors. See Figures C-4a through C-4c for receptor locations.

The ISCST3 model was run using the meteorological data from Little Rock, Arkansas, and Los Angeles, California, and the results are shown in Tables C-2a and C-2b. The results indicated that the standard deviation of the differences in air concentrations is greatest between source No. 2 and source No. 3. This difference is due to the orientation of the source. This occurs for both the Cartesian receptor grid and the polar receptor grid at both meteorological locations. This shows that the model is sensitive to the orientation of the rectangular area source.

Standard deviations are significantly smaller when source No. 1 is compared to source Nos. 2 or 3. This shows that the differences in Unitized Air Concentration (UAC) between the square source and the two rectangular sources are less than the differences between the two rectangular sources. A square area source also contributes the least amount of impact of orientation. Since no information on source shape or orientation is available, a square source will minimize the errors caused by different source shapes and orientations.

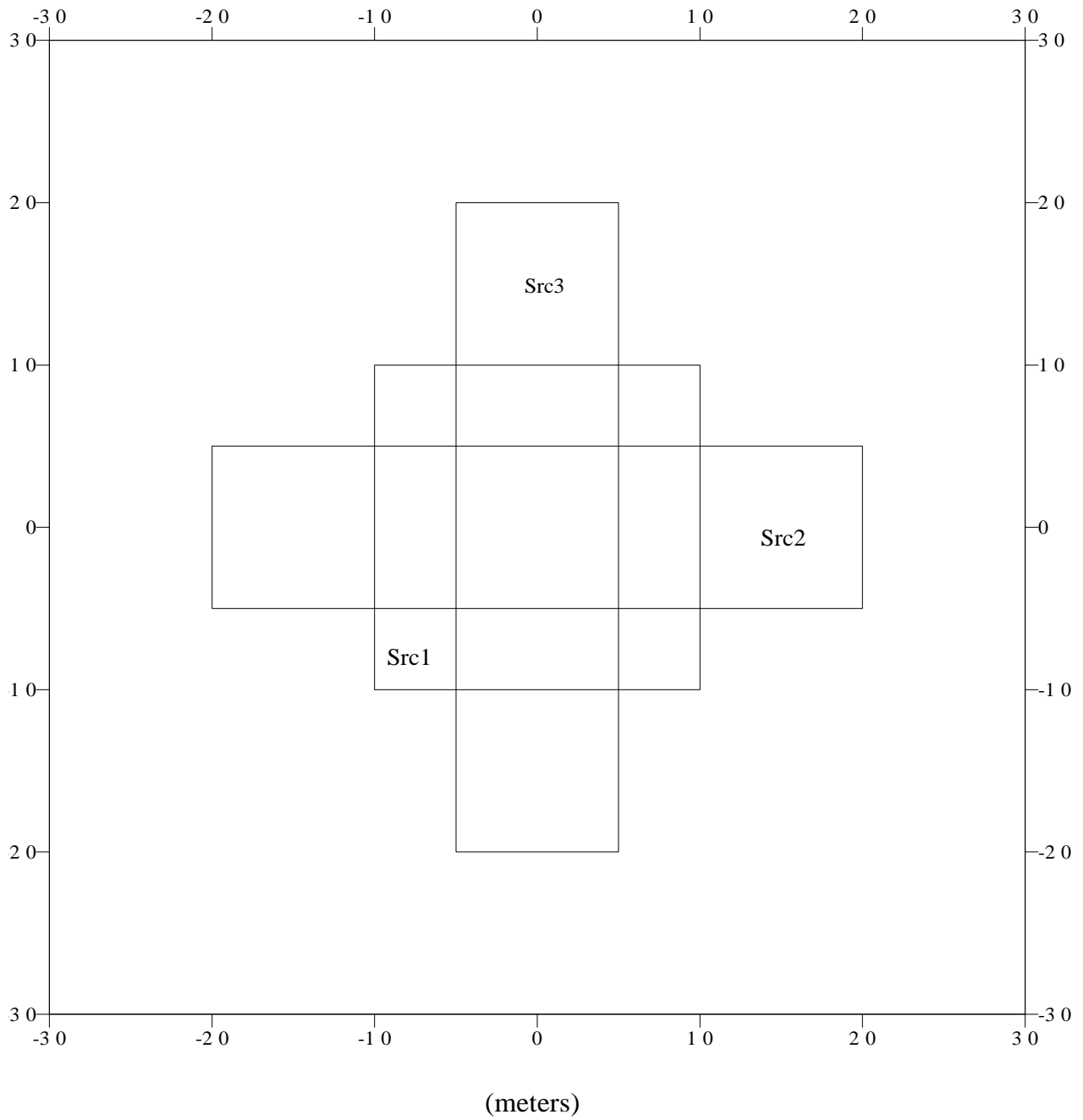
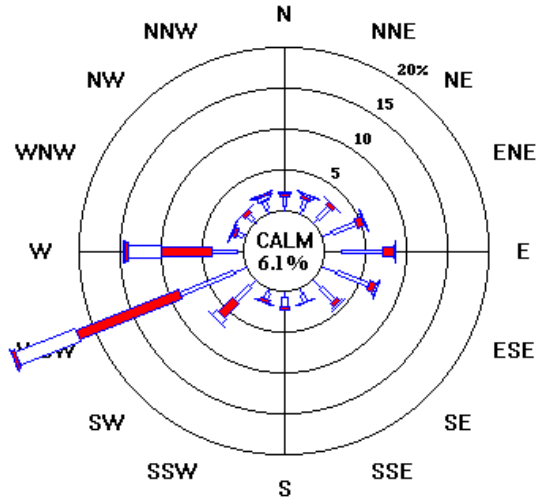
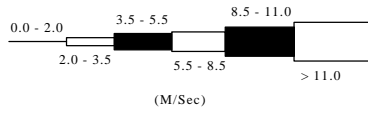


Figure C-2. Source Shapes and Orientations.

Los Angeles, California



Little Rock, Arkansas

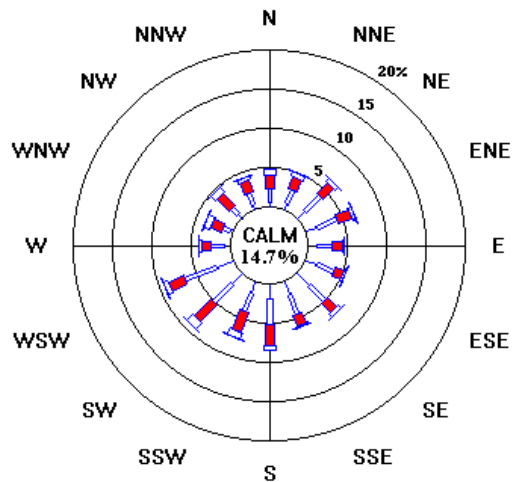
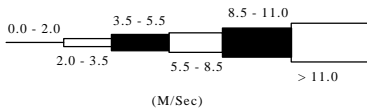


Figure C-3. Wind Roses

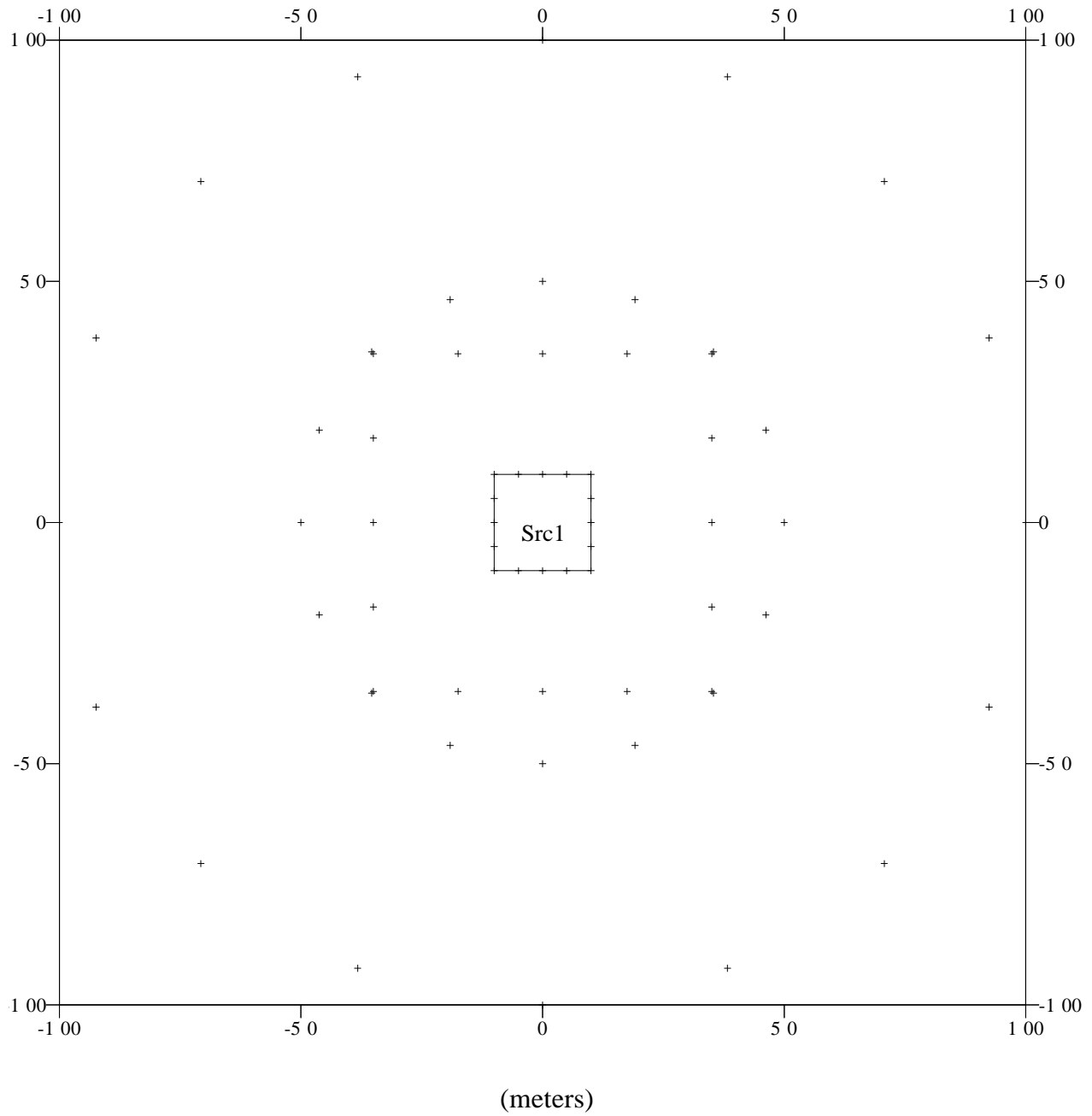


Figure C-4a. Receptor Locations (Source No. 1).

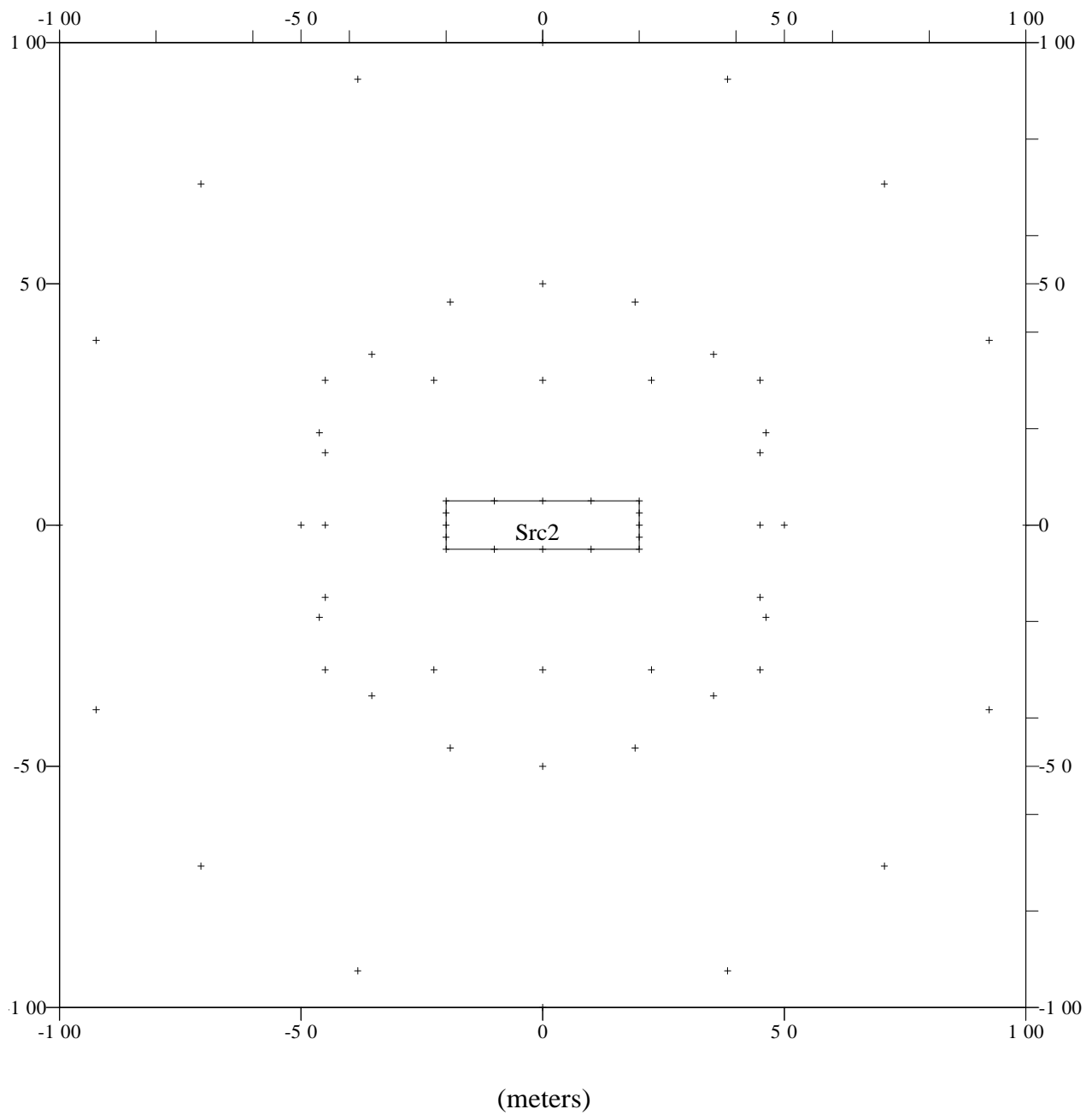


Figure C-4b. Receptor Locations (Source No. 2).

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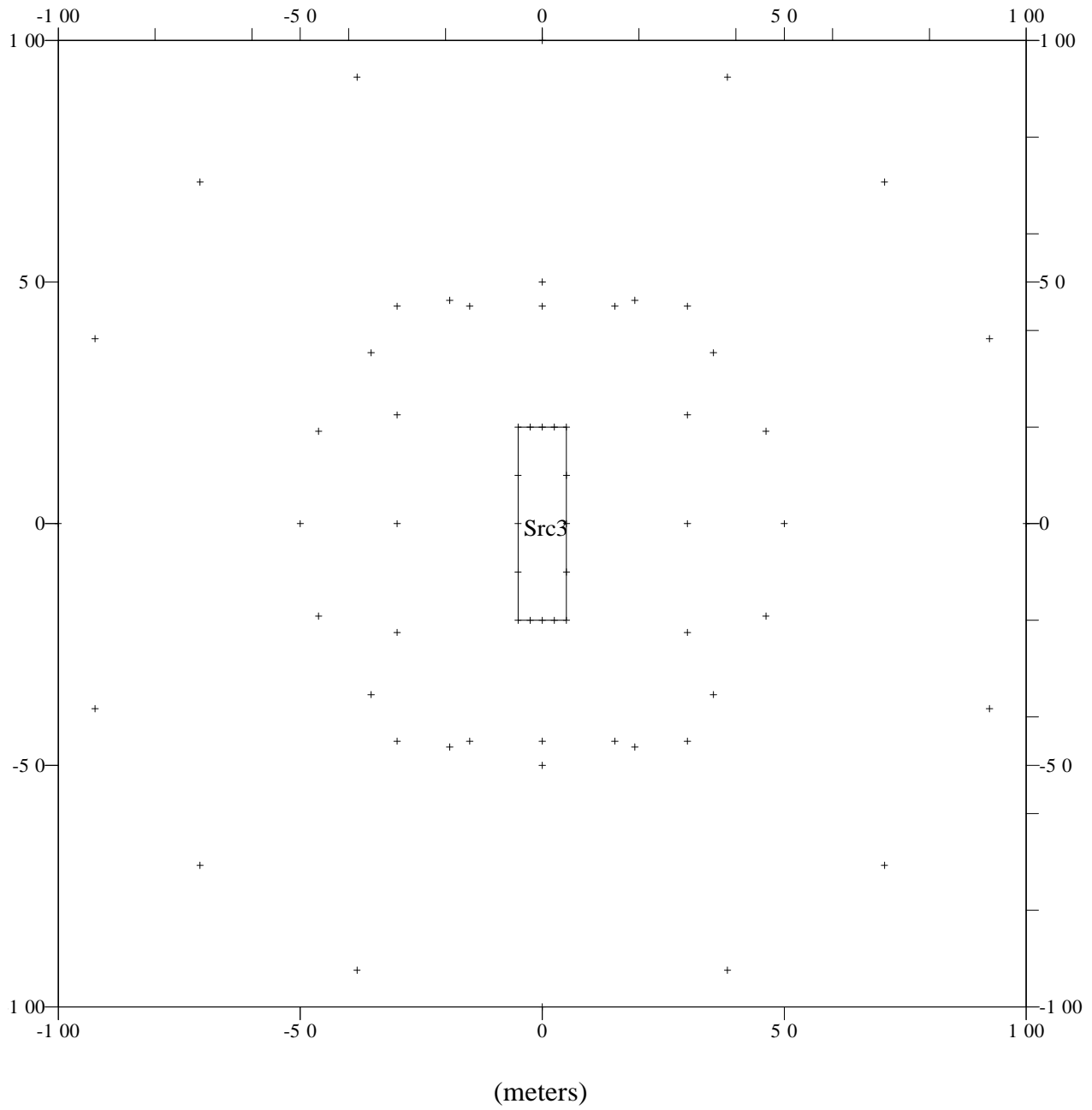


Figure C-4c. Receptor Locations (Source No. 3).

Table C-2a. Comparisons of Unitized Air Concentrations ($\mu\text{g}/\text{m}^3/\mu\text{g}/\text{s}\cdot\text{m}^2$) for Different Source Shapes and Orientations (Little Rock, Arkansas)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Polar Receptor Grid									Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC						
19	46	0.190	19	46	0.199	19	46	0.211	0.010	5%	0.021	11%	0.012	6%
38	92	0.050	38	92	0.051	38	92	0.051	0.001	1%	0.001	2%	0.000	1%
35	35	0.249	35	35	0.243	35	35	0.278	-0.007	-3%	0.028	11%	0.035	14%
71	71	0.067	71	71	0.067	71	71	0.069	-0.001	-1%	0.001	2%	0.002	3%
46	19	0.321	46	19	0.361	46	19	0.256	0.041	13%	-0.065	-20%	-0.105	-29%
92	38	0.095	92	38	0.098	92	38	0.088	0.003	3%	-0.007	-7%	-0.010	-10%
50	0	0.124	50	0	0.128	50	0	0.147	0.004	3%	0.023	19%	0.020	15%
100	0	0.030	100	0	0.030	100	0	0.033	0.000	-1%	0.003	9%	0.003	11%
46	-19	0.085	46	-19	0.096	46	-19	0.084	0.011	12%	-0.001	-1%	-0.011	-12%
92	-38	0.023	92	-38	0.024	92	-38	0.023	0.001	2%	-0.001	-2%	-0.001	-5%
35	-35	0.106	35	-35	0.109	35	-35	0.103	0.003	3%	-0.003	-3%	-0.006	-6%
71	-71	0.030	71	-71	0.030	71	-71	0.029	0.000	0%	0.000	-1%	-0.001	-2%
19	-46	0.117	19	-46	0.113	19	-46	0.128	-0.005	-4%	0.011	9%	0.016	14%
38	-92	0.033	38	-92	0.032	38	-92	0.034	-0.001	-4%	0.001	2%	0.002	7%
0	-50	0.122	0	-50	0.117	0	-50	0.143	-0.005	-4%	0.021	17%	0.026	22%
0	-100	0.035	0	-100	0.033	0	-100	0.037	-0.002	-5%	0.002	5%	0.004	11%
-19	-46	0.134	-19	-46	0.128	-19	-46	0.150	-0.006	-4%	0.016	12%	0.022	17%
-38	-92	0.038	-38	-92	0.036	-38	-92	0.038	-0.002	-4%	0.001	2%	0.002	6%
-35	-35	0.161	-35	-35	0.158	-35	-35	0.170	-0.003	-2%	0.009	6%	0.012	8%
-71	-71	0.043	-71	-71	0.043	-71	-71	0.045	0.000	1%	0.001	3%	0.001	3%
-46	-19	0.159	-46	-19	0.185	-46	-19	0.140	0.026	16%	-0.019	-12%	-0.045	-24%
-92	-38	0.044	-92	-38	0.046	-92	-38	0.043	0.002	4%	-0.002	-4%	-0.004	-8%
-50	0	0.103	-50	0	0.114	-50	0	0.107	0.011	11%	0.004	4%	-0.007	-6%
-100	0	0.027	-100	0	0.027	-100	0	0.027	0.000	2%	0.000	1%	0.000	0%
-46	19	0.126	-46	19	0.145	-46	19	0.118	0.019	15%	-0.008	-6%	-0.027	-18%
-92	38	0.035	-92	38	0.036	-92	38	0.034	0.001	4%	-0.001	-4%	-0.003	-7%
-35	35	0.152	-35	35	0.160	-35	35	0.153	0.008	5%	0.001	0%	-0.007	-5%
-71	71	0.041	-71	71	0.042	-71	71	0.041	0.001	3%	0.001	2%	-0.001	-2%
-19	46	0.173	-19	46	0.179	-19	46	0.187	0.007	4%	0.014	8%	0.008	4%
-38	92	0.047	-38	92	0.047	-38	92	0.048	0.000	0%	0.001	3%	0.001	3%
0	50	0.224	0	50	0.191	0	50	0.276	-0.032	-14%	0.052	23%	0.085	44%
0	100	0.068	0	100	0.061	0	100	0.074	-0.008	-11%	0.006	9%	0.014	22%
Standard Deviation:									0.012	7%	0.018	9%	0.028	14%

(continued)

Table C-2a (continued)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Cartesian Receptor Grid									Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC						
-10	-10	3.014	-20	-5	2.675	-5	-20	2.673	-0.339	-11%	-0.341	-11%	-0.002	0%
-5	-10	4.266	-10	-5	4.219	-2.5	-20	3.451	-0.047	-1%	-0.815	-19%	-0.769	-18%
0	-10	4.354	0	-5	4.307	0	-20	3.526	-0.047	-1%	-0.827	-19%	-0.781	-18%
5	-10	3.961	10	-5	4.069	2.5	-20	3.152	0.109	3%	-0.809	-20%	-0.918	-23%
10	-10	2.175	20	-5	1.899	5	-20	2.011	-0.276	-13%	-0.164	-8%	0.112	6%
10	-5	5.211	20	-2.5	3.875	5	-10	5.567	-1.337	-26%	0.355	7%	1.692	44%
10	0	5.968	20	0	4.704	5	0	5.913	-1.264	-21%	-0.055	-1%	1.209	26%
10	5	6.012	20	2.5	4.918	5	10	5.834	-1.094	-18%	-0.178	-3%	0.916	19%
10	10	4.946	20	5	4.468	5	20	4.344	-0.477	-10%	-0.602	-12%	-0.125	-3%
5	10	6.804	10	5	6.758	2.5	20	5.550	-0.047	-1%	-1.254	-18%	-1.208	-18%
0	10	6.846	0	5	6.830	0	20	5.604	-0.016	0%	-1.242	-18%	-1.226	-18%
-5	10	6.157	-10	5	6.353	-2.5	20	4.954	0.196	3%	-1.203	-20%	-1.399	-22%
-10	10	3.245	-20	5	2.793	-5	20	3.052	-0.451	-14%	-0.193	-6%	0.259	9%
-10	5	4.923	-20	2.5	3.801	-5	10	5.166	-1.121	-23%	0.244	5%	1.365	36%
-10	0	5.169	-20	0	4.032	-5	0	5.287	-1.137	-22%	0.118	2%	1.255	31%
-10	-5	4.809	-20	-2.5	3.727	-5	-10	4.991	-1.081	-22%	0.182	4%	1.264	34%
-35	-35	0.164	-45	-30	0.158	-30	-45	0.132	-0.006	-4%	-0.032	-19%	-0.026	-16%
-17.5	-35	0.219	-22.5	-30	0.247	-15	-45	0.167	0.027	12%	-0.052	-24%	-0.079	-32%
0	-35	0.243	0	-30	0.284	0	-45	0.179	0.041	17%	-0.063	-26%	-0.104	-37%
17.5	-35	0.186	22.5	-30	0.192	15	-45	0.147	0.006	3%	-0.039	-21%	-0.045	-23%
35	-35	0.108	45	-30	0.088	30	-45	0.100	-0.020	-19%	-0.008	-7%	0.012	14%
35	-17.5	0.141	45	-15	0.105	30	-22.5	0.160	-0.036	-25%	0.019	14%	0.055	52%
35	0	0.277	45	0	0.164	30	0	0.401	-0.113	-41%	0.124	45%	0.236	144%
35	17.5	0.503	45	15	0.396	30	22.5	0.466	-0.107	-21%	-0.037	-7%	0.070	18%
35	35	0.254	45	30	0.263	30	45	0.200	0.009	3%	-0.054	-21%	-0.063	-24%
17.5	35	0.315	22.5	30	0.373	15	45	0.234	0.058	18%	-0.081	-26%	-0.139	-37%
0	35	0.417	0	30	0.445	0	45	0.341	0.028	7%	-0.076	-18%	-0.104	-23%
-17.5	35	0.272	-22.5	30	0.286	-15	45	0.214	0.014	5%	-0.057	-21%	-0.071	-25%
-35	35	0.155	-45	30	0.131	-30	45	0.146	-0.024	-15%	-0.009	-6%	0.015	11%
-35	17.5	0.211	-45	15	0.155	-30	22.5	0.232	-0.056	-27%	0.022	10%	0.078	50%
-35	0	0.213	-45	0	0.145	-30	0	0.298	-0.068	-32%	0.084	40%	0.153	106%
-35	-17.5	0.265	-45	-15	0.193	-30	-22.5	0.264	-0.073	-27%	-0.002	-1%	0.071	37%
Standard Deviation:									0.463	15%	0.435	17%	0.747	41%

Table C-2b. Comparisons of Unitized Air Concentrations ($\mu\text{g}/\text{m}^3/\mu\text{g}/\text{s}\cdot\text{m}^2$) for Different Source Shapes and Orientations (Los Angeles, California)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Polar Receptor Grid									Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC						
19	46	0.059	19	46	0.065	19	46	0.069	0.006	9%	0.010	17%	0.005	7%
38	92	0.016	38	92	0.016	38	92	0.016	0.000	-1%	0.000	3%	0.001	4%
35	35	0.188	35	35	0.168	35	35	0.284	-0.020	-11%	0.096	51%	0.116	69%
71	71	0.046	71	71	0.045	71	71	0.052	-0.001	-3%	0.006	13%	0.007	16%
46	19	0.582	46	19	0.607	46	19	0.461	0.025	4%	-0.121	-21%	-0.146	-24%
92	38	0.172	92	38	0.174	92	38	0.161	0.003	2%	-0.011	-6%	-0.014	-8%
50	0	0.278	50	0	0.293	50	0	0.293	0.014	5%	0.015	5%	0.001	0%
100	0	0.068	100	0	0.067	100	0	0.074	-0.001	-2%	0.005	8%	0.007	10%
46	-19	0.061	46	-19	0.062	46	-19	0.087	0.002	3%	0.026	43%	0.025	40%
92	-38	0.015	92	-38	0.015	92	-38	0.016	0.000	0%	0.002	10%	0.002	11%
35	-35	0.062	35	-35	0.068	35	-35	0.062	0.006	10%	0.000	0%	-0.006	-9%
71	-71	0.016	71	-71	0.017	71	-71	0.017	0.001	4%	0.001	3%	0.000	-1%
19	-46	0.080	19	-46	0.076	19	-46	0.087	-0.004	-4%	0.007	9%	0.011	14%
38	-92	0.023	38	-92	0.022	38	-92	0.024	-0.001	-5%	0.001	3%	0.002	8%
0	-50	0.086	0	-50	0.084	0	-50	0.096	-0.003	-3%	0.009	11%	0.012	15%
0	-100	0.023	0	-100	0.024	0	-100	0.024	0.000	1%	0.001	3%	0.000	2%
-19	-46	0.099	-19	-46	0.092	-19	-46	0.108	-0.006	-7%	0.009	9%	0.016	17%
-38	-92	0.028	-38	-92	0.027	-38	-92	0.028	-0.001	-2%	0.000	1%	0.001	3%
-35	-35	0.122	-35	-35	0.119	-35	-35	0.143	-0.003	-2%	0.021	18%	0.024	20%
-71	-71	0.033	-71	-71	0.032	-71	-71	0.034	0.000	-1%	0.001	4%	0.002	5%
-46	-19	0.218	-46	-19	0.223	-46	-19	0.226	0.005	2%	0.008	4%	0.003	2%
-92	-38	0.060	-92	-38	0.061	-92	-38	0.061	0.001	1%	0.001	1%	0.000	0%
-50	0	0.320	-50	0	0.378	-50	0	0.278	0.057	18%	-0.042	-13%	-0.099	-26%
-100	0	0.093	-100	0	0.098	-100	0	0.087	0.005	6%	-0.006	-6%	-0.011	-11%
-46	19	0.264	-46	19	0.273	-46	19	0.260	0.009	3%	-0.005	-2%	-0.013	-5%
-92	38	0.074	-92	38	0.075	-92	38	0.073	0.001	1%	-0.001	-2%	-0.002	-2%
-35	35	0.137	-35	35	0.123	-35	35	0.164	-0.014	-10%	0.027	20%	0.041	33%
-71	71	0.037	-71	71	0.035	-71	71	0.039	-0.002	-5%	0.002	4%	0.003	9%
-19	46	0.063	-19	46	0.066	-19	46	0.073	0.003	4%	0.010	15%	0.007	11%
-38	92	0.017	-38	92	0.017	-38	92	0.018	0.000	-2%	0.001	3%	0.001	5%
0	50	0.067	0	50	0.058	0	50	0.080	-0.008	-12%	0.014	21%	0.022	37%
0	100	0.020	0	100	0.018	0	100	0.021	-0.002	-9%	0.001	6%	0.003	15%
Standard Deviation:									0.013	6%	0.030	14%	0.040	18%

(continued)

Table C-2b (continued)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Cartesian Receptor Grid									Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC						
-10	-10	3.225	-20	-5	3.241	-5	-20	2.674	0.016	1%	-0.551	-17%	-0.567	-17%
-5	-10	4.025	-10	-5	4.333	-2.5	-20	3.119	0.308	8%	-0.906	-23%	-1.214	-28%
0	-10	3.952	0	-5	4.297	0	-20	3.050	0.345	9%	-0.902	-23%	-1.247	-29%
5	-10	3.431	10	-5	3.871	2.5	-20	2.564	0.440	13%	-0.867	-25%	-1.307	-34%
10	-10	1.683	20	-5	1.592	5	-20	1.511	-0.091	-5%	-0.172	-10%	-0.081	-5%
10	-5	5.931	20	-2.5	4.787	5	-10	5.570	-1.143	-19%	-0.360	-6%	0.783	16%
10	0	6.636	20	0	5.882	5	0	5.644	-0.754	-11%	-0.992	-15%	-0.238	-4%
10	5	6.640	20	2.5	6.294	5	10	5.524	-0.346	-5%	-1.116	-17%	-0.770	-12%
10	10	5.600	20	5	5.866	5	20	4.325	0.266	5%	-1.275	-23%	-1.541	-26%
5	10	6.893	10	5	8.126	2.5	20	4.939	1.232	18%	-1.955	-28%	-3.187	-39%
0	10	6.860	0	5	8.285	0	20	4.913	1.424	21%	-1.947	-28%	-3.371	-41%
-5	10	6.031	-10	5	7.442	-2.5	20	4.156	1.411	23%	-1.875	-31%	-3.286	-44%
-10	10	3.393	-20	5	3.497	-5	20	2.702	0.103	3%	-0.691	-20%	-0.794	-23%
-10	5	5.649	-20	2.5	5.102	-5	10	5.015	-0.547	-10%	-0.634	-11%	-0.088	-2%
-10	0	5.944	-20	0	5.373	-5	0	5.167	-0.572	-10%	-0.777	-13%	-0.205	-4%
-10	-5	5.663	-20	-2.5	5.028	-5	-10	5.104	-0.635	-11%	-0.559	-10%	0.076	2%
-35	-35	0.124	-45	-30	0.139	-30	-45	0.095	0.014	11%	-0.029	-23%	-0.043	-31%
-17.5	-35	0.158	-22.5	-30	0.183	-15	-45	0.123	0.025	16%	-0.035	-22%	-0.060	-33%
0	-35	0.172	0	-30	0.199	0	-45	0.121	0.028	16%	-0.050	-29%	-0.078	-39%
17.5	-35	0.123	22.5	-30	0.124	15	-45	0.100	0.001	0%	-0.024	-19%	-0.024	-20%
35	-35	0.064	45	-30	0.053	30	-45	0.063	-0.011	-17%	-0.001	-2%	0.010	19%
35	-17.5	0.095	45	-15	0.076	30	-22.5	0.119	-0.019	-20%	0.024	25%	0.043	57%
35	0	0.592	45	0	0.377	30	0	0.696	-0.215	-36%	0.104	18%	0.319	85%
35	17.5	0.829	45	15	0.739	30	22.5	0.683	-0.090	-11%	-0.146	-18%	-0.055	-7%
35	35	0.192	45	30	0.304	30	45	0.101	0.112	58%	-0.091	-47%	-0.203	-67%
17.5	35	0.109	22.5	30	0.195	15	45	0.072	0.086	78%	-0.037	-34%	-0.122	-63%
0	35	0.125	0	30	0.144	0	45	0.100	0.019	15%	-0.025	-20%	-0.044	-31%
-17.5	35	0.113	-22.5	30	0.160	-15	45	0.077	0.047	42%	-0.035	-31%	-0.082	-52%
-35	35	0.139	-45	30	0.166	-30	45	0.089	0.026	19%	-0.050	-36%	-0.077	-46%
-35	17.5	0.387	-45	15	0.335	-30	22.5	0.370	-0.053	-14%	-0.017	-4%	0.036	11%
-35	0	0.603	-45	0	0.472	-30	0	0.603	-0.131	-22%	0.000	0%	0.131	28%
-35	-17.5	0.318	-45	-15	0.275	-30	-22.5	0.316	-0.043	-13%	-0.002	-1%	0.041	15%
Standard Deviation:									0.542	24%	0.614	15%	1.026	33%

C.3 Receptor Locations and Spacings

A sensitivity analysis was conducted using the ISCST3 model to determine what receptor locations and spacings should be used in the risk analysis for five types of waste management units (WMUs). A discussion of the analysis follows.

Because it takes a substantial amount of time for the ISCST3 model to execute, it was necessary to choose a limited number of receptors to be used in the dispersion modeling analysis. The larger the number of receptor points, the longer the run time. However, modeling fewer receptors may result in the omission of the maximum point for assessing exposure impacts. Therefore, a sensitivity analysis was conducted to determine the number of receptors needed for the model run and to locate ideal receptor placements.

A wind rose was plotted for each of the 29 meteorological stations to be used in the risk analysis for a 5-year time period in order to choose two meteorological stations for this sensitivity analysis. Little Rock, Arkansas, and Los Angeles, California, meteorological stations were selected for the sensitivity analysis. The wind roses show that Little Rock has very evenly distributed wind directions, and Los Angeles has a predominant southwest to west wind (Figure C-3). Little Rock and Los Angeles were chosen to determine if a higher density of receptors should be placed downwind of a site near Los Angeles, as compared to a site near Little Rock. Similarly, the 5th, 50th, and 95th percentile of sizes of LAUs were used in the sensitivity analysis to determine whether sizes of units can affect receptor locations and spacings. The areas of the 5th, 50th, and 95th percentile of sizes of LAUs are 1,200 m², 100,000 m², and 1,700,000 m², respectively.

The dispersion modeling was conducted using two sets of receptor grids. The first set of receptor points (Cartesian receptor grid) was placed around the modeled source with distances of 25, 50, 75, and 150 meters from the edge of the unit. Square-shaped ground-level area sources were used in the modeling. Therefore, these receptors are located on five squares surrounding the source. The second set of receptor points (polar receptor grid) was placed outside of the first set of receptors to 10 kilometers from the center of the source. Since the ISCST3 model's area source algorithm does not consider elevated terrain, receptor elevations were not input in the modeling.

In this sensitivity analysis, both downwind and lateral receptor spacings were investigated for three unit sizes using 5 years of meteorological data from Little Rock and Los Angeles. For the first set of receptor points (i.e., Cartesian receptor grid), five downwind distances of 25, 50, 75, and 150 meters from the edge of the source were used. For lateral receptor spacing, choices of 64, 32, and 16 equally spaced receptor points for each square were used in the modeling to determine the number of receptors needed to catch the maximum impacts. (See Figures C-5a through C-5c for Cartesian receptor locations and spacings [50th percentile]). For the second set of receptor points (i.e., polar receptor grid), about 20 downwind distances (i.e., receptor rings) were used. Receptor lateral intervals of 22.5° and 10° were used to determine whether 22.5° spacing can catch the maximum impacts. With a 22.5° interval, there are 16 receptors on each ring. There are 36 receptors on each ring for the 10° interval. See Figures C-6a and C-6b for polar receptor locations (5th percentile).

The results (Figures C-7a through C-7f) show that the maximum downwind concentrations decrease sharply from the edge of the area source to 150 meters from the source. The maximum concentrations decrease more sharply for a smaller area source than for a larger one. This means that more close-to-source receptors are generally needed for a small area source than for a large one.

The results also show that the maximum impacts are generally higher for a dense receptor grid (i.e., 64 or 32 receptors on each square) than for a scattered receptor grid (i.e., 16 receptors on each square). However, the differences of the maximum receptor impacts are not significant between a dense and a scattered receptor grid (Figures C-7a through C-7f). It should be noted that the above conclusions apply to both Little Rock and Los Angeles. This means that the distribution of wind directions does not play an important role in determining receptor lateral spacings.

Figures C-8a through C-8f compare the maximum concentrations at each ring for 22.5° and 10° intervals. The results show that the differences of the maximum concentrations are greater for close-to-source receptors than for further out receptors, and the differences are greater for larger area sources than for smaller area sources. The differences of the maximum concentrations for 22.5° and 10° intervals are generally small, and the concentrations tend to be the same at 10 kilometers. The conclusions were drawn from both Little Rock and Los Angeles meteorological data.

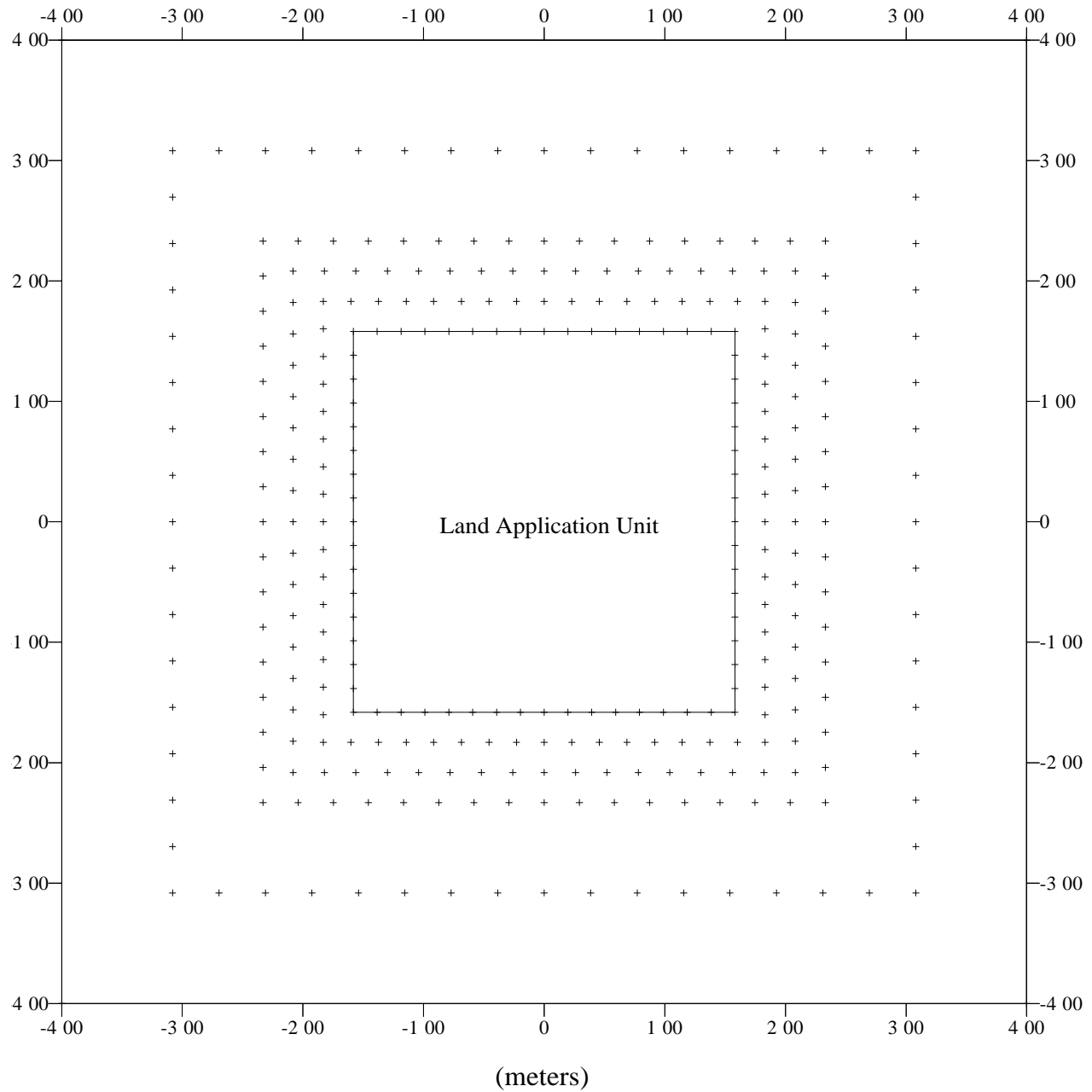


Figure C-5a. Cartesian Receptor Grid (64 receptors each square).

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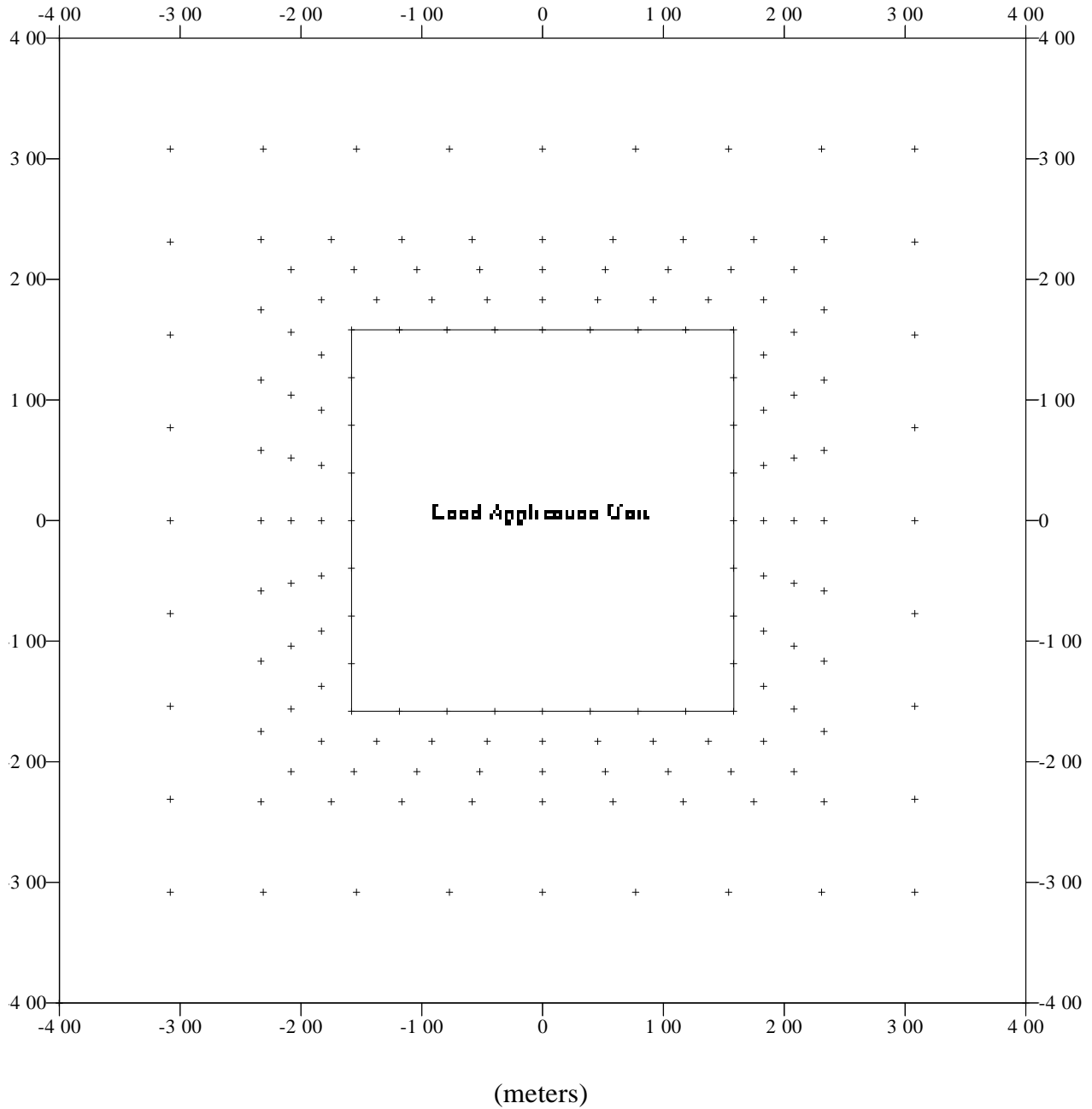


Figure C-5b. Cartesian Receptor Grid (32 receptors each square).

US EPA ARCHIVE DOCUMENT

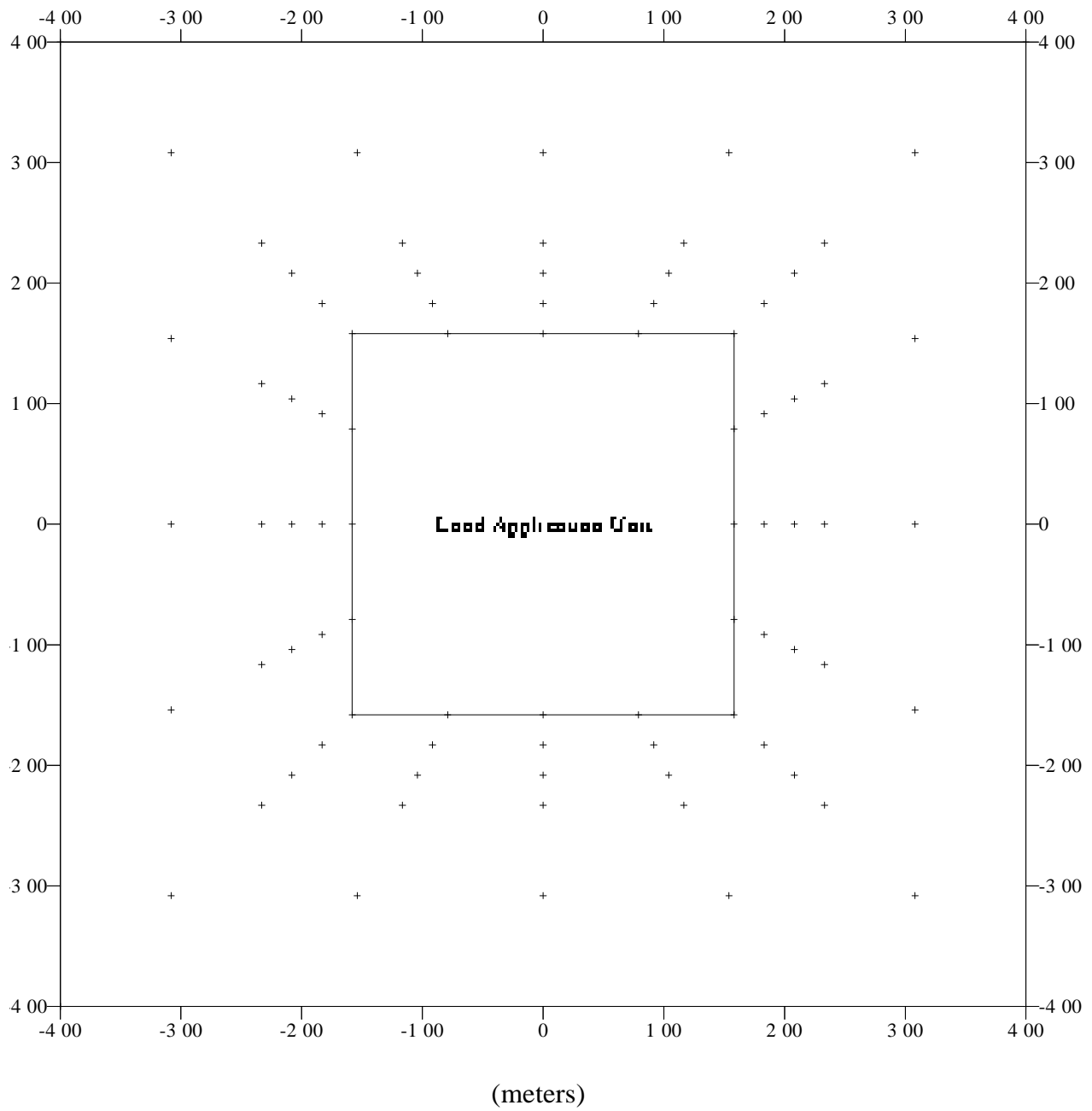


Figure C-5c. Cartesian Receptor Grid (16 receptors each square).

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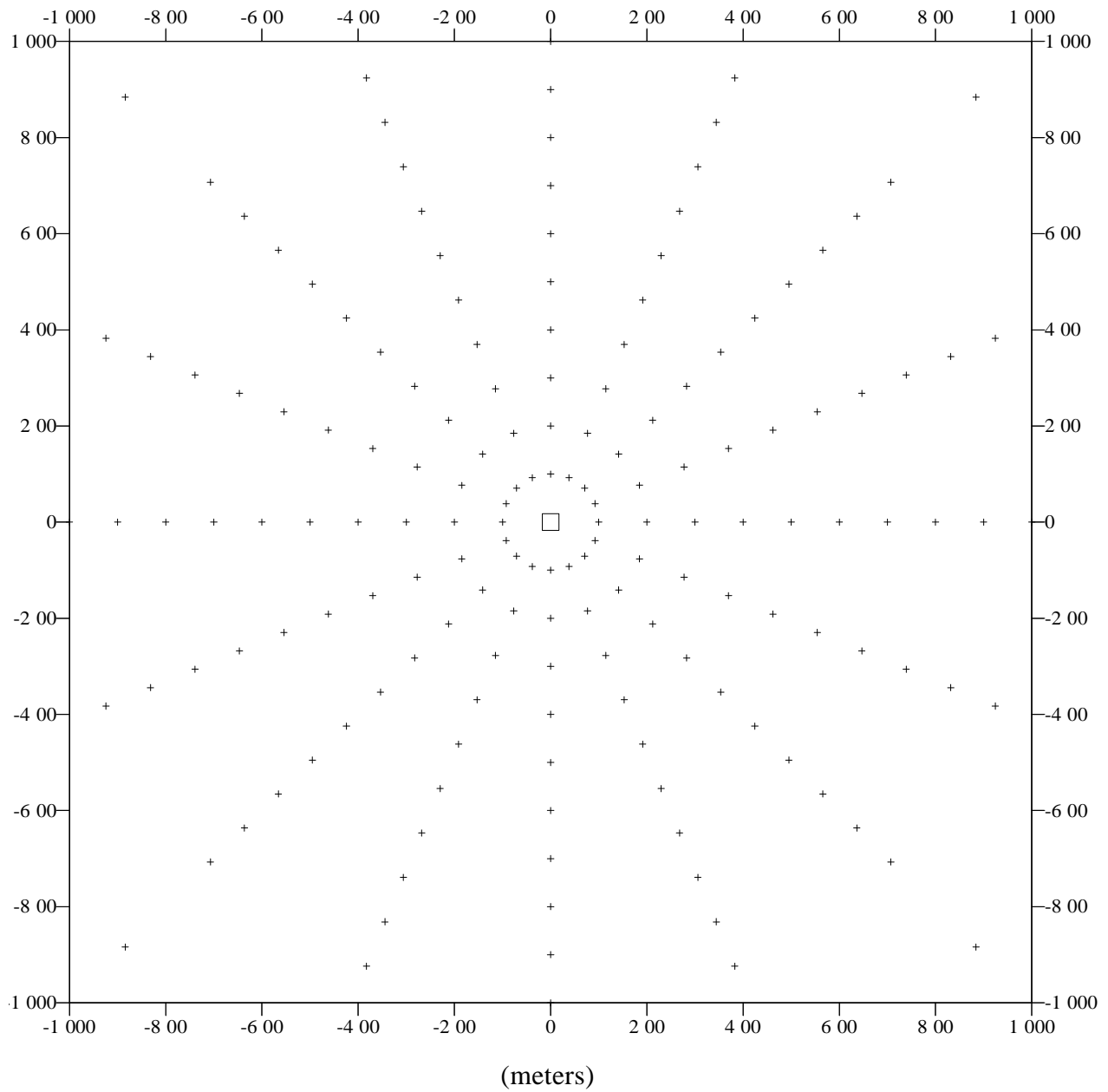


Figure C-6a. Polar Receptor Grid (22.5 degree).

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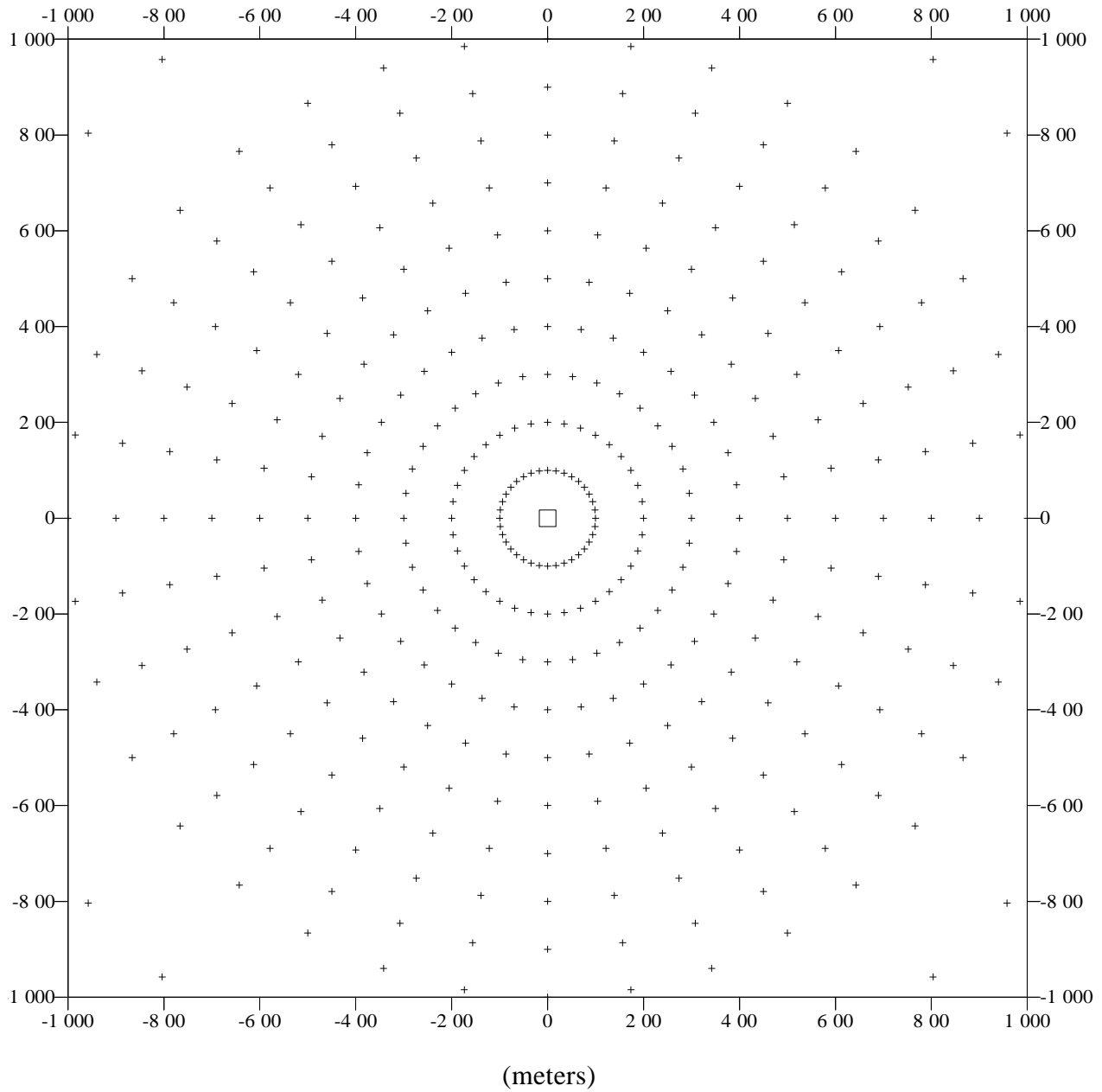


Figure D-6b. Polar Receptor Grid (10 degree).

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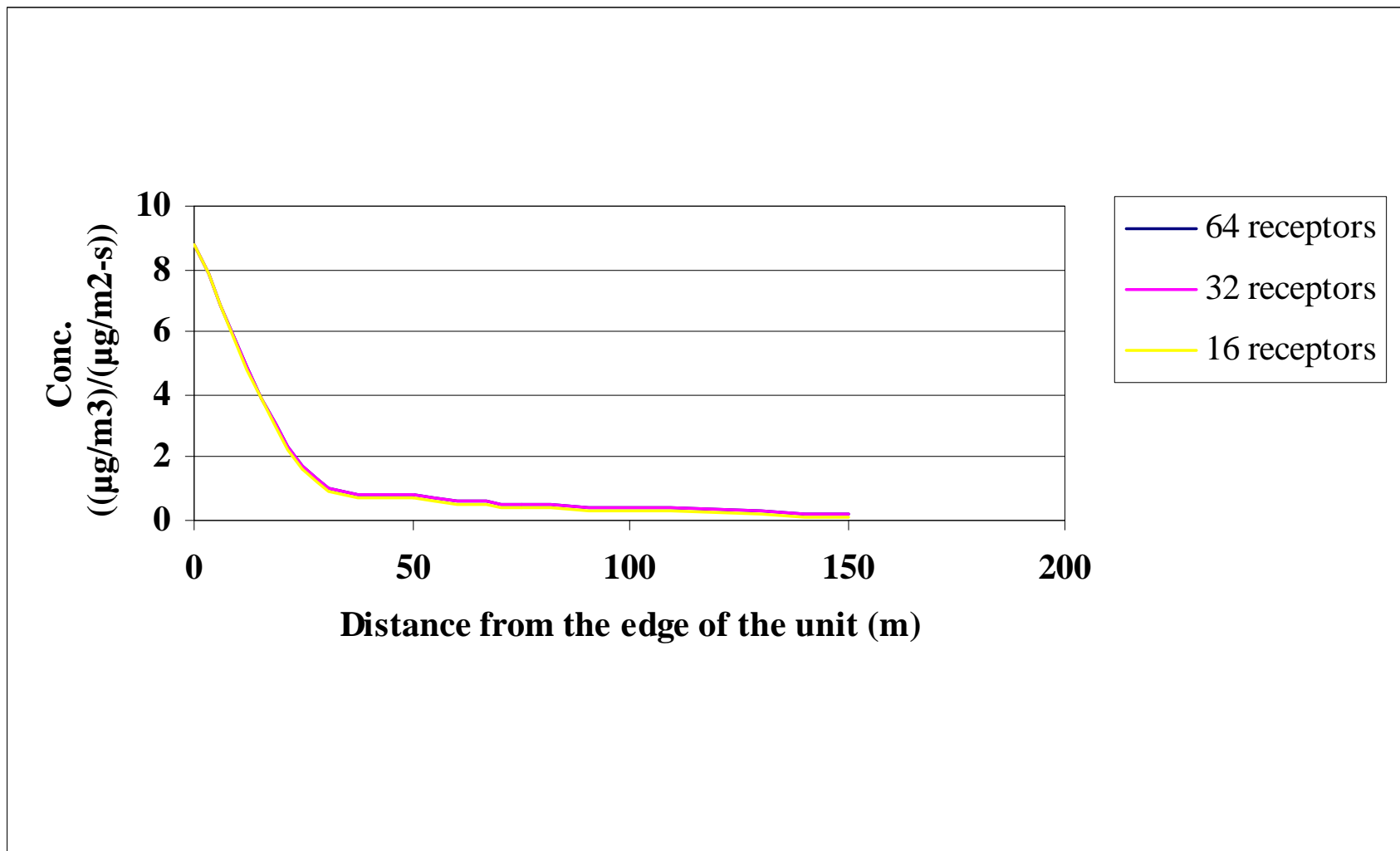


Figure C-7a. Maximum Concentrations (5th Percentile, LAU, Los Angeles, CA)

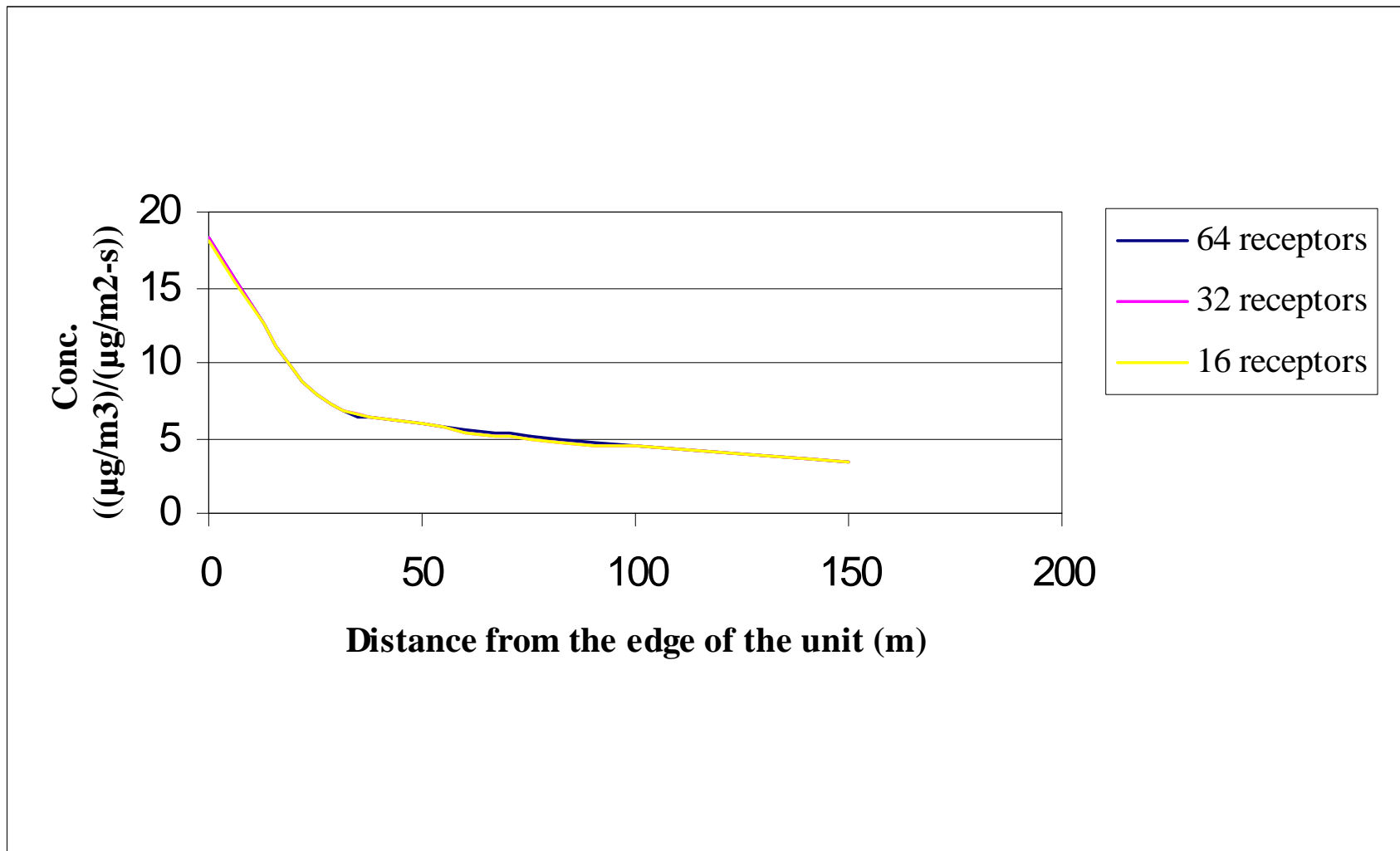


Figure C-7b. Maximum Concentrations (50th Percentile, LAU, Los Angeles, CA)

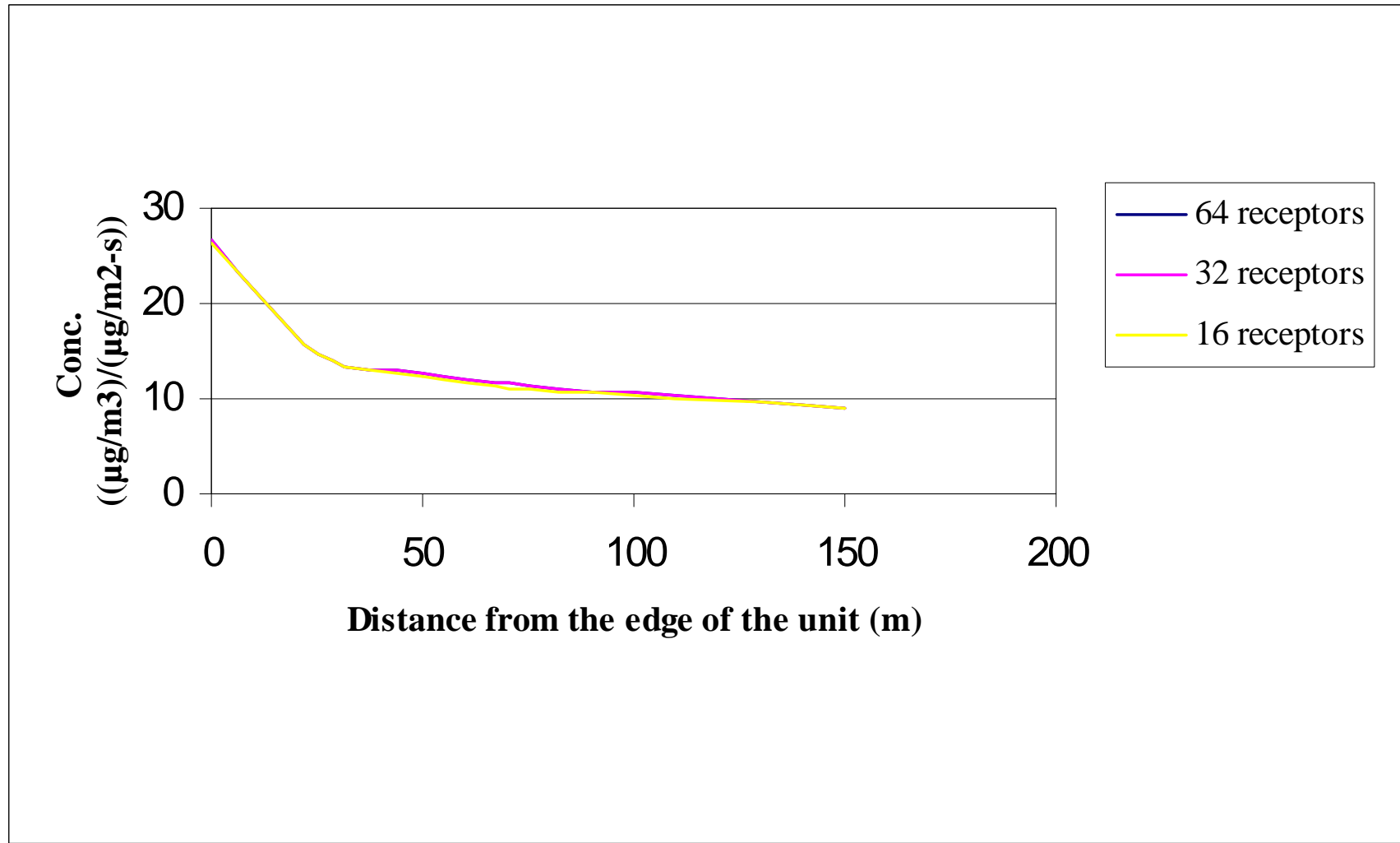


Figure C-7c. Maximum Concentrations (95th Percentile, LAU, Los Angeles, CA)

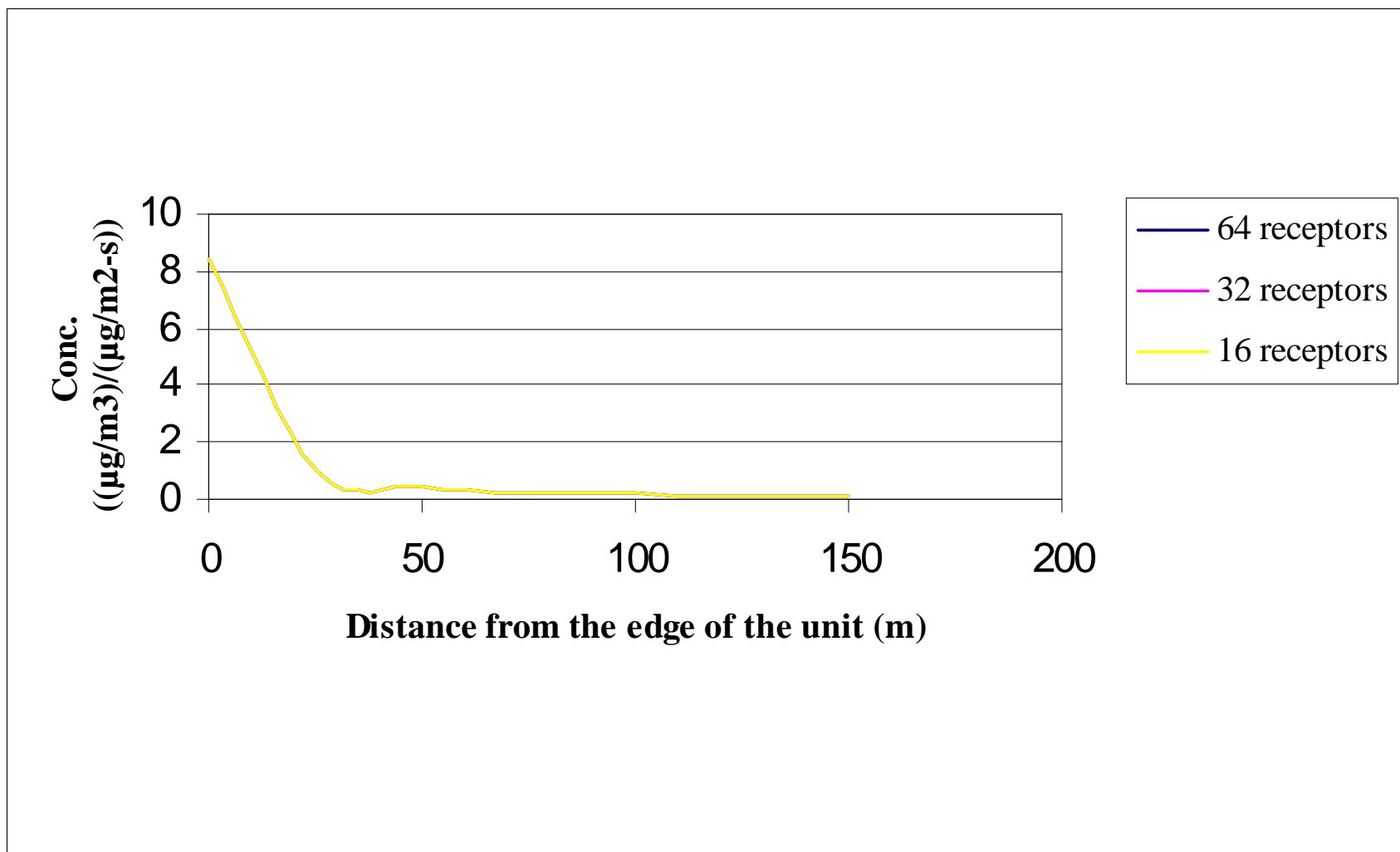


Figure C-7d. Maximum Concentrations (5th Percentile, LAU, Little Rock, AR)

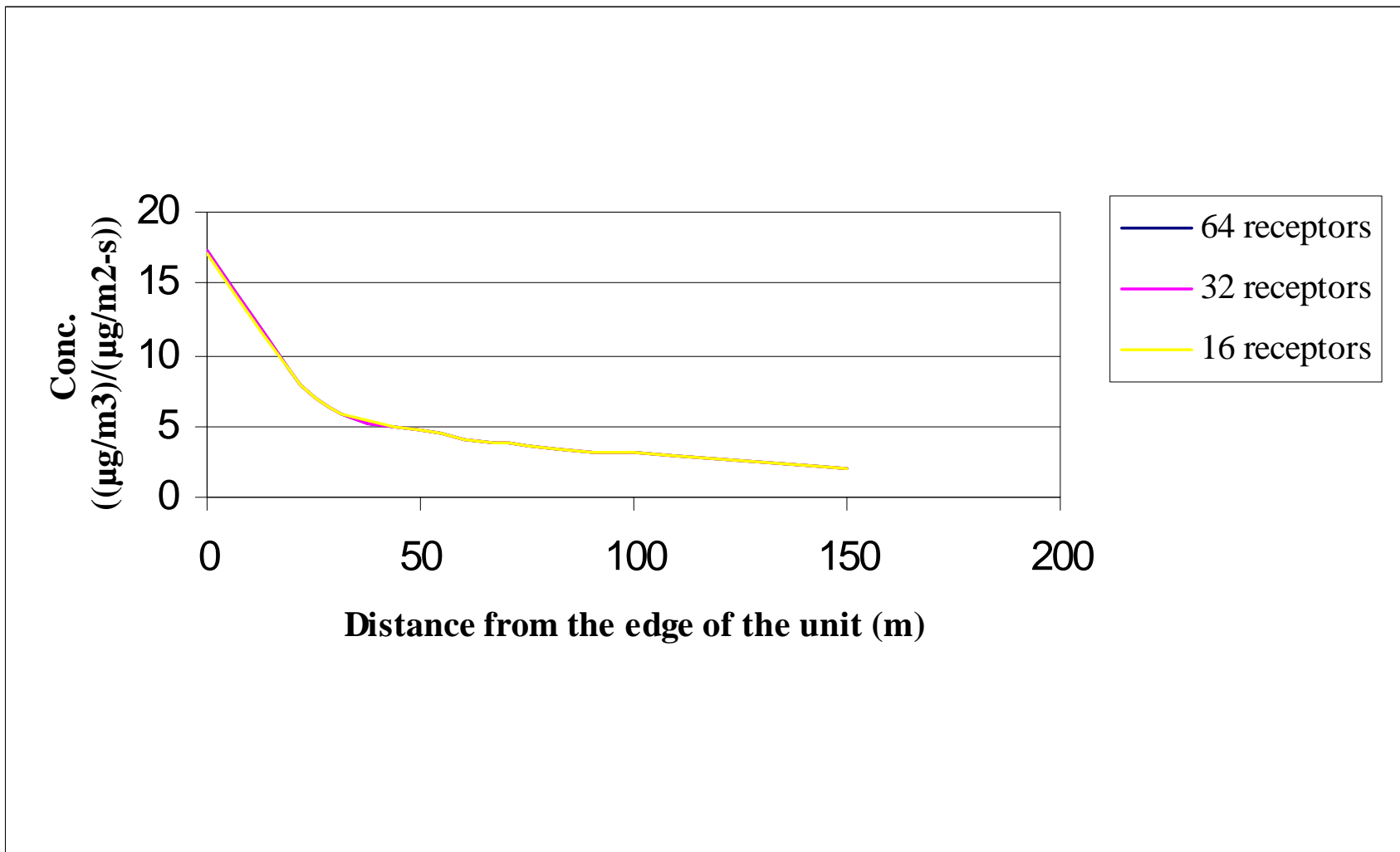


Figure C-7e. Maximum Concentrations (50th Percentile, LAU, Little Rock, AR)

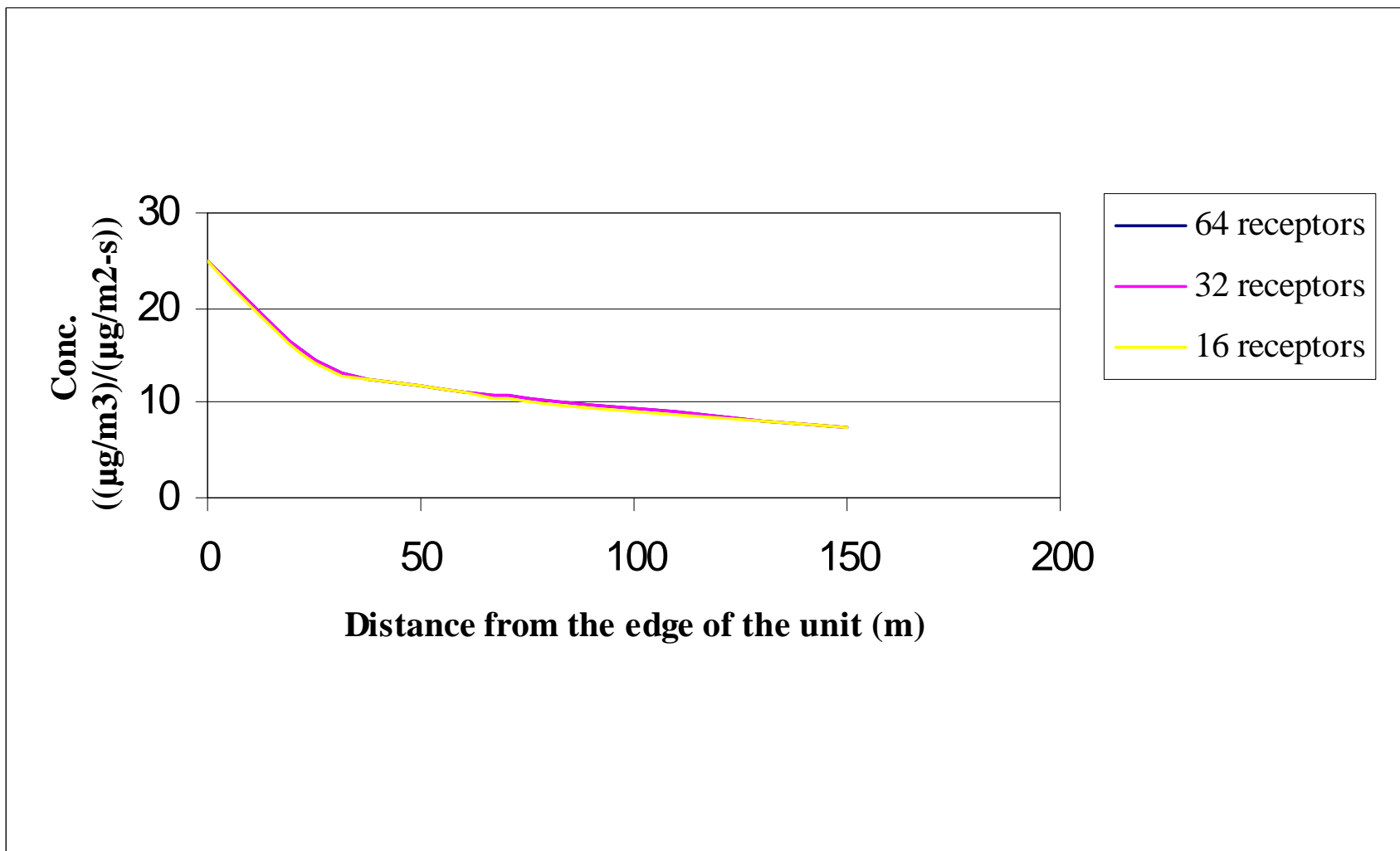


Figure C-7f. Maximum Concentrations (95th Percentile, LAU, Little Rock, AR)

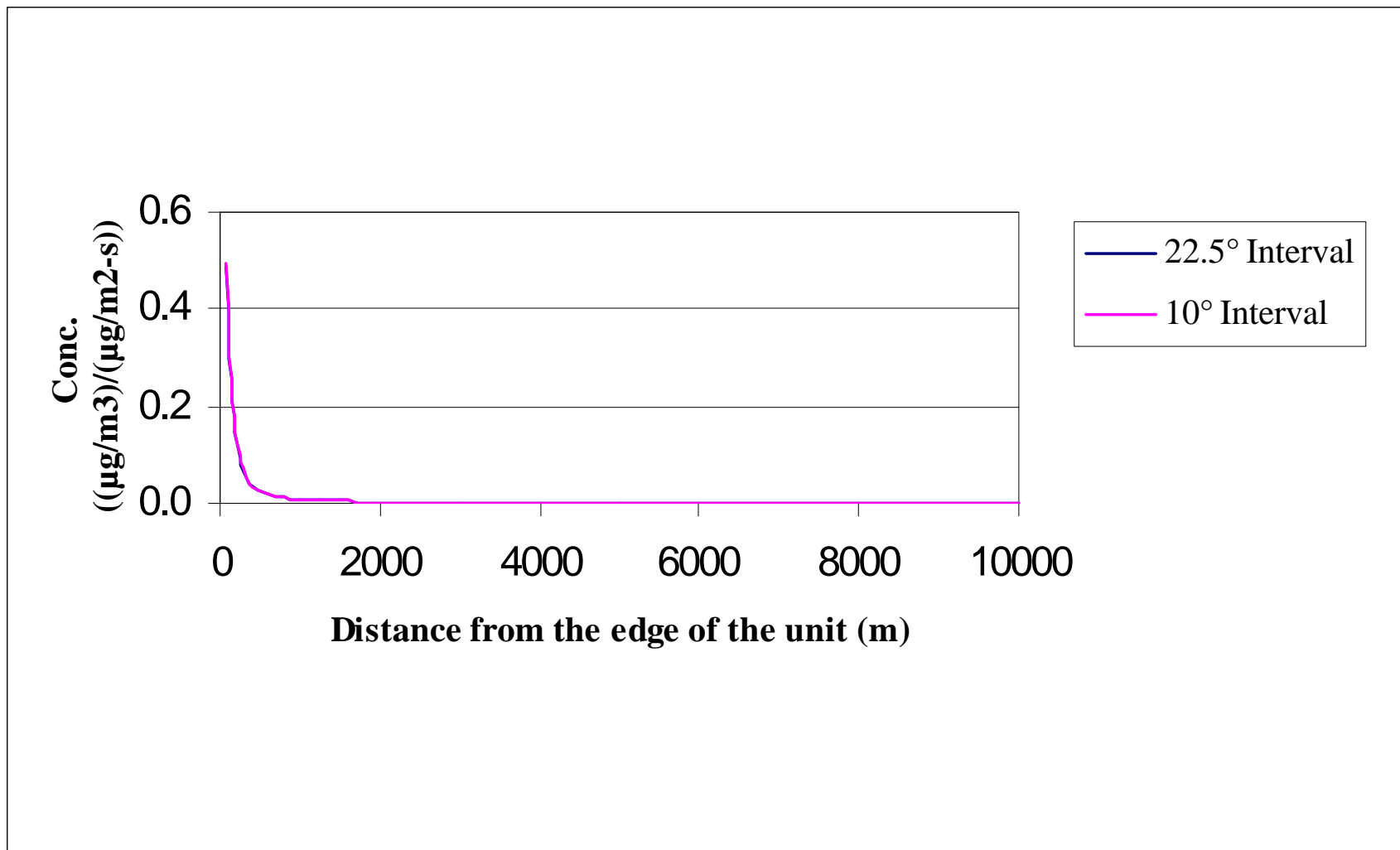


Figure C-8a. Maximum Concentrations (5th Percentile, LAU, Lose Angeles, CA)

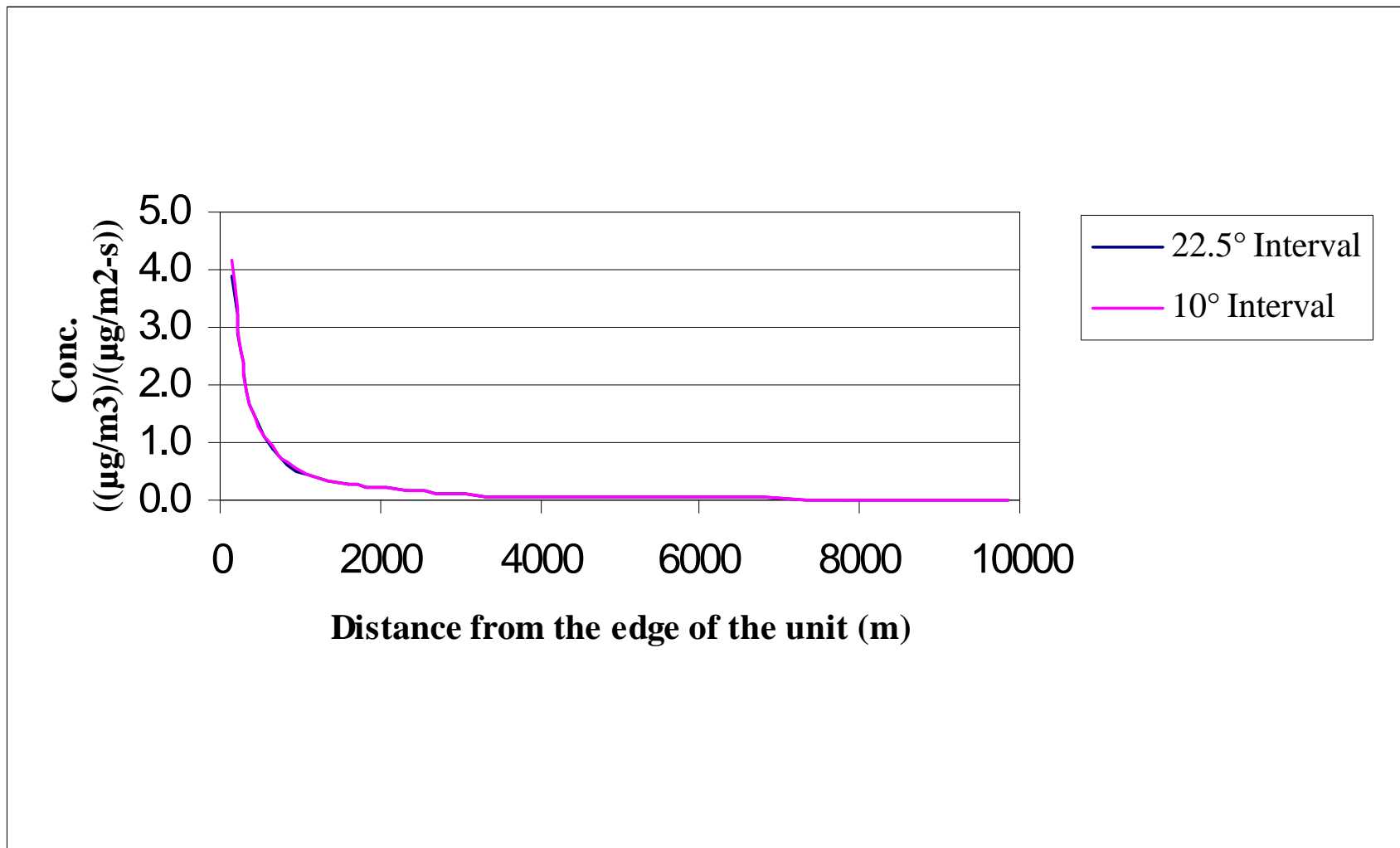


Figure C-8b. Maximum Concentrations (50th Percentile, LAU, Los Angeles, CA)

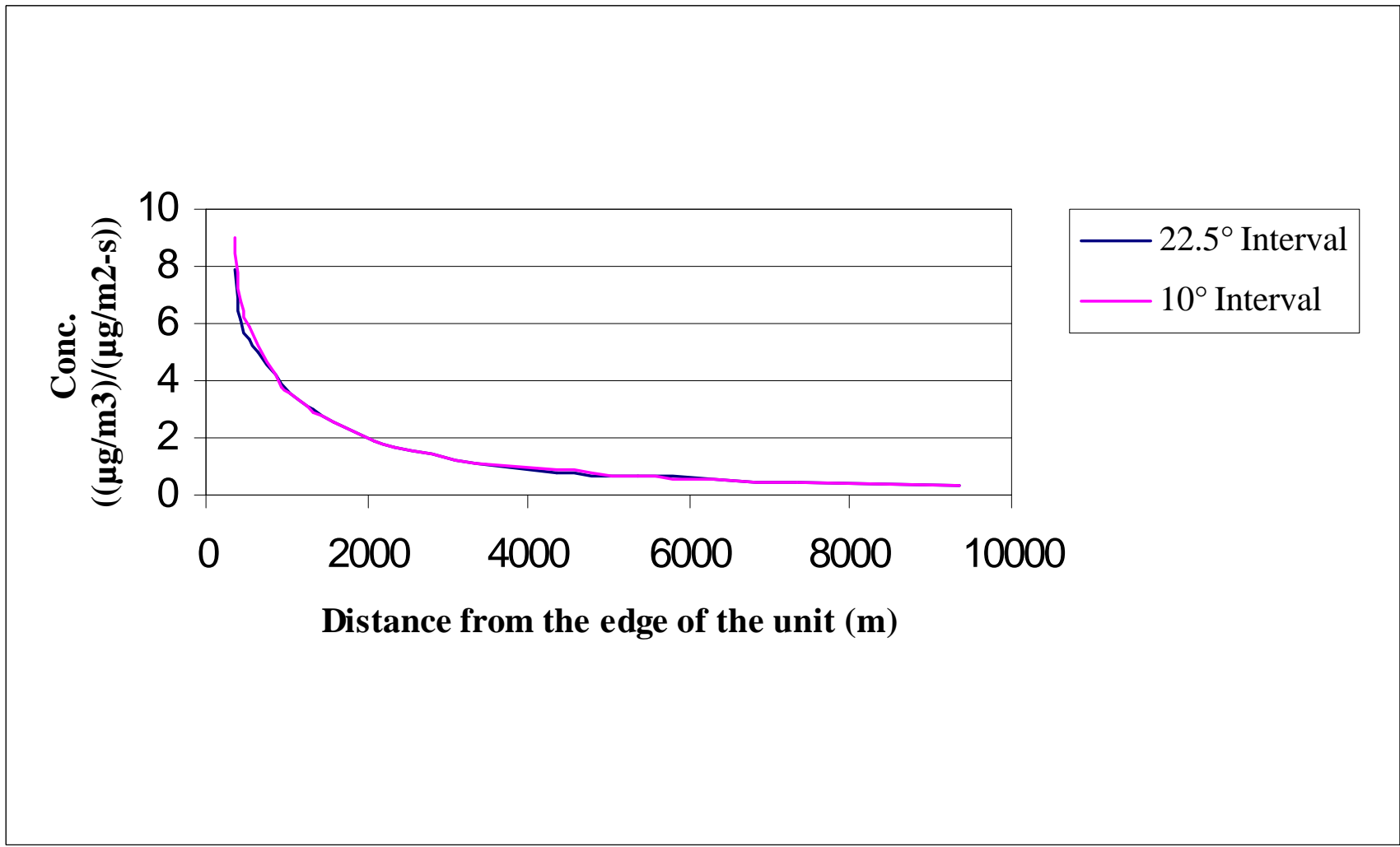


Figure C-8c. Maximum Concentrations (95th Percentile, LAU, Los Angeles, CA)

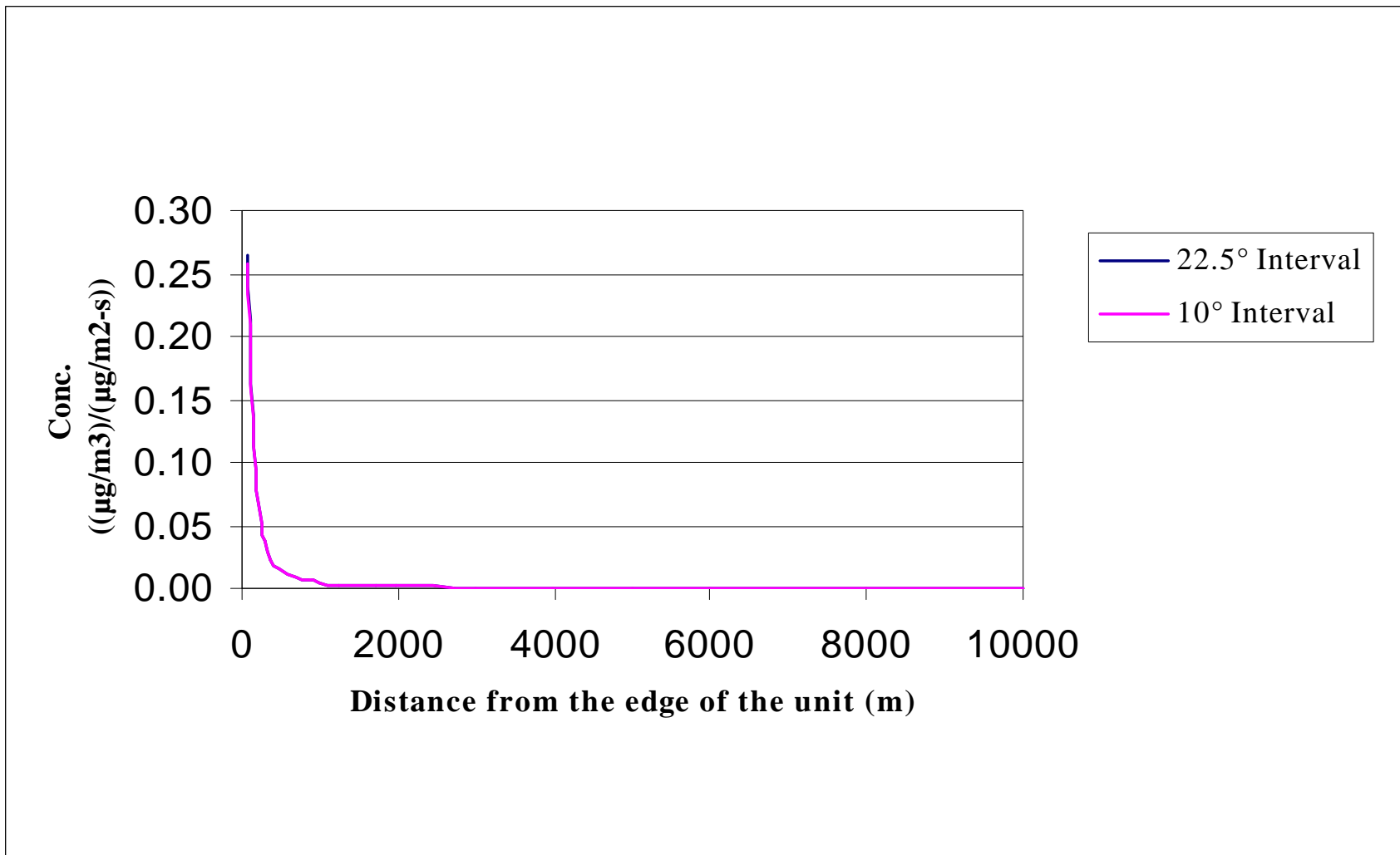


Figure C-8d. Maximum Concentrations (5th Percentile, LAU, Little Rock, AR)

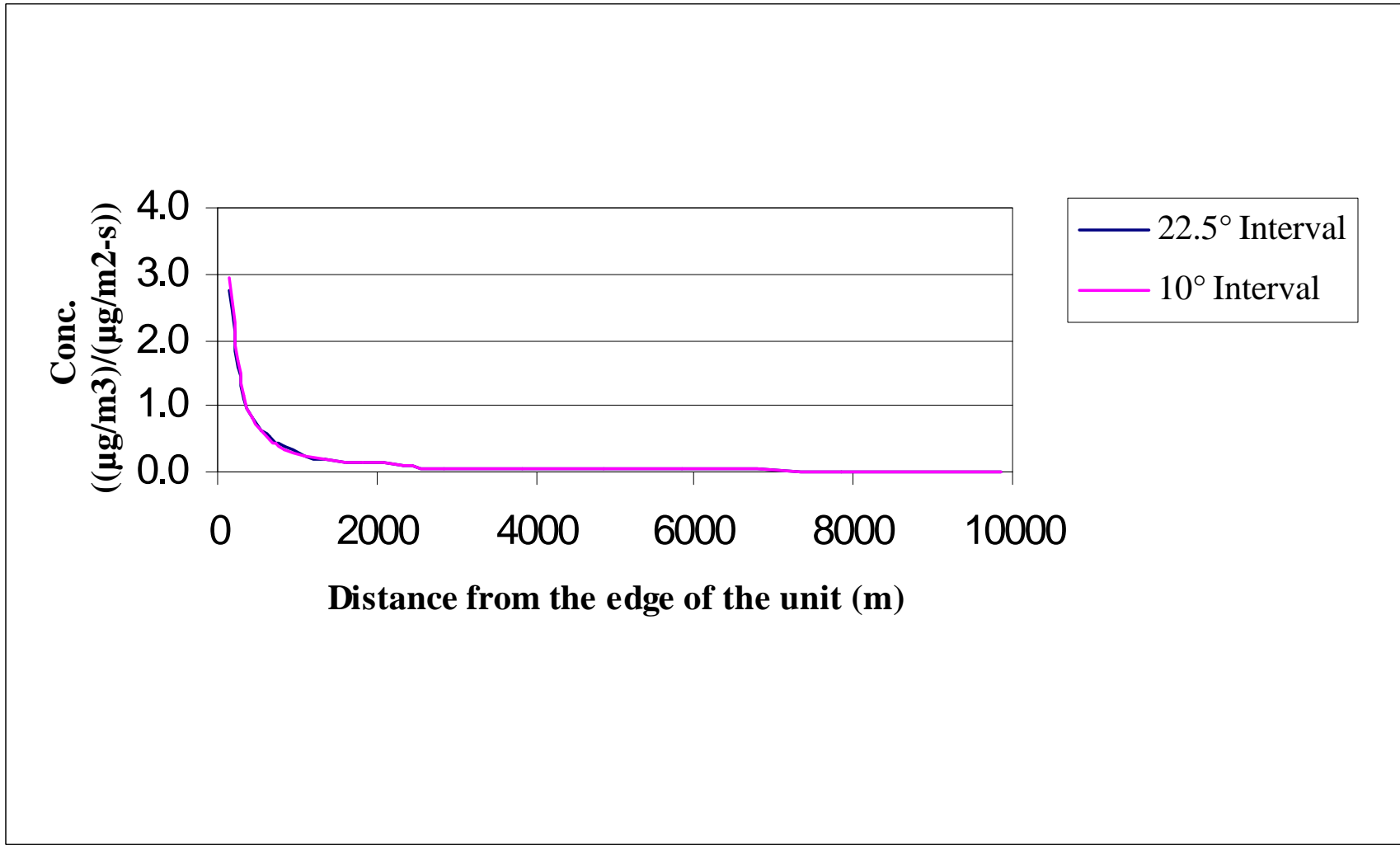


Figure C-8e. Maximum Concentrations (50th Percentile, LAU, Little Rock, AR)

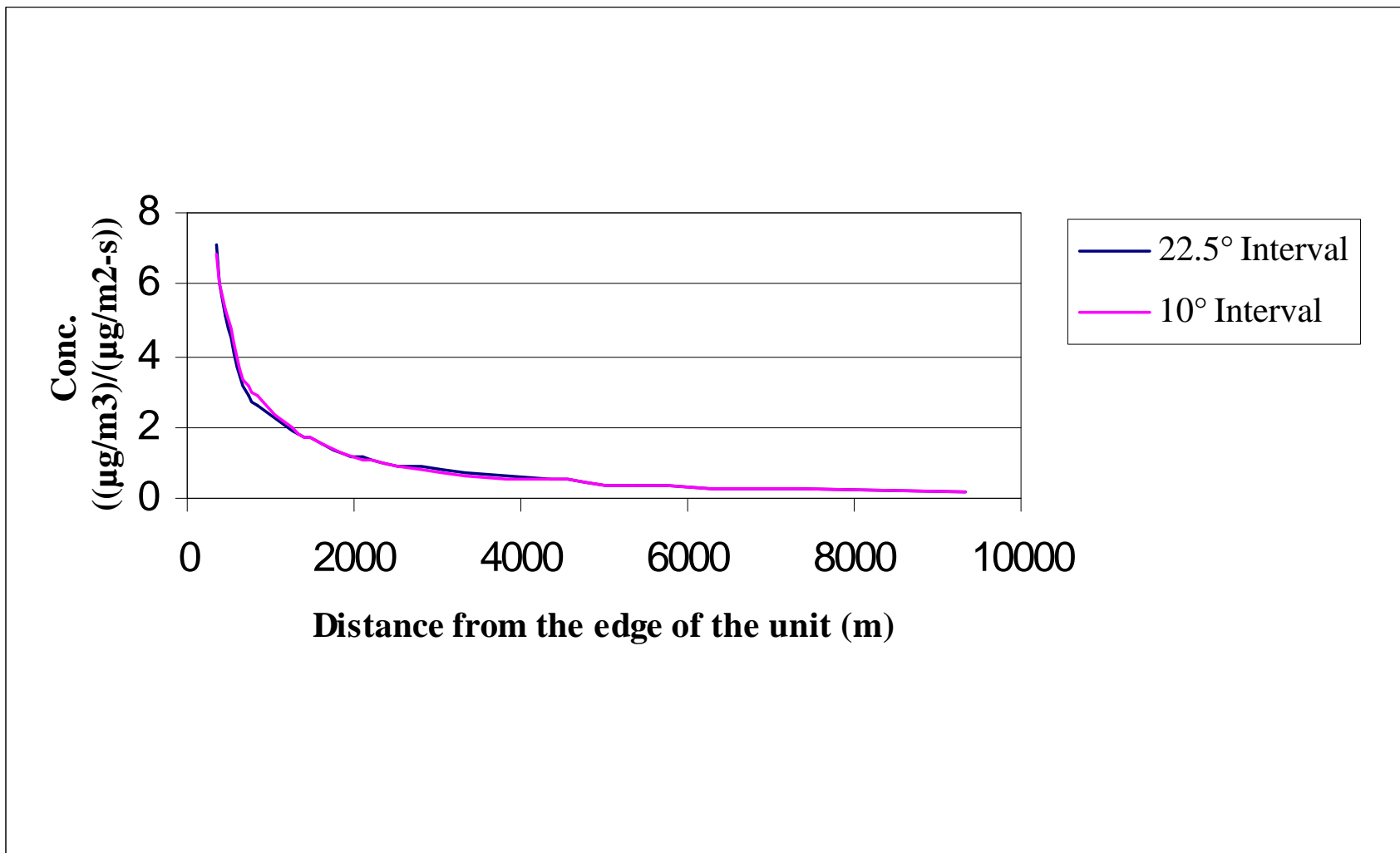


Figure C-8f. Maximum Concentrations (95th Percentile, LAU, Little Rock, AR)

C.4 An Analysis on Windroses at the 29 Sites

The hourly meteorological data from the 29 meteorological stations used in the Air Characteristic Study were used to generate windroses. A windrose consists of 16 directions, with the angle between any two adjacent directions being 22.5° . The prevailing wind directions for the 29 meteorological stations were counted to estimate the number of entries in each wind directions category. The results are presented in Figure C-9.

The narrowness of the most frequent wind directions for each of the 29 meteorological stations was examined. Based on the narrowness (or angles) of the most frequent wind directions, four categories were used to distinguish the windroses for the 29 meteorological stations. The four categories of windroses are:

- Narrowly distributed: most frequent wind directions no greater than 45°
- Moderately distributed: most frequent wind directions no greater than 90°
- Evenly distributed: no obvious predominant wind directions
- Bimodally distributed: most frequent wind directions are from two opposite directions.

The number of meteorological stations in each category is given in Table C-3. Figure C-10 gives some examples of windroses for each category. The windroses for the 29 meteorological stations are available and can be provided upon request.

An examination of the windroses and the maximum unitized annual average air concentrations from the Air Characteristic Study revealed that the sites with high concentrations are those with narrowly distributed wind directions. Simply put, persistent wind direction consistently blows pollutants from the source to the same receptors. Therefore, the more often the wind blows in a certain direction, the more likely high cumulative concentrations will occur at sites in that direction.

Air concentrations from a source are inversely proportion to windspeed. Given the same distribution of wind directions, a site with lower windspeed will have higher concentrations. The windroses show that, in the prevailing wind direction, the percentage of light wind occurring at a site with narrowly distributed wind directions is often higher than that at a site with evenly distributed wind directions. Therefore, we can conclude that a site with narrowly distributed wind directions will most likely produce the highest long-term average air concentrations.

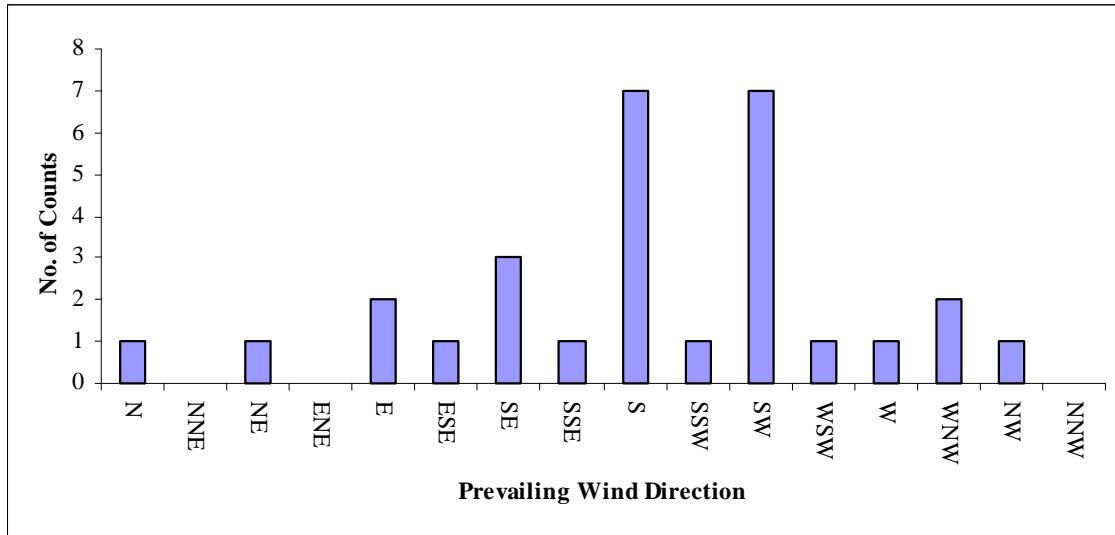
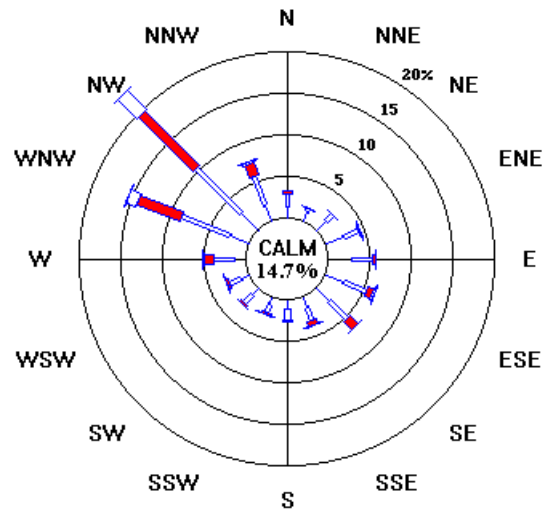
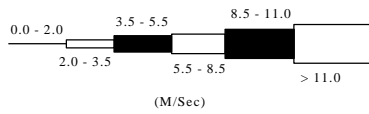


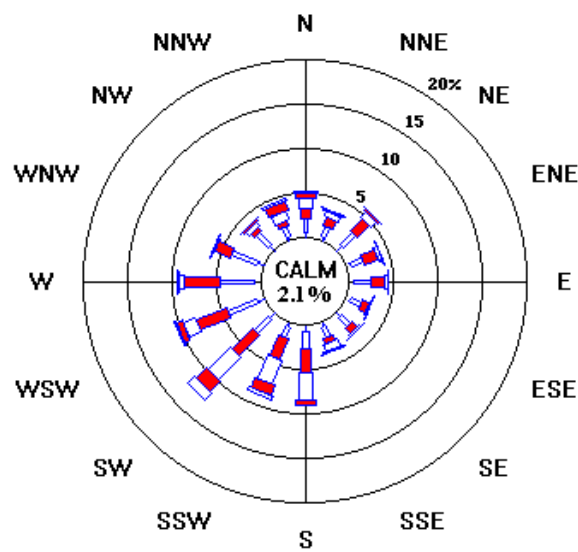
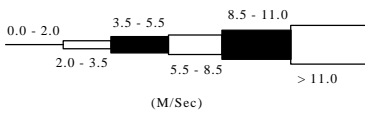
Figure C-9. Counts of Prevailing Wind Directions in Each Direction

Table C-3. No. of Met Stations with Different Shapes of Windroses

Shape of Windrose	No. of Stations
Narrowly distributed	10
Moderately distributed	4
Evenly distributed	6
Bi-modally distributed	9



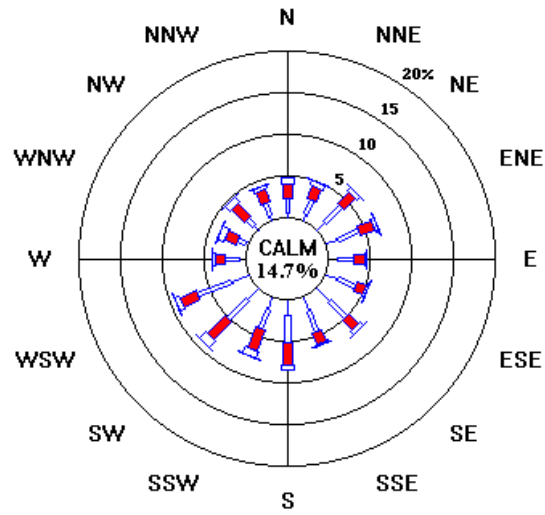
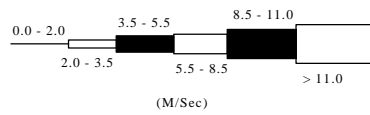
Narrowly Distributed Windrose



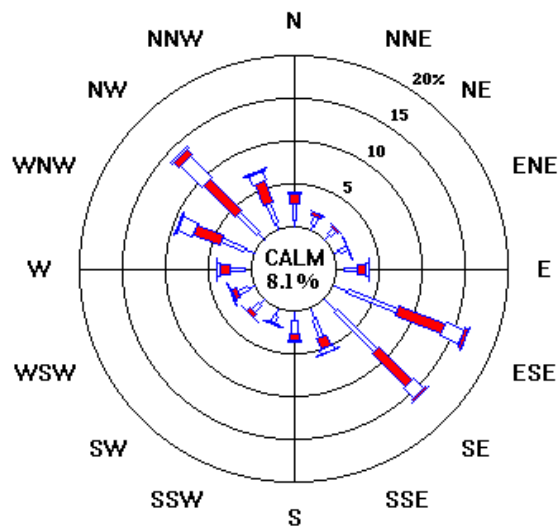
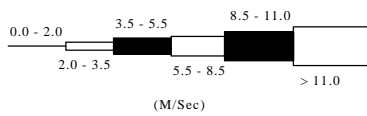
Moderately Distributed Windroses

Figure C-10. Examples of Different Shapes of Windroses

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Evenly Distributed Windrose



Bi-modally Distributed Windrose

Figure C-10 (Continued). Examples of different Shapes of Windroses

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Appendix D

Derivation of Chronic Inhalation Noncancer and Cancer Health Benchmark Values

NONCARCINOGENS

DERIVATION OF INHALATION REFERENCE CONCENTRATIONS

2-Chlorophenol

CAS #95-57-8

RfC:	0.0014 mg/m ³
Basis for RfC:	Route-to-route extrapolation from the RfD
Critical Study:	Exon, J.H., and L.D. Koller. 1982. Effects of transplacental exposure to chlorinated phenols. <i>Environ Health Perspect</i> 46:137-140 (as cited in U.S. EPA, 1998).
Critical Dose:	5 mg/kg/d [X] NOAEL [] LOAEL
Critical Effect:	Increase in conception rate and number of stillbirths and decrease in size of litters
Species:	Rat
Route of Exposure:	Drinking water
Duration:	10 weeks
Uncertainty Factor:	1000: 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations 10 for use of a subchronic study
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{RfD} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 0.005 \text{ mg/kg/d} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 0.0014 \text{ mg/m}^3$$

where:

70 kg = default adult human body weight

20 m³/d = default human daily rate of inhalation

Calculations assume 100% absorption.

Summary of Study:

The RfD is based on a NOAEL of 5 mg/kg/d with a LOAEL of 50 mg/kg/d for reproductive effects in a subchronic drinking water study in rats (Exon and Koller, 1982, as cited in U.S. EPA, 1998). In this study, groups of 12 to 20 weanling female Sprague-Dawley rats were exposed to 0, 5, 50, or 500 ppm of 2-chlorophenol in the drinking water and bred after 10 weeks of 2-chlorophenol treatment. Treatment was continued during breeding, gestation, and weaning. The weanling rats were evaluated for percent conception, litter size, birth weight, weaning weight, number of stillbirths, and hematology (hematocrit, hemoglobin levels, red and white cell counts, and mean corpuscular volume). The evaluations revealed an increase in the conception rate and in the number of stillborns as well as a decrease in the size of the litters in the rats exposed to 500 ppm, which can be converted to a dosage of 50 mg/kg/d—the LOAEL. No effects

were observed at 50 ppm, which can be converted to a dosage of 5 mg/kg/d. Dividing the NOAEL of 5 mg/kg/d by an uncertainty factor of 1,000 (10 factors each for animal to human extrapolation, interspecies variability, and the use of subchronic data), yields the RfD of 0.005 mg/kg/d (EPA, 1998).

Rationale for Route-to-Route Extrapolation:

A first pass in the liver or respiratory tract is not expected to contribute to the toxicity of 2-chlorophenol because it has been demonstrated that the toxic action of the lower chlorinated phenols is due to the undissociated molecule. In studies with rats, it was observed that the toxicity of chlorophenols administered via subcutaneous and intraperitoneal routes is similar to that which is observed in orally administered chlorophenols (Deichmann and Keplinger, 1981). Since the dermal irritation index for 2-chlorophenol is low, no significant portal of entry effect is expected from inhalation exposure to 2-chlorophenol (HSDB, 1998).

Consequently, route-specific difference in toxicity is not expected for 2-chlorophenol. Therefore, in accordance with EPA guidelines (U.S. EPA, 1994), the oral toxicity data for 2-chlorophenol are adequate for use in the calculation of an inhalation RfC for the substance.

Strengths and Uncertainties:

The strength of the RfC is that it is based on an RfD on IRIS that has undergone rigorous EPA peer review.

The major uncertainty of the RfC is the lack of inhalation toxicity studies in humans or animals and the use of default values in the route-to-route extrapolation.

References:

Deichmann, W.B., and M.L. Keplinger. 1981. Aromatic Hydrocarbons. In: G.D. Clayton and F.E. Clayton (eds). *Patty's Industrial Hygiene and Toxicology*. 3rd revised edition. Volume 2A: Toxicology. New York: John Wiley and Sons, pp. 3325-3415.

Exon, J.H., and L.D. Koller. 1982. Effects of transplacental exposure to chlorinated phenols. *Environ Health Perspect* 46:137-140 (as cited in U.S. EPA, 1998).

Hazardous Substances Databank (HSDB): 2-Chlorophenol. 1998. Online database. National Library of Medicine, Bethesda, MD.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). 2-Chlorophenol. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

Cobalt
CAS #7440-48-4

RfC:	0.00001 mg/m ³
Critical Study:	National Toxicology Program (NTP). 1996. NTP Technical Report on the toxicology and carcinogenesis studies of cobalt sulfate heptahydrate (CAS No.10026-24-1) in F344/N rats and B6C3F ₁ mice (inhalation studies). Draft. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication no. 96-3961. NTP TR-471.
Critical Dose:	0.11 mg/m ³ [] NOAEL [X] LOAEL
Critical Effect:	Hyperplasia of the lateral wall of the nose and atrophy of the olfactory epithelium
Species:	Rat
Route of Exposure:	Inhalation
Duration:	104 weeks
Uncertainty Factor:	300: 10 for use of a LOAEL 10 for protection of sensitive human subpopulations 3 for extrapolation from animals to humans with the use of a LOAEL adjusted for human equivalent concentration
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{LOAEL}_{\text{HEC}} \div \text{UF} = 0.004 \text{ mg/m}^3 \div 300 = 0.00001 \text{ mg/m}^3$$

Summary of Study:

Groups of 50 male and 50 female rats and mice were exposed to aerosols containing 0, 0.3, 1.0, and 3.0 mg/m³ cobalt sulfate heptahydrate (0, 0.11, 0.4, and 1.1 mg cobalt/m³) 6 hours/day, 5 days per week, for 104 weeks (NTP, 1996). Survival of both rats and mice were similar to controls. The respiratory tract was the primary site of nonneoplastic lesions and neoplasms. Cobalt sulfate heptahydrate caused a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract. Olfactory epithelial atrophy and hyperplasia of the lateral wall of the nose were increased significantly at all dose levels in both male and female rats, and severity of these lesions increased with increasing exposure concentration. Olfactory epithelial atrophy was seen at 0.4 and 1.1 mg cobalt/m³ in male and female mice. Hyperplasia of the adrenal medulla was increased significantly in male rats at 0.11 mg cobalt/m³ and cytoplasmic vacuolization of the bronchus was increased significantly in male and female mice at this concentration.

A LOAEL of 0.11 mg cobalt/m³ was selected based on respiratory effects (olfactory epithelial atrophy and hyperplasia of the lateral wall of the nose) observed at this lowest dose in rats. This LOAEL was adjusted for continuous exposure (0.02 mg/m³). A LOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-5 (insoluble, approximately spherical particles). An uncertainty factor of 300 was applied: 10 for use of a LOAEL, 10 for protection of sensitive human subpopulations, and 3 for extrapolation from animals to humans with the use of a LOAEL adjusted for human equivalent concentration.

Conversion Factors:

Dose levels are:

% cobalt in cobalt sulfate heptahydrate = 38% = 0.38.

0.3 mg CoSO₄•7H₂O/m³ x 0.38 = 0.11 mg Co/m³; 1.0 mg CoSO₄•7H₂O/m³ = 0.4 mg Co/m³;

3.0 mg CoSO₄•7H₂O/m³ = 1.1 mg Co/m³.

LOAEL_{ADJ} = 0.11 mg Co/m³ x (6 h/24 h) x (5/7 d) = 0.02 mg Co/m³

LOAEL_{HEC} = LOAEL_{ADJ} x RDDDR_r

LOAEL_{HEC} = 0.02 mg Co/m³ x 0.209 = 0.004 mg Co/m³

where

LOAEL_{ADJ} is the adjusted LOAEL, LOAEL_{HEC} is the human equivalent concentration LOAEL, RDDDR_r (regional deposited dose ratio) is a multiplicative factor used to adjust an observed inhalation particulate concentration mass median aerodynamic diameter of an animal to the predicted inhalation particulate exposure concentration for a human; based on mass median aerodynamic diameter MMAD = 1.5 μm, geometric standard deviation (sigma g) = 2.2, mean body weight of female rat = 237 g, extrathoracic respiratory effects; RDDDR calculated using EPA RDDDR Program (computer disk).

Additional Information:

Human studies support the critical endpoint (the respiratory system) identified in the NTP (1996) study. Respiratory irritation, wheezing, asthma, pneumonia, and fibrosis have been reported in epidemiological studies at concentrations ranging from 0.007 to 0.893 mg cobalt/m³ as cobalt dust (Antilla et al., 1986; Davison et al., 1983; Demedts et al., 1984; Hartung et al., 1982; Shirakawa et al., 1988, 1989; Van Cutsem et al., 1987, as cited in ATSDR, 1992). Sprince et al. (1988) reported a strong relationship between work-related wheezing and cobalt dust exposure in workers engaged in tungsten carbide production. The relative odds for work-related wheezing doubled when cobalt exposure exceeded 0.050 mg/m³ compared with exposure to less than 0.050 mg/m³.

Animal studies have also shown a variety of respiratory effects. Exposure of rats and rabbits to mixed cobalt oxides resulted in lesions of the alveolar region of the respiratory tract (Johanssen et al., 1984, 1987, as cited in ATSDR, 1992). Lifetime exposure of hamsters to cobalt oxide resulted in emphysema (Wehner et al., 1977, as cited in ATSDR, 1992). An NTP subchronic (13-week) study reported squamous metaplasia of the larynx, histiocytic infiltrates in the lung, and acute inflammation of the nose in rats and mice (NTP, 1991). A LOAEL of 0.3 mg cobalt sulfate heptahydrate/m³ was identified for this endpoint. Asthma and sensitization were reported in workers exposed to 0.007 mg Co/m³ as cobalt dust (Shirakawa et al., 1988). The LOAEL of 0.007 mg Co/m³ identified in the Shirakawa et al. (1988) occupational study is similar to the

LOAEL_{HEC} calculated in the NTP study; therefore, the use of the cobalt sulfate heptahydrate study to derive the RfC would not be expected to be overly conservative.

Strengths and Uncertainties:

The strengths of the RfC are that it was based on a well-designed chronic study from the NTP that involved extensive clinical and pathological examinations in two species and the critical effect noted in the study has been observed in numerous other human and animal studies. Respiratory effects have been reported in workers and animals exposed to a variety of cobalt compounds, including cobalt dust. Similar respiratory effects have been reported across several species exposed to various cobalt compounds at similar exposure levels (adjusting to mg Co/m³ and human equivalent concentrations).

The major uncertainty of the RfC is the lack of a NOAEL from this study or other studies. The high incidence of adverse effects at the lowest dose tested presents uncertainties as to the true NOAEL for cobalt.

References:

- Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological profile for cobalt. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.
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- California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.
- Davison, A.G., P.L. Haslam, B. Corrin, et al. 1983. Interstitial lung disease and asthma in hard-metal workers: Bronchoalveolar lavage, ultrastructural and analytical findings and results of bronchial provocation tests. *Thorax* 38:119-128 (as cited in ATSDR, 1992).
- Demedts, M., B. Gheysens, J. Nagels, et al. 1984. Cobalt lung in diamond polishers. *Am Rev Respir Dis* 130:130-135 (as cited in ATSDR, 1992).
- Hartung, M., K.H. Schaller, and E. Brand. 1982. On the question of the pathogenetic importance of cobalt for hard metal fibrosis of the lung. *Int Arch Occup Env Health* 50:53-57 (as cited in ATSDR, 1992).
- Johanssen, A., P. Camner, C. Jarstrand, et al. 1984. Lung morphology and phospholipids after experimental inhalation of soluble cadmium, copper, and cobalt. *Environ Res* 34:295-309 (as cited in ATSDR, 1992).

Johanssen, A., B. Robertson, and P. Camner. 1987. Nodular accumulation of type II cells and inflammatory lesions caused by inhalation of low cobalt concentrations. *Environ Res* 43:227-243 (as cited in ATSDR, 1992).

National Toxicology Program (NTP). 1991. NTP report on the toxicity studies of cobalt sulfate heptahydrate in F344/N rats and B6C3F₁ mice (inhalation studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication no. 91-3124. NTP TOX-5.

National Toxicology Program (NTP). 1996. NTP Technical Report on the toxicology and carcinogenesis studies of cobalt sulfate heptahydrate (CAS No.10026-24-1) in F344/N rats and B6C3F₁ mice (inhalation studies). Draft. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication no. 96-3961. NTP TR-471.

Shirakawa, T., Y. Kusaka, N. Fujimura, et al. 1988. The existence of specific antibodies to cobalt in hard metal asthma. *Clin Allergy* 18:451-460 (as cited in ATSDR, 1992).

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U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Van Cutsem, E.J., J.L. Ceuppens, L.M. Lacquet, et al. 1987. Combined asthma and alveolitis induced by cobalt in a diamond polisher. *Eur J Respir Dis* 70:54-61 (as cited in ATSDR, 1992).

Wehner, A.P., R.H. Busch, R.J. Olson, et al. 1977. Chronic inhalation of cobalt oxide and cigarette smoke by hamsters. *Am Ind Hyg Assoc J* 38:338-346 (as cited in ATSDR, 1992).

Cresols
CAS #1319-77-3

RfC:	0.0004 mg/m ³
Critical Study:	Uzhdavini, E.R., K. Astaf'yeva, A.A. Mamayeva, and G.Z. Bakhtizina. 1972. [Inhalation toxicity of o-cresol]. Trudy Ufimskogo Nauchno-Issledovatel'skogo Instituto Gigiyeny Profzabolevaniya, 7:115-9. (Russian) [as cited in CalEPA, 1997, and U.S. EPA 1985, 1986]
Critical Dose:	9 mg/m ³ [] NOAEL [X] LOAEL
Critical Effect:	Alterations in bone marrow cellularity
Species:	Rat
Route of Exposure:	Inhalation
Duration:	4 months
Uncertainty Factor:	3000: 10 for use of a LOAEL 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations 3 for extrapolation from subchronic to chronic exposure
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{LOAEL}_{\text{HEC}} \div \text{UF} = 1.3 \text{ mg/m}^3 \div 3000 = 0.0004 \text{ mg/m}^3$$

Summary of Study:

Male and female rats were exposed to 0 or 9.0 mg/m³ o-cresol via inhalation, first for 2 months (6 h/d, 5 d/wk) and then for 2 more months (4 h/d, 5 d/wk) (Uzhdavini et al., 1972, as cited in CalEPA, 1997). The following endpoints were examined: elemental conditioned defensive reflex, white blood cell levels, bone marrow elements, and liver function (as indicated indirectly by hexobarbital narcosis). Both exposed and control animals showed some loss of the defensive reflex, with the effect occurring in all exposed animals before the end of the second month and in control animals at later times. White blood cell counts were elevated in male animals, peaking at the end of the exposure period and returning to normal 1 month after cessation of exposure. Exposed animals also showed a statistically significant change in the leukoid-to-erythroid ratio in the bone marrow. Liver toxicity was suggested by an extension of hexobarbital narcosis duration in treated animals. A LOAEL of 9 mg/m³ for hematological effects was identified.

The LOAEL of 9 mg/m³ was adjusted for continuous exposure (1.3 mg/m³). A LOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 3000 was applied: 10 for use of a

LOAEL, 10 for extrapolation from humans to animals, 10 for human variability, and 3 for extrapolation from subchronic to chronic exposure.

Conversion Factors:

$$\text{LOAEL}_{\text{ADJ}} = 9 \text{ mg/m}^3 \times (5/24 \text{ h}) \times (5/7 \text{ d}) = 1.3 \text{ mg/m}^3$$

$$\text{LOAEL}_{\text{HEC}} = \text{LOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{LOAEL}_{\text{HEC}} = \text{LOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{LOAEL}_{\text{HEC}} = 1.3 \text{ mg/m}^3 \times 1 = 1.3 \text{ mg/m}^3$$

where

$\text{LOAEL}_{\text{ADJ}}$ is the adjusted LOAEL, $\text{LOAEL}_{\text{HEC}}$ is the human equivalent concentration LOAEL, RGDR is the regional gas dose ratio (animal:human), and $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficient; $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

Additional Information:

In humans, inhalation exposure is reported to cause respiratory effects, including the development of pneumonia, pulmonary edema, and hemorrhage (Clayton and Clayton, 1981). Irritation of the nose and throat, nasal constriction, and dryness was reported in 8 of 10 individuals briefly exposed to 6 mg/m³ (Uzhdavini et al., 1972, as cited in CalEPA 1997).

Signs of respiratory irritation (as indicated by increased paratid gland secretions) were observed in cats exposed to 5 to 9 mg/m³ o-cresol for 30 minutes (Uzhdavini et al., 1972, as cited in CalEPA 1997). Exposure of mice to 50 mg/m³ o-cresol for 2 h/d for 1 month did not affect mortality; however, heart muscle degeneration and degeneration of nerve cells and glial elements were reported (Uzhdavini et al., 1972, as cited in CalEPA, 1997, U.S. EPA, 1985).

Strengths and Uncertainties:

Major areas of uncertainty are the lack of quantitative human data, the scarcity of animal inhalation data, and the lack of a NOAEL for this study. Also, the data presented were incomplete, the number of animals used is not known, exposure and control conditions were not described, statistical analyses were not provided, and the purity of the compound tested could not be ascertained. However, the Uzhdavini et al. (1972) study was used by CalEPA in the derivation of their chronic inhalation reference exposure level (REL) and is the only study with quantitative data on cresols available at this time.

References:

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Clayton, G.D., and F.E. Clayton (eds). 1981. *Patty's Industrial Hygiene and Toxicology*. 3rd revised edition. Volume 2A: Toxicology. New York: John Wiley and Sons, pp. 2597-2601.

U.S. Environmental Protection Agency. 1985. Health and environmental effects profile for cresols. Cincinnati, OH: Environmental Criteria and Assessment Office, Office of Research and Development, U.S. EPA. EPA/600/x-85-358.

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U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Uzhdavini, E.R., K. Astaf'yeva, A.A. Mamayeva, and G.Z. Bakhtizina. 1972. [Inhalation toxicity of o-cresol]. Trudy Ufimskogo Nauchno-Issledovatel'skogo Instituto Gigiyeny Profzabolevaniya, 7:115-9. [as cited in CalEPA, 1997, and U.S. EPA 1985, 1986]

3,4-Dimethylphenol CAS # 95-65-8

RfC: Data are inadequate to support the derivation of an RfC at this time.

Supporting Data:

An RfD of 0.001 mg/kg/d is listed in IRIS (U.S. EPA, 1998), based on a subchronic feeding study in rats. Changes in blood pressure and body weight and histopathological changes in liver, kidney and spleen were reported (Veldre and Janes, 1979). Route-to-route extrapolation of an RfC from the RfD is not recommended because of the potential for respiratory tract effects following inhalation exposure and first-pass effects following ingestion exposure.

Although dimethylphenols have been detected in tobacco smoke, automobile exhausts, and exhausts from stationary sources, they have not been detected in ambient air (U.S. EPA, 1986). 3,4-Dimethylphenol is not likely to occur at detectable concentrations in ambient air because it is a solid at ambient temperatures and has a low vapor pressure. Consequently, inhalation exposures are unlikely to be important for the general population. Skin absorption and ingestion, which can be evaluated by the RfD, are likely to be the predominant exposure pathways.

Very little toxicity or metabolism data specific to 3,4-dimethylphenol are available. Dimethylphenols and related compounds (phenol and methylphenols [cresols]) are rapidly absorbed following ingestion, inhalation, or skin contact and are corrosive to skin, eyes, mucous membranes, and the respiratory tract. Therefore, portal-of-entry effects are likely to be important and cannot be addressed from route-to-route extrapolation. First-pass effects also may be important. These compounds are metabolized predominantly to glucuronide and sulfate conjugates and excreted in the urine (U.S. EPA, 1986). Skowronski et al. (1994) suggested that a lack of first-pass metabolism in the liver may contribute to the toxicity of phenol following skin absorption; therefore, differences in metabolism following ingestion and inhalation exposures also could affect toxicity.

References:

Skowronski, G.A., A.M. Kadry, R.M. Turkall, et al. 1994. Soil decreases the dermal penetration of phenol in male pig in vitro. *J Toxicol Environ Health* 41:467-479.

U.S. Environmental Protection Agency. 1986. Health and environmental effects profile for dimethylphenols. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/x-86/256.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA /600//8-90-066F.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). 3,4-Dimethylphenol. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

Veldre, I.A., and H.J. Janes. 1979. Toxicological studies of shale oils, some of their components and commercial products. *Environ Health Perspect* 30:141-146 (as cited in U.S. EPA, 1998).

1,4-Dioxane
CAS # 123-91-1

RfC:	0.8 mg/m ³
Critical Study:	Torkelson, T.R., B.K.J. Leong, R.J. Kociba, et al. 1974. 1,4-Dioxane. II. Results of a 2-year inhalation study in rats. <i>Toxicol Appl Pharmacol</i> 30:287-298.
Critical Dose:	400 mg/m ³ [X] NOAEL [] LOAEL
Critical Effect:	No effect on liver, kidney, or hematological endpoints
Species:	Rat
Route of Exposure:	Inhalation
Duration:	2 years
Uncertainty Factor:	100: 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 83.3 \text{ mg/m}^3 \div 100 = 0.8 \text{ mg/m}^3 \text{ (0.2 ppm)}$$

Summary of Study:

Groups of Wistar rats were exposed to 0 or 111 ppm (0 or 400 mg/m³) 1,4-dioxane 7 h/d, 5 d/wk for 2 years (Torkelson et al., 1974). Animals were observed for signs of toxicity, including behavioral changes, eye and nasal irritation, respiratory distress, and skin condition. Body weight was measured weekly. Hematological measurements were made at 16 and 23 months and included serum glutamic-pyruvic transaminase (SGPT) activity, blood urea nitrogen (BUN), alkaline phosphatase (AP) activity, and total protein determinations. At sacrifice, gross necropsy of all animals was performed, and organs were examined for tumors. Histological examination of tissues was conducted.

No significant differences in survival, body weight, general appearance, or behavior were reported. Packed cell volume (PCV), red blood cells, and hemoglobin were slightly, but significantly ($p < 0.05$), increased and white blood cells were significantly decreased in exposed males; however, the study authors note that these differences were within normal physiological levels and not considered of toxic importance. Slightly decreased BUN and AP values observed in exposed males were not considered to be biologically significant by the investigators based on the fact that an increase, not a decrease, in these parameters would indicate kidney or liver damage. Increased total protein in exposed males was also reported but not considered to be biologically significant. No significant differences in liver, kidney, or spleen weights, or gross or

microscopic alterations were observed. Tumor incidence (including hepatic and nasal) was not significantly different in any of the organs examined.

The NOAEL of 400 mg/m³ was adjusted for continuous exposure (83.3 mg/m³). A NOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrapulmonary effects). An uncertainty factor of 100 was applied: 10 for extrapolation from humans to animals and 10 for human variability.

Conversion Factors:

$$0.4 \text{ mg/L} \times 1,000 \text{ L/m}^3 = 400 \text{ mg/m}^3$$

$$\text{NOAEL}_{\text{ADJ}} = 400 \text{ mg/m}^3 \times (7/24 \text{ hr}) \times (5/7 \text{ d}) = 83.3 \text{ mg/m}^3$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 83.3 \text{ mg/m}^3 \times 1 = 83.3 \text{ mg/m}^3$$

where

NOAEL_{ADJ} is the adjusted NOAEL, NOAEL_{HEC} is the human equivalent concentration NOAEL, RGDR is the regional gas dose ratio (animal:human), and (H_{b/g})_A/(H_{b/g})_H is the ratio of blood:gas partition coefficient; (H_{b/g})_A/(H_{b/g})_H defaults to 1 where H_{b/g} values are not known.

Additional Information:

The major metabolite of 1,4-dioxane in rats is beta-hydroxyethoxyacetic acid (HEAA), which is excreted in the urine (Braun and Young, 1977). Results from a study by Young et al. (1978) show that the fate of 1,4-dioxane in rats is markedly dose-dependent due to a limited capacity to metabolize dioxane to HEAA. Exposure to 1,4-dioxane by ingestion results in saturation of metabolism above a single dose of 100 mg/kg, or as low as 10 mg/kg when administered in multiple doses. When rats were exposed to 50 ppm for 6 hours, nearly all the inhaled 1,4-dioxane was also metabolized to HEAA (99%); the plasma half-life was 1.1 hours (Young et al., 1978). The correlation of the dose-dependent fate of 1,4-dioxane with the results of toxicological studies in rats supports the conclusion that there is an apparent threshold for the toxic effects of dioxane that coincides with saturation of the metabolic pathway for its detoxification (Young et al., 1978). 1,4-Dioxane and HEAA were also found in the urine of dioxane plant workers exposed to an average concentration of 1.6 ppm (TWA) for 7.5 hours (Young et al., 1976, 1977).

In a study by Kociba et al. (1974), Sherman rats were exposed to 0, 0.01, 0.1, or 1.0% 1,4-dioxane in drinking water for up to 2 years. No hematologic changes were reported. Histopathologic examination revealed hepatocellular and renal tubular degenerative changes, accompanied by regenerative activity, in rats exposed to the two highest dose levels, but not at the low dose (Kociba et al., 1974). The lack of hematological effects observed in the ingestion study suggests that the toxicity of 1,4-dioxane may be route-specific. Studies suggest that the inhalation of 1,4-dioxane may lead to adverse effects, but good dose-response data are not available. The toxicity of 1,4-dioxane may be a function of the saturation of the mechanism of metabolism (Young et al., 1978).

Strengths and Uncertainties:

The strengths of the RfC are that it is based on a lifetime study, with a large number of toxic endpoints examined and a large sample size (n=192-288). The weaknesses of the inhalation benchmark value include the use of a free-standing NOAEL, that only one exposure level was used in the Torkelson et al. (1974) study, the limited human data, the limited inhalation data in animals, and the lack of developmental and reproductive studies.

References:

Braun, W.H., and J.D. Young. 1977. Identification of beta-hydroxyethoxyacetic acid the major urinary metabolite of 1,4-dioxane in the rat. *Toxicol Appl Pharmacol* 39:33-38.

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Kociba, R.J., S.B. McCollister, C. Park, et al. 1974. 1,4-Dioxane. I. Results of a 2-year ingestion study in rats. *Toxicol Appl Pharmacol* 30:275-286.

Torkelson, T.R., B.K.J. Leong, R.J. Kociba, et al. 1974. 1,4-Dioxane. II. Results of a 2-year inhalation study in rats. *Toxicol Appl Pharmacol* 30:287-298.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Young, J.D., W.H. Braun, and P.J. Gehring. 1978. Dose-dependent fate of 1,4-dioxane in rats. *J Toxicol Environ Health* 4:709-726.

Young, J.D., W.H. Braun, P.J. Gehring, et al. 1976. 1,4-Dioxane and beta-hydroxyethoxyacetic acid excretion in urine of humans exposed to dioxane vapors. *Toxicol Appl Pharmacol* 38:643-646.

Young, J.D., W.H. Braun, L.W. Rumpy, et al. 1977. Pharmacokinetics of 1,4-dioxane in humans. *J Toxicol Environ Health* 3:507-520.

2-Ethoxyethanol Acetate

CAS # 111-15-9

RfC:	0.3 mg/m ³
Basis for RfC:	Calculated from RfC for 2-ethoxyethanol
Critical Study:	Barbee, S.J., J.B. Terrill, D.J. DeSousa, and C.C. Conaway. 1984. Subchronic inhalation toxicology of ethylene glycol monoethyl ether in the rat and rabbit. <i>Environ Health Perspect</i> 57: 157-163.
Critical Dose:	68 mg/m ³ (2-ethoxyethanol) [X] NOAEL [] LOAEL
Critical Effect:	Decreased testis weight, seminiferous tubule degeneration, and decreased hemoglobin (2-ethoxyethanol)
Species:	Rabbit
Route of Exposure:	Inhalation
Duration:	13 weeks
Uncertainty Factor:	300: 3 for extrapolation from animals to humans 10 for intraspecies extrapolation 10 for use of a subchronic study
Modifying Factor:	1

Summary of Study:

Groups of New Zealand rabbits (10/sex/group) were exposed to 0, 25, 103 or 403 ppm (0, 92, 380, or 1,485 mg/m³) 2-ethoxyethanol 6 h/d, 5 d/wk for 13 weeks (Barbee et al., 1984). The duration-adjusted exposure concentrations were 0, 16, 68, and 265 mg/m³, respectively. Groups of Sprague-Dawley rats were similarly exposed to 2-ethoxyethanol (0, 25, 103, or 403 ppm). Physical examination and body weight measurements were conducted weekly. Hematological, chemical, and histopathological changes (including bone marrow of the sternum) were also assessed. No respiratory effects (nasal turbinates, tracheas, and lungs were examined) were observed in either species.

In both male and female rabbits, body weight was significantly depressed at 403 ppm. Significantly decreased testes weight was observed in rabbits exposed to 403 ppm. Pathological changes in the testes were characterized as minimal to slight focal degeneration of the seminiferous tubules with loss of epithelium in 3 of 10 rabbits. Spermatogenic activity in the affected males was judged by overall organ morphology and deemed normal. Additionally, both sexes exhibited significantly decreased hemoglobin, hematocrit, and erythrocyte count at 403 ppm.

Hematological, reproductive, and developmental effects were also observed in pregnant Wistar rats (Doe, 1984). Neurobehavioral and neurochemical effects were reported in the offspring of female Sprague-Dawley rats, as well as in the dams (Nelson et al., 1981, 1982a, 1982b).

Based on the observed testicular and hematopoietic effects at 403 ppm (Barbee et al., 1984), the $\text{NOAEL}_{\text{HEC}}$ and $\text{LOAEL}_{\text{HEC}}$ for 2-ethoxyethanol in the rabbit are identified as 68 and 265 mg/m^3 , respectively. The NOAEL was adjusted for intermittent exposure. A $\text{NOAEL}_{\text{HEC}}$ was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 300 was applied (10 for intraspecies extrapolation, 10 for use of a subchronic study, and 3 to account for interspecies extrapolation). An RfC of 0.2 mg/m^3 was calculated for 2-ethoxyethanol (U.S. EPA, 1998).

Calculations and Conversion Factors:

Dose levels are:

25 ppm 2-ethoxyethanol $\times (90.12/24.45) = 92 \text{ mg}/\text{m}^3$ 2-ethoxyethanol; 103 ppm = 380 mg/m^3 ;
403 ppm = 1,485 mg/m^3 .

$$\text{NOAEL}_{\text{ADJ}} = 380 \text{ mg}/\text{m}^3 \times (6/24 \text{ h}) \times (5/7 \text{ d}) = 68 \text{ mg}/\text{m}^3$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 68 \text{ mg}/\text{m}^3 \times 1 = 68 \text{ mg}/\text{m}^3$$

where

$\text{NOAEL}_{\text{ADJ}}$ is the adjusted NOAEL , $\text{NOAEL}_{\text{HEC}}$ is the human equivalent concentration NOAEL , RGDR is the regional gas dose ratio (animal:human), and $(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficients; $(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

$$\text{RfC for 2-ethoxyethanol} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 68 \text{ mg}/\text{m}^3 \div 300 = 0.2 \text{ mg}/\text{m}^3$$

$$\text{RfC (2-ethoxyethanol)} \times [\text{MW (2-ethoxyethanol acetate)} \div \text{MW (2-ethoxyethanol)}] =$$

$$\text{RfC (2-ethoxyethanol acetate)}$$

$$0.2 \text{ mg}/\text{m}^3 \times (132.16/90.12) = 0.3 \text{ mg}/\text{m}^3 = \text{RfC (2-ethoxyethanol acetate)}$$

Additional Information:

The RfC of 0.2 mg/m^3 for 2-ethoxyethanol (U.S. EPA, 1998) is based on hematological and male reproductive effects in New Zealand rabbits. It is assumed that 2-ethoxyethanol acetate would be equitoxic to 2-ethoxyethanol on a molar basis, since both compounds are metabolized to ethoxyacetic acid by the alcohol dehydrogenase pathway (DOL-OSHA, 1993). The RfC for 2-ethoxyethanol acetate was calculated from the RfC for 2-ethoxyethanol of 0.2 mg/m^3 (U.S. EPA, 1998), resulting in an RfC of 0.3 mg/m^3 for 2-ethoxyethanol acetate.

Strengths and Uncertainties:

The strength of the RfC is that it is based on an RfC in IRIS that has undergone Agency review. The major uncertainty of the RfC for 2-ethoxyethanol acetate is that it is based on an RfC for 2-ethoxyethanol and converted using the molecular weight ratio. However, it is assumed that 2-ethoxyethanol acetate would be equitoxic to 2-ethoxyethanol on a molar basis.

References:

- Barbee, S.J., J.B. Terrill, D.J. DeSousa, and C.C. Conaway. 1984. Subchronic inhalation toxicology of ethylene glycol monoethyl ether in the rat and rabbit. *Environ Health Perspect* 57: 157-163.
- Doe, J.E. 1984. Ethylene glycol monoethyl ether and ethylene glycol monoethyl ether acetate teratology studies. *Environ Health Perspect* 57:33-41.
- Department of Labor, Occupational Safety and Health Administration (DOL-OSHA). 1993. Federal Register: Occupational exposure to 2-methoxyethanol, 2-ethoxyethanol and their acetates (glycol ethers); Proposed rule. 58FR15526. March 23, 1993.
- Nelson, B.K., W.S. Brightwell, J.V. Setzer, et al. 1981. Ethoxyethanol behavioral teratology in rats. *Neurotoxicology* 2:231-249.
- Nelson, B.K., W.S. Brightwell, and J.V. Setzer. 1982a. Prenatal interactions between ethanol and the industrial solvent 2-ethoxyethanol in rats: Maternal and behavioral teratogenic effects. *Neurobehav Toxicol Teratol* 4(3):387-394.
- Nelson, B.K., W.S. Brightwell, J.V. Setzer, and T.L. O'Donohue. 1982b. Prenatal interactions between ethanol and the industrial solvent 2-ethoxyethanol in rats: Neurochemical effects in the offspring. *Neurobehav Toxicol Teratol* 4(3):395-401.
- Research Triangle Institute (RTI). 1996. Assessment of Risks from the Management of Used Solvents. U.S. Environmental Protection Agency, Office of Solid Waste, Washington, DC. July 1996. [In support of 61FR42317]
- U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.
- U.S. Environmental Protection Agency. 1996. Federal Register: Hazardous Waste Management System; Identification and Listing of Hazardous Waste; Solvents; CERCLA Hazardous Substance Designation and Reportable Quantities; Proposed Rule. 61FR42317-354. August 14, 1996.
- U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS) file for 2-ethoxyethanol.

Ethylene glycol CAS # 107-21-1

RfC:	0.6 mg/m ³
Critical Study:	Wills, J.H., F. Coulston, E.S. Harris, et al. 1974. Inhalation of aerosolized ethylene glycol by man. <i>Clin Toxicol</i> 7:463-476.
Critical Dose:	67 mg/m ³ [X] NOAEL [] LOAEL
Critical Effect:	Throat and upper respiratory tract irritation
Species:	Humans
Route of Exposure:	Inhalation
Duration:	30 days
Uncertainty Factor:	100: 10 for protection of sensitive human subpopulations 10 for use of a subchronic study
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{NOAEL}_{\text{ADJ}} \div \text{UF} = 55.8 \text{ mg/m}^3 \div 100 = 0.6 \text{ mg/m}^3$$

Summary of Study:

Twenty volunteer male prisoners were exposed to ethylene glycol in mean daily concentrations between 3 and 67 mg/m³ for 30 days, 20 h/d, without effect (Wills et al., 1974). Irritation was noted after 15 minutes at an exposure concentration of 188 mg/m³ and was judged intolerable at 244 mg/m³. No effects were observed in clinical serum enzyme levels for liver and kidney toxicity, hematotoxicity, or psychological responses. The irritation resolved soon after exposure with no effects noted after a 6-week followup period.

A NOAEL of 67 mg/m³ was selected and adjusted for continuous exposure (55.8 mg/m³). An uncertainty factor of 100 was applied: 10 for use of a subchronic study (30 day-duration) and 10 for protection of sensitive human subpopulations.

Conversion Factors:

$$\text{NOAEL}_{\text{ADJ}} = 67 \text{ mg/m}^3 \times 20/24 \text{ h} = 55.8 \text{ mg/m}^3$$

Additional Information:

Animal studies are inconclusive regarding the respiratory effects of ethylene glycol. Suber et al. (1989, as cited in ATSDR, 1997) report thickened respiratory epithelium with enlarged goblet cells in rats that inhaled ethylene glycol over 90 days. Another study in rhesus monkeys and rats showed no respiratory effects from continuous exposure to propylene glycol for 13 to 18 months (Robertson et al., 1947, as cited in ATSDR, 1997). Developmental effects have been seen in

animal studies. Tyl et al. (1995a, 1995b, as cited in CalEPA, 1997) reported reduced ossification in humerus, zygomatic arch, and the metatarsals in fetuses of rats and mice exposed to ethylene glycol on days 6 through 15 of gestation.

Strengths and Uncertainties:

The major strength of the RfC is that it was based on human data with controlled inhalation exposures and the observation of a NOAEL. The major uncertainty to the RfC is the lack of chronic inhalation studies in humans and confirming studies in animals.

References:

Agency for Toxic Substances and Disease Registry. 1997. Toxicological profile for ethylene glycol and propylene glycol. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Robertson, O.H., C.G. Loosli, and T.T. Puck. 1947. Test for chronic toxicity of propylene glycol and triethylene glycol on monkeys and rats by vapor inhalation and oral administration. *J Pharmacol Exper Therap* 91:52-76 (as cited in ATSDR 1997).

Suber, R.L., R.D. Deskin, I. Nikiforov, et al. 1989. Subchronic nose-only inhalation study of propylene glycol in Sprague-Dawley rats. *Food Chem Toxicol* 27(9):573-584 (as cited in ATSDR, 1997).

Tyl, R.W., B. Ballantyne, L.C. Fisher, et al. 1995a. Evaluation of the developmental toxicity of ethylene glycol aerosol in CD-1 mice by nose-only exposure. *Fundam Appl Toxicol* 27:49-62 (as cited in CalEPA, 1997).

Tyl, R.W., B. Ballantyne, L.C. Fisher, et al. 1995b. Evaluation of the developmental toxicity of ethylene glycol aerosol in CD rat and CD-1 mouse by whole-body exposure. *Fundam Appl Toxicol* 24:57-75 (as cited in CalEPA, 1997).

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Wills, J.H., F. Coulston, E.S. Harris, et al. 1974. Inhalation of aerosolized ethylene glycol by man. *Clin Toxicol* 7:463-476.

Methanol
CAS # 67-56-1

RfC:	13 mg/m ³
Critical Study:	Rogers, J.M., M.L. Mole, N. Chernoff, et al. 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose-response modeling for estimation of benchmark doses. <i>Teratology</i> 47(3):175-188.
Critical Dose:	1,310 mg/m ³ [X] NOAEL [] LOAEL
Critical Effects:	Developmental malformations (increased cervical ribs, exencephaly, and cleft palate)
Species:	Mouse
Route of Exposure:	Inhalation
Duration:	Gd 6-15
Uncertainty Factor:	100: 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 1310 \text{ mg/m}^3 \div 100 = 13 \text{ mg/m}^3 \text{ (10 ppm)}$$

Summary of Study:

Groups of pregnant CD-1 mice were exposed to 1,000, 2,000, 5,000, 7,500, 10,000, or 15,000 ppm methanol (1,310, 2,620, 6,552, 9,828, 13,104, or 19,656 mg/m³) for 7 h/d on days 6 through 15 of gestation (Rogers et al., 1993). Three groups of controls were used. Sham-exposed controls were exposed to filtered air. Additional control groups remained in their cages and received food and water ad libitum or were food-deprived for 7 h/d (to match the food deprivation experienced by the exposed mice). Dams were observed twice daily and weighed on alternate days during the exposure period. Blood methanol concentrations were determined in three mice per exposure level on gestation days 6, 10, and 15. On day 17, the remaining mice were weighed and sacrificed and the gravid uteri removed. Implantation sites, live and dead fetuses, and resorptions were counted, and fetuses were examined externally and weighed as a litter. Half of each litter were examined for skeletal morphology and the other half of each litter were examined for internal soft tissue anomalies.

One dam died in each of the three highest exposure groups, but no dose-response relationship was evident for maternal death. The sham-exposed and food-deprived controls, as well as all methanol-exposed dams, gained less weight than did unexposed dams fed ad libitum, but methanol did not exacerbate this effect. Significant increases in the incidence of exencephaly

and cleft palate were observed at 6,552 mg/m³ and above, increased embryo/fetal death at 9,828 mg/m³ and above (including an increasing incidence of full-litter resorptions), and reduced fetal weight at 13,104 mg/m³ and above. A dose-related increase in cervical ribs (small ossification sites lateral to the seventh cervical vertebra) was significant at 2,620 mg/m³ and above. Therefore, a NOAEL of 1,310 mg/m³ for developmental toxicity in mice was identified in this study.

Because this is a developmental study, the NOAEL of 1,310 mg/m³ was not adjusted for continuous exposure. A NOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 100 was applied: 10 for extrapolation from humans to animals and 10 for human variability.

Conversion Factors:

Dose levels are:

(1,000 ppm x 32.04)/ 24.45 = 1,310 mg/m³; 2,000 ppm = 2,620 mg/m³; 5,000 ppm = 6,552 mg/m³; 7,500 ppm = 9,828 mg/m³; 10,000 ppm = 13,104 mg/m³; 15,000 ppm = 19,656 mg/m³

NOAEL_{HEC} = NOAEL x RGDR

NOAEL_{HEC} = NOAEL x (H_{b/g})_A/(H_{b/g})_H

NOAEL_{HEC} = 1310 mg/m³ x 1 = 1310 mg/m³

where

NOAEL_{HEC} is the human equivalent concentration NOAEL, RGDR is the regional gas dose ratio (animal:human) and (H_{b/g})_A/(H_{b/g})_H is the ratio of blood:gas partition coefficient; (H_{b/g})_A/(H_{b/g})_H defaults to 1 where H_{b/g} values are not known.

Additional Information:

Developmental effects were also reported in a study by Nelson et al. (1985). Pregnant Sprague-Dawley rats were exposed to methanol at concentrations of 0, 5,000, 10,000, and 20,000 ppm (0, 6,552, 13,104, and 26,208 mg/m³) 7 h/d on days 1 through 19 of gestation (high dose rats were exposed on Gd 7-15 only). Dams were sacrificed on Day 20. Half of the fetuses were examined for visceral defects, and the other half were examined for skeletal defects. No effect on the numbers of corpora lutea or implantations or the percentage of dead or resorbed fetuses was observed. At the two highest concentrations, a dose-related decrease in fetal weights was reported. The highest concentration of methanol produced slight maternal toxicity and a high incidence of congenital malformations (p<0.001), predominantly extra or rudimentary cervical ribs and urinary or cardiovascular defects. Similar malformations were seen in the 10,000 ppm group, but the incidence was not significantly different from controls. No adverse effects were noted in the 6552 mg/m³ group (Nelson et al., 1985).

Strengths and Uncertainties:

The major strengths of the Rogers et al. (1993) study are the identification of a NOAEL and the demonstration of a dose-response relationship. The study was well performed, large numbers of animals were used (n=20-44 per group), and effects at six exposure concentrations were examined. The results are also supported by an additional developmental study (Nelson et al., 1985).

The major uncertainties of the RfC are the lack of human data for chronic inhalation exposure and the lack of comprehensive, long-term multiple dose studies.

References:

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Nelson, B.K., W.S. Brightwell, D.R. MacKenzie, et al. 1985. Teratological assessment of methanol and ethanol at high inhalation levels in rats. *Fundam Appl Toxicol* 5:727-736.

Rogers, J.M., M.L. Mole, N. Chernoff, et al. 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose-response modeling for estimation of benchmark doses. *Teratology* 47(3):175-188.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

2-Methoxyethanol Acetate CAS # 110-49-6

RfC:	0.03 mg/m ³
Basis for RfC:	Calculated from RfC for 2-methoxyethanol
Critical Study:	Miller, R.R., J.A. Ayres, J.T. Young, and M.J. McKenna. 1983. Ethylene glycol monomethyl ether. I. Subchronic vapor inhalation study with rats and rabbits. <i>Fund Appl Toxicol</i> 3(1):49-54.
Critical Dose:	17 mg/m ³ (2-methoxyethanol) [X] NOAEL [] LOAEL
Critical Effect:	Testicular effects (2-methoxyethanol)
Species:	Rabbit and rat
Route of Exposure:	Inhalation
Duration:	13 weeks
Uncertainty Factor:	1,000: 10 for protection of sensitive humans 10 for use of a subchronic study 10 to account for extrapolation from animals to humans and for database deficiencies
Modifying Factor:	1

Summary of Study:

Groups of New Zealand white rabbits (5/sex/dose) and Sprague-Dawley rats (10/sex/dose) were exposed to 0, 30, 100, or 300 ppm 2-methoxyethanol (0, 93, 311, or 934 mg/m³) 6 h/d, 5 d/wk for 13 weeks (Miller et al., 1983). The duration-adjusted exposure concentrations were 0, 17, 56, and 167 mg/m³, respectively. Toxicity was assessed by clinical observations, body and organ weights, hematology, clinical chemistry, urinalysis (rats only), and gross and histopathological examination of major organs including respiratory tract, heart, liver, kidney, bone marrow, testes, uterus, and ovaries.

Effects reported in both sexes of rabbits exposed to 300 ppm included reduced body weight, hematological changes (pancytopenia), lymphoid tissue atrophy (thymus), and a significant decrease in testicular weight with small flaccid testes in the males. A slight to moderate decrease in testes size was also reported in 2/5 and 4/5 male rabbits exposed to 30 and 100 ppm, respectively. Microscopic lesions included degenerative changes in the germinal epithelium of the testes in 3/3, 3/5, and 1/5 male rabbits exposed to 300, 100, and 30 ppm, respectively. The decrease in testes weight was considered to be concentration-dependent in the male rabbits. No effects on the reproductive organs of the female rabbits were found. Thymus weights were significantly decreased in both sexes exposed to 300 ppm. Based upon the testicular effects in

rabbits, a NOAEL of 30 ppm ($\text{NOAEL}_{\text{ADJ}}=17 \text{ mg/m}^3$) and a LOAEL of 100 ppm ($\text{LOAEL}_{\text{ADJ}}=56 \text{ mg/m}^3$) were identified.

The authors reported a significant decrease in body weight in the male rats exposed to 300 ppm and in the females exposed to 100 ppm or more. Effects reported in both sexes of rats exposed to 300 ppm included hematological changes (pancytopenia), lymphoid tissue atrophy, a decrease in liver weight, and changes in clinical chemistry parameters. In the 300 ppm group (male and female rats), the mean values for total serum protein, albumin, and globulins were lower than the control values. A significant decrease in testicular weight and small flaccid testes were also reported in the male rats exposed to 300 ppm. Microscopic examination showed moderate to severe degeneration of the germinal epithelium in the seminiferous tubules at the highest exposure. There were no microscopic changes in the testes in the animals exposed to 100 or 30 ppm. The authors found no effects in the reproductive organs of the female rats.

Based on the observed testicular effects at 100 ppm (Miller et al., 1983), a NOAEL of 30 ppm for 2-methoxyethanol in the rabbit was identified ($\text{NOAEL}_{\text{HEC}} = 17 \text{ mg/m}^3$). The NOAEL was adjusted for intermittent exposure. A $\text{NOAEL}_{\text{HEC}}$ was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 1000 was applied (10 for protection of sensitive humans, 10 for use of a subchronic study, and 10 to account for extrapolation from animals to humans and for database deficiencies). An RfC of 0.02 mg/m^3 was calculated for 2-methoxyethanol (U.S. EPA, 1998).

Calculations and Conversion Factors:

Dose levels are:

30 ppm 2-methoxyethanol $\times (76.09/24.45) = 93 \text{ mg/m}^3$ 2-methoxyethanol; 100 ppm = 311 mg/m^3 ; 300 ppm = 934 mg/m^3 .

$$\text{NOAEL}_{\text{ADJ}} = 93 \text{ mg/m}^3 \times (6/24 \text{ hours}) \times (5/7 \text{ days}) = 17 \text{ mg/m}^3$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 17 \text{ mg/m}^3 \times 1 = 17 \text{ mg/m}^3$$

where

$\text{NOAEL}_{\text{ADJ}}$ is the adjusted NOAEL, $\text{NOAEL}_{\text{HEC}}$ is the human equivalent concentration NOAEL, RGDR is the regional gas dose ratio (animal:human), and $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficients; $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

$$\text{RfC for 2-methoxyethanol} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 17 \text{ mg/m}^3 \div 1,000 = 0.02 \text{ mg/m}^3$$

$$\text{RfC (2-methoxyethanol)} \times [\text{MW (2-methoxyethanol acetate)} \div \text{MW (2-methoxyethanol)}] = \text{RfC (2-methoxyethanol acetate)}$$

$$0.02 \text{ mg/m}^3 \times (118.13/76.09) = 0.03 \text{ mg/m}^3 = \text{RfC (2-methoxyethanol acetate)}$$

Additional Information:

The RfC of 0.02 mg/m^3 for 2-methoxyethanol (U.S. EPA, 1998) is based on male reproductive effects in New Zealand rabbits. It is assumed that 2-methoxyethanol acetate would be equitoxic to 2-methoxyethanol on a molar basis, since both compounds are metabolized to methoxyacetic

acid by the alcohol dehydrogenase pathway (DOL-OSHA, 1993). The RfC for 2-methoxyethanol acetate was calculated from the RfC for 2-methoxyethanol of 0.02 mg/m³ (U.S. EPA, 1998), resulting in an RfC of 0.03 mg/m³ for 2-methoxyethanol acetate.

Strengths and Uncertainties:

The strength of the RfC is that it is based on an RfC in IRIS that has undergone Agency review. The major uncertainty of the RfC for 2-methoxyethanol acetate is that it is based on an RfC for 2-methoxyethanol and converted using the molecular weight ratio. However, it is assumed that 2-methoxyethanol acetate would be equitoxic to 2-methoxyethanol on a molar basis.

References:

- Department of Labor, Occupational Safety and Health Administration (DOL-OSHA). 1993. Federal Register: Occupational exposure to 2-methoxyethanol, 2-ethoxyethanol and their acetates (glycol ethers); Proposed rule. 58FR15526. March 23, 1993.
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- Research Triangle Institute (RTI). 1996. Assessment of Risks from the Management of Used Solvents. U.S. Environmental Protection Agency, Office of Solid Waste, Washington, DC. July 1996. [In support of 61FR42317]
- U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.
- U.S. Environmental Protection Agency. 1996. Federal Register: Hazardous Waste Management System; Identification and Listing of Hazardous Waste; Solvents; CERCLA Hazardous Substance Designation and Reportable Quantities; Proposed Rule. 61FR42317-354. August 14, 1996.
- U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS) file for 2-methoxyethanol.

Vanadium
CAS # 7440-62-2

RfC:	0.00007 mg/m ³
Critical Study:	Zenz, C., and B.A. Berg. 1967. Human responses to controlled vanadium pentoxide exposure. <i>Arch Environ Health</i> 14:709-712.
Critical Dose:	0.06 mg vanadium/m ³ [] NOAEL [X] LOAEL
Critical Effect:	Upper respiratory symptoms (coughing)
Species:	Human
Route of Exposure:	Inhalation
Duration:	8 hours
Uncertainty Factor:	300: 3 for use of a minimal LOAEL in a human study 10 for use of an acute study 10 for protection of sensitive human subpopulations
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{LOAEL}_{\text{ADJ}} \div \text{UF} = 0.02 \text{ mg/m}^3 \div 300 = 0.00007 \text{ mg/m}^3$$

Summary of Study:

Nine human volunteers were exposed to vanadium pentoxide at levels of 0.1, 0.25, and 1.0 mg/m³ (0.06, 0.1, and 0.6 mg vanadium/m³) for 8 hours (Zenz and Berg, 1967). Subjects exposed to 0.06 mg vanadium/m³ experienced no symptoms during or immediately after exposure. Within 24 hours, considerable mucus formed, which resulted in slight coughing that increased after 48 hours, subsided within 72 hours, and disappeared completely after 4 days. All individuals exposed to 0.1 mg vanadium/m³ developed a loose cough the following morning, while those individuals exposed to 0.6 mg vanadium/m³ developed frequent coughing that persisted for 8 days. There were no other signs of irritation, fever, or increased pulse rate.

A LOAEL of 0.06 mg/m³ was selected, since the subjects experienced slight coughing at this dose. This LOAEL was adjusted for exposure duration (0.02 mg/m³). An uncertainty factor of 300 was applied: 3 for use of a minimal LOAEL in a human study, 10 for use of an acute study (8 hours' duration), and 10 for protection of sensitive human subpopulations, since this study involved only nine volunteers who cannot be assumed to be the most sensitive individuals to this type of effect.

Conversion Factors:

Dose levels are:

% vanadium in vanadium pentoxide = 56.02% = 60% = 0.6. $0.1 \text{ mg V}_2\text{O}_5/\text{m}^3 \times 0.6 = 0.06 \text{ mg vanadium}/\text{m}^3$; $0.25 \text{ mg V}_2\text{O}_5/\text{m}^3 = 0.15 \text{ mg vanadium}/\text{m}^3$; $1 \text{ mg V}_2\text{O}_5/\text{m}^3 = 0.6 \text{ mg vanadium}/\text{m}^3$.

$\text{LOAEL}_{\text{ADJ}} = 0.06 \text{ mg}/\text{m}^3 \times 8/24 \text{ h} = 0.02 \text{ mg}/\text{m}^3$

Additional Information:

Other human studies support the critical effect (upper respiratory symptoms, coughing) reported in the Zenz and Berg (1967) study. Workers exposed to vanadium pentoxide for durations ranging from 1 day to 6 or more years showed mild respiratory distress, such as cough, wheezing, chest pain, runny nose, or sore throat (Levy et al., 1984; Musk and Tees, 1982; Orris et al., 1983; Sjoeborg, 1956; Thomas and Stiebris, 1956, as cited in ATSDR, 1992); quantitative data were not available for these occupational studies. Animal studies also support the human findings; monkeys, rats, and rabbits experienced respiratory effects from inhalation exposure to vanadium pentoxide (Knecht et al., 1985; Lee and Gillies, 1986; Sjoeborg, 1950; as cited in ATSDR, 1992).

Strengths and Uncertainties:

The major strength of the RfC is that it was based on human data with a critical effect that has been reported in numerous other studies. ATSDR's acute inhalation MRL ($0.0002 \text{ mg}/\text{m}^3$) is also based on the Zenz and Berg (1967) study. The RfC developed for the Air Characteristic Study ($0.00007 \text{ mg}/\text{m}^3$) is also supported by the Sjoeborg (1950) study. Dyspnea was reported in rabbits exposed to $0.8 \text{ mg V}/\text{m}^3$ (as vanadium pentoxide) 1 h/d for 8 months. Using the Sjoeborg (1950) study, adjusting for intermittent exposure ($\text{LOAEL}_{\text{ADJ}} = 0.03 \text{ mg V}/\text{m}^3$), and applying an uncertainty factor of 3,000 for animal-to-human extrapolation, use of a LOAEL, human variability, and extrapolation of subchronic to chronic duration would result in an RfC ($0.00001 \text{ mg}/\text{m}^3$) similar to the RfC based on the Zenz and Berg (1967) study. Although the Zenz and Berg (1967) study was of brief duration (8 hours), the lowest LOAEL among available data was identified in this study and quantitative human data are generally preferred over animal data (when available). Therefore, the RfC of $0.00007 \text{ mg}/\text{m}^3$ based on the Zenz and Berg (1967) data is used in the Air Characteristic Study.

The major uncertainty to the RfC is the short duration (8 hours) and the limited number of individuals (9) in the Zenz and Berg (1967) study. In addition, a NOAEL was not identified in the study.

References:

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological profile for vanadium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Knecht, E.A., W.J. Moorman, J.C. Clark, et al. 1985. Pulmonary effects of acute vanadium pentoxide inhalation in monkeys. *Am Rev Respir Dis* 132:1181-1185 (as cited in ATSDR, 1992).

Lee, K.P., and P.J. Gillies. 1986. Pulmonary response and intrapulmonary lipids in rats exposed to bismuth orthovanadate dust by inhalation. *Environ Res* 40:115-135 (as cited in ATSDR, 1992).

Levy, B.S., L. Hoffman, and S. Gottsegen. 1984. Boilmakers' bronchitis. *J Occup Med* 26: 567-570 (as cited in ATSDR, 1992).

Musk, A.W., and J.G. Tees. 1982. Asthma caused by occupational exposure to vanadium compounds. *Med J Aust* 1:183-184 (as cited in ATSDR, 1992).

Orris, P., J. Cone, and S. McQuilkin. 1983. Health hazard evaluation report HETA 80-096-1359, Bloomington, IL. Washington, DC: U.S. Department of Health and Human Services, National Institute of Occupational Safety and Health. NTIS-PB85-163574 (as cited in ATSDR, 1992).

Sjoeberg, S.G. 1950. Vanadium pentoxide dust - a clinical and experimental investigation on its effects after inhalation. *Acta Med Scand Supp.* 238:1-188 (as cited in ATSDR, 1992).

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Thomas, D.L., and K. Stiebris. 1956. Vanadium poisoning in industry. *The Medical Journal of Australia* 1:607-609 (as cited in ATSDR, 1992).

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Zenz, C., and B.A. Berg. 1967. Human responses to controlled vanadium pentoxide exposure. *Arch Environ Health* 14:709-712.

CARCINOGENS

**DERIVATION OF INHALATION UNIT RISK FACTORS
AND CANCER SLOPE FACTORS**

Bromodichloromethane

CAS #75-27-4

Inhalation Unit Risk Factor:	1.8E-05 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	6.2E-02 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Tubular cell adenoma and tubular cell adenocarcinoma
Species:	Mice
Route of Exposure:	Gavage, corn oil
Duration:	2 years

Basis for Toxicity Values:

EPA has not developed an inhalation reference concentration (RfC) for bromodichloromethane. An oral reference dose (RfD) value of 0.02 mg/kg/d, based on a chronic gavage study in mice for renal cytomegaly is available on IRIS for bromodichloromethane (U.S. EPA, 1998).

Based on inadequate human data and sufficient evidence of carcinogenicity in animals, EPA considers bromodichloromethane a probable human carcinogen (Class B2) by the oral route and has calculated an oral cancer slope factor (CSF) of 0.062 ($\text{mg}/\text{kg}/\text{d}$)⁻¹ for the substance. In a National Toxicology Program (NTP) study, 2-year gavage administration of bromodichloromethane to both sexes of F344/N rats and B6C3F1 resulted in compound-related statistically significant increases in tumors of the kidney in male mice, the liver in female mice, and the kidney and large intestine in male and female rats (NTP, 1987, as cited in U.S. EPA, 1998).

In male mice, the incidences of tubular cell adenomas and the combined incidence of tubular cell adenomas and adenocarcinomas of the kidneys were significantly increased in the high-dose animals. In female mice, there were significant increases of hepatocellular adenomas and hepatocellular carcinomas. The combined incidence of hepatocellular adenomas or carcinomas in vehicle control, low-dose, and high-dose groups were 3/50, 18/48, and 29/50, respectively.

In male and female rats, the incidences of tubular cell adenomas, adenocarcinomas, and the combined incidence of adenomas and adenocarcinomas of the kidneys were statistically significantly increased only in the high-dose groups. The combined incidence of tubular cell adenomas or adenocarcinomas in vehicle control, low-dose, and high-dose groups were 0/50, 1/49, and 13/50 for males and 0/50, 1/50, and 15/50 for females, respectively.

Tumors of the large intestines, namely adenocarcinomas and adenomatous polyps, were significantly increased in male rats in a dose-dependent manner. These large intestinal tumors, however, were observed only in high-dose female rats (adenocarcinomas 0/46, 0/50, 6/47; adenomatous polyps 0/46, 0/50, 7/47 in the vehicle control, low-dose and high-dose groups, respectively). The combined incidence of large intestine adenocarcinomas and/or adenomatous polyps in vehicle control, low-dose, and high-dose groups were 0/50, 13/49, and 45/50 for males and 0/46, 0/50, and 12/47 for females. The combined tumor incidences in the large intestine and kidney in male and female rats at control, low dose, and high dose were 0/50, 13/49, 46/50 and

0/46, 1/50, 24/48, respectively. Under the conditions of this bioassay, the NTP concluded there was clear evidence of carcinogenicity of bromodichloromethane in male and female F344/N rats and B6C3F1 mice (U.S. EPA, 1998).

The mechanism for the carcinogenicity of bromodichloromethane appears to be genotoxic carcinogenesis, independent of liver activation and, hence, route-independent. In one genotoxicity assay, bromodichloromethane was mutagenic in *Salmonella typhimurium* strain TA100 in the absence of liver homogenate in a vapor phase test performed in a desiccator. Positive results for mutagenicity were reported for bromodichloromethane in other *S. typhimurium* assays in which the TA100 and TA1537 strains were used without rat liver homogenate activation. Bromodichloromethane also induced weak mutagenic effects in *Saccharomyces cerevisiae* strains D7 and XV185-14C in the absence of liver homogenate (U.S. EPA, 1998; HSDB, 1998).

Thus, inhalation exposure to bromodichloromethane is likely to lead to carcinogenic consequences not dissimilar from that from oral exposure. Therefore, in accordance with current EPA guidelines, it is considered appropriate to calculate an inhalation unit risk factor for bromodichloromethane from the oral CSF listed for that substance in IRIS (U.S. EPA, 1994, 1996).

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{day} = 0.062 (\text{mg}/\text{kg}/\text{d})^{-1} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 1.8\text{E}-05(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS (U.S. EPA, 1998) and HEAST (U.S. EPA, 1997) that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table D-1, Figure D-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

References:

Hazardous Substances Databank (HSDB): Bromodichloromethane. 1998. Online database. National Library of Medicine, Bethesda, MD.

National Toxicology Program (NTP). 1987. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Bromodichloromethane (CAS no. 75-27-4) in F344/N Rats and B6C3F1 Mice (gavage studies). NTP Tech. Report Series No.321. U.S. Dept. Health and Human Services, Public Health Service, National Institute of Health (as cited in U.S. EPA, 1998).

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Chlorodibromomethane

CAS #124-48-1

Inhalation Unit Risk Factor:	2.4E-05 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	8.4E-02 (mg/kg/d) ⁻¹
Critical Effects:	Hepatocellular adenoma or carcinoma
Species:	Mice
Route of Exposure:	Gavage
Duration:	2 years

Basis for Toxicity Values:

EPA has not developed an inhalation reference concentration (RfC) for chlorodibromomethane. An oral reference dose (RfD) value of 0.02 mg/kg/d, based on a subchronic gavage study in rats for hepatic lesions is available on IRIS for chlorodibromomethane (U.S. EPA, 1998)

Based on inadequate human data and limited evidence of carcinogenicity in animals, EPA considers chlorodibromomethane a possible human carcinogen (Class C) by the oral route and has calculated an oral cancer slope factor (CSF) of 0.084 (mg/kg/d)⁻¹ for the substance. In the study, 2-year gavage administration of chlorodibromomethane to both sexes of B6C3F1 mice caused increased incidence of adenomas and carcinomas in female mice and a significantly increased incidence of hepatocellular carcinomas in high-dose male mice (NTP, 1985, as cited in U.S. EPA, 1998). Drinking water administration of chlorodibromomethane to both sexes of CBAXC57B1/6 mice also resulted in significantly increased incidence of tumors (U.S. EPA, 1998).

The mechanism for the carcinogenicity of chlorodibromomethane appears to be genotoxic carcinogenesis, independent of liver activation and, hence, route-independent. In one genotoxicity assay, chlorodibromomethane produced reverse mutations in *Salmonella typhimurium* strain TA100 in a vapor-phase test performed in a desiccator. Positive results for gene conversion in *Saccharomyces cerevisiae* strain D4 without, but not with, hepatic homogenates, and negative results for mutation in strain XV185-14C both with and without hepatic homogenates have been reported for chlorodibromomethane. In others tests, chlorodibromomethane produced sister chromatid exchange in cultured human lymphocytes and in bone marrow cells of mice treated orally (U.S. EPA, 1998; HSDB, 1998).

Thus, inhalation exposure to chlorodibromomethane is likely to lead to carcinogenic consequences not dissimilar from that from oral exposure. Therefore, in accordance with current EPA guidelines, it is considered appropriate to calculate an inhalation unit risk factor for chlorodibromomethane from the oral CSF listed for that substance in IRIS (U.S. EPA, 1994, 1996).

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = \\ 0.084 (\text{mg}/\text{kg}/\text{d})^{-1} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 2.4\text{E-}05(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS (EPA, 1998) and HEAST (U.S. EPA, 1997) that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table D-1, Figure D-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

References:

Hazardous Substances Databank (HSDB): Chlorodibromomethane. 1998. Online database. National Library of Medicine, Bethesda, MD.

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7,12-Dimethylbenz[*a*]anthracene
CAS # 57-97-6

Unit Risk Factor:	2.4E-02 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	8.4E+01 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Malignant angioendothelioma of the mesenteric intestine
Species:	Mouse (albino)
Route of Exposure:	Diet
Duration:	60 weeks

Basis for Toxicity Values:

There are no human data available that may be used to address the carcinogenicity of 7,12-dimethylbenz[*a*]anthracene (DMBA). However, DMBA belongs to a class of chemicals known as polycyclic aromatic hydrocarbons (PAHs), which are components of coal tar and incomplete combustion. Many of the PAHs have been demonstrated to be carcinogenic to rats and mice following oral exposure, skin painting, intrapulmonary injection, inhalation, subcutaneous injection, and intraperitoneal injection; however, most of these studies are not considered suitable for quantitative risk assessment. Nevertheless, the data do indicate that the carcinogenic potencies vary and that DMBA is considered one of the most potent PAHs (Pitot and Dragan, 1996).

DMBA is not listed in EPA's IRIS (U.S. EPA, 1998) or HEAST (U.S. EPA, 1997) databases and was not included in EPA's (1993) *Provisional Guidance for Quantitative Risk Assessment of PAHs*. However, the California Environmental Protection Agency (CalEPA) has developed a unit risk factor (URF) and cancer slope factor (CSF) for DMBA in support of the Air Toxics Hot Spots Program (CalEPA, 1994a, 1994b, 1997). The CalEPA URF and inhalation CSF are listed above and are recommended as interim values.

The CalEPA developed an "expedited" approach for deriving cancer potency values in order to implement Proposition 65 (Hoover et al., 1995). The expedited approach was used for DMBA. Under the expedited approach, instead of conducting a comprehensive literature review, cancer dose response data are taken from the Carcinogenic Potency Database (CPDB) (Gold and Zeiger, 1997). The linearized multistage model is automatically used to derive cancer potency estimates for low-dose exposures, and pharmacokinetic adjustments are not made.

Only one study was listed in the CPDB (Chouroulinkov et al., 1967). Female albino mice were fed DMBA for 60 weeks at a dose rate of 0.39 mg/kg/d. No tumors were reported in 40 control mice. Malignant angioendotheliomas of the intestine were reported in 49 of 75 test animals. Twenty test animals also had nonmalignant forestomach papillomas.

Additional Information:

The CPDB summarizes the results of 5,152 cancer tests on 1,298 chemicals. Carcinogenic potency estimates are presented as TD₅₀ values. TD₅₀ is defined as *that dose-rate in mg/kg body wt/d which, if administered chronically for the standard lifespan of the species, will halve the probability of remaining tumorless throughout that period* (Gold and Zeiger, 1997). The TD₅₀ is analogous to the dose that is lethal to 50 percent of test animals (LD₅₀). A low TD₅₀ indicates high potency, just as a low LD₅₀ indicates high acute toxicity.

Some studies have reported high correlations between various measures of cancer potency and the maximum tolerated dose or maximum dose tested in the carcinogenicity studies (Gaylor, 1989; Krewski et al., 1993). The correlation of TD₅₀ values as reported in the CPDB and inhalation CSFs derived from IRIS or HEAST was evaluated as a possible means to estimate the CSF from the TD₅₀. Forty-five chemicals were identified that had both a TD₅₀ and an inhalation CSF (see Table D-2, Figure D-2). The correlation coefficient for the regression is 0.95. The TD₅₀ reported for DMBA is 0.084 mg/kg/d (Gold and Zeiger, 1997). Based on a linear regression of log TD₅₀ as the independent variable and log (1/CSF) as the dependent variable, an inhalation CSF of 55 (mg/kg/d)⁻¹ and a URF of 1.6E-02 (μg/m³)⁻¹ are predicted. These values are in close agreement with the CalEPA values of 84 (mg/kg/d)⁻¹ and 2.4E-02(μg/m³)⁻¹, respectively.

References:

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2,4-Dinitrotoluene CAS #121-14-2

Unit Risk Factor:	1.9E-04 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	6.8E-01 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Hepatocellular carcinoma, liver neoplastic nodules, benign and malignant mammary gland tumors.
Species:	Female Sprague-Dawley rats
Route of exposure:	Diet
Duration:	2 years

Basis for Toxicity Values:

There are no human data available that may be used to address the carcinogenicity of 2,4-dinitrotoluene. 2,4-Dinitrotoluene is not listed in EPA's IRIS (U.S. EPA, 1998) or HEAST (U.S. EPA, 1997) databases. However, an oral CSF of 0.68 ($\text{mg}/\text{kg}/\text{d}$)⁻¹ is available in IRIS for a mixture of 2,4- and 2,6-dinitrotoluene. The mixture was 98% 2,4-dinitrotoluene and 2% 2,6-dinitrotoluene. The oral CSF for the mixture is proposed as an interim value for the inhalation CSF for 2,4-dinitrotoluene.

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS and HEAST that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table D-1, Figure D-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

Dose-Response Data:

The oral CSF listed in HEAST was based on a study by Ellis et al. (1979). Sprague-Dawley rats were fed dietary concentrations of 0, 15, 100, and 700 ppm and Swiss mice were fed 0, 100, 700, and 5,000 ppm for 2 years. Mortality was high in all treatment groups. A statistically significant increase in liver tumors was observed in both male and female rats and a statistically significant increase in benign mammary gland tumors was observed in female rats. In addition, an increased incidence of kidney tumors was observed in the mid-dose male mice. Data used to derive the CSF were based on liver and mammary tumors in female rats and are presented below as reported in IRIS.

Administered Dose (ppm)	Human Equivalent Dose (mg/kg/d)	Tumor Incidence
0	0	11/23
15	0.129	12/35
100	0.927	17/27
700	7.557	34/35

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \text{ } \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 0.68 \text{ (mg/kg/d)}^{-1} \times 1 \text{ mg}/1,000 \text{ } \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 1.9\text{E-}04(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

The California Environmental Protection Agency (CalEPA) adopted a URF of 8.9E-05 (μg/m³)⁻¹ and an inhalation CSF of 3.1E-01 (mg/kg/d)⁻¹ for practical grade 2,4-dinitrotoluene based on a potency factor derived by EPA (U.S. EPA, 1987) (CalEPA, 1997). These values were based on a feeding study using Sprague-Dawley rats (Lee et al., 1978). Liver and mammary tumors in female rats were used to develop the CSF and results were very similar to the Ellis et al. (1979) study discussed above.

The Carcinogenic Potency Database (CPDB) summarizes the results of 5,152 cancer tests on 1,298 chemicals (Gold and Zeiger, 1997). Carcinogenic potency estimates are presented as TD₅₀ values. TD₅₀ values are defined as *that dose-rate in mg/kg body wt/day which, if administered chronically for the standard lifespan of the species, will halve the probability of remaining tumorless throughout that period* (Gold and Zeiger, 1997). The TD₅₀ is analogous to the dose that is lethal to 50% of test animals (LD₅₀). A low TD₅₀ indicates high potency, just as a low LD₅₀ indicates high acute toxicity.

Some studies have reported high correlations between various measures of cancer potency and the maximum tolerated dose or maximum dose tested in the carcinogenicity studies (Gaylor, 1989; Krewski et al., 1993). The correlation of TD₅₀s as reported in the CPDB and inhalation CSFs derived from IRIS or HEAST was evaluated as a possible means to estimate the CSF from the TD₅₀. Forty-five chemicals were identified that had both a TD₅₀ and an inhalation CSF (see Table D-2, Figure D-2). The correlation coefficient for the regression is 0.95. The reported TD₅₀ is 9.35 mg/kg/d (Gold and Zeiger, 1997). Based on a linear regression of log TD₅₀ as the independent variable and log (1/CSF) as the dependent variable, an inhalation CSF of 0.53 (mg/kg/d)⁻¹ and a URF of 1.5E-04 (μg/m³)⁻¹ are predicted. These values are in close agreement with the oral CSF listed in IRIS for a mixture of 2,4- and 2,6-dinitrotoluene and the CalEPA values.

References:

California Environmental Protection Agency (CalEPA). 1997. Air Toxics Hot Spots Program Risk Assessment Guidelines: Technical Support Document for Determining Cancer Potency Factors. Draft for Public Comment. Office of Environmental Health Hazard Assessment.

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3-Methylcholanthrene CAS # 56-49-5

Unit Risk Factor:	2.1E-03 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	7.4E+00 ($\text{mg}/\text{kg}/\text{day}$) ⁻¹
Critical Effects:	Mammary gland adenocarcinomas
Species:	Wistar rats
Route of Exposure:	Gavage
Duration:	26 to 52 weeks

Basis for Toxicity Values:

There are no human data available that may be used to address the carcinogenicity of 3-methylcholanthrene (3-MC). However, 3-MC belongs to a class of chemicals known as polycyclic aromatic hydrocarbons (PAHs), which are components of coal tar and incomplete combustion. Many of the PAHs have been demonstrated to be carcinogenic to rats and mice following oral exposure, skin painting, intrapulmonary injection, inhalation, subcutaneous injection, and intraperitoneal injection; however, most of these studies are not considered suitable for quantitative risk assessment. Nevertheless, the data do indicate that the carcinogenic potencies vary and that 3-MC is considered one of the most potent PAHs (Pitot and Dragan, 1996).

3-MC is not listed in EPA's IRIS (U.S. EPA, 1998) or HEAST (U.S. EPA, 1997) databases and was not included in EPA's (1993) *Provisional Guidance for Quantitative Risk Assessment of PAHs*. However, the California Environmental Protection Agency (CalEPA) has developed a unit risk factor (URF) and cancer slope factor (CSF) for 3-MC in support of the Air Toxics Hot Spots Program (CalEPA, 1994a, 1994b, 1997). The CalEPA URF and inhalation CSF are listed above and recommended as interim values.

The CalEPA developed an "expedited" approach for deriving cancer potency values in order to implement Proposition 65 (Hoover et al., 1995). The expedited approach was used for 3-MC. Under the expedited approach, instead of conducting a comprehensive literature review, cancer dose response data are taken from the Carcinogenic Potency Database (CPDB) (Gold and Zeiger, 1997). The linearized multistage model is automatically used to derive cancer potency estimates for low-dose exposures, and pharmacokinetic adjustments are not made.

Fifteen studies (4 diet and 11 gavage) were listed in the CPDB (Gold and Zeiger, 1997). All of the studies included a control group and one treatment group. No tumors were reported in any of the dietary studies; however, a significant increase in tumors was reported in all of the gavage studies. Doses for the gavage studies ranged from 2.46 mg/kg/d to 12.2 mg/kg/d. Adenocarcinomas of the mammary gland were reported in nine studies and two studies identified unspecified mammary tissue tumors. Tumor incidence ranged from 67% to 100%.

Additional Information:

The CPDB summarizes the results of 5,152 cancer tests on 1,298 chemicals. Carcinogenic potency estimates are presented as TD₅₀s. TD₅₀s are defined as *that dose-rate in mg/kg body wt/day which, if administered chronically for the standard lifespan of the species, will halve the probability of remaining tumorless throughout that period* (Gold and Zeiger, 1997). The TD₅₀ is analogous to the dose that is lethal to 50% of test animals (LD₅₀). A low TD₅₀ indicates high potency, just as a low LD₅₀ indicates high acute toxicity.

Some studies have reported high correlations between various measures of cancer potency and the maximum tolerated dose or maximum dose tested in the carcinogenicity studies (Gaylor, 1989; Krewski et al., 1993). The correlation of TD₅₀s as reported in the CPDB and inhalation CSFs derived from IRIS or HEAST was evaluated as a possible means to estimate the CSF from the TD₅₀. Forty-five chemicals were identified that had both a TD₅₀ and an inhalation CSF (see Table D-2, Figure D-2). The correlation coefficient for the regression is 0.95. The TD₅₀ reported for 3-MC is 0.491 mg/kg/d (Gold and Zeiger, 1997). Based on a linear regression of log TD₅₀ as the independent variable and log (1/CSF) as the dependent variable, an inhalation CSF of 9.6 (mg/kg/d)⁻¹ and a URF of 2.7E-03 (μg/m³)⁻¹ are predicted. These values are in close agreement with the CalEPA values of 7.4 (mg/kg/d)⁻¹ and 2.1E-03 (μg/m³)⁻¹, respectively.

References:

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U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

o-Toluidine (2-Methylaniline)
CAS # 95-53-4

Unit Risk Factor:	6.9E-05 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	2.4E-01 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Skin fibromas - also increased incidence of other tumor types including sarcomas, mesotheliomas, carcinomas, hemangiosarcomas, and hepatocellular carcinomas of various tissues.
Species:	F-344 rats and B6C3F1 mice
Route of Exposure:	Diet
Duration:	2 years

Basis for Toxicity Values:

There is limited evidence that o-toluidine is carcinogenic in humans; however, data are inadequate for a quantitative risk assessment (U.S. EPA, 1987). o-Toluidine is not listed in EPA's IRIS (U.S. EPA, 1998) but an oral CSF is included in HEAST (U.S. EPA, 1997). The oral CSF of 2.4E-01 mg/kg/d is proposed as an interim value for the inhalation CSF.

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS and HEAST that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table D-1, Figure D-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

Dose-Response Data:

The oral CSF listed in HEAST was based on a study by Hecht et al. (1982). Groups of 30 male F344 rats were fed dietary concentrations of 0 or 4,000 ppm o-toluidine hydrochloride for 73 weeks followed by 20 weeks of observation. An increased incidence of skin fibromas, mammary fibroadenomas, spleen fibromas, and peritoneal sarcomas was reported. Skin fibromas gave the greatest response and were used to derive the CSF. The data are summarized below as reported in U.S. EPA (1987).

Experimental Dose o-Toluidine•HCl (mg/rat/d)	Transformed Dose o-Toluidine (mg/kg/d)	Incidence
0	0	1/27
62	80	25/30

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} =$$
$$0.24 (\text{mg}/\text{kg}/\text{d})^{-1} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 6.9\text{E-}05(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

The National Cancer Institute (NCI) also has conducted a cancer bioassay of o-toluidine hydrochloride (NCI, 1979). F344 rats were fed diets containing 0, 3,000, and 6,000 ppm and B6C3F₁ mice were fed diets containing 0, 1,000, and 3,000 ppm for 2 years. Multiple site sarcomas, subcutaneous fibromas, and multiple site mesotheliomas were observed in male rats. Female rats had multiple site sarcomas, mammary fibroadenomas, splenic sarcomas, and urinary bladder carcinomas. Multiple site hemangiosarcomas were seen in male mice and hepatocellular carcinomas and adenomas were seen in female mice. U.S. EPA (1987) reported that the Hecht et al. (1982) study was selected over the NCI (1979) study because the former resulted in a higher cancer potency estimate.

References:

Hecht, S.S., K. El-Bayoumy, A. Rivenson, and E. Fiala. 1982. Comparative carcinogenicity of o-toluidine hydrochloride and o-nitrosotoluene in F-344 Rats. *Cancer Letters* 16:103-108.

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**Table D-1. Correlation of Oral and Inhalation Cancer Slope Factors
Reported in IRIS and HEAST**

CAS #	Chemical	Oral CSF	Inh CSF	log Oral CSF	log Inh. CSF
79-06-1	Acrylamide	4.5	4.5	0.6532	0.6532
107-13-1	Acrylonitrile	0.54	0.24	-0.2676	-0.6198
309-00-2	Aldrin	17	17	1.2304	1.2304
140-57-8	Aramite	0.025	0.025	-1.6021	-1.6021
7440-38-2	Arsenic	1.5	15	0.1761	1.1761
103-33-3	Azobenzene	0.11	0.11	-0.9586	-0.9586
71-43-2	Benzene	0.029	0.029	-1.5376	-1.5376
92-87-5	Benzidine	230	235	2.3617	2.3711
7440-41-7	Beryllium	4.3	8.4	0.6335	0.9243
111-44-4	Bis(2-chloroethyl)ether	1.1	1.16	0.0414	0.0645
542-88-1	Bis(chloromethyl)ether	220	217	2.3424	2.3365
108-60-1	Bis(2-chloro-1-methylethyl)ether	0.07	0.035	-1.1549	-1.4559
75-25-2	Bromoform	0.0079	0.0039	-2.1024	-2.4145
56-23-5	Carbon tetrachloride	0.13	0.053	-0.8861	-1.2757
57-74-9	Chlordane	1.3	1.3	0.1139	0.1139
510-15-6	Chlorobenzilate	0.27	0.27	-0.5686	-0.5686
67-66-3	Chloroform	0.0061	0.08	-2.2147	-1.0969
74-87-3	Chloromethane	0.013	0.0063	-1.8861	-2.2007
50-29-3	DDT	0.34	0.34	-0.4685	-0.4685
96-12-8	1,2-Dibromo-3-chloropropane	1.4	0.0024	0.1461	-2.6162
106-93-4	1,2-Dibromoethane	85	0.77	1.9294	-0.1135
107-06-2	1,2-Dichloroethane	0.091	0.091	-1.0410	-1.0410
75-35-4	1,1-Dichloroethylene	0.6	0.175	-0.2218	-0.7570
542-75-6	1,3-Dichloropropene	0.18	0.13	-0.7447	-0.8861
60-57-1	Dieldrin	16	16	1.2041	1.2041
122-66-7	1,2-Diphenylhydrazine	0.8	0.77	-0.0969	-0.1135
106-89-8	Epichlorohydrin	0.0099	0.0042	-2.0044	-2.3768
75-21-8	Ethylene oxide	1.02	0.35	0.0086	-0.4559
319-84-6	HCH alpha	6.3	6.3	0.7993	0.7993
319-85-7	HCH beta	1.8	1.8	0.2553	0.2553
608-73-1	HCH tech.	1.8	1.8	0.2553	0.2553
76-44-8	Heptachlor	4.5	4.5	0.6532	0.6532
1024-57-3	Heptachlor epoxide	9.1	9.1	0.9590	0.9590
118-74-1	Hexachlorobenzene	1.6	1.6	0.2041	0.2041
87-68-3	Hexachlorobutadiene	0.078	0.077	-1.1079	-1.1135
67-72-1	Hexachloroethane	0.014	0.014	-1.8539	-1.8539

(continued)

Table D-1. (continued)

CAS #	Chemical	Oral CSF	Inh CSF	log Oral CSF	log Inh. CSF
302-01-2	Hydrazine	3	17.1	0.4771	1.2330
75-09-2	Methylene chloride	0.0075	0.0016	-2.1249	-2.7959
101-14-4	4,4'-Methylenebis(2-chloroaniline)	0.13	0.13	-0.8861	-0.8861
924-16-3	<i>N</i> -Nitrosodi- <i>n</i> -butylamine	5.4	5.6	0.7324	0.7482
55-18-5	<i>N</i> -Nitrosodiethylamine	150	151	2.1761	2.1790
62-75-9	<i>N</i> -Nitrosodimethylamine	51	49	1.7076	1.6902
930-55-2	<i>N</i> -Nitrosopyrrolidine	2.1	2.13	0.3222	0.3284
1336-36-3	PCBs	2	0.4	0.3010	-0.3979
75-56-9	Propylene oxide	0.24	0.013	-0.6198	-1.8861
630-20-6	1,1,1,2,-Tetrachloroethane	0.026	0.026	-1.5850	-1.5850
79-34-5	1,1,2,2,-Tetrachloroethane	0.2	0.2	-0.6990	-0.6990
8001-35-2	Toxaphene	1.1	1.1	0.0414	0.0414
79-00-5	1,1,2-Trichloroethane	0.057	0.056	-1.2441	-1.2518
88-06-2	2,4,6,-Trichlorophenol	0.011	0.011	-1.9586	-1.9586
75-01-4	Vinyl chloride	1.9	0.3	0.2788	-0.5229

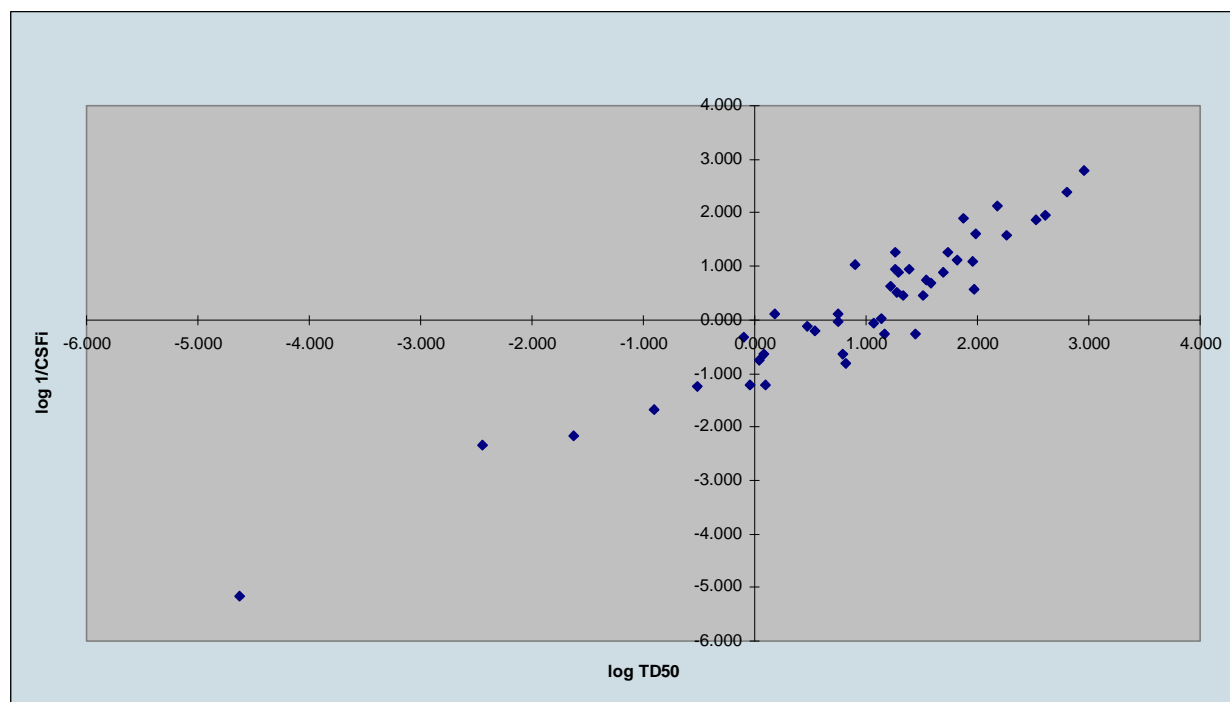


Figure D-1. Correlation of Oral and Inhalation Cancer Slope Factors.

Table D-2. Correlation of TD_{50s} Reported in the Cancer Potency Database and Inhalation Cancer Slope Factors Reported in IRIS and HEAST.

TD ₅₀ Geo Mean ^b	Inh CSF ^c	CSF test species ^d	1/CSF	log TD ₅₀ ^e (X)	log 1/CSF (Y)		
18.53	0.053	b	18.87	1.268	1.276		
32.28	0.34	b	2.94	1.509	0.469		
*	17	m	0.059	0.104	-1.230		
*	1.16	m	0.86	1.068	-0.064		
*	0.98	m	1.020	1.143	0.009		
*	1.3	m	0.77	0.476	-0.114		
*	0.27	m	3.70	1.973	0.569		
*	0.08	m	12.50	1.956	1.097		
*	0.175	m	5.71	1.539	0.757		
*	0.13	m	7.692	1.695	0.886		
*	16	m	0.0625	-0.040	-1.204		
*	6.3	m	0.159	0.821	-0.799		
*	1.8	m	0.556	1.444	-0.255		
*	1.8	m	0.556	1.170	-0.255		
*	4.5	m	0.222	0.083	-0.653		
*	0.014	m	71.429	2.529	1.854		
*	0.0016	m	625.000	2.963	2.796		
*	5.6	m	0.179	0.037	-0.748		
*	0.026	m	38.4615	2.260	1.585		
*	0.2	m	5	1.583	0.699		
*	1.1	m	0.909	0.746	-0.041		
*	0.056	m	17.857	1.740	1.252		
*	0.0077	r	129.870	2.185	2.114		
*	4.5	r	0.222	0.789	-0.653		
*	0.24	r	4.167	1.228	0.620		
*	0.025	r	40	1.985	1.602		
*	0.11	r	9.09	1.382	0.959		
*	217	r	0.00	-2.447	-2.336		
*	0.11	r	9.091	1.267	0.959		
*	0.004	r	250.00	2.812	2.398		
*	0.77	r	1.299	0.182	0.114		
*	0.091	r	10.99	0.905	1.041		
*	0.77	r	1.298701	0.747	0.114		
*	0.35	r	2.857	1.328	0.456		
*	1.6	r	0.625	0.545	-0.204		
*	0.077	r	12.987	1.818	1.114		
*	17.1	r	0.058	-0.510	-1.233		
*	0.13	r	7.692	1.286	0.886		
*	151	r	0.007	-1.625	-2.179		
*	49	r	0.020	-0.907	-1.690		
*	2.13	r	0.469	-0.097	-0.328		
*	0.013	r	76.9231	1.872	1.886		

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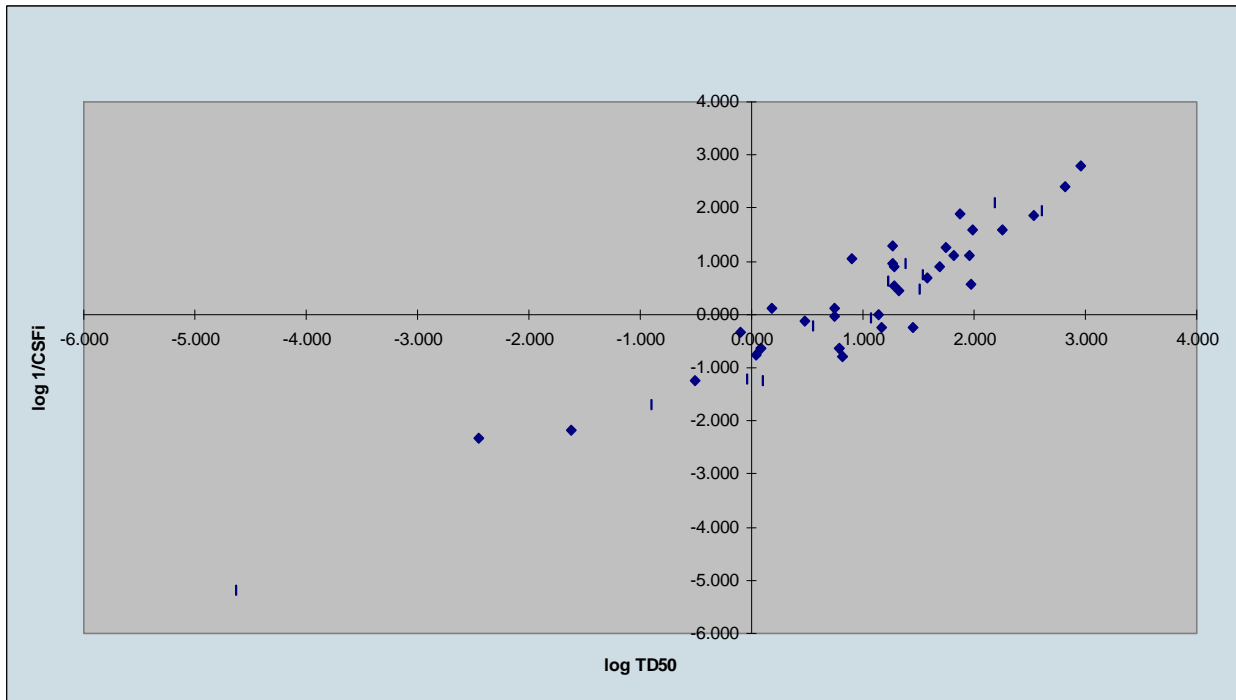


Figure D-2. Correlation of TD_{50} and inhalation cancer slope factors.