US EPA ARCHIVE DOCUMENT

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7.0 Human Health Toxicity Assessments

To characterize the risk from human exposures to the constituents of concern, toxicity information on each chemical of concern is integrated with the results of the exposure assessment. A chemical's ability to cause an adverse health effect depends on the toxicity of the chemical, the chemical's route of exposure to an individual (either through ingestion or inhalation), the duration of exposure, and the dose received (the amount that a human ingests or inhales). For a risk assessment, the toxicity of a constituent is defined by a human health benchmark for each route of exposure. Essentially, a benchmark is a quantitative value used to predict a chemical's possible toxicity and ability to induce a health effect at certain levels of exposure. These health benchmarks are derived from toxicity data based on animal studies or human epidemiological studies. Each benchmark represents a dose-response estimate that relates the likelihood and severity of adverse health effects to exposure and dose. Because individual chemicals cause different health effects at different doses, benchmarks are chemical-specific.

Human health benchmarks for chronic oral and inhalation exposures were needed to conduct the risk characterization. This section presents the noncancer and cancer benchmarks used to evaluate human health effects that may result from exposure to constituents modeled for this risk assessment. Human health benchmarks and their sources are discussed in Section 7.1 and the benchmarks for each constituent are provided. Section 7.2 summarizes the health benchmarks identified from alternative (non-EPA) sources. Chronic health benchmarks derived for this risk assessment are provided in Section 7.3. Appendix Q contains detailed information on the scientific basis of each human health benchmark. For each constituent, noncancer and cancer effects and the toxicological studies, calculations, and methods used to derive the benchmarks are described.

7.1 Chronic Health Benchmarks Used in This Risk Assessment

Chronic human health benchmarks were used in this risk assessment to evaluate potential noncancer and cancer risks. EPA uses reference doses (RfDs) and reference concentrations (RfCs) to evaluate noncancer risk from oral and inhalation exposures, respectively. Oral cancer slope factors (CSFs), inhalation unit risk factors (URFs), and inhalation CSFs are used to evaluate risk for carcinogens. The benchmarks are chemical-specific and do not vary between receptors (i.e., residents, farmers, recreational fishers) or age groups.

The RfD and RfC are estimates (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 appreciable risk of deleterious noncancer effects during a lifetime. It is not a direct estimator of risk but rather a reference point to gauge the potential effects. At exposures increasingly greater than the RfD (or RfC), the potential for adverse health effects increases.

Lifetime exposure above the RfD (or RfC) does not imply that an adverse health effect would necessarily occur (U.S. EPA, 2000b).

The RfD and RfC are the primary benchmarks used to evaluate noncarcinogenic hazards posed by environmental exposures to chemicals. They are based on the "threshold" approach, which is the theory that there is a "safe" exposure level (i.e., a threshold) that must be exceeded before an adverse noncancer effect occurs. RfDs and RfCs do not provide true dose-response information in that they are estimates of an exposure level or concentration that is believed to be below the threshold level or no observed adverse effects level (NOAEL). The degree of uncertainty and confidence levels in RfDs varies and is based on different toxic effects.

The CSF is an upper-bound estimate (approximating a 95 percent confidence limit) of the increased human cancer risk from a lifetime exposure to an agent. This estimate is usually expressed in units of proportion (of a population) affected per milligram of agent per kilogram body weight per day (mg/kg-d). The unit risk, which is calculated from the slope factor, is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water or 1 $\mu g/m^3$ in air. That is, if unit risk = 1.5×10^{-6} µg/L, then 1.5 excess tumors are expected to develop per 1,000,000 people if exposed daily for a lifetime to 1 µg of the chemical in 1 L of drinking water (U.S. EPA, 2000b). Unlike RfDs and RfCs, CSFs and URFs do not represent "safe" exposure levels; rather, they relate levels of exposure with a probability of effect or risk.

Several sources of human health benchmarks are available. Health benchmarks for this risk assessment were obtained primarily from the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST). IRIS and HEAST are maintained by EPA, and their values were used in this analysis whenever available.

IRIS is EPA's electronic database containing consensus scientific positions on potential adverse human health effects that may occur from chronic exposure to environmental contaminants (U.S. EPA, 2000b). Each chemical file contains descriptive and quantitative information on potential health effects. Health benchmarks for chronic noncarcinogenic health effects include RfDs and RfCs. Cancer classifications, oral CSFs, and oral and inhalation URFs are included for carcinogenic effects. IRIS is EPA's official repository of Agency-wide consensus information on human health risk.

HEAST is a comprehensive listing of provisional noncarcinogenic and carcinogenic health benchmarks (RfDs, RfCs, CSFs, and URFs) derived by EPA (U.S. EPA, 1997a). HEAST benchmarks are considered secondary to those contained in IRIS. Although the health benchmarks in HEAST have undergone review and have the concurrence of individual EPA program offices, either they have not been reviewed as extensively as those in IRIS or they do not have as complete a data set as is required to be listed in IRIS. HEAST benchmarks have not been updated in several years and are not recognized as Agency-wide consensus information.

Other provisional EPA benchmarks, Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels (MRLs), California Environmental Protection Agency (CalEPA) chronic inhalation reference exposure levels (RELs), and CalEPA cancer potency factors were

US EPA ARCHIVE DOCUMENT

used when values were not available from IRIS or HEAST; the hierarchy for selecting alternative human health benchmarks is explained in Section 7.2. A health benchmark developed by EPA is considered "provisional" if the value has had some form of Agency review but does not represent Agency-wide consensus (i.e., it does not appear on IRIS). At the time each provisional health benchmark was derived, all available toxicological information was evaluated, the value was calculated using the most current methodology, and a consensus was reached on the value by an individual EPA program office (but not Agency-wide) (U.S. EPA, 1997a). All health benchmarks not identified from IRIS, including MRLs and CalEPA cancer potency factors and RELs, were treated as provisional health benchmarks for this risk assessment. Because chronic inhalation benchmarks were not available, RfCs were developed for nickel-soluble salts and nickel oxide for use in this risk assessment.

EPA's Superfund Technical Support Center (National Center for Environmental Assessment or NCEA) derives provisional RfCs, RfDs, CSFs, and URFs for certain chemicals. These provisional health benchmarks are published in NCEA risk assessment issue papers. Because these values have not undergone EPA's formal review process, they do not represent EPA-verified benchmarks.

EPA has also derived provisional health benchmark values in other risk assessment documents such as Health Assessment Documents (HADs), Health Effect Assessments (HEAs), Health and Environmental Effects Profiles (HEEPs), Health and Environmental Effects Documents (HEEDs), Drinking Water Criteria Documents (DWCDs), and Ambient Water Quality Criteria Documents (AWQCDs). Evaluations of potential carcinogenicity of chemicals in support of reportable quantity adjustments were published by EPA's Carcinogen Assessment Group (CAG) and may include cancer potency factor estimates. Health benchmark values identified in these EPA documents are not recognized as Agency-wide consensus information, however.

ATSDR calculates minimal risk levels that are substance-specific health guidance levels for noncarcinogenic endpoints. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified exposure duration. MRLs are derived for acute, intermediate, and chronic exposure durations for oral and inhalation routes of exposure. Inhalation and oral MRLs are similar to EPA's RfCs and RfDs, respectively; however, MRLs are intended to serve as screening levels. When based on the same critical study, the inhalation and oral MRLs have similar toxicity endpoints but may apply different uncertainty factors in contrast to EPA's RfDs and RfCs. MRLs are available on ATSDR's web site and are presented in detail in individual ATSDR Toxicological Profiles.

CalEPA has developed cancer potency factors for chemicals regulated under California's Hot Spots Air Toxics program (CalEPA, 1999a). The cancer potency factors are analogous to EPA's oral and inhalation CSFs. CalEPA has also developed chronic inhalation reference exposure levels, analogous to U.S. EPA's RfCs, for 120 substances (CalEPA, 1997, 1999b). CalEPA used EPA's 1994 inhalation dosimetry methodology to derive inhalation RELs. The cancer potency factors and inhalation RELs have undergone internal peer review by various California agencies and have been the subject of public comment.

US EPA ARCHIVE DOCUMENT

To assess less than lifetime cancer risks (e.g., child) and address population variability (e.g., body weight differences among adults), inhalation CSFs were used in this risk assessment. Inhalation CSFs were used to account for age-specific differences and population variability in inhalation rate and body weight as well as exposure duration and frequency. Inhalation URFs are not dependent on exposure factors (e.g., inhalation rate) and therefore cannot be used to address population variability or age-specific differences in exposure scenarios. Inhalation CSFs are not available from IRIS, so they were calculated for use in this risk assessment based on inhalation URFs (which are available from IRIS). The inhalation CSFs were calculated using the following equation:

$$\text{inh CSF} = \text{inh URF} \times 70\ \text{kg} \div 20\ \text{m}^3/\text{d} \times 1{,}000\ \mu\text{g/mg} \quad . \tag{7-1}$$

For one constituent (tetrachloroethylene), the oral CSF was calculated from the oral URF using the following equation:

$$\text{oral CSF} = \text{oral URF} \times 70\ \text{kg} \div 2\ \text{L/d} \times 1{,}000\ \mu\text{g/mg} \quad . \tag{7-2}$$

Figure 7-1 illustrates the approach used to identify or develop the chronic health benchmarks used in this analysis. Because IRIS is EPA's official repository of Agency-wide consensus human health risk information, benchmarks from IRIS were used whenever available. Benchmarks from HEAST were used if none were available from IRIS. If health benchmarks were not available from IRIS or HEAST, benchmarks from alternative sources were sought.

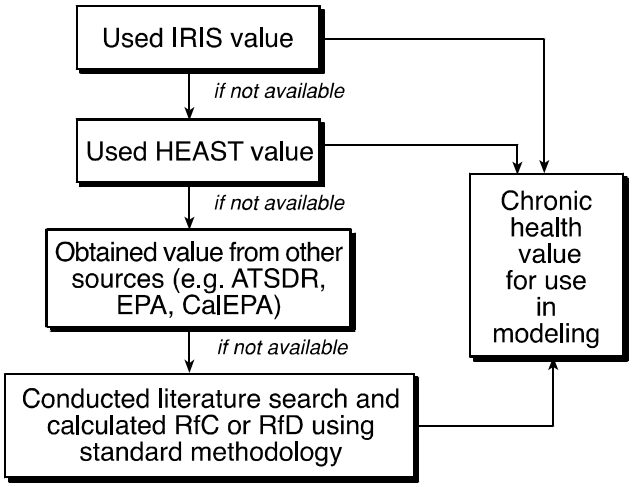

Figure 7-1. Approach used to select chronic health benchmark values.

The chronic human health benchmarks used in this risk analysis are summarized in Table 7-1. The Chemical Abstract Service Registry Number (CASRN), constituent name, cancer classification, RfD (in units of mg/kg-d), RfC (mg/m^3), noncancer target organs, oral and inhalation CSF (mg/kg-d^{-1}), inhalation URF [(µg/m^3)$^{-1}$], and reference for each benchmark are provided in this table. "RfD target" and "RfC target" refer to the target organ (e.g., kidney, liver) or critical effect used as the basis for the RfD or RfC. The critical effect for a few benchmarks is listed as "no effect" and refers to the fact that no adverse effects were observed in the principal study. For dibutyl phthalate, the RfD was based on increased mortality at higher dose levels; therefore, the target organ was classified as "death." A key to the references cited and abbreviations used is provided at the end of the table.

Recent evaluations conducted by EPA's Office of Water conclude that protecting against chloroform's noncancer health effects protects against excess cancer risk. EPA now believes that the noncancer health effects resulting from inhalation of chloroform would precede the development of cancer and would occur at lower doses than tumor (cancer) development.

US EPA ARCHIVE DOCUMENT

US EPA ARCHIVE DOCUMENT

Table 7-1. Chronic Health Benchmarks Used in Paints Risk Assessment

CASRN	Constituent	Class	Ref[a]	RfD (mg/kg-d)	RfD Target	RfD Ref	Oral CSF $(mg/kg\text{-}d)^{-1}$	CSF Ref	RfC (mg/m^3)	RfC Target	RfC Ref	inh URF $(\mu g/m^3)^{-1}$	Inh URF Ref	inh CSF $(mg/kg\text{-}d)^{-1}$	Inh CSF Ref
79-06-1	Acrylamide	B2	I	2.0E-04	Neuro	I	4.5E+00	I	7.0E-04	Neuro	C97	1.3E-03	I	4.5E+00	H
107-13-1	Acrylonitrile	B1	I	1.0E-03	Repro	H	5.4E-01	I	2.0E-03	Respir	I	6.8E-05	I	2.4E-01	H
7440-36-0	Antimony			4.0E-04	Hemato	I			2.0E-04[b]	Respir	I				
7440-39-3	Barium	D	I	7.0E-02	Kidney	I			5.0E-04	Repro	H				
71-43-2	Benzene	A	I				5.5E-02	I				7.8E-06	I	2.7E-02	Calc
71-36-3	Butyl alcohol, n-	D	I	1.0E-01	Neuro	I									
85-68-7	Butylbenzylphthalate	C	I	2.0E-01	Liver	I									
7440-43-9	Cadmium	B1	I	5.0E-04[c] 1.0E-03	Kidney	I			2.0E-05	Kidney, respir	C99b	1.8E-03	I	6.3E+00	Calc
67-66-3	Chloroform	B2	I	1.0E-02	Liver	I			1.0E-01	Liver	A				
16065-83-1	Chromium (III)	D	I	1.5E+00	No effect	I									
18540-29-9	Chromium (VI)	A	I	3.0E-03	No effect	I			1.0E-04[d]	Respir	I	1.2E-02	I	4.1E+01	H
7440-48-4	Cobalt			6.0E-02	Hemato	N			1.0E-05	Respir	AC				
7440-50-8	Copper	D	I	[e]					2.0E-05	Respir	C97				
108-39-4	Cresol, m-	C	I	5.0E-02	Body wt, neuro	I									
95-48-7	Cresol, o-	C	I	5.0E-02	Body wt, neuro	I									
106-44-5	Cresol, p-	C	I	5.0E-03	Neuro, respir	H									
117-81-7	Di(2-ethylhexyl)phthalate	B2	I	2.0E-02	Liver	I	1.4E-02	I	1.0E-02	Respir	SF				

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US EPA ARCHIVE DOCUMENT

Table 7-1. (continued)

CASRN	Constituent	Class	Ref[a]	RfD (mg/kg-d)	RfD Target	RfD Ref	Oral CSF $(mg/kg\text{-}d)^{-1}$	CSF Ref	RfC (mg/m^3)	RfC Target	RfC Ref	inh URF $(\mu g/m^3)^{-1}$	Inh URF Ref	inh CSF $(mg/kg\text{-}d)^{-1}$	Inh CSF Ref
84-74-2	Dibutylphthalate	D	I	1.0E-01	Death	I									
75-09-2	Dichloromethane (methylene chloride)	B2	I	6.0E-02	Liver	I	7.5E-03	I	3.0E+00	Liver	H	4.7E-07	I	1.6E-03	Calc
105-67-9	Dimethylphenol, 2,4-			2.0E-02	Hemato, neuro	I									
100-41-4	Ethylbenzene	D	I	1.0E-01	Kidney, liver	I			1.0E+00	Develop	I				
107-21-1	Ethylene glycol			2.0E+00	Kidney	I			6.0E-01	Respir	AC				
50-00-0	Formaldehyde	B1	I	2.0E-01	Body wt	I						1.3E-05	I	4.5E-02	H
7439-92-1	Lead	B2	I	[f]											
7487-94-7	Mercuric chloride (inorganic mercury)	C	I	3.0E-04	Immuno	H									
7439-97-6	Mercury (elemental)	D	I						3.0E-04	Neuro	I				
67-56-1	Methanol			5.0E-01	Liver, neuro	I			1.3E+01	Develop	AC				
78-93-3	Methyl ethyl ketone (MEK)	D	I	6.0E-01	Develop	I			1.0E+00	Develop	I				
108-10-1	Methyl isobutyl ketone (MIBK)			8.0E-02	Liver, kidney, neuro	H			8.0E-02	Liver, kidney	H				
22967-92-6	Methylmercury	C	I	1.0E-04	Develop	I									
80-62-6	Methyl methacrylate	E	I	1.4E+00	No effect	I			7.0E-01	Respir	I				

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US EPA ARCHIVE DOCUMENT

Table 7-1. (continued)

CASRN	Constituent	Class	Ref[a]	RfD (mg/kg-d)	RfD Target	RfD Ref	Oral CSF $(mg/kg\text{-}d)^{-1}$	CSF Ref	RfC (mg/m^3)	RfC Target	RfC Ref	inh URF $(\mu g/m^3)^{-1}$	Inh URF Ref	inh CSF $(mg/kg\text{-}d)^{-1}$	Inh CSF Ref
7440-02-0	Nickel			2.0E-02	Body wt, organ wt	I			8.0E-05	Respir	D				
1313-99-1	Nickel oxide								1.5E-04	Respir	D				
87-86-5	Pentachlorophenol	B2	I	3.0E-02	Kidney, liver	I	1.2E-01	I	1.0E-01	Kidney, liver, develop	C97	5.1E-06	C99a	1.8E-02	Calc
108-95-2	Phenol	D	I	6.0E-01	Develop	I									
7782-49-2	Selenium	D	I	5.0E-03	Respir	I			2.0E-02	Liver, cardio, neuro	C99b				
7440-22-4	Silver	D	I	5.0E-03	Skin	I			2.0E-02	Skin	C97				
100-42-5	Styrene			2.0E-01	Hemato, liver	I			1.0E+00	Neuro	I				
127-18-4	Tetrachloroethylene	B2	O	1.0E-02	Liver	I	5.2E-02	O	3.0E-01	Neuro	A	5.8E-07	O	2.0E-03	Calc
7440-31-5	Tin			6.0E-01	Kidney, liver	H									
108-88-3	Toluene	D	I	2.0E-01	Kidney, liver	I			4.0E-01	Neuro, respir	I				
108-05-4	Vinyl acetate			1.0E+00	Body wt, kidney	H			2.0E-01	Respir	I				
1330-20-7	Xylene (mixed isomers)	D	I	2.0E+00	Body wt, neuro	I			4.0E-01	Neuro	A				
108-38-3	Xylene, m-			2.0E+00	Body wt, neuro	H			4.0E-01[g]	Neuro	A				

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US EPA ARCHIVE DOCUMENT

Table 7-1. (continued)

CASRN	Constituent	Class	Ref[a]	RfD (mg/kg-d)	RfD Target	RfD Ref	Oral CSF $(mg/kg\text{-}d)^{-1}$	CSF Ref	RfC (mg/m^3)	RfC Target	RfC Ref	inh URF $(\mu g/m^3)^{-1}$	Inh URF Ref	inh CSF $(mg/kg\text{-}d)^{-1}$	Inh CSF Ref
95-47-6	Xylene, o-			2.0E+00	Body wt, neuro	H			4.0E-01[g]	Neuro	A				
106-42-3	Xylene, p-			2.0E+00[h]	Body wt, neuro	I			4.0E-01[g]	Neuro	A				
7440-66-6	Zinc	D	I	3.0E-01	Hemato	I			9.0E-04	Respir, immuno	C97				

Body Wt = Body weight.
CASRN = Chemical Abstract Service registration number.
CSF = Cancer slope factor.
RfC = Reference concentration.
RfD = Reference dose.
URF = Unit risk factor.

[a] Sources:
- I = IRIS (U.S. EPA, 2000b)
- H = HEAST (U.S. EPA, 1997a).
- A = ATSDR minimal risk levels (MRLs) (ATSDR, 2000).
- SF = Superfund Risk Issue Paper (U.S. EPA, 1996a).
- FR = 63 FR 64371-0402 (U.S. EPA, 1998a)
- C99a = CalEPA cancer potency factor (CalEPA, 1999a)
- C99b = CalEPA chronic REL (CalEPA, 1999b)
- C97 = CalEPA chronic REL (CalEPA, 1997)
- N = NCEA Risk Assessment Issue Paper (U.S. EPA, 1997b)
- AC = Developed for the Air Characteristic Study (U.S. EPA, 1999)
- D = Developed for this risk assessment.
- O = Other source (see Sections 7.1.1 and 7.1.2).

[b] RfC is for antimony trioxide.
[c] RfD = 5.0E-04 mg/kg-d for water; RfD = 1.0E-03 mg/kg-d for food.
[d] RfC is for chromium (VI) particulates.
[e] MCL of 1.3 mg/L is available for copper (no RfD) (U.S. EPA, 2000a).
[f] A 400-ppm screening level for lead in soil (U.S. EPA, 1998b) and a 0.015-mg/L action level in drinking water (U.S. EPA, 2000a) were used in this risk assessment in lieu of human health benchmarks (which are not available).
[g] RfC is for total xylenes.
[h] RfD is for total xylenes.

Although EPA has not finalized an RfC for chloroform, ATSDR has developed an inhalation MRL and this noncancer health benchmark was used in the paints listing risk analysis.

7.2 Alternative Chronic Health Benchmarks Identified

As discussed in the previous section, if IRIS or HEAST chronic health benchmarks were not available, benchmarks from alternative sources were sought. Provisional EPA benchmarks, ATSDR MRLs, CalEPA chronic RELs (CalEPA, 1997, 1999b), and CalEPA cancer potency factors (CalEPA, 1999a) were included whenever available. The derivation of each alternative benchmark was evaluated to confirm that appropriate, high-quality data had been used and that methodologies in accordance with current EPA guidance (U.S. EPA, 1994, 1996b) had been applied. When more than one alternative human health benchmark was available, the data (e.g., critical study) and methodologies used to derive each benchmark were evaluated to ensure that the most appropriate value was used in the risk analysis. Alternative human health benchmarks were identified for the following 14 constituents

- Cadmium (7440-43-9)
- Chloroform (67-66-3)
- Cobalt (7440-48-4)
- Copper (7440-50-8)
- Di(2-ethylhexyl)phthalate (117-81-7)
- Ethylene glycol (107-21-1)
- Methanol (67-56-1)
- Nickel (7440-02-0)
- Pentachlorophenol (87-86-5)
- Selenium (7782-49-2)
- Silver (7440-22-4)
- Tetrachloroethylene (127-18-4)
- Xylenes (1330-20-7)
- Zinc (7440-66-6).

A provisional RfD calculated by EPA's NCEA was identified for cobalt (U.S. EPA, 1997b). For chloroform, tetrachloroethylene, and total xylenes, ATSDR's chronic inhalation MRLs were used. A provisional RfC for di(2-ethylhexyl) phthalate was identified in a Superfund risk issue paper (U.S. EPA, 1996a). Interim RfCs for cobalt, ethylene glycol, and methanol were developed in EPA's Air Characteristic Study (U.S. EPA, 1999). CalEPA chronic RELs were identified for acrylamide, cadmium, copper, pentachlorophenol, selenium, silver, and zinc (CalEPA, 1997, 1999b).

Alternative cancer benchmarks were also identified. For tetrachloroethylene, an oral CSF and an inhalation URF were found in EPA's *Health Assessment Document* and *Addendum*, respectively (U.S. EPA, 1985, 1986). A CalEPA inhalation URF was identified for pentachlorophenol (CalEPA, 1999a). Table 7-2 summarizes the alternative health benchmarks identified for this analysis, as well as the target organs affected and the source of the benchmarks.

EPA considered the use of alternative human health benchmarks (e.g., ATSDR MRLs, CalEPA reference exposure levels) in the paints listing risk analysis when benchmarks were not available from IRIS or HEAST. If target waste concentrations were driven by an alternative health benchmark, the benchmark underwent further technical review by EPA. Based on this review, alternative health benchmarks for di(2-ethylhexyl)phthalate (inhalation URF and CSF), formaldehyde (RfC), and phenol (RfC) were removed from use in the risk analysis.

US EPA ARCHIVE DOCUMENT

Table 7-2. Alternative Chronic Health Benchmarks

CASRN	Chemical Name	Benchmark and Benchmark Value	Target Organ	Source
79-06-1	Acrylamide	RfC = 7.04E-04 mg/m^3	Neurological	CalEPA chronic REL based on Burek et al., 1980
7440-43-9	Cadmium	RfC = 2.0E-05 mg/m^3	Renal, respiratory	CalEPA chronic REL based on Lauwerys et al., 1974
67-66-3	Chloroform	RfC = 0.1 mg/m^3	Hepatic	ATSDR chronic inhal MRL based on Bomski et al., 1967
7440-48-4	Cobalt	RfD = 0.06 mg/kg-d	Hematological	NCEA provisional RfD
7440-48-4	Cobalt	RfC = 1.0E-05 mg/m^3	Respiratory	Air Characteristic RfC based on NTP 1991, 1996a; Bucher et al., 1990
7440-50-8	Copper	RfC = 2.0E-05 mg/m^3	Respiratory	CalEPA chronic REL based on Gleason, 1968
117-81-7	Di(2-ethylhexyl) phthalate	RfC = 0.01 mg/m^3	Respiratory	SF provisional RfC based on Klimisch et al., 1992
107-21-1	Ethylene glycol	RfC= 0.6 mg/m^3	Respiratory	Air Characteristic RfC based on Wills et al., 1974
67-56-1	Methanol	RfC = 13 mg/m^3	Developmental	Air Characterization RfC based on Rogers et al., 1993
87-86-5	Pentachlorophenol	RfC = 0.1 mg/m^3	Renal, hepatic, developmental	CalEPA chronic REL based on route-to-route extrapolation of U.S. EPA RfD
87-86-5	Pentachlorophenol	Inh URF = 5.1E-6 per mg/m^3		CalEPA cancer potency factor
7782-49-2	Selenium	RfC = 0.02 mg/m^3	Hepatic, cardiovascular, neurological	CalEPA chronic REL citing Dudley and Miller, 1941
7440-22-4	Silver	RfC = 0.02 mg/m^3	Skin	CalEPA chronic REL based on route-to-route extrapolation of U.S. EPA RfD
127-18-4	Tetrachloroethylene	RfC = 0.04 ppm (0.3 mg/m^3)	Neurological	ATSDR chronic inhal MRL based on Ferroni et al., 1992.
127-18-4	Tetrachloroethylene	Oral CSF = 0.052 per mg/kg-d		Calculated from oral URF cited in HAD (U.S. EPA, 1985)
127-18-4	Tetrachloroethylene	Inh URF =5.8E-07 mg/m^3		Inhalation URF cited in HAD addendum (U.S. EPA, 1986)

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US EPA ARCHIVE DOCUMENT

Table 7-2. (continued)

CASRN	Chemical Name	Benchmark and Benchmark Value	Target Organ	Source
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
108-38-3	Xylene, m-	RfC = 0.1 ppm (0.4 mg/m^3)	Neurological	ATSDR chronic inhal MRL for total xylenes based on Uchida et al., 1993
95-47-6	Xylene, o-	RfC = 0.1 ppm (0.4 mg/m^3)	Neurological	ATSDR chronic inhal MRL for total xylenes based on Uchida et al., 1993
106-42-3	Xylene, p-	RfC = 0.1 ppm (0.4 mg/m^3)	Neurological	ATSDR chronic inhal MRL for total xylenes based on Uchida et al., 1993
7440-66-6	Zinc	RfC = 0.0009 mg/m^3	Respiratory, immunological	CalEPA chronic REL based on Malo et al., 1993

7.3 Chronic Health Benchmarks Derived for This Risk Assessment

Because chronic inhalation benchmarks were not available, RfCs were developed for nickel-soluble salts and nickel oxide for use in this risk assessment. Available toxicological data were examined to identify studies with the highest NOAEL or lowest observable adverse effects level (LOAEL) for the most sensitive or most relevant species. Appropriate inhalation studies were identified for nickel sulfate hexahydrate and nickel oxide (NTP, 1996b, 1996c). The critical studies were well-designed and included several exposure concentrations (three plus controls), exposure was of chronic duration (2 years) and by a relevant route (inhalation), and the results showed statistically significant dose-response relationships. 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, 1994).

Table 7-3 summarizes the chronic health benchmarks derived for use in this analysis, the method of development and critical studies used, and the target organs identified. Details on the derivation of these benchmark values are provided in Appendix Q.

Table 7-3. Chronic Health Benchmarks Derived for This Risk Assessment

CASRN	Chemical Name	Benchmark and Benchmark Value	Target Organ	Method of Derivation
7440-02-0	Nickel soluble salts	RfC = 8.0E-05 mg/m^3	Respiratory	Standard RfC derivation based on NTP, 1996b
1313-99-1	Nickel oxide	RfC = 1.5E-04 mg/m^3	Respiratory	Standard RfC derivation based on NTP, 1996c

US EPA ARCHIVE DOCUMENT

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US EPA ARCHIVE DOCUMENT

8.0 Generating Results

The final step of the risk assessment process is to characterize the risk posed to receptors (e.g., residents, farmers, and fishers). In this step, the preceding components of the risk assessment, estimates of toxicity (the health benchmarks) and exposure assessments, are summarized and integrated into quantitative expressions of risk. For this risk assessment, estimates of dose and toxicity were used to calculate individual excess lifetime carcinogenic risk estimates and noncancer hazard quotients for the 43 constituents in combined solids, emission control dust, and wastewater. Section 8.1 describes the risk calculations completed for this analysis that were based on unit waste concentrations (e.g., 1 mg/kg). Section 8.2 describes how these estimates of risk were used to calculate target waste concentrations using scaling factors and how waste volumes were then used to estimate protective waste concentrations in paint waste streams. This chapter also presents the methodology used to determine when target waste concentrations exceeded solubility limits (Section 8.3).

8.1 Human Health Risk Characterization

The goal of this risk assessment was to generate risk-based constituent concentrations that can be present in waste and remain below a specified target risk level. To accomplish this, it was necessary to first predict the risk of managing 1 mg/kg of a constituent in each of the waste management units (landfills, treatment tanks, and surface impoundments). Thus, from the probabilistic analysis, a distribution of risk estimates was generated based on a single unit waste concentration. The risk from this unit concentration was then used to scale to the concentration in the unit using the target risk level (Section 8.2).

The target risk level for this assessment was either

- An excess individual lifetime cancer risk of 1 chance in 100,000 of developing cancer (1E-5) for constituents that can produce cancer health effects

or

- A measure of projected intake levels to safe intake levels, a hazard quotient, of 1 for constituents that can produce noncancer health effects.

Not only can exposure to a constituent create both cancer and noncancer health impacts, but the type and magnitude of the exposure will differ depending on whether the constituent was ingested or inhaled. As such, the cancer and noncancer health impacts for both ingestion and inhalation of the constituent were calculated. Because there is a different risk resulting from the type of health impact (cancer vs. noncancer) and route of exposure (ingestion vs. inhalation),

US EPA ARCHIVE DOCUMENT

different risk endpoints were generated for each constituent in each waste management unit. The risk endpoints are listed in Table 8-1.

The risks resulting from exposures to the air pathway and groundwater pathway were evaluated separately. Estimated exposures from air pathways occur during the operating or post-closure life of the unit while risks via the groundwater pathways are, for the most part, not projected to occur within the same time frame. In addition, the location of aboveground receptors was randomly selected and did not necessarily coincide with the location of the groundwater plume. Therefore, the risks from these two pathways were not added together. As a result, each of the risk endpoints was estimated twice, once for aboveground exposure and once for groundwater exposure. Sections 8.1.1 through 8.1.5 provide further details on how each risk endpoint was determined.

8.1.1 Lifetime Excess Cancer Risk

Cancer risk was characterized using lifetime excess cancer risk estimates to represent the excess probability of developing cancer over a lifetime as a result of exposure to the constituent of interest. Lifetime excess cancer risk estimates use the lifetime average daily dose as the exposure metric. Lifetime excess cancer risk estimates are the product of the LADD for a specific receptor and the corresponding cancer slope factor, as shown in Equation 8-1. Lifetime excess cancer risk estimates are calculated separately for inhalation and ingestion exposures because they are based on separate routes of exposure and use different CSFs.

$$\text{Lifetime excess cancer risk} = \text{LADD} \times \text{CSF} \qquad (8\text{-}1)$$

where

LADD = lifetime average daily dose (mg/kg BW/d)
CSF = cancer slope factor $(\text{mg/kg BW/d})^{-1}$.

8.1.2 Total Lifetime Excess Cancer Risk

Constituent-specific individual lifetime excess cancer risks were generated for each receptor for inhalation and ingestion pathway exposures. These pathway-specific lifetime excess cancer risks were then summed to generate a total lifetime excess cancer risk for each receptor.

8.1.3 Ingestion Hazard Quotient by Pathway

Noncancer risk is characterized through the use of hazard quotients, which are generated by dividing an average daily dose for ingestion pathways by the corresponding reference dose.[1] The ingestion hazard quotient uses the ADD as the exposure metric. An HQ establishes whether a particular individual has experienced exposure that places him or her either above or below a

[1] Hazard quotients calculated for lead are based on soil screening levels (400 ppm) and action levels in drinking water (0.015 mg/L) for soil ingestion and groundwater pathways, respectively.

US EPA ARCHIVE DOCUMENT

Table 8-1. Risk Endpoints Used for Risk Categories

Risk Category	Risk Endpoints	Definition
Carcinogens	Lifetime excess cancer risk - inhalation	Lifetime excess cancer risk resulting from inhalation exposure to a single chemical
	Lifetime excess cancer risk - ingestion	Lifetime excess cancer risk resulting from ingestion exposure to a single chemical
	Total lifetime excess cancer risk	Lifetime excess cancer risk resulting from multiple pathway exposures to a single chemical
Noncarcinogens	Ingestion hazard quotient by pathway	Ingestion pathway noncancer risk characterization for a single chemical for a single ingestion pathway component (e.g., soil ingestion)
	Ingestion hazard quotient	Ingestion pathway noncancer risk characterization from exposure to all ingestion pathway components for a single chemical
	Inhalation hazard quotient	Inhalation pathway noncancer risk characterization for a single chemical

threshold of concern for a specific health effect. Therefore, unlike cancer risk estimates, HQs are not probability statements. Rather, the reference dose represents a “no-effects” level that is presumed to be without appreciable risk from chronic exposures over a lifetime. The RfD may be derived from human or animal studies and may include uncertainty factors to account for deficiencies in the available studies. Equation 8-2 shows the calculation for the ingestion hazard quotient. This calculation was completed for each pathway considered (e.g., beef ingestion). These metrics provide an estimate of the degree to which specific ingestion pathways contribute to the overall ingestion pathway noncancer risk.

$$HQ_i = \frac{ADD_i}{RfD} \tag{8-2}$$

where

ADD_i = average daily dose for each ingestion pathway (mg/kg-d)
i = pathway
RfD = reference dose (mg/kg-d).

US EPA ARCHIVE DOCUMENT

8.1.4 Ingestion Hazard Quotient

The overall hazard quotient due to ingestion (HQ_{ingest}) is calculated using the sum of the ADD estimates for each pathway considered. Essentially the same equation for the pathway-specific HQs is used for the overall HQ as shown in Equation 8-3.

$$HQ_{ingest} = \frac{ADD_{ingest}}{RfD} \tag{8-3}$$

where

$$ADD_{ingest} = \Sigma\, ADD_i.$$

The hazard quotients for each receptor that were summed to result in HQ_{ingest} are provided in Table 8-2.

Table 8-2. Ingestion HQs Summed for Total Ingestion HQ for Each Receptor

Receptor	HQ Soil	HQ Above-ground Produce	HQ Below-ground Produce	HQ Beef	HQ Milk	HQ Fish	HQ Drinking Water
Adult resident	✓						
Child resident	✓						
Farmer	✓	✓	✓	✓	✓		
Child farmer	✓	✓	✓	✓	✓		
Fisher	✓					✓	
Adult resident[a]							✓
Child resident[a]							✓

[a] Groundwater pathways were considered separately for the adult resident and the child resident because the time frame for groundwater exposure is often not consistent with that of other exposure pathways. Furthermore, aboveground receptors are randomly located and do not necessarily coincide with the location of the groundwater plume.

8.1.5 Inhalation Hazard Quotient

The inhalation hazard quotient, HQ_{inhal}, is similar to the HQ_{ingest} in that it represents a ratio of an exposure to a reference value. However, unlike the ingestion HQ, which uses the ADD as the exposure metric, the HQ_{inhal} uses an air concentration as the exposure metric. This

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concentration is compared to a reference concentration. As with the reference dose, the RfC represents a "no-effects" level that is presumed to be without appreciable risk of adverse effects from chronic exposures over a lifetime. The RfC may be derived from human or animal studies and may include uncertainty factors to account for deficiencies in the available studies. Inhalation hazard quotient is calculated as follows:

$$HQ_{inhal} = \frac{C_{air}}{RfC} \tag{8-4}$$

where

C_{air} = ambient air concentration (mg/m^3)
RfC = reference concentration (mg/m^3).

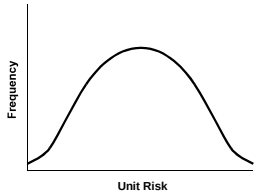
8.2 Calculating Risk-Based Waste Concentrations

The goal of this risk-based listing analysis was to calculate target waste concentrations that would be protective of human health. To obtain these target waste concentrations, scaling factors were calculated based on the risk estimates described in Section 8.1. Risk-based waste concentrations were developed through a series of steps. Figure 8-1 presents the steps used to calculate distributions of protective waste concentrations in the probabilistic analysis based on Equations 8-6 and 8-7. As depicted in the figure, the first step in the analysis was to calculate a distribution of risk, based on one unit waste concentration. This risk calculation step is described in Section 8.1. The next step was to calculate a distribution of scaling factors by dividing the target risk level by the distribution of unit risk for each of the 10,000 iterations in the Monte Carlo analysis. The distribution of scaling factors is then multiplied by the original unit waste concentration in the model resulting in a distribution of target waste concentrations for the entire WMU. The final step was to calculate a distribution of target waste concentrations in paint waste from which various percentile values were selected (e.g., 50th, 90th, and 95th). For the deterministic analysis, these calculations were performed twice, once for the central tendency analysis and once for the high-end analysis.

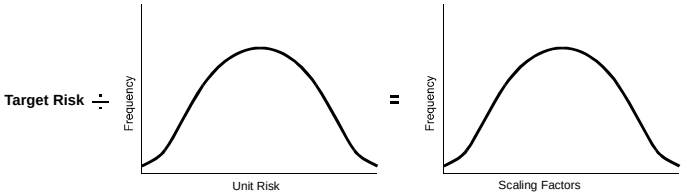
The following sections describe how scaling factors were developed and used to determine target waste and target leachate concentrations. Specifically, Section 8.2.1 provides a detailed explanation of how scaling factors were determined in order to scale the unit concentration modeled in the entire WMU to a concentration in the entire WMU that would be protective of human health. Section 8.2.2 describes how waste volume data were then used to calculate protective waste concentrations in paint waste streams based on the protective concentration in the WMU. Section 8.2.3 describes how target leachate concentrations were calculating based on the groundwater model results.

US EPA ARCHIVE DOCUMENT

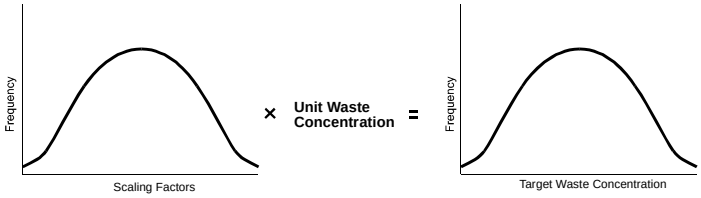
Step 1. Calculate Distribution of Unit Risk



Step 2. Divide target risk by each risk estimate in the distribution to result in a distribution of scaling factors.



Step 3. Multiply scaling factors by unit waste concentration.



Step 4. Calculate target waste concentrations in paint waste streams.

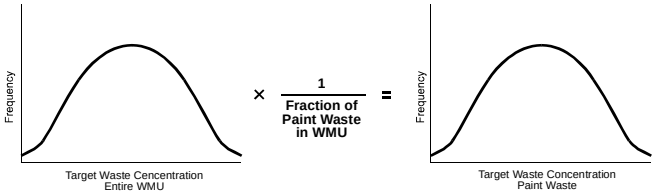


Figure 8-1. Process for calculating distributions of protective waste concentrations.

Note: The fraction of paint waste in the WMU was based on empirical data provided by paint manufacturing facilities.

US EPA ARCHIVE DOCUMENT

8.2.1 Identify Waste Concentration Scaling Factors

For this risk assessment, scaling factors were developed to scale unit (i.e., a unit of measure, this is the initial assumed waste concentration of 1 mg/kg) waste concentrations to target waste concentrations (i.e., the waste concentration that generates a risk of 1×10^{-5} or a hazard quotient of 1). This scaling approach is allowable since all of the modeling results in the analysis were linear with respect to the initial unit waste concentration of 1 mg/kg. For example, if the unit waste concentration is doubled in a WMU, then the resulting risk estimates would also double. This relationship is illustrated in Equation 8-5.

$$\frac{C_{WMU}}{UWC} = \frac{Risk_{target}}{Risk_{UWC}} \; OR \; \frac{HQ_{target}}{HQ_{UWC}} \tag{8-5}$$

where

C_{WMU} = target waste concentration in a WMU (mg/kg) or (mg/L)

UWC = unit waste concentration used in the source model (mg/kg) or (mg/L)

$Risk_{target}$ = target total lifetime cancer risk (unitless)

$Risk_{UWC}$ = total lifetime cancer risk based on unit waste concentrations (unitless)

HQ_{target} = target hazard quotient (unitless)

HQ_{UWC} = inhalation or ingestion hazard quotient based on unit waste concentrations (unitless).

Based on the relationship in Equation 8-5, target waste concentrations in the WMUs were calculated by multiplying a scaling factor by the unit waste concentration modeled in the WMUs. The following equations show how the scaling factors and target waste concentrations in the WMUs were calculated:

$$SF = \frac{Risk_{target}}{Risk_{UWC}} \; or \; \frac{HQ_{target}}{HQ_{UMC}} \tag{8-6}$$

and

$$C_{WMU} = SF \times UWC \tag{8-7}$$

where

SF = scaling factor (unitless).

US EPA ARCHIVE DOCUMENT

8.2.1.1 <u>Aboveground Waste Concentration Scaling Factors</u>. Two types of models were executed independently in order to estimate exposures to aboveground receptors for each WMU. First, the source models were used to estimate chemical emission rates from each WMU based on a unit concentration in the waste (e.g., 1 mg/kg). In addition, the fate and exposure model was used to estimate carcinogenic risks and noncancer hazards based on unit emission rates (e.g., 1 g/s-m^2). Thus, the risk and hazard estimates from the fate and transport model required an additional scaling step, such that

$$Risk_{UWC} = \frac{Q_{wmu} \times Risk_{UER}}{UER} \tag{8-8}$$

and

$$HQ_{UWC} = \frac{Q_{WMU} \times HQ_{UER}}{UER} \tag{8-9}$$

where

Q_{wmu}	=	WMU emission rate from the source model based on unit waste concentration (g/s-m^2)
$Risk_{UER}$	=	total lifetime cancer risk based on a unit emission rate (unitless)
HQ_{UER}	=	inhalation or ingestion hazard quotient based on unit emission rate (unitless)
UER	=	unit emission rate used in the fate and exposure model (g/s-m^2).

Given Equations 8-8 and 8-9, the scaling factors equation was modified for the aboveground model to take both scaling steps into account, such that

$$SF = \frac{Risk_{target} \times Unit\ Emission\ Rate}{Risk_{unit\ emission\ rate} \times Q_{wmu}} \tag{8-10}$$

and

$$SF = \frac{HQ_{target} \times Unit\ Emission\ Rate}{HQ_{UER} \times Q_{wmu}} \quad . \tag{8-11}$$

US EPA ARCHIVE DOCUMENT

8.2.1.2 <u>Groundwater Waste Concentration Scaling Factors</u>. Similar to the aboveground model, two separate models were executed independently to estimate exposures due to contaminated groundwater. First, the source model was used to estimate chemical concentrations in leachate from each WMU (i.e., landfills and surface impoundments) based on a unit waste concentration. In addition, a groundwater model was used to estimate the amount of dilution and attenuation that would occur between the release point from the WMU and the receptor well. Specifically, a dilution and attenuation factor (DAF) was calculated from the results of the groundwater model such that

$$DAF = \frac{ULC}{C_{well_{ULC}}} \tag{8-12}$$

where

ULC = unit leachate concentration used in the groundwater model (mg/L)

C_{well_ULC} = concentration predicted in the receptor well based on the unit leachate concentration (mg/L).

The DAF estimated from the groundwater model was multiplied by the leachate concentration from the source model to calculate the well concentration based on the unit concentration in the WMU. These well concentrations were used to calculate exposure and risk estimates to receptors. Thus, unlike the aboveground modeling, the risk estimates calculated for groundwater exposure were already based on the unit waste concentration in the WMU. Therefore, for the groundwater modeling, no additional adjustments were made to the equation for the scaling factor as given in Equation 8-6.

8.2.2 Calculate Protective Waste Concentrations for Paint Waste Streams

The previous sections discussed how scaling factors were used to calculate target waste concentrations in each WMU. However, the goal of the analysis was to calculate concentrations that would be protective of human health and the environment in paint waste streams. For the analysis, it was assumed that the mass of constituents in WMUs was based only on the amount of paint waste disposed of in the WMUs. In other words, all nonpaint waste in each WMU was assumed to have zero concentrations for each constituent. A dilution calculation was used to estimate target waste concentrations in paint waste streams given the target waste concentrations in the WMU, the volume of the WMU, and the volume of paint waste disposed of in the WMU. This approach is acceptable because the source models assume waste to be uniformly mixed throughout each WMU.

First, the fraction of paint waste in the WMU was calculated using the following equation:

$$f_{wmu} = \frac{V_{paint\ waste}}{V_{wmu}} \tag{8-13}$$

US EPA ARCHIVE DOCUMENT

where

f_{wmu} = fraction of paint waste in the WMU (unitless)

$V_{paint\ waste}$ = annual volume of paint waste disposed of in a WMU (m^3/yr)

V_{WMU} = annual capacity of the WMU (m^3/yr).

For landfills, the annual capacity of the WMU was simply one-thirtieth of the volume of the landfill, and the analysis assumed the entire volume was filled over the 30-year lifetime of the unit. For tanks and surface impoundments, the annual capacity was based on the flow rate of waste into the WMUs.

When calculating target waste concentrations for solid waste disposed of in a landfill, the bulk density was also taken into account. Therefore, the protective concentration in paint waste was calculated using the following equation:

$$C_w = C_{WMU} \times \frac{1}{f_{wmu}} \times \frac{BD_{WMU_{sw}}}{BD_w}. \qquad (8\text{-}14)$$

where

C_W = target waste concentration in paint waste (mg/kg)

BD_{WMU} = bulk density of mixed waste in the WMU (g/cm^3)

BD_W = bulk density of paint waste (g/cm^3).

For aqueous waste in tanks and surface impoundments, the bulk density was assumed to be 1 for waste in the WMUs as well as for paint waste. Therefore, no adjustment for bulk density was needed and the equation for the target waste concentration in aqueous paint waste was reduced to

$$C_W = C_{WMU} \times \frac{1}{f_{wmu}} \quad . \qquad (8\text{-}15)$$

Waste concentrations that produce a target risk value were calculated for each of the 10,000 iterations of the Monte Carlo simulation and for central tendency and high-end deterministic analyses. Protective waste concentrations were determined as the maximum waste concentration for each constituent that will result in risk of 1E-5 or HQ of 1 for exposed receptors at the 90th percentile level for the probabilistic analysis and at the central tendency and

US EPA ARCHIVE DOCUMENT

high-end levels for the deterministic analyses. This was done for each receptor-pathway combination (e.g., adult farmer-ingestion hazard quotient).

8.2.3 Calculate Protective Leachate Concentrations for Paint Waste Streams

The target waste concentrations calculated for the groundwater pathway described in Section 8.2 were also used to calculate protective leachate concentrations. For the models used in this analysis and with all other parameters held constant, leachate concentrations and waste concentrations are directly and linearly related to each other through the source partition model. Thus, the leachate concentration associated with the target waste concentration in paint waste was calculated as follows:

$$C_L = \frac{(C_{L_{WMU}})\,(C_W)}{C_{WMU}} \tag{8-16}$$

where

C_L = leachate concentration (mg/L) associated with the target waste concentration in paint waste

C_{L_WMU} = leachate concentration (mg/L) associated with the target waste concentration in a WMU

C_W = target waste concentration in paint waste (mg/kg)

C_{WMU} = target concentration in a WMU (mg/kg).

For the probabilistic analysis, this was done for each of the 10,000 iterations of the Monte Carlo simulation and the 90th percentile value identified for each receptor pathway combination (e.g., adult resident-ingestion). For the deterministic analysis, it was done for the central tendency and high-end calculations for each receptor-pathway combination.

8.3 Evaluate Solubility Limits

The results for all WMU types evaluated in this assessment were calculated as described in Section 8.2.1 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 phase rather than the organic phase; therefore, this is the most realistic scenario.

For landfills, some of the calculated waste concentrations based on aqueous-phase emissions may exceed limits based on the solubility of the chemical, such as the soil saturation concentration, the aqueous phase concentration in the soil pore spaces, and the aqueous phase concentration in the leachate. These solubility limits represent the maximum possible aqueous-phase concentration in soil; once this is exceeded, free (organic-phase) product may occur in the soil. These limitations on calculations are incorporated into the landfill partition model. If any of

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these limitations are exceeded, the model will report the corresponding error. For this reason, the landfill partition model was used to test each protective waste concentration to determine if solubility limits were violated. For this test, the landfill partition model input parameter values were set to central tendency values. Target waste concentrations exceeding solubility limits are noted in the results tables in Appendix A by an "S".

For tanks and surface impoundments, some of the calculated waste concentrations based on aqueous-phase emissions may exceed solubility limits. Literature value solubility limits were compared to target waste concentrations to determine when they were exceeded. Target waste concentrations exceeding solubility limits are noted in the results tables in Appendix A by an "S".

Note that solubility and physical limits are somewhat site- and waste-specific; actual limits will vary with conditions. Therefore, calculated waste concentrations may exceed limits in some situations but not in others, which introduces some degree of uncertainty in the analysis.

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9.0 Ecological Risk Assessment of Paint Wastes

Paint waste management activities can impact not only the health of individuals living near a WMU, they can also have adverse effects on nonhuman organisms and natural systems. For example, wildlife can come into contact with constituents released from WMUs by swimming or living in contaminated waters or by drinking or catching prey such as fish from contaminated waters. Plants that grow in soils containing constituents of concern can take them into their leaves and stems through root uptake, which can have detrimental effects on the plants as well as on the animals that eat the plants. Microorganisms and small invertebrates that live in close contact with the soil (e.g., worms) can accumulate COCs through contact with contaminated soil. Therefore, it is important to evaluate risks posed to ecological receptors as well as those posed to humans. Protection of human health does not necessarily protect ecological receptors. Some chemicals are more toxic to nonhumans; wildlife species generally have higher metabolic rates than humans and, therefore, eat, drink, and breathe proportionately more contaminants than humans; and nonhuman organisms live in closer association with their immediate environment and often cannot avoid contamination or replace destroyed food sources as humans can (Suter, 1993).

This section describes the screening ecological risk assessment (SERA) developed to evaluate the potential ecological risks associated with the management of paint manufacturing wastes in landfills, surface impoundments, and treatment tanks. This section complements the human health risk analysis described in previous sections; therefore, detailed information on management practices and fate and transport modeling of paint waste constituents has not been included. The SERA compares modeled media concentrations to media concentrations developed to be protective of ecological receptors. The protective media concentrations for ecological receptors in soil, surface water, or sediment are referred to as chemical stressor concentration limits (CSCLs). The CSCLs are medium- and receptor-specific and represent environmental concentrations below which adverse effects are unlikely for both direct and indirect exposure. Adverse effects are effects such as a decrease in the rate of reproductive or developmental success or a decrease in yield for plants. The particular effects assessed for this SERA are discussed in detail in Section 9.2.1.

The ratio of a modeled medium concentration and a CSCL is defined as the hazard quotient (HQ) and provides the risk metric for the paint wastes SERA. An exceedance of the target HQ of 1 suggests that the modeled waste management practices may not be protective of ecological receptors. Because the CSCLs are based on de minimis ecological effects, HQs of 1 and lower are presumed to indicate that the potential for adverse effects is negligible for the receptor for which the CSCL was derived. In cases where an HQ exceeded the target of 1, a Tier 2 analysis was performed to determine the waste concentration at which the maximum HQ was 1 (i.e., all HQs would meet the target).

The remainder of this section is organized as follows. Section 9.1 describes the overall technical approach for the SERA. Sections 9.2 through 9.4 describe the three basic phases of the risk assessment process: problem formulation, analysis, and risk characterization. Appendix R presents the data inputs for the analysis including toxicological benchmarks, bioaccumulation factors (BAFs), exposure factors, dietary fractions, and the CSCLs by environmental medium (e.g., soil, water, sediment), receptor, and constituent. The ecological HQs are discussed in Section 2.0 and presented in Appendix B.

9.1 Technical Approach

The technical approach for the SERA follows EPA's *Guidelines for Ecological Risk Assessment* (U.S. EPA, 1998). These guidelines describe the three basic phases that frame the ecological risk assessment process: problem formulation, analysis, and risk characterization. The framework advocated in the guidelines has been adopted by EPA for all ecological risk assessment work conducted in support of regulatory determinations. Briefly, these phases can be summarized as follows:

- **Problem Formulation Phase -** Defines the problem by answering these questions: (1) What are the constituents of concern? (2) Once released, what is the environmental behavior of the constituents (e.g., persistence, bioaccumulation, speciation)? (3) Given the source characterization (e.g., size, geographic location), what ecosystems and ecological receptors are potentially at risk? (4) What adverse ecological effects are possible following exposure? The three key activities in this phase are: selection of assessment endpoints, development of a conceptual model, and preparation of an analysis plan.

- **Analysis Phase -** Provides estimates of the constituent concentrations in the environment to which ecological receptors are exposed (i.e., exposure profile) and develops CSCLs from data on adverse ecological effects on various receptors (e.g., developmental toxicity in amphibians).

- **Risk Characterization Phase -** Compares the modeled media concentrations to the CSCLs to estimate the potential for adverse ecological effects (i.e., the HQ approach). Includes a risk description of the assessment (e.g., limitations) and discusses the likely ecological significance of HQ exceedances.

9.2 Problem Formulation

As described in EPA's *Guidelines for Ecological Risk Assessment* (U.S. EPA, 1998), a successful problem formulation "depends upon the quality of three products: (1) assessment endpoints that adequately reflect management goals and the ecosystem they represent, (2) conceptual models that describe key relationships between a stressor and assessment endpoint or among several stressors and assessment endpoints, and (3) an analysis plan." The analysis plan outlines the technical approach for the SERA. In general, the analysis plan called for the selection of appropriate endpoints and receptors, the derivation of CSCLs based on best available toxicological data, and the comparison of CSCLs with modeled media concentrations. The

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analysis plan was implemented as proposed and as described in the remainder of this section. The two remaining components of the problem formulation phase, development of the assessment endpoints and the conceptual model, are described in the following subsections.

9.2.1 Selection of Assessment Endpoints

Perhaps the most important step in the problem formulation phase is the selection of assessment endpoints, defined as "explicit expressions of the actual environmental value that is to be protected" (U.S. EPA, 1998). The assessment endpoints serve as a critical link between the ecological risk assessment and the management goal, which, for the paint wastes SERA, may be stated as follows: to determine whether paint waste management practices are likely to cause adverse effects to the environment. The assessment endpoints must be ecologically relevant to the ecosystem(s) they represent and susceptible to the stressors of concern, in this case the constituents in paint manufacturing wastes. Candidates for assessment endpoints often include threatened or endangered species, critical habitats and ecosystems, commercially or recreationally important species, functional attributes that support food sources or flood control, or aesthetic values, such as the existence of charismatic species such as eagles (U.S. EPA, 1998). Regardless of the assessment endpoint(s) chosen for this analysis, it should be emphasized that each assessment endpoint is defined by two key elements: (1) a valued ecological entity (e.g., a species) and (2) an attribute of that entity that is important to protect (e.g., reproductive fitness).

For the paint wastes SERA, the assessment endpoints were chosen to (1) maintain the structure and function of soil, aquatic, and benthic (sediment) communities; (2) maintain viable mammalian, avian, and herpetofaunal wildlife populations; and (3) maintain primary producers (e.g., plants) in terrestrial and aquatic systems. These endpoints are consistent with the assessment endpoints selected for the proposed Hazardous Waste Identification Rule (HWIR) and other listing determination risk assessments. Because the paint wastes listing risk assessment is a national scale analysis, ecological exposures are presumed to occur anywhere in the contiguous United States. Consequently, a suite of assessment endpoints was chosen based on

- Relevance to habitats and land use surrounding waste management units
- Representation of various trophic levels and feeding strategies within terrestrial and aquatic food chains
- Susceptibility to chemicals based on exposure and/or toxicological sensitivity.

In Table 9-1, the assessment endpoints, or values to be protected, are defined in terms of: (1) the significance of an ecological entity (i.e., the reason we would want to protect it), (2) the ecological receptor(s) representing that entity, (3) the characteristic about the entity that is important to protect, and (4) the measures of effect used to quantify impact. The intent of including multiple receptors is that, by protecting producers (i.e., plants) and consumers (i.e., predators) at different trophic levels, as well as certain structural components (e.g., benthic community), a degree of protection may be inferred for the ecosystem as a whole.

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Table 9-1. Assessment Endpoints and Measures of Effects

Assessment Endpoint	Examples of Ecological Significance	Representative Receptors	Characteristics	Measure of Effect
Viable mammalian wildlife populations	■ Multiple trophic levels represented ■ Represent species with large foraging ranges ■ Represent species with longer life spans ■ Variety of dietary exposures represented	Deer mouse, meadow vole, red fox	Reproductive and developmental success	Chronic or subchronic MATC for developmental and reproductive effects
Viable avian wildlife populations		Red-tailed hawk, northern bobwhite	Reproductive and developmental success	Chronic or subchronic MATC for developmental and reproductive effects
Protection of amphibian and reptile populations ("herps") against acute effects	■ Species represent unique habitat niches ■ Many species are particularly sensitive to exposure	Frog, newt, snake, turtle	Lethality and percent deformity	Acute LC_{50}s for developmental effects resulting from early life stage exposures
Sustainable soil community structure and function	■ Represent base food web in terrestrial systems ■ Habitat vital to decomposers and soil aerators ■ Crucial to nutrient cycling	Nematodes, soilmites, springtails, annelids arthropods	Growth, survival, and reproductive success	95% of species below no effects concentration at 50th percentile confidence interval
Maintain terrestrial primary producers (plant community)	■ Primary producers ■ Act as food base for herbivores ■ Constitute essential habitat for virtually all receptor groups (e.g., nests)	Soy beans, alfalfa, rye grass	Growth, yield, germination	10th percentile from LOEC data distribution
Sustainable aquatic community structure and function	■ Important food source for animals that live in waterbody margins ■ Diverse aquatic life important to maintain biotic integrity	Fish (salmonids), aquatic invertebrates (daphnids)	Growth, survival, reproductive success	National Ambient Water Quality Criteria for aquatic life (95% species protection)
Sustainable benthic community structure and function	■ Provide habitat for reproductive life stages (e.g., eggs, larval forms) ■ Act to process nutrients and decompose organic matter	Protozoa, flat worms, ostracods	Growth, survival, reproductive success	10th percentile from LOEC data distribution
Maintain primary aquatic producers (algal and plant community)	■ Primary producers ■ Base food source in the aquatic system	Algae and vascular aquatic plants	Growth, mortality, biomass, root length	EC_{20} for algae; lowest LOEC for aquatic plants

EC_{20} = Effective concentration for 20% of the population.
LC_{50} = Lethal concentration for 50% of the population.
LOEC = Lowest observed effects concentration.
MATC = Maximum acceptable toxicant concentration.

In addition to evaluating representative species populations and communities, a screening assessment can consider the potential effects on endpoints with site-specific or localized significance, such as managed lands (e.g., National Wildlife Refuges), critical habitats (e.g., wetlands), and threatened and endangered species. The locations of paint waste management units are not specified for this assessment; therefore, the occurrence of such receptors at paint WMUs cannot be determined.

9.2.2 Conceptual Model

Subsequent to assessment endpoint selection, the conceptual model for the analysis was developed. The conceptual model establishes the exposure scenarios, pathways, and receptors of concern. For the paint wastes SERA, the conceptual model addresses three waste management scenarios: landfills, surface impoundments, and tanks. Two waste streams, emission control dust and combined solids, were modeled for landfills, and aqueous wastes were modeled for surface impoundments and for treatment tanks. Release mechanisms modeled in the assessment include aerial deposition of particulates and volatiles from WMUs to terrestrial and aquatic habitats and erosion runoff from terrestrial habitats into aquatic habitats. The ecological assessment was based on the source characterization described in Section 4.0, and the media concentrations were modeled using the same models and methods as those used in the human health assessment, as described in Section 5.0.[1]

To evaluate ecological exposures, a two-tiered assessment was used. Initially, ecological exposures were assessed by assuming a 750,000-ppm paint waste concentration for all constituents. EPA selected this concentration because 750,000 ppm is thought to be substantially higher than the actual concentration in paint waste of any constituent evaluated in this assessment. A second tier assessment was performed for any constituent that did not screen out of the analysis at the 750,000-ppm level. In Tier 2, constituent-specific waste concentrations were calculated. These Tier 2 waste concentrations are the concentrations that would result in a maximum HQ of 1 and would, therefore, be considered protective of ecological receptors.

The SERA conceptual model considers the potential for adverse effects to a suite of ecological receptors that may occur in terrestrial, freshwater, and wetland habitats including, for example, mammals, birds, and soil and benthic fauna. The habitats and receptors considered in this study are consistent with the national assessment strategy developed to support HWIR. Because the HWIR risk assessment framework included intensive literature searches and data collection efforts, and because it was intended to support national studies of waste management practices, this assessment has adopted the HWIR framework as the basis for selecting receptors and habitats. The process of selecting receptors, habitats, and exposure pathways for the SERA conceptual model is discussed in the following sections.

9.2.2.1 <u>Selection of Representative Receptors</u>. Once the assessment endpoints are established, receptors are selected based on the environmental conditions, or habitats, at the site

[1] The same constituents were assessed for the human health and the ecological assessments, with the exception of nickel oxide. Ecotoxicological data for nickel oxide were not identified for any receptors; therefore, it could not be assessed.

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to be evaluated. For example, if streams and forests are located near the site, then receptors that use these habitats are selected. Because the paint wastes SERA is not a site-based analysis, specific habitat types that are potentially affected cannot be determined. Instead, it is assumed that paint waste WMU sites and surrounding lands support terrestrial and aquatic receptors at all trophic levels, including top predators (apex species) and representing all significant feeding strategies. By evaluating receptors that are typical of different types of habitats, the paints assessment is relevant to terrestrial, wetland, and aquatic systems. Selection of receptors is also constrained by available toxicological and exposure factor data. Extensive literature and database searches were performed for toxicological benchmark data and for exposure factor data (e.g., body weights, ingestion rates, and dietary composition) for over 60 ecological receptors under the HWIR ecological risk assessment. Because this compilation of data reflects currently available data for receptors that are relevant to the paint wastes SERA, receptors were selected from the HWIR receptor databases. Two types of receptors were needed to address the assessment endpoints selected for the SERA: receptor populations, represented by individual species such as the raccoon or the white-tailed deer, and receptor communities consisting of several taxa making up a particular system, such as the soil community, which includes several different species of invertebrate organisms. Receptor communities include the following:

- Aquatic community—freshwater aquatic invertebrates and fish
- Sediment community—benthic (sediment-dwelling) invertebrates
- Soil community—soil-dwelling invertebrates
- Algae and aquatic plants—floating and sediment-rooted primary producers
- Terrestrial plants—vascular plants rooted in soil (e.g., trees or crops)
- Amphibians—vertebrates generally characterized by an initial stage of life as an aquatic larva and metamorphosis into a lunged adult (e.g., frogs, salamanders, and newts).

Receptor populations assessed in this SERA include 45 species of mammals and birds. These receptor species are listed in Table 9-2, along with information about each species' eating strategy and trophic level.

9.2.2.2 <u>Identification of Exposure Pathways</u>. Ecological exposure pathways for this SERA were identified based on the selected management scenarios for active landfills, surface impoundments, and treatment tanks and likely routes of exposure for receptors assigned to simple food webs. Airborne chemical constituents may be deposited onto adjacent soils, plants, or surface waters. In addition, constituents may be carried into nearby surface waters and sediments through erosion and runoff from contaminated soil. As shown in Figure 9-1, receptors may be exposed to contaminated media and/or prey and plants in both terrestrial and aquatic systems. Uptake of COCs can occur as direct biological uptake through contact with

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Table 9-2. Receptor Species for Paint Wastes SERA

Species	Scientific Name	Dietary Composition[a]	Functional Group	Trophic Level[b]	References
American kestrel	*Falco sparverius*	Terrestrial	Carnivore	T2	Lane and Fischer, 1997 Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
American robin	*Turdus migratorius*	Terrestrial	Omnivore	T2	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
American woodcock	*Scolopax minor*	Terrestrial	Omnivore	T2	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Bald eagle	*Haliaeetus leucocephalus*	Terrestrial Aquatic	Carnivore	T3	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Beaver	*Castor canadensis*	Terrestrial Aquatic	Herbivore	T1	Jenkins and Busher, 1979 Stokes and Stokes, 1986 Whitaker, 1997
Belted kingfisher	*Ceryle alcyon*	Aquatic	Omnivore	T2	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Black bear	*Ursus americanus*	Terrestrial	Omnivore	T3	Schaefer and Sargent, 1990 Stokes and Stokes, 1986 Whitaker, 1997
Black-tailed jackrabbit	*Lepus californicus*	Terrestrial	Herbivore	T1	MacMahon, 1985 Sample et al., 1997 Whitaker, 1997
Burrowing owl	*Speotyto cunicularia*	Terrestrial	Omnivore	T2	Sample et al., 1997 Stokes and Stokes, 1996 Terres, 1980
Canada goose	*Branta canadensis*	Terrestrial Aquatic	Herbivore	T1	Niering, 1985 Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Cerulean warbler	*Dendroica cerulea*	Terrestrial	Insectivore	T2	Evans and Fischer, 1997 Stokes and Stokes, 1996 Terres, 1980
Cooper's hawk	*Accipiter cooperi*	Terrestrial	Carnivore	T3	Sample et al., 1997 Stokes and Stokes, 1996 Terres, 1980

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US EPA ARCHIVE DOCUMENT

Table 9-2. (continued)

Species	Scientific Name	Dietary Composition[a]	Functional Group	Trophic Level[b]	References
Coyote	*Canis latrans*	Terrestrial	Omnivore	T3	Bekoff, 1977 Sample et al., 1997 Stokes and Stokes, 1986 Whitaker, 1997
Deer mouse	*Peromyscus maniculatus*	Terrestrial	Omnivore	T2	Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Eastern cottontail rabbit	*Sylvilagus floridanus*	Terrestrial	Herbivore	T1	Chapman et al., 1980 Stokes and Stokes, 1986 Whitaker, 1997
Great blue heron	*Ardea herodias*	Aquatic	Omnivore	T2	Niering, 1985 Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Great Basin pocket mouse	*Perognathus parvus*	Terrestrial	Omnivore	T2	Sample et al., 1997 Whitaker, 1997
Green heron	*Butorides virescens*	Aquatic	Omnivore	T2	Niering, 1985 Sample et al., 1997 Stokes and Stokes, 1996 Terres, 1980
Herring gull	*Larus argentatus*	Terrestrial Aquatic	Omnivore	T2	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Kit fox	*Vulpes velox*	Terrestrial	Carnivore	T3	MacMahon, 1985 McGrew, 1979 Sample et al., 1997 Whitaker, 1997
Least weasel	*Mustela nivalis*	Terrestrial	Carnivore	T2	Stokes and Stokes, 1986 Whitaker, 1997
Lesser scaup	*Aythya affinis*	Terrestrial Aquatic	Omnivore	T2	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Little brown bat	*Myotis lucifugus*	Terrestrial	Insectivore	T2	Sample et al., 1997 Whitaker, 1997
Loggerhead shrike	*Lanius ludovicianus*	Terrestrial	Carnivore	T2	Hall et al., 1997 Stokes and Stokes, 1996 Terres, 1980

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US EPA ARCHIVE DOCUMENT

Table 9-2. (continued)

Species	Scientific Name	Dietary Composition[a]	Functional Group	Trophic Level[b]	References
Long-tailed weasel	*Mustela frenata*	Terrestrial	Carnivore	T2	Sample et al., 1997 Stokes and Stokes, 1996 Sutton and Sutton, 1985
Mallard	*Anas platyrhynchos*	Terrestrial Aquatic	Omnivore	T2	Niering, 1985 Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Marsh wren	*Cistothorus palustris*	Terrestrial	Carnivore	T2	Niering, 1985 Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Meadow vole	*Microtus pennsylvanicus*	Terrestrial	Herbivore	T1	Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Mink	*Mustela vison*	Terrestrial Aquatic	Carnivore	T2	Niering, 1985 Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Mule deer	*Odocoileus hemionus*	Terrestrial	Herbivore	T1	Anderson and Wallmo, 1984 Sample et al., 1997 Whitaker, 1997 Whitney, 1985
Muskrat	*Ondatra zibethicus*	Terrestrial Aquatic	Herbivore	T1	Niering, 1985 Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997 Willner et al., 1980
Northern bobwhite	*Colinus virginianus*	Terrestrial	Omnivore	T2	Stokes and Stokes, 1986 Terres, 1980 U.S. EPA, 1993a
Osprey	*Pandion haliaetus*	Aquatic	Carnivore	T3	Stokes and Stokes, 1986 Terres, 1980 U.S. EPA, 1993a
Pine vole	*Microtus pinetorum*	Terrestrial	Herbivore	T1	Sample et al., 1997 Whitaker, 1997
Prairie vole	*Microtus ochrogaster*	Terrestrial	Herbivore	T1	U.S. EPA, 1993a Whitaker, 1997

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Table 9-2. (continued)

Species	Scientific Name	Dietary Composition[a]	Functional Group	Trophic Level[b]	References
Raccoon	*Procyon lotor*	Terrestrial Aquatic	Omnivore	T2	Lotze and Andersen, 1979 Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Red fox	*Vulpes vulpes*	Terrestrial	Omnivore	T3	Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Red-tailed hawk	*Buteo jamaicensis*	Terrestrial	Carnivore	T3	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
River otter	*Lutra canadensis*	Terrestrial Aquatic	Carnivore	T2	Niering, 1985 Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Short-tailed shrew	*Blarina brevicauda*	Terrestrial	Omnivore	T2	Stokes and Stokes, 1986 U.S. EPA, 1993a Whitaker, 1997
Short-tailed weasel	*Mustela erminea*	Terrestrial	Carnivore	T2	King, 1983 Sample et al., 1997 Whitaker, 1997
Spotted sandpiper	*Actitis macularia*	Terrestrial	Carnivore	T2	Stokes and Stokes, 1996 Terres, 1980 U.S. EPA, 1993a
Tree swallow	*Tachycineta bicolor*	Terrestrial	Omnivore	T2	Sample et al., 1997 Stokes and Stokes, 1996 Terres, 1980
Western meadowlark	*Sturnella neglecta*	Terrestrial	Omnivore	T2	Sample et al., 1997 Stokes and Stokes, 1996 Terres, 1980
White-tailed deer	*Odocoileus virginianus*	Terrestrial	Herbivore	T1	Smith, 1991 Stokes and Stokes, 1986 Whitaker, 1997

[a] Dietary composition indicates whether the receptor's diet was assumed to come predominantly from a terrestrial or aquatic source for this analysis.

[b] Trophic level: T1 = prey, not a predator; T2 = both a predator and prey; T3 = a top predator, not prey.

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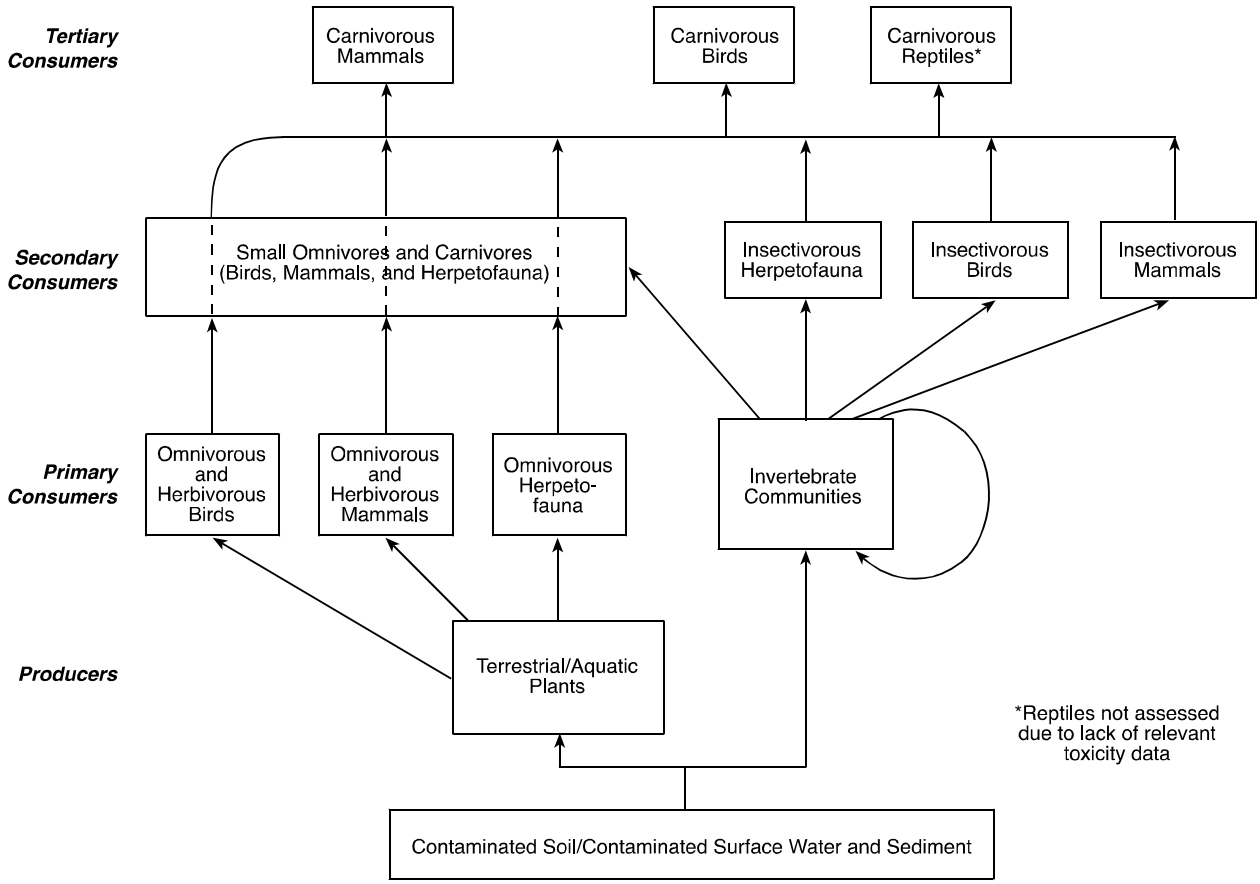

Figure 9-1. General food web model for aquatic and terrestrial systems.

contaminated media and as biological uptake through the food chain from ingesting contaminated food and water. The exposure pathways represented in this analysis are

- Root uptake of constituents in soils or sediment by plants
- Direct contact with contaminated surface water by aquatic animals (e.g., fish, amphibians)
- Direct contact with contaminated sediments by benthic invertebrates
- Direct contact with contaminated soils by soil invertebrates
- Ingestion of constituents in surface water, soils, sediments, plants, and prey by terrestrial animals.

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Exposure routes that will not be addressed in the screening assessment are

- Dermal absorption of constituents in surface water or soil by terrestrial wildlife
- Inhalation of volatile constituents in air.

Dermal absorption of constituents is considered to be an insignificant exposure pathway for potentially exposed wildlife receptors. The reason dermal absorption is considered insignificant is that wildlife receptors have dense undercoats or down that effectively prevents chemicals from reaching the skin and significantly reduces the total surface area of exposed skin (Peterle, 1991; U.S. ACE, 1996). In addition, previous studies indicate that exposures due to dermal absorption are insignificant compared to ingestion for terrestrial receptors (Peterle, 1991). Dermal absorption, therefore, is not assessed.

Inhalation of volatile compounds is not assessed for wildlife receptors either. It is not assessed because concentrations of volatile chemicals released from WMUs or surrounding soil to the ambient air are drastically reduced, even near the soil surface (U.S. ACE, 1996), and significant concentrations of volatile organic compounds (VOCs) would be required to induce noncarcinogenic effects in wildlife based on inhalation toxicity data for laboratory rats and mice (U.S. ACE, 1996).

9.3 Analysis Phase

This section describes the methodology used to estimate CSCLs for receptors evaluated in this screening analysis. The fate and transport model used to estimate constituent concentrations in plants, soil, sediment, and surface water (i.e., the exposure profile) is discussed in Section 5.0 in this report.

The methodology used to develop CSCLs for this SERA is explained in detail in Sections 9.3.1 and 9.3.2. The CSCLs for all ecological receptors addressed in the SERA are presented in Appendix R. It is useful to think about the CSCLs developed in terms of either population-type concentration limits or community-type concentration limits (see text box). The population-type CSCLs reflect exposures via ingestion of contaminated media and food items (e.g., plants, prey). The community-type CSCLs reflect direct exposures to contaminated media. It should be noted that the CSCLs for receptor

Population-type CSCLs: Receptor populations are terrestrial bird and mammal species, such as the red tailed hawk or the raccoon, exposed to COCs through the ingestion pathway. Available toxicological benchmarks for these receptors consist of ingestion doses reported in the literature. However, this SERA evaluated risks based on media concentrations; therefore, protective media concentrations for these receptors had to be calculated based on the protective dose (benchmark), what the receptor eats, and to what degree each COC is transferred from contaminated media into the various food items (e.g., plants or small prey species).

Community-type CSCLs: Receptor communities comprise groups of species, such as benthic invertebrates or terrestrial plants, that are exposed through direct contact with contaminated media. Available toxicological benchmarks for direct exposure for receptor communities consist of constituent concentrations in various media. These benchmarks can be directly used as CSCLs. For example, the water CSCLs for the aquatic community consist of ambient water quality criteria, which are expressed as milligrams of constituent per liter of water (mg/L).

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communities are not truly community-level concentration limits in the sense that they do not consider predator-prey interactions. Rather, they are based on the theory that protection of 95 percent of the species in the community will provide a sufficient level of protection for the community (see, for example, Stephan et al., 1985, for additional detail).

9.3.1 CSCL Development for Receptor Populations

The calculation of ecological screening factors for receptor populations is based on the implicit assumption that each receptor species forages only within the contaminated area, regardless of the size of its home range. For smaller animals, this assumption has little impact on the estimates of exposure. However, for larger animals with more extensive foraging areas, this assumption may overestimate exposure if the animal's foraging patterns tend to be evenly spread over the home range. Thus, it is important to recognize both the explicit and implicit sources of protection in this methodology.

For amphibian populations, a CSCL for water was derived as the geometric mean of acute studies that include the following information:

- Test organism
- Toxicological endpoint
- Exposure duration
- Life stage at which exposure occurred (e.g., embryo, tadpole).

Appropriate toxicity data for amphibians included reproductive effects, developmental effects, or lethality from studies conducted for an exposure duration of less than 8 days. Limiting the study duration to short exposures allows the use of a larger data set in deriving the CSCLs. However, it is important to point out that this CSCL should be construed as only "protective" of gross effects to amphibian populations (e.g., lethality to 50 percent of the population). Therefore, results must be interpreted carefully.

The ecotoxicological benchmarks used to calculate CSCLs are presented in Appendix R along with information about the studies from which the benchmarks were derived. The remainder of this section outlines the basic technical approach used to convert avian or mammalian benchmarks (in daily doses) to CSCLs (in units of concentration) that will be compared with the modeled media concentrations that result from paint waste management practices.

For populations of mammals and birds, the overall approach used to establish ecotoxicological benchmarks is similar to the methods used to establish RfDs for humans as described in IRIS. Each method uses a hierarchy for the selection of toxicity data and extrapolates from a test species to the species of interest. However, there are fundamental differences in the goals of noncancer risk assessments for humans and ecological receptors. Risk assessments of humans seek to protect the individual while risk assessments of ecological receptors typically seek to protect populations or communities of important species. The procedures used to develop benchmarks (i.e., RfDs) for the protection of human health are very sensitive by design and go beyond the need to sustain the reproductive fitness in a local

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population (U.S. EPA, 1992). Consequently, benchmarks for mammals and birds were established using three key guidelines:

- First, because population viability was selected as an assessment endpoint, the benchmarks were developed from measures of reproductive/developmental success or, if unavailable, other effects that could conceivably impair population dynamics.

- Second, the population-level benchmark was preferred over population-inference benchmarks. Population-level benchmarks are based on studies of effects on an entire population (i.e., many interacting individuals) while population-inference benchmarks are based on studies of individuals with protection of the population being inferred from protection of the individual (i.e., MATC for individual organisms on reproductive endpoints). Although relatively few population-level benchmarks have been developed to date, these benchmarks are considered to be more rigorous than the point estimates gleaned from toxicity studies.

- Third, uncertainty factors (UFs) were generally not applied to address interindividual variability. For example, a UF of 10 was not applied to subchronic studies because reproductive and developmental toxicity studies are frequently short-term.

Once the appropriate ecotoxicological studies were identified for mammals and/or birds, the CSCLs were calculated for each medium of interest using a three-step process:

1. Scale benchmark from study species to receptor species.
2. Identify uptake/accumulation factors.
3. Calculate protective concentration for receptor (i.e., CSCL).

Step 1: Scale Benchmark from Study Species to Receptor Species

The benchmark chosen to represent the mammalian or avian receptor was extrapolated from the study species to the receptor species ($MATC_{RS}$) within the same general group (mammals or birds) using a cross-species scaling equation (Sample et al., 1996). For population-inference benchmarks for mammals, the extrapolation is performed using Equation 9-1:

$$MATC_{RS} = MATC_{SS} \times \left(\frac{bw_{SS}}{bw_{RS}}\right)^{1/4} \qquad (9\text{-}1)$$

where

$MATC_{SS}$ = MATC for the study species
bw_{SS} = body weight of the study species
bw_{RS} = body weight of the receptor species.

US EPA ARCHIVE DOCUMENT

This is the default methodology EPA proposed for adjusting animal data to an equivalent human dose for carcinogenicity assessments and reportable quantity documents. The method applies a scaling factor of 0.75 to the receptor species body weight.

For avian species, new research suggests that the cross-species scaling equation used for mammals is not appropriate (Mineau et al., 1996). Mineau et al. (1996) used a database that characterized acute toxicity of pesticides to avian receptors of various body weights. The results of the regression analysis revealed that applying mammalian scaling equations may not predict sufficiently protective doses for avian species. Mineau et al. (1996) suggested that a scaling factor of 1 applied to the receptor species body weight provides a better dose estimate for birds, as shown in Equation 9-2. This recommendation was adopted for avian receptors in this assessment.

$$MATC_{RS} = MATC_{SS} \times \left(\frac{bw_{SS}}{bw_{RS}}\right)^{0} \qquad (9\text{-}2)$$

Scaled benchmarks used to calculate CSCLs for mammals and birds are presented in Appendix R.

Step 2: Identify Uptake/Accumulation Factors

As suggested in Figure 9-1, movement of contaminants through the food web is an important exposure vector for mammals and birds. Consequently, estimates of chemical accumulation in the tissues of plants and prey items are required. For receptors likely to rely on aquatic systems for food (e.g., kingfisher), bioaccumulation factors (BAFs) and/or bioconcentration factors (BCFs) are required for aquatic biota such as fish, benthos, and aquatic plants. These data are identified in the open literature or, as for organic constituents in fish, they were estimated as described in Section 5.3.2.

For receptors found primarily in terrestrial systems, biological uptake factors reporting the relationship between tissue concentrations and soil concentrations are required for terrestrial plants, soil invertebrates, earthworms, and vertebrates. For the most part, these data were identified in the literature using the same sources as those used to develop BAFs for the HWIR analysis. BAFs for earthworms and small mammals were estimated using methods presented in Sample et al. (1998a, 1998b). Root uptake factors for organic constituents were calculated based on methods in EPA's *Parameter Guidance Document* (U.S. EPA, 1997).

In general, BAFs were identified in the open literature and EPA references or were calculated based on the relationship between log K_{ow} and accumulation in lipid tissue. To ensure that the SERA is protective, a default value of 1 was assigned for each uptake/accumulation factor that could not be derived through estimation methods or identified in the literature. The BAFs used to calculate CSCLs for mammals and birds are presented in Appendix R.

US EPA ARCHIVE DOCUMENT

Step 3: Calculate Protective Concentration for Receptor

Based on the $MATC_{RS}$, the CSCL for a receptor that relies on aquatic biota as the primary food source was calculated as a function of the receptor's body weight, the receptor's ingestion rate for food and water, and the bioaccumulation potential of the constituent, as shown in Equation 9-3:

$$CSCL_{water} = \frac{MATC_{RS} \text{ x } bw}{(I_{food} \sum BAF_j \text{ x } F_j \text{ x } AB_j) + (I_{water})} \tag{9-3}$$

where

bw	=	body weight (kg)
I_{food}	=	total daily intake of aquatic biota (kg WW/d)
BAF_j	=	bioaccumulation factor for food item j (L/kg WW))
F_j	=	fraction of diet consisting of food item j (unitless)
AB_j	=	absorption of chemical in the gut from food item j (assumed = 1)
I_{water}	=	total daily water intake (L/d).

For terrestrial systems, Equation 9-3 was simply modified to account for soil intake:

$$CSCL_{soil} = \frac{MATC_{RS} \times bw}{(I_{food} \sum BCF_j \text{ x } F_j \text{ x } AB_j) + (I_{soil})} \tag{9-4}$$

where

bw	=	body weight (kg)
I_{food}	=	total daily food intake of terrestrial biota (kg/d)
BCF_j	=	bioconcentration factor for food item j (assumed unitless)
F_j	=	fraction of diet consisting of food item j (unitless)
AB_j	=	absorption of chemical in the gut from food item j (assumed = 1)
I_{soil}	=	total daily soil intake (kg/d).

For some receptors, sediment ingestion may be a significant exposure pathway. Therefore, sediment ingestion CSCLs were calculated for receptors that obtain food from aquatic habitats as shown in Equation 9-5:

$$CSCL_{SedIng} = \frac{MATC_{RS} \times bw}{I_{SedIng}} \tag{9-5}$$

where I_{sedIng} is the total daily sediment ingestion (kg/d).

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Information sources to develop the input values for body weight (bw), ingestion rates (I_{xx}), and dietary fractions (F_j) were taken from the extensive HWIR databases. The HWIR databases were developed using EPA's *Wildlife Exposure Factors Handbook* (U.S. EPA, 1993a) and augmented by substantial literature review and synthesis of a variety of information sources. Appendix R presents the exposure factors and their respective sources for each species.

For dietary fractions (Fj), an extensive database of diet items and dietary fractions was used to construct diets for each receptor. The database was compiled from a wide range of sources on feeding habits, including primary sources for wildlife exposure factors (U.S. EPA, 1993a; Sample et al., 1997), species monographs, zoological profiles, and field guides. Information from species monographs and zoological profiles was emphasized over field guides. In general, dietary information from the various sources was quite consistent. The dietary fraction data consist of percentages that each potential diet item accounts for in the diet. For example, dietary fractions for the Eastern box turtle are as follows:

Diet Item	% of Diet
Worms	3 to 60
Forage	13 to 39
Fruits	5 to 33
Other invertebrates	8 to 22
Small herpetofauna	0 to 10
Small mammals	0 to 10

The data are reported in the database as potential minimum and maximum proportions of each species' diet. Each species' dietary composition was established by selecting items based on their relative biological uptake potential as well as their potential dietary proportion. This was done by multiplying a receptor's maximum dietary fraction for each potential diet item by the corresponding BAF for that item. (BAFs vary by constituent, so this procedure was executed for each constituent.) The receptor's potential diet items were then ranked based on the product of the dietary fraction and the BAF. The top-ranked diet item was selected first, and its dietary fraction was entered for the receptor's dietary composition. Then the second-ranked item was selected, and so on, until a complete diet (i.e., dietary fractions equaled 1) was compiled. In this manner, exposure was maximized based on dietary intake rate as well as constituent uptake rate. Amphibians are included in the dietary composition for both aquatic and terrestrial diets, and they were therefore included in both Equations 9-3 and 9-4. However, the water CSCL equation is based on the wet weight of food items, and the soil CSCL equation is based on the dry weight of food items. The bioaccumulation factors reported for amphibians are based on dry weight measurements; in order to use amphibian BAFs in the water CSCL equation, they were converted to wet weight assuming 64 percent moisture content (U.S. EPA, 1993a). Dietary fractions for each species are presented in Appendix R.

US EPA ARCHIVE DOCUMENT

9.3.2 CSCL Development for Receptor Communities

For receptor communities, the specific methods used to calculate the CSCLs varied with the receptor taxon. CSCLs were derived for receptor communities in direct contact with contaminated media (i.e., terrestrial and aquatic plants, soil biota, sediment biota, and fish/aquatic invertebrates). The major source of ecotoxicity data was the primary literature. Secondary sources of data included documents and databases developed by EPA, other government agencies such as the National Oceanographic and Atmospheric Administration (NOAA), and other research facilities such as Oak Ridge National Laboratories (ORNL). Appendix R, Tables R-3 through R-7, present the benchmarks used as CSCLs for each receptor discussed in the following sections. The approach for calculating ecological CSCLs for each receptor community is described below.

9.3.2.1 <u>Aquatic Community</u>. For aquatic biota in freshwater systems, the final chronic value (FCV) developed for the National Ambient Water Quality Criteria (NAWQC) was chosen as the CSCL. If an AWQC was not available, the continuous chronic criterion (CCC) developed for the Great Lakes Water Quality Initiative (GLWQI) was used as the CSCL (U.S. EPA, 1995, 1996). If neither of these criteria was available, a secondary chronic value (SCV) was calculated using the Tier II methods developed through the Great Lakes Initiative (Stephan et al., 1985; Suter and Tsao, 1996; RTI, 1995a, b). See the text box for more information about FCVs, CCCs, and SCVs.

The secondary chronic value is calculated using methods analogous to, but less rigorous than, those applied in calculating the final chronic value. While the final chronic value requires data on species from

Calculation of FCV, CCC, and SCV

FCV and CCC

- If acceptable chronic toxicity data are available on at least one species representing each of the eight different data requirements, the FCV (CCC) is essentially the concentration corresponding to a cumulative probability of 0.05 for the appropriate species.
- If the chronic toxicity data do not meet the eight family requirements, the FCV (CCC) is calculated as follows:
  1. Calculate a final acute value (FAV) that meets the eight species requirements.
  2. Estimate an acute-to-chronic ratio as the ratio of at least three comparable (e.g., same-species) acute and chronic toxicity studies.
  3. Divide the FAV by 2.
  4. Divide the result of Step 3 by the acute-to-chronic ratio.

SCV—Calculated using methods analogous to those for FCV (CCC), except the Tier II methods—

- Require chronic data on at least one of the eight species requirements.
- Use a secondary acute value (SAV) in place of the FAV.
- Are derived based on a statistical analysis of NAWQC data conducted by Host et al. (1991).

Host et al. (1991) developed adjustment factors (AFs) depending on the number of taxonomic families represented in the database. The Tier II methodology was designed to generate SCVs that are below FCVs (for a complete data set) with a 95 percent confidence limit.

For a complete review of calculation methods, refer to Stephan et al. (1985).

US EPA ARCHIVE DOCUMENT

each of eight taxonomic families, derivation of a secondary chronic value requires chronic data on only one of the eight families. In addition, the secondary chronic value uses a secondary acute value in place of the final acute value and is derived based on a statistical analysis of AWQC data conducted by Host et al. (1991). Host et al. (1991) developed adjustment factors depending on the number of taxonomic families that are represented in the database.

9.3.2.2 <u>Dissolved Surface Water CSCLs</u>. Conversion factors were available for several metal constituents to convert CSCLs for total metal concentrations in the water column to CSCLs for total dissolved concentrations (U.S. EPA, 1999a). Dissolved metals are more bioavailable to exposed organisms and therefore provide more meaningful CSCLs for metal COCs. Although the CSCLs for total concentrations (i.e., the values from the NAWQC and GLWQI discussed above) are still deemed scientifically defensible by EPA, the Agency recommends the use of dissolved metal concentrations when they are available (Prothro, 1993).

Methods are currently available to develop dissolved CSCLs for metals only in the freshwater community. Dissolved CSCLs were derived from total water CSCLs using a conversion factor. The conversion factors applicable to chronic criteria in freshwater are presented in Table 9-3. The conversion factors were developed by EPA's Office of Water using a series of filtration experiments that measured the difference between filtered and unfiltered concentrations of metals in surface waters. Dissolved CSCLs were derived by multiplying the total CSCL by the conversion factor:

$$\text{Metal CSCL}_{\text{dissolved}} = (\text{Metal CSCL}_{\text{total}}) \times (\text{Conversion Factor}) \qquad (9\text{-}6)$$

where

Metal CSCL $_{total}$ = either a final or secondary chronic value in freshwater
Conversion Factor = fraction of dissolved metal.

9.3.2.3 <u>Algae and Aquatic Plants</u>. For algae and aquatic plants, toxicological data are available in the open literature and in data compilations such as the *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision* (Suter and Tsao, 1996). Studies on freshwater vascular plants are seldom available; however, toxicity data are available from standard algal tests. In order of preference, the CSCLs for algae and aquatic plants are based on either (1) a lowest observed effects concentration (LOEC) for vascular aquatic plants or (2) effective concentration (EC_{xx}) for a species of freshwater algae, generally a species of green algae.

9.3.2.4 <u>Benthic Community</u>. The benthic community consists of invertebrates that live primarily in the sediment (e.g., worms, amphipods). Two methods were applied in developing the CSCL for the benthic community. The first and preferred method used measured sediment concentrations that resulted in minimal effects to the composition and abundance of the sediment community. The sediment criteria were derived from the upper limit of the range of sediment contaminant concentrations dominated by no-effects data on survival, species diversity, and abundance endpoints. Measurements to derive the CSCLs were taken at the national scale and reflected a variety of sediment types and benthic community species. The second CSCL

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Table 9-3. Conversion Factors for Dissolved Metal

Constituent	Conversion Factor[a]
Cadmium[b]	1.1017 [ln(hardness)(0.041838)]
Chromium III[b]	0.860
Chromium VI	0.962
Copper[b]	0.960
Lead[b]	1.4620 [ln(hardness)(0.145712)]
Nickel[b]	0.997
Zinc[b]	0.986

[a] Conversion factor for chronic CSCLs in freshwater.
[b] Dependent on the water hardness (assumed to be 100 mg $CaCO_3$/L for this analysis).

derivation method used the equilibrium partitioning (EqP) relationship between sediments and surface waters to predict a protective concentration for the benthic community. Equilibrium partitioning refers to a chemical's tendency to partition to the sediment substrate and the water filling the sediment pore spaces. This method was used only for nonionic organic constituents. The two CSCL derivation methods are discussed in more detail the following sections.

Measured Sediment CSCLs. The premier sources of measured sediment toxicity data are the NOAA and the Florida Department of Environmental Protection sediment documents. NOAA annually collects and analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States as part of the National Status and Trends Program. Data collected by NOAA include measured sediment concentrations and the corresponding measures of toxicity in resident species such as amphipods, arthropods, and bivalves on a variety of community-based endpoints (e.g., abundance, mortality, species composition, and species richness). These data are used by NOAA to estimate the 10th percentile effects (i.e., low effects) concentration and a median effects concentration for adverse effects in the sediment community. These values are not NOAA standards; rather, they are used to rank sites based on the potential for adverse ecological effects.

In contrast, the Florida Department of Environmental Protection sediment criteria were developed from the NOAA data to approximate a probable effects level (PEL) estimated from NOAA's median data and a threshold effects level (TEL) estimated from NOAA's low effects data. PELs and TELs correspond to the statistically derived upper limit of contaminated sediment concentrations that demonstrate probable effects and no effects to the benthic community, respectively. Generally, Florida Department of Environmental Protection values are more conservative than NOAA values. Even though these criteria were developed for a marine community, researchers have demonstrated that marine TELs have good correlation with no-effects levels found for freshwater systems (Smith et al., 1996). In order of preference, TELs were adopted as CSCLs if available; if not, NOAA's low effects values were used. The Florida

US EPA ARCHIVE DOCUMENT

Department of Environmental Protection criteria were chosen above the NOAA criteria for the following reasons:

- The same database was used for both the NOAA criteria and the Florida Department of Environmental Protection criteria development.
- In most cases, the Florida Department of Environmental Protection criteria were more conservative than the NOAA criteria because a larger portion of the low-effects data were used in benchmark development.
- The marine TELs developed by the Florida Department of Environmental Protection were found to be analogous to TELs observed in freshwater organisms (Smith et al., 1996).

Estimated Sediment CSCLs. When measured effects data were not available for organic constituents using the TEL or NOAA's low effects approach, the EqP approach was used to estimate the sediment CSCL (U.S. EPA, 1993b). The EqP approach uses a surface water final chronic value or secondary chronic value to estimate sediment CSCLs. (See text box on page 9-20 for definitions of FCV and SCV.) The approach is based on the partitioning relationships among surface water, pore water, and organic carbon in sediment. This method assumes that the equilibrium partitioning between the sediment and the water column is a function of the fraction of organic carbon in the sediment. Equations 9-7 and 9-8 were used to calculate the sediment CSCL, depending on whether an FCV or an SCV was available. In calculating sediment CSCLs for nonionic chemicals, the fraction organic carbon was assumed to be 1 percent of the total organic carbon; organic carbon partitioning coefficients were adopted as reported in Jones et al. (1997).

$$\text{Sediment CSCL} = f_{oc} \times K_{oc} \times \text{FCV} \tag{9-7}$$

$$\text{Sediment CSCL} = f_{oc} \times K_{oc} \times \text{SCV} \tag{9-8}$$

where

Sediment CSCL	=	protective concentration in sediment (mg/L)
f_{oc}	=	fraction organic carbon (unitless)
K_{oc}	=	organic carbon (unitless)
FCV or SCV	=	final or secondary chronic value (mg/L).

9.3.2.5 <u>Terrestrial Plant Community</u>. For the terrestrial plant community, CSCLs for soil were derived according to the methodology presented in the *Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision* (Efroymson et al., 1997a). The authors derive ecologically relevant benchmarks by rank-ordering the phytotoxicity data according to the lowest observed effects concentrations. This approach was adopted and CSCLs were selected at the lowest LOEC for constituents with 10 or fewer values. For constituents with more than 10 LOEC values, the 10th percentile LOEC was

US EPA ARCHIVE DOCUMENT

selected. Because the toxicity endpoints reflect endpoints such as plant growth and yield reduction, the CSCLs are presumed to be relevant to sustaining "healthy" plant communities.

9.3.2.6 Soil Community. For the soil community, recommended benchmark values were taken from Efroymson et al. (1997b). These benchmarks were developed from lowest-observed-effects-levels (LOELs) for earthworms and microbial endpoints. Earthworms have been recognized to play important roles in promoting soil fertility, releasing nutrients, and providing aeration and aggregation of soil, as well as being an important food source for higher trophic level organisms. In addition, their constant contact with soil media and their permeable epidermis make them more susceptible to contaminant exposures. Likewise, microbial communities play a key functional role in soil fertility, decomposition processes, and nutrient cycling, providing nutrients in available forms to plants. Microbial CSCLs were used only when a significantly higher sensitivity to a particular constituent was indicated for soil microbes than for earthworms. This was the case for nickel and zinc.

For cadmium and lead, sufficient data were available in the literature to derive benchmarks based on a community-level approach similar to that used to develop NAWQC. This is the preferred method for soil CSCL development when sufficient data are available because it is designed to protect the structure and function of the soil community. As such, these CSCLs more fully represent the soil community than does use of a single species (e.g., earthworms) to infer effects on the community. The community CSCL derivation methods used for cadmium and lead are reviewed fully in the HWIR data documentation, Section 14.0 (U.S. EPA, 1999b).

Two key uncertainties were noted in the development of community-based CSCLs. First, the ecotoxicity data used in the method were based on no-observed-effects concentrations. The CSCLs developed using the earthworm/microbial method for the soil community were based on low-effects levels (i.e., some effect was observed at this concentration). Because these CSCLs were based on no-effects soil concentrations (i.e., no effects were observed at these concentrations), some added conservatism was generated in the soil community CSCLs for lead and cadmium. Protecting a receptor at a no-effects level is more conservative in that no effect, rather than some effect, is expected. Second, the species designed to represent key compartments in the soil community did not include microbes. This introduces some uncertainty in the soil CSCL because microflora make up approximately 80 to 90 percent of the biomass in soil, and microflora are responsible for the majority of the biological activity in soil (e.g., nitrogen mineralization).

9.3.3 Tier 2 Analysis

A tiered approach was used in the SERA to determine the waste concentrations at which all constituent-receptor-media combinations screen out (i.e., are considered protective). The Tier 1 analysis assesses all of the constituents, pathways, and receptors selected for the risk analysis, as described in Section 9.2. Tier 2 assesses only those scenarios that do not screen out in the Tier 1 analysis. The screening criterion is a hazard quotient of 1; thus, any constituent-receptor-media combination for which the Tier 1 HQ is greater than 1 is assessed further in Tier 2.

US EPA ARCHIVE DOCUMENT

Tier 2 methods consisted of varying the waste concentration in the WMU to determine the concentration at which the maximum HQ is 1. The relationship between waste concentrations and risk (HQ) is linear. Therefore, the waste concentrations at which the maximum HQ does not exceed 1 is calculated as given by Equation 9-9:

$$\frac{HQ_{\max}}{HQ_{target}} = \frac{750{,}000\ ppm}{Conc_{target}} \tag{9-9}$$

where

HQ_{max}	=	Tier 1 maximum HQ
HQ_{target}	=	Tier 2 target HQ of 1
750,000 ppm	=	Tier 1 waste concentration
$Conc_{target}$	=	Tier 2 target waste concentration that results in all HQs ≤ 1.

Equation 9-9 is rearranged to solve for $Conc_{target}$ as follows:

$$Conc_{target} = \frac{750{,}000 \times HQ_{target}}{HQ_{\max}} \tag{9-10}$$

For example, using Equation 9-10 and the relevant HQ_{max} reported in Section 2.0, the landfill dust target waste concentration for lead in soil that would protect mammals is 250,000 ppm, as shown in Table 9-4. This calculation was performed using the HQ_{max} for the 90th percentile media concentrations.

9.4 Risk Characterization

EPA defines risk characterization in terms of

- The risk estimation that predicts the likelihood of adverse ecological effects
- The risk description that synthesizes the overall conclusion of the assessment and addresses uncertainty, assumptions, and limitations.

Section 2.2 includes summary tables of the HQs and analysis of each HQ exceeding 1 for Tier 1 and the calculated waste concentrations that result in maximum HQs of 1 for Tier 2. The discussion here presents the risk description and the assumptions and issues associated with the risk estimation.

9.4.1 Risk Description

Ecological risk is estimated in Tier 1 by calculating constituent-specific HQs for each receptor exposed in each medium. HQs are calculated by dividing the modeled media

US EPA ARCHIVE DOCUMENT

Table 9-4. Sample Target Waste Calculation

Tier 1 Waste Concentration (ppm)	HQ_{target}	HQ_{max}	$Conc_{target}$ (ppm)
750,000	1	3E+00[a1]	250,000

[a] Taken from Table 2-6 in Section 2.0.

concentrations by the corresponding CSCL. The media concentrations were modeled using a waste concentration of 750,000 ppm. EPA selected this concentration because 750,000 ppm is thought to be substantially higher than the actual concentration in paint waste of any constituent evaluated in this assessment. Media concentrations are generated as distributions, and the 90th percentile values were used to calculate the HQs.

For screening assessments that are based on an HQ approach, the comparison of modeled exposure concentrations to CSCLs in the risk estimation has a binary outcome: either the constituent concentration is above the screening criterion (HQ >1) or the concentration is equal to or below the criterion (HQ ≤ 1). Because the CSCLs were based on de minimis ecological effects, it is presumed that a hazard quotient below 1 indicates a low potential for adverse ecological effects for those receptors included in the analysis for which data were available. However, caution should be exercised in extrapolating to particular ecosystems or to receptors not explicitly modeled in this framework. The nature of a screening analysis suggests a few general caveats in interpreting the results:

1. Screening assessments are, by definition, based on assumptions that ensure a high degree of protection for ecological receptors. Some of these assumptions may result in CSCLs that are below the environmental concentrations that could be tolerated by wildlife. For example, representative species are assumed to obtain 100 percent of their food in a contaminated area. Consequently, a simple exceedance of the target HQ does not necessarily warrant additional investigation. Each HQ should be considered within the context of data quality and level of conservatism implicit in the CSCL.

2. Because the methodology is based on the exceedance of a target HQ of 1, the outcome of the screen is binary: HQ > 1 or HQ ≤ 1. Although large exceedances suggest a greater relative potential for ecological damage, an HQ of 50 is not necessarily five times the impact of an HQ of 10.

3. The potential for adverse ecological effects (as indicated by an HQ exceedance) should not be confused with the ecological significance of those effects. Regardless of the magnitude of an HQ exceedance, screening results can only suggest ecological impacts; they do not demonstrate actual ecological effects, nor do they indicate whether those effects will have significant implications for ecosystems and their components.

US EPA ARCHIVE DOCUMENT

4. Ecological receptors selected for the screening methodology were chosen to represent relatively common populations and communities of wildlife that could be assumed to inhabit areas surrounding the facilities. Threatened and endangered species were not evaluated in the analysis.

5. The protection of sensitive habitat types is not evaluated in this analysis. For example, managed lands (e.g., National Wildlife Refuges) and critical habitats (e.g., wetlands) may be more sensitive or vulnerable to adverse impacts. Critical habitats are widely recognized as serving significant ecological functions (e.g., maintenance of water quality). A risk assessment of particular sensitive habitats requires site-specific information; because the paint wastes risk assessment does not include information on actual WMU locations, this analysis does not address potential risk to sensitive habitats.

Rather than estimating risk, the Tier 2 analysis calculates waste concentrations that result in HQs of 1 or lower. This phase of the analysis was performed for the constituent-receptor-medium combinations that had HQs greater than 1 in the Tier 1 risk estimation phase. The Tier 2 calculated concentrations are WMU-specific concentrations that are assumed to be protective of all ecological receptors exposed at the particular WMU.

9.4.2 Exposure Issues

9.4.2.1 <u>Co-occurrence of Receptor and Constituent of Concern</u>. As a simplification for national scale analyses (i.e., no site-based data), co-occurrence was typically assumed. However, the prior probability that a receptor will be found in a contaminated sector is not known nor is it known whether a receptor will forage for food in contaminated areas or if those areas do, in fact, support the type of habitat needed by the receptor.

9.4.2.2 <u>Conceptual Site Model</u>. As described in Section 4.3, the conceptual site model consists of a buffer area, agricultural field, and a waterbody. The calculation of HQs for soil exposures uses the modeled soil concentrations from the agricultural field. These are the soil concentrations relevant to human exposure since human receptors are assumed not to occupy the buffer. However, ecological receptors are likely to forage and feed in the buffer area, and soil concentrations can potentially be higher in the buffer area than in the agricultural field. Therefore, by basing the ecological HQs on agricultural field soil concentrations, the analysis may underestimate potential exposure.

9.4.2.3 <u>Assumptions on Dietary Exposure</u>. The assessment assumed maximum intake of contaminated prey in the diets of primary and secondary consumers (i.e., 100 percent of the diet originates from the contaminated area). Obviously, under field conditions, many receptors are opportunistic feeders with substantial variability in both the type of food items they consume as well as their seasonal patterns of feeding and foraging. Consequently, the exclusive diet of contaminated food items tends to provide a very conservative estimate of potential risks.

9.4.2.4 <u>Bioavailability of Constituents of Concern</u>. For the purposes of this analysis, all forms of a constituent were assumed to be equally bioavailable; therefore, the actual

US EPA ARCHIVE DOCUMENT

exposures that may occur in the field may be overestimated or underestimated. This assumption is appropriate for a conservative analysis; however, both the chemical form and the environmental conditions influence bioavailability and, ultimately, the expression of adverse effects.

9.4.2.5 Multiple Constituent Exposures. The risk of each constituent was considered separately in this analysis. However, exposure to multiple constituents is highly likely. The synergism or antagonism between different constituent combinations may elicit unexpected adverse impacts to ecosystems. Hence, a single-constituent analysis may underestimate or overestimate risks associated with multiple chemical stressors.

9.4.3 CSCL Development Issues

CSCLs were developed for constituents when sufficient data were available. In many cases, sufficient data were unavailable for a receptor/constituent combination; therefore, the potential risk to a receptor could not be assessed. In particular, insufficient data were available to derive chronic effects CSCLs for amphibians. Because the risk results can only be interpreted within the context of available data, the absence of data cannot be construed to mean that adverse ecological effects will not occur.

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US EPA ARCHIVE DOCUMENT

10.0 Analysis of Variability and Uncertainty

> **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 section discusses the methods that were used in the paints listing risk assessment to account for 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.

This discussion describes the treatment of variability and uncertainty in reference to some parameters used to describe human exposures and risk. Treatment of variability using a Monte Carlo simulation forms the basis for the human health risk distributions, which in turn are the basis for calculating protective waste and leachate concentrations. Previous sections of this technical background document describe how distributions were generated and point values estimated for input parameters. They also describe how these values were used in the models and calculations to produce national level distributions of waste and leachate concentrations that are protective of human health. Uncertainty necessitated the use of assumptions, default values, and imputation techniques in this study. This discussion focuses on how this treatment of variability and uncertainty affects the results.

The ecological risk assessment used predicted media concentrations using the same methods and data as for the human health risk assessment. Therefore, uncertainty and variability associated with source partition and fate and transport modeling are common to both human health and ecological assessments. Variability and uncertainty unique to the ecological risk assessment are described in Section 9.0.

10.1 Variability

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

US EPA ARCHIVE DOCUMENT

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.

For example, the meteorological parameters used in dispersion modeling, such as windspeed and wind direction, are measured hourly by the National Weather Service at many locations throughout the United States, and statistics about these parameters are well documented. Although the distributions of these parameters may be well 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. Much of the temporal variation is accounted for by using models such as ISCST3 that calculate concentrations hourly and sum these hourly values to provide annual concentration estimates. Additionally, using meteorological data from multiple monitoring stations located throughout the United States can account 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 the data into discrete elements of the analysis. For example, use of a refined receptor grid accounts for spatial variability in concentrations around a WMU. Variability in WMU characteristics is accounted for using large databases of individual WMU characteristics that represent the range of possible WMU characteristics.

Spatial variability in environmental setting was accounted for using 49 different locations around the continental United States. Because paint facilities and, therefore, the disposal of wastes generated during manufacturing occur nationally, this analysis characterized environmental conditions that influence the fate and transport of constituents in the environment using regional databases. The location of paint manufacturing facilities and the paint production volume by state was known. Additionally, it was assumed that nonhazardous waste from paint manufacturing facilities would be disposed of within reasonable transport distances of the facility. Therefore, locations for modeling were selected first for states according to the volume of paint manufactured and then by the general location of paint manufacturing facilities within the state. Locations were then weighted within the Monte Carlo analysis according to state paint production volumes.

The risk assessment components discussed include

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

10.1.1 Source Characterization and Emissions Modeling

The specific WMUs in which paint wastes are disposed of were not known; however, EPA determined that wastes could be disposed of in any off-site industrial landfill, treatment tank, or surface impoundment. For this analysis, national databases containing information on

US EPA ARCHIVE DOCUMENT

various WMUs and their design and operating characteristics were used to characterize the variability in WMUs. The *Industrial Subtitle D Survey* (Schroeder et al., 1987) was used to characterize industrial landfills and surface impoundments. The TSDF survey data (U.S. EPA, 1987) were used to characterize treatment tank characteristics. Using the information contained in these databases, three distributions of WMUs were developed—one for each WMU type. These distributions were used in the probabilistic analysis to capture the national variation in WMU physical and operating characteristics.

Source partition modeling was performed for 49 different locations, which allowed variation in location-dependent parameters (e.g., soil, temperature, precipitation) to be considered explicitly in the modeling. Variation in these parameters influenced variation in predicted air emissions, leachate, and infiltration rates. The values of many model input parameters used to characterize WMU and waste characteristics were varied using statistical distributions. These meteorological data sets were combined with WMU physical characteristics data (e.g., surface area) to provide unit air concentrations (UACs) or used with emissions data to estimate air concentrations for landfills, treatment tanks, and surface impoundments.

In the Monte Carlo analysis, the WMU characteristics from national databases, environmental conditions from 49 locations, and randomly selected parameter values for waste characteristics were combined to produce the 10,000 iterations of the source partition model calculations. The source model calculations generated the distribution of environmental releases used in the fate and transport modeling.

10.1.2 Fate and Transport Modeling

The parameter values required to model contaminant fate and transport were obtained from regional databases. The treatment of regional variation in location-dependent parameters used in fate and transport modeling is discussed in the following sections.

10.1.3 Air Dispersion Modeling

To capture geographic variation, dispersion modeling was conducted using meteorological datasets from 49 different meteorological stations around the continental United States. This provided regional representation of the variability in meteorological data. For landfills, these datasets were combined with 21 surface areas representing the distribution of WMU size to provide 1,029 different sets of UACs to use with emissions data to estimate air concentrations. For treatment tanks, these datasets were combined with 31 surface area-height combinations representing the distribution of WMU dimensions to provide 1,519 different sets of UACs to use with emissions data to estimate air concentrations. For surface impoundments, these datasets were combined with 20 surface areas representing the distribution of WMU size to provide 980 different sets of UACs to use with emissions data to estimate air concentrations.

The location of receptors was an important source of variability addressed in the exposure modeling. Previous EPA studies have provided data on distances between WMUs and nearest human receptors and on distances between landfills and nearest residential wells. Because EPA is interested in protecting people residing near WMUs, these data were used to develop

US EPA ARCHIVE DOCUMENT

distributions for distance to receptor to capture variation in resident location. Individuals may potentially be located in any direction and at various distances from a facility, and this analysis explicitly incorporated this consideration. For the air pathways, a receptor grid was established to locate individuals in any of 16 directions and at varying distances between 50 and 550 m from the edge of the WMU. The Monte Carlo analysis used a normal distribution to assign probability to various distances from the WMU, giving greater weight to the central tendency distance of 300 m, and a uniform distribution to assign direction, giving equal probability to a receptor being located in any direction. For the groundwater pathway, downgradient distance was varied as was the location within the lateral extent of the groundwater plume.

Obviously, 49 meteorological stations do not represent every site-specific condition that could exist in the continental United States. In selecting the meteorological stations, consideration was given to representing different Bailey ecological regions and to not excluding from the analysis those areas with unique dispersion characteristics (e.g., coastal areas). Therefore, it is believed that these 49 stations provide a reasonable representation of the variability in meteorological conditions for the U.S. climate regions.

10.1.4 Soil and Water Modeling

Soil characteristics were based on the location of the 49 meteorological stations used in the modeling. Soil characteristics within 20 miles of the meteorological station location were used to determine the soil characteristics for watershed modeling. Precipitation was also varied based on the 49 different meteorological locations. This approach captured the national distribution of soil types and accounted for regional variation in soil characteristics.

Waterbody characteristics were not varied in the fate and transport modeling. However, in addition to variation in soil type and precipitation, watershed modeling also took into account regional variation in agricultural field size, which can affect constituent loading to the waterbody via runoff and erosion (see Section 10.1.6). Otherwise, regional variations in waterbody were not accounted for in this analysis.

10.1.5 Groundwater Modeling

To capture regional differences in aquifer types for use in the groundwater modeling, aquifers typical of the 49 meteorological station locations were characterized. For each location, aquifer types typical of the region were identified. If more than one aquifer type was associated with a given location, equal weight was assigned to each aquifer type for use in groundwater modeling. This approach captured the national distribution of aquifer types and ensured that all aquifer types were included in the modeling.

Within each aquifer type, aquifer characteristics (e.g., aquifer thickness or vadose zone depth) are variable. To account for this variability, vadose parameters were varied within each aquifer type using data from EPA Composite Model with Transformation Products (EPACMTP). Correlated aquifer parameters were varied together for each aquifer type to preserve the correlation of those parameters.

US EPA ARCHIVE DOCUMENT

10.1.6 Terrestrial and Aquatic Food Chain

Constituent concentrations calculated for the agricultural field are influenced by the size of the agricultural field. Regional differences in agricultural field size were incorporated into the modeling by characterizing agricultural field size based on the median agricultural field size in counties within 20 miles of the meteorological station location. Therefore, regional differences in agricultural field size were reflected in 49 different sizes representative of different regions of the nation.

To the extent that agricultural field size affects runoff and erosion of constituents into one of the waterbodies modeled in the assessment, the variation in agricultural field size also had an effect on regional characterization of runoff and erosion loadings to the waterbody. Otherwise, no regional variations were considered for the aquatic food chain modeling.

10.1.7 Exposure Modeling

Individual physical characteristics, activities, and behavior are quite different. As such, the exposure factors that influence the exposure of an individual, including inhalation rate, ingestion rate, body weight, and exposure duration, are quite variable. To include this variability explicitly in the analysis, statistical distributions for these variables were used for each receptor in the analysis: adult and child residents, adult and child farmer, and fisher. For adults, a single exposure factor distribution was used for males and females. For child exposures, one age group (1 to 6) was considered, representing age at the start of exposure, because, for most health effects, this age group is most sensitive. Exposure parameter data were taken from the *Exposure Factors Handbook* (U.S. EPA, 1997a, 1997b, 1997c) and used to establish statistical distributions of values for each exposure parameter for each receptor.

10.1.8 Summary of Variability Considerations

In summary, a distribution of protective waste and leachate concentrations was developed that includes specific consideration of the variability in

- WMU and waste characteristics
- Regional-specific environmental conditions
- Location of receptors
- Exposure factors for each receptor.

Taken together, these provide national distributions of a risk-specific waste and leachate concentration across all facilities of a specified type.

10.2 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 (i.e., better data, better models). EPA typically classifies the major areas of uncertainty in risk assessments as scenario uncertainty, model uncertainty, and

US EPA ARCHIVE DOCUMENT

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. Parameter uncertainty is the lack of knowledge regarding the true value of a parameter used in the analysis.

Although some aspects of uncertainty were directly addressed in this analysis, much of the uncertainty associated with this analysis could only be addressed qualitatively. Significant sources of uncertainty are presented in this section. 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.

10.2.1 Scenario Uncertainty

Sources of scenario uncertainty include the assumptions and modeling decisions that are made to represent an exposure scenario. The lack of information or resources to define and model actual exposure conditions introduced uncertainty into this analysis.

Professional judgment, often coupled with an evaluation of the results of a sensitivity analysis, was used to decide which parameters to include in describing exposure conditions and behaviors. Scenario uncertainties that are important to understand in interpreting the results of this study are discussed in the following subsections.

10.2.1.1 Paint Waste Characteristics. Very little data were available on the physical and chemical characteristics of paint waste. To address this lack, assumptions on the waste characteristics were based on general knowledge of generic industrial wastes. In this analysis, except for constituent concentration, which was calculated, it was assumed that the paint waste in the WMU mixed with other generic industrial wastes. Therefore, general waste characteristics, including default assumptions for the waste parameters (e.g., bulk density, moisture, pH), were used.

10.2.1.2 Characteristics and Location of Waterbodies. One aspect of the site layout of particular relevance to aquatic food chain modeling is the location and characteristics of the waterbodies. The size of the waterbody impacts constituent concentration predicted for that waterbody. The waterbody characteristics selected were for a third-order stream, intended to represent a small but fishable waterbody. This small size would tend to ensure that calculated waste concentrations would be protective of routes of exposure from surface water. The location of the waterbody was also assumed to be either at the edge of the agricultural field or at the edge of the buffer area.

10.2.1.3 Receptor Populations Evaluated. The land use around the waste management units that manage the paint waste streams is unknown. For this analysis, it was assumed that the land could be used for residential, agricultural, or recreational purposes. As such, human receptors evaluated were an adult and child resident, an adult farmer, the child of a farmer, and a resident who is a recreational fisher at a nearby waterbody. Risk estimates presented in this document address hypothetical chronic exposures for these receptors and are designed to provide

US EPA ARCHIVE DOCUMENT

a realistic range of potential scenarios. Not all potential scenarios were evaluated; for example, infants (0- to 1-yr-olds) were not evaluated.

10.2.1.4 <u>Exposure Uncertainty</u>. Exposure modeling relies heavily on default assumptions concerning population activity patterns, mobility, dietary habits, body weights, and other factors. As described earlier in the variability section, the Monte Carlo analysis for the adult and child exposure scenario addressed the possible variability in the exposure modeling by using distributions of values for exposure factors. There are some uncertainties, however, in the data that are used. Although it is possible to study various populations to determine various exposure parameters (e.g., age-specific soil ingestion rates or intake rates for food) or to assess past exposures (epidemiological studies) or current exposures, risk assessment is about prediction. Therefore, long-term exposure monitoring in this context is infeasible. The *Exposure Factors Handbook* (U.S. EPA 1997a,b,c) provides the current state-of-the-science concerning exposure modeling and assumptions and is used throughout this document. To the extent that actual exposure scenarios vary from the assumptions in this risk assessment, risks could be underestimated or overestimated. However, although there could be individuals living near a paint waste management unit who have higher exposures than those predicted, it is more likely that actual exposures for most of these individuals would fall within the predicted range and, moreover, would be similar to what was modeled.

10.2.1.5 <u>Natural Background Exposures</u>. In certain cases, EPA performs a risk assessment on wastes that contain contaminants that also are present in the environment as a result of both natural processes and anthropogenic activities. Under these circumstances, receptors potentially receive a “background” exposure that may be greater than the exposure resulting from release of contaminants from the waste. For national analyses like this assessment, the inclusion of background concentrations as part of the analysis is not feasible due to the variability of background concentrations nationwide and the lack of data on national background concentrations for each constituent. Not including the exposure an individual may already have to a constituent of concern (i.e., exposure to background concentrations) does not change the “marginal” increase in risk a person may have due to possible exposures to constituents in paint waste.

10.2.2 Model Uncertainty

Model uncertainty is associated with all models used in all phases of a risk assessment because models and their mathematical expressions are simplifications of reality that are used to approximate real-world conditions and processes and their relationships. 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. Models do not include all parameters or equations necessary to express reality because of the inherent complexity of the natural environment and the lack of sufficient data to describe the natural environment. Because this is a probabilistic assessment that predicts what may occur with the management of certain paint wastes under assumed scenarios, it is not possible to compare the results of our models (sometimes referred to as model validation) to any specific situation that may exist. 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

US EPA ARCHIVE DOCUMENT

and not in others. A similar problem can occur when a model that is applicable under average conditions is used for conditions that differ from the average. In addition, in some instances, choosing the correct model form is difficult when conflicting theories seem to explain a phenomenon equally well. In other instances, EPA does not have established model forms from which to choose to address certain phenomena, such as facilitated transport. Models used in this risk assessment were selected based on science, policy, and professional judgment. These models were selected because they provide the information needed for this analysis and because they are generally considered to be state-of-the-science. Even though the models used in the risk analyses are used widely and have been accepted for numerous applications, they each retain significant sources of uncertainty. Evaluated as a whole, the sources of model uncertainty in this analysis could result in either an overestimation or underestimation of risk. Specific areas of modeling uncertainty in this analysis are as follows:

- There were multiple constituents identified as materials used in paint manufacturing that were not modeled in this risk assessment due to a lack of information on how they behave when introduced to the environment. The fate and transport modeling was limited to those constituents for which (1) the physical/chemical parameters necessary to run the models were available and (2) adequate information on toxicity to understand potential health impacts from exposure. In selecting constituents of concern, multiple constituents were identified that were complex inorganic compounds containing more than one metal of concern and organometallic compounds (compounds containing both a metal and organic constituents) that can be used in manufacturing paint. For example, compounds such as lead chromate molybdate, lead naphthenate, and zinc phosphate may be used as ingredients in paint. An adequate set of both the physical/chemical parameters and toxicity information for modeling fate and transport and predicting risk to human health was lacking for these metal complexes. Due to this absence of data, the risk presented by these multiple compounds was simulated by modeling the ionic form of the metal. For example, the model predictions for lead were used to represent the complex lead inorganic metal compounds and lead organometallic compounds that may be used in paints. Since so little is known about these complex metal compounds and what their fate may be in the environment, the modeling may over- or underestimate the actual risks. In addition, for metals, transformations may take place because the pH of the waste or media can change the state of the metal, sometimes to a less toxic form and sometimes to a more toxic form. This risk assessment did not model transforma-tion products or changes in metal species.

- Exposure modeling relies heavily on default assumptions concerning population activity patterns, mobility, dietary habits, body weights, and other factors. There are some uncertainties associated with some of the data used for these parameters. Although it is possible to study various populations to determine various exposure parameters (e.g., age-specific soil ingestion rates or intake rates for food) or to assess past exposures (epidemiological studies) or current exposures, risk assessment is about prediction. Therefore, long-term exposure monitoring in this context is infeasible. The *Exposure Factors Handbook* provides the current state-

US EPA ARCHIVE DOCUMENT

of-the-science concerning exposure modeling and assumptions and was used in this risk assessment. To the extent that actual exposure factors vary from the assumptions in this risk assessment, risks could be underestimated or overestimated.

- In modeling the fate and transport of chemicals in groundwater, complex hydrogeology such as karst or highly fractured aquifers was not assessed. Some fraction of the groundwater settings in this analysis have fractured flow. In general, fractured flow in groundwater can channel the contaminant plume, thus allowing it to move faster and in a more concentrated state than in a nonfractured flow environment. As a result, the modeling may under- or overestimate the concentrations in the groundwater.

- There is uncertainty in predicting the movement of contaminants over long periods of time. The risk to receptors for the groundwater pathway was evaluated over a time period of 10,000 years. There are significant uncertainties concerning how exposure and environmental assumptions will change over time, and the modeling methodology does not change these assumptions over this 10,000-year period.

10.2.2.1 <u>Air Dispersion Modeling</u>. The ISCST3 model was used to calculate the dispersion of particle and vapor emissions from a WMU. This model has many capabilities needed for this assessment, such as the ability to model area sources. For dispersion modeling of this type, ISCST3 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. Deposition and associated plume depletion are important for particulates and vapors and were explicitly incorporated into this analysis. Currently, algorithms specifically designed to model the dry deposition of gases have not been verified for the specific compounds in question (primarily volatile organics). In place of algorithms, a transfer coefficient was used to model the dry deposition of gases. A concern with this approach is that the deposition is calculated outside of the model. As a result, the mass deposited on the ground from the plume and is not subtracted from the air concentrations estimated by ISCST3. This results in a slight nonconservation of mass in the system.

Other uncertainties introduced into the analysis in dispersion modeling are related to WMU shape. A 20-sided polygon shape approximating a circle was selected because it minimized the error introduced by not knowing the orientation of the WMU shape to wind direction.

10.2.2.2 <u>Mercury Modeling</u>. Mercury concentrations in the environment affect all receptor populations. Important among these are residential fishers. An important exposure pathway for mercury is the aquatic food chain pathway (e.g., waterbody concentrations - fish tissue concentrations - human consumption of fish); however, other exposure pathways have been evaluated as well, including terrestrial food chain pathways and soil ingestion.

US EPA ARCHIVE DOCUMENT

A number of uncertainties are introduced into the risk assessment for mercury modeling because of a lack of data or inability to capture real-world complexities in the model formulations. Mercury was modeled based on assumptions about WMU-specific emission rates. The form of mercury emitted by a given WMU is thought to be a determining factor in the fate and transport of mercury in the atmosphere. In this analysis, two forms of mercury were modeled: elemental mercury and divalent mercury. Modeling was conducted for two assumptions: (1) that all of the mercury released to the atmosphere is elemental mercury and (2) that all of the mercury released is divalent mercury. For groundwater modeling, mercury was assumed to be divalent mercury.

In addition to uncertainties in the type of mercury emissions, atmospheric dispersion and deposition modeling conducted for this analysis did not account for atmospheric processes that would alter the vapor/particle partitioning or the other transformations of the mercury species, which introduces uncertainties in mercury species air concentrations and deposition rates. There are other uncertainties related to deposition of mercury as well. These include the lack of mercury-specific modeling of wet and dry removal processes (e.g., gas scavenging rates and gas deposition velocities) and the use of air model algorithms that are not fully mass conserving with respect to that portion of divalent mercury vapor that is dry deposited.

The behavior of mercury species in the soil and water environments is complex. There are a variety of uncertainties related to the fate and transport of mercury in watershed soils and surface water. Among these are uncertainties involving the transport of mercury deposited in upland areas of a watershed to surface water and transformation of mercury in soil and subsequent volatilization and release to the atmosphere. Also uncertain is the disposition of mercury to surface water, methylation and demethylation processes, sequestering in the water column and sediments, and uptake in aquatic organisms. In particular, methylation rates are highly variable and depend on the characteristics of the particular waterbody, and, in this analysis, waterbody characteristics were not varied. Modeling the aquatic food chain pathway was based on the modeling of divalent mercury concentrations in soil and water.

10.2.2.3 <u>Groundwater Modeling</u>. In the groundwater model, EPACMTP, it is assumed that the soil and aquifer are uniform porous media. EPACMTP does not model preferential pathways such as fractures, macropores, or facilitated transport, which may affect migration of strongly sorbing constituents such as metals. EPACMTP also does not model colloidal transport or the geochemical interactions between different contaminants in the leachate. Any of these factors could result in underpredicting contaminant concentrations at the receptor well. Conversely, the EPACMTP modeling incorporates the following assumptions: (1) transverse dispersion is negligible in the unsaturated zone, potentially resulting in an overestimation of risks; (2) receptors use the uppermost aquifer, rather than a deeper aquifer, as a domestic source of drinking water, which overestimates risks where the uppermost aquifer is not used; and (3) hydrogeologic conditions that influence contaminant fate and transport are uniform spatially as well as temporally (that is, in the time period over which the model is executed, 10,000 years), potentially resulting in an underestimation or overestimation of receptor well concentrations.

10.2.2.4 <u>Assumption of Additivity of Chemicals in Characterizing Risk</u>. Both cancer and noncancer risks were evaluated on a chemical-specific basis within the analysis. Additive

US EPA ARCHIVE DOCUMENT

effects from multiple-chemical exposures were not calculated. Chemical mixtures can display both synergistic and antagonist behavior with regard to risk. In general, however, the overall risks of a mixture are very likely to be greater than that of exposure to a single chemical. Therefore, not adding risks across the chemicals is an area of uncertainty that leads to an underestimate of total risk. The additive effects from multiple-chemical exposure were not calculated because information was not available on the concentrations or co-management of particular constituents. Whether or not a particular chemical mixture poses an additive risk depends on the targets (tissue, organ, or organ system) and the mechanisms of action of the individual chemicals. Without information on the co-management of constituents, it was not feasible to consider additive risks.

10.2.2.5 Human Health Benchmarks. Sources of uncertainty in toxicological benchmarks include one or more of the following: extrapolation from laboratory animal data to humans, variability of response within the human population, extrapolation of responses at high experimental doses under controlled conditions to low doses under highly variable environmental conditions, and adequacy of the database (number of studies available, toxic endpoints evaluated, exposure routes evaluated, sample sizes, length of study, etc.). Toxicological benchmarks are designed to be conservative (that is, to potentially overestimate risk) because of the uncertainties and challenges associated with condensing toxicity data into a single quantitative expression.

Cancer Slope Factors. Cancer slope factors 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 and reference concentrations are derived is 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, 2000a). RfDs and RfCs are based on the no observed adverse effect 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:

US EPA ARCHIVE DOCUMENT

- 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, for example, 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 (U.S. EPA, 1994). Therefore, the RfD (or RfC) is derived by using the following formula:

$$\text{RfD} = \text{NOAEL}/(\text{UF} \times \text{MF}). \qquad (10\text{-}1)$$

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

Human Health Benchmarks and Children. EPA recognizes that significant uncertainties exist regarding the estimation of lifetime cancer risks in children. EPA estimated the risk of developing cancer from the estimated lifetime average daily dose and the slope of the dose-response curve. A CSF is derived from either human or animal data and is taken as the upper bound on the slope of the dose-response curve in the low-dose region, generally assumed to be linear, expressed as a lifetime excess cancer risk per unit exposure. Individuals exposed to carcinogens in the first few years of life may be at increased risk of developing cancer.

The noncancer toxicological effects in children are also an area of uncertainty. Hazard quotients for children are based on comparing childhood exposure, for which age-specific data (e.g., food consumption rates) are available, with adult toxicity measures (e.g., RfDs), for which adequate age-specific dose-response data are often lacking. This mismatch could result in great uncertainty in the estimation of hazard quotients for children. This could sometimes result in an overestimation of children's risk and sometimes in an underestimation. This issue is still under investigation in the scientific community and no consensus has been reached.

10.2.2.6 Lead Risk Characterization. Lead exposures were evaluated based on soil screening levels for the air pathway and drinking water criteria (maximum contaminant levels or MCLs) for the groundwater pathway. These screening levels are used to determine levels of lead that may be in environmental media and not pose a risk to public health. These media criteria are

US EPA ARCHIVE DOCUMENT

thought to be conservative in nature. If these levels indicate that the lead risk could be high enough to warrant establishing protective waste and leachate concentrations for lead, future analyses may reevaluate lead exposures using blood lead level as an indicator.

10.2.3 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 because of limitations in measurement technology. 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.

10.2.3.1 Waste Management Unit Parameters. As discussed in Section 4.4, 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* (Schroeder et al., 1987) was used to characterize landfills and surface impoundments. 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; thus, the number of highly aerated biological treatment tanks may be underestimated. Underestimation of the number of highly aerated treatment tanks would result in lower emissions estimates and higher protective waste concentrations. Because the tank data were also used to characterize surface impoundment aeration characteristics, these uncertainties also affect surface impoundment results.

Source characterization also required making assumptions about the way WMUs are operated. Surface impoundments were assumed to be closed after 50 years and the site cleaned of all residual constituent contamination.

10.2.3.2 Distribution Coefficients, K_d. The distribution coefficient, K_d, which is used in the source partition model, the groundwater model, and in modeling constituent concentration in surficial soils, is an important parameter for modeling the fate and transport of metals in the environment. In previous analyses, K_d values were calculated using MINTEQ but, because of comments on the validity of some of the data upon which MINTEQ calculations are based, EPA decided, for this analysis, that K_d values would be derived from literature values. A comprehensive review of the literature was undertaken to compile K_d data for an earlier rulemaking (U.S. EPA, 2000b). Despite this substantial earlier effort, considerable uncertainty

US EPA ARCHIVE DOCUMENT

remains in the literature-based values of K_d used in this analysis because data concerning K_d values for particular constituents reported in the literature were limited. In addition, reported values often were not accompanied by qualifying information. Conditions that affect K_d values (e.g., constituent concentration, metal species evaluated, pH, experimental technique) are often not reported in the literature, making interpretation of results difficult. For these reasons, substantial uncertainty concerning the values of K_d remain.

10.2.3.3 Watershed Universal Soil Loss Equation (USLE) Parameters. A combination of region-specific and national default parameters was used along with USLE to model soil erosion losses from watersheds to waterbodies. The USLE calculations are particularly sensitive to site-specific values; thus, uncertainty is associated with using regional and national parameter values. Many of the ULSE parameters were based on the regional meteorological and regional soil data used in other parts of the analysis. These include soil erodibility factor (K), rainfall erosivity, and slope. Other parameters were based on national default values (e.g., cover and management factors) or default relationships with other factors (e.g., length was determined as a function of slope).

10.2.3.4 Biotransfer Factors for Cows. The uptake of chemicals into dairy cows was estimated using biotransfer factors (BTFs) that enabled calculation of individual contaminants in tissue as a consequence of feed, soil, and water ingestion. Chemical concentrations in feed, soil, and water are multiplied by their respective ingestion rates and by contaminant-specific BTFs and then summed to obtain the concentration of individual contaminants in tissue. Feed-to-animal tissue BTFs are generally the only BTFs available in the scientific literature. The absence of soil and water to tissue BTFs necessitates the assumption that contaminant transfer from soil and water to tissue is similar to that for food to tissue (i.e., the same set of BTFs must be used for each exposure route). This assumption may be nonconservative in that BTFs in drinking water may be higher than those in feed because chemicals may be more bioavailable in water. Although this assumption may be nonconservative, the degree of uncertainty is unknown and cannot be predicted.

10.2.3.5 Exposure Factors. For most exposure factors addressed, data analyses involved fitting distributions of data summaries from the EFH (U.S. EPA, 1997a, 1997b, 1997c), in most cases by fitting distributions to selected percentiles. It is assumed that little information is lost by fitting to percentiles versus fitting to raw data. However, some believe that such analyses should always be based on raw data, synthesizing all credible sources.

The datasets for time spent in shower clearly are affected by rounding and grouping of data. The fitting methods do not account for these sources of uncertainty.

Three standard two-parameter probability statistical distributions (gamma, lognormal, and Weibull) were used for this analysis. These distributions are special cases of a three-parameter distribution (generalized gamma) that contains them and allows for a likelihood ratio test of the fit of the two-parameter models. Other statistical distributions are possible (e.g., U.S. EPA, 2000c), but the technique used in this analysis offered considerable improvement over using a lognormal model in all cases and was appropriate for this analysis. In support of this conclusion, a comparison of results showed that the three-parameter generalized gamma distribution did not

US EPA ARCHIVE DOCUMENT

significantly improve on goodness of fit over the two-parameter distributional forms in 58 of 59 cases at the 5 percent level of significance.

Although they offer significant improvement in objectivity over visual estimation, goodness-of-fit tests used to determine which statistical distribution to use for a particular parameter are themselves subject to some uncertainty that should to be considered in their application to exposure factors. One area of concern is uncertainty about how the survey statistics in the Exposure Factors Handbook (EFH) (U.S. EPA, 1997a, 1997b, 1997c) were calculated. All of the statistics that have been used to assess goodness of fit assume a random sample, which may or may not be a valid assumption for EFH data. Specifically, many of the EFH data sources are surveys that, in many cases, do not involve purely random samples. Rather, they use clustering and stratification, primarily for economic reasons.

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US EPA ARCHIVE DOCUMENT