DERM Technical Report:

Development of Cleanup Target Levels (CTLs) for Chapter 24, Miami-Dade County Code

Prepared for the
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I. Introduction

This document describes the procedures used to develop groundwater, surface water, and soil Cleanup Target Levels (CTLs), provides the equations used for calculating these values, and identifies the sources of input values for these equations. In addition, this document presents information regarding the derivation of site-specific soil CTLs, including methodology for selection of the appropriate input values for their calculation.

Groundwater CTLs (GCTLs), which are based upon protection of human health and aesthetic (organoleptic) considerations, are consistent with the numerical standards set forth in Section 24-43.3(2)(h) of Chapter 24 of the Code of Miami-Dade County ("Code"), Chapter 62-550, Florida Administrative Code (F.A.C.), *Drinking Water Standards, Monitoring, and Reporting*, and the methodology provided in Chapter 62-777, F.A.C. Freshwater and marine surface water CTLs, which are based upon the protection of human health and aquatic species and aesthetic considerations, are consistent with the numerical standards set forth in Section 24-42(4) of the Code and the methodologies employed in Chapter 62-302, F.A.C. and Chapter 62-777, F.A.C.

Soil CTLs (SCTLs), which are based upon direct human contact (i.e., direct exposure) and leachability potential (i.e., leachability), were developed using an approach that is based largely on earlier efforts made by the United States Environmental Protection Agency, USEPA (1996a, 1996b), and is consistent with that employed by the Florida Department of Environmental Protection (FDEP) for setting Soil Cleanup Target Levels under Chapter 62- 777, F.A.C.. Although direct human contact SCTLs for various exposure scenarios can be calculated using the methodology presented here, this report focuses on only two scenarios: exposure from residential and from commercial/industrial land use. SCTLs are based on default assumptions and are intended to be broadly applicable. Site-specific characteristics can be used to develop site-specific SCTLs. Methods for calculating these site-specific SCTLs are discussed.

Be advised that this document must be used in conjunction with the provisions set forth in Section 24-44(2) of the Code and the other technical guidance documents provided in the Supporting Information for the Implementation of the Risk Based Corrective Action Provisions for Miami-Dade County. A copy of these documents may be downloaded from the DERM web page: http://www.miamidade.gov/derm/land/trends_risk_based.asp.

II. General Concepts and Approaches

A. Risk or Hazard

1. Cancer Risks

Regulatory agencies currently view risks from carcinogens differently from non-cancer health effects. For most chemicals, carcinogenicity is assumed not to have a threshold, and even very small doses are assumed to pose some (albeit small) risk of cancer. In this view, safety must be defined as some risk (i.e., probability) of cancer so small as to be considered insignificant. For Chapter 24 as well as Chapter 62-777, F.A.C., a lifetime excess cancer risk of 1×10^{-6} (one in a million) is used for calculating CTLs for carcinogens. The USEPA has developed measurements of cancer potency of carcinogens, which are termed cancer slope factors (CSFs). CSFs are calculated through various low-dose extrapolation procedures and represent the increase in lifetime cancer risk per unit dose, with the CSF in units of 1/(mg/kg-day).

There are cases in which carcinogenicity can be assumed to occur only after some dose or threshold is reached, depending on the mode of action by which the contaminant is thought to cause cancer. For example, chloroform is classified by the USEPA as probable human carcinogen, but a recent review of chloroform carcinogenicity studies has prompted the Agency to conclude that cancer occurs only at relatively high exposures. The USEPA considers the chloroform oral Reference Dose (RfDo) developed to protect against non-cancer endpoints adequate to also protect from cancer.

2. Non-cancer Hazards

All non-cancer health effects are assumed to have a dose threshold. That is, it is assumed that below some dose, the effect does not occur. A chemical can often produce many different types of adverse health effects, each with its own threshold. If the threshold for the most sensitive health effect can be identified — the effect that occurs at the lowest dose — limiting exposure to produce doses below that threshold should protect against all of the effects of the chemical. This concept is the basis for the USEPA reference dose (RfD). The USEPA examines toxicity data for a chemical, identifies the most sensitive effect, and then determines a dose sufficiently low enough to prevent that effect from occurring in the most sensitive individuals. Because environmental exposures can be long term, the dose is actually a dosing rate (amount of chemical per day), and it is intended to protect against toxicity for exposures that range up to a lifetime. Reference doses are specific to the route of exposure (ingestion, dermal contact, or inhalation). Therefore, the development of CTLs for each medium must use the RfDs for the relevant route(s) of exposure

developed by the USEPA or through route-to-route extrapolation, as discussed in the following section.

For hazard calculations, the projected exposure dose divided by the applicable reference dose is termed the hazard quotient. CTLs are calculated based on a hazard quotient of 1. This means that the chemical dose implicit in the standard is equivalent to the maximum safe dose developed for that chemical by the USEPA for lifetime exposure.

It is important to point out that the toxicity values developed by the USEPA — the reference doses and cancer slope factors — are developed conservatively. That is, in view of uncertainties in the risk assessment process, they typically have a "safety buffer" built in. As a result, it is more accurate to state, for example, that a CTL corresponds to a risk "that is less than one in a million" rather than to state that it poses a risk "equal to one in a million."

B. Toxicity Values

1. Primary Sources

Calculation of a risk-based CTL requires a chemical-specific toxicity value, either a RfD or a CSF. The toxicity values and their sources/bases are provided in Tables 5a and 5b. When available, these toxicity values are taken from various USEPA sources. These sources, in order of preference for CTL development, are:

1) Integrated Risk Information System (IRIS).
2) National Center for Environmental Assessment (NCEA) provisional toxicity values.
3) Health Effects Assessment Summary Tables (HEAST).
4) Office of Pesticide Programs (OPP), Reference Dose Tracking Report; or Office of Water, Drinking Water Regulations and Health Advisories; or upper intake limits developed by the National Academy of Sciences (NAS, 2001); or withdrawn values from IRIS or HEAST.

Note: The last category consists of several sources of roughly equal preference.

2. Secondary Sources

Alternative approaches can be used when no toxicity values for a given chemical are available from any of the primary sources discussed above. Among the chemicals listed herein, some toxicity values had to be extrapolated using a combination of several approaches, including route-to-route extrapolation, surrogate values, the toxic equivalency factor (TEF) approach, and extrapolation from occupational exposure limits. Most of the toxicity values not available from the USEPA were derived using route-to-route extrapolation. A few more were based on surrogate

values and the TEF approach. Only one CTL was developed using occupational exposure limits. Each of these extrapolation methods is described in the following sections.

a) Route-to-route Extrapolation

Often, inhalation and dermal toxicity criteria are not available. In these cases, route-to-route extrapolation can be used to expand upon published toxicity values for one route of exposure to develop toxicity values for other routes. For example, the oral toxicity value can be used to derive corresponding inhalation or dermal values (see Appendix B). Intake from different routes is not necessarily equivalent, and information regarding toxicokinetics of the chemical (or assumptions in this regard) must be taken into account when performing route-to-route extrapolation. Further, route-to-route extrapolation is not appropriate when there is evidence that the toxicity value serving as the basis for extrapolation is likely to be route-specific. If a CSF or a RfD is known or presumed to be route-specific, it should not be regarded as suitable for route-to-route extrapolation.

While the USEPA originally recommended route-to-route extrapolation as a means of developing toxicity values (e.g., in USEPA, 1989a), more recently they have discouraged its use, citing the uncertainties involved (see, for example, the discussion in USEPA, 1996b). While these uncertainties cannot be denied, when route-to-route extrapolation is performed with knowledge of the disposition and toxicity of the chemical, these uncertainties are hardly disproportionate to the uncertainties associated with other aspects in the calculation of CTLs. Further, when the alternative is to omit a particular route of exposure from the CTL calculation, in effect assuming that risk from this route is zero, this too is a source of uncertainty. In fact, for some chemicals, the absence of a toxicity value can mean that the dominant source of risk is ignored. In light of this, the cause of minimizing uncertainty is arguably best served by judicial use of route-to-route extrapolation in CTL development.

b) Surrogate Chemicals

Alternative approaches for developing toxicity values include the use of "surrogate values" (i.e., toxicity values for substances from the same chemical class and with similar toxicological properties). The use of these surrogate toxicity values offers a means to provide some estimate of risk, and of acceptable concentrations, for chemicals with little or no toxicity information. However, this approach carries with it significant uncertainty because small changes in chemical structure can produce profound differences in toxicity (compare the toxicity of CO and CO_2, acetate and fluoroacetate, ethanol and methanol, for example). Table 9 below lists the chemicals for which surrogate toxicity values are used in the development of CTLs presented in this report,

the surrogate value, and the source of the surrogate value. It should be noted that all of the chemicals in question are considered non-carcinogens and therefore only surrogate reference doses are used.

Table 9
Surrogate Toxicity Values

Contaminant	Surrogate Oral RfD (mg/kg-d)	Surrogate Contaminant
acenaphthylene	3.0E-02	pyrene [a]
benzo(g,h,i)perylene	3.0E-02	pyrene [a]
chlorophenol, 3-	5.0E-03	chlorophenol, 2-
chlorophenol, 4-	5.0E-03	chlorophenol, 2-
dichlorophenol, 2,3-	3.0E-03	dichlorophenol, 2,4-
dichlorophenol, 2,5-	3.0E-03	dichlorophenol, 2,4-
dichlorophenol, 2,6-	3.0E-03	dichlorophenol, 2,4-
dichlorophenol, 3,4-	3.0E-03	dichlorophenol, 2,4-
hexachlorocyclohexane, delta	3.0E-04	hexachlorocyclohexane, gamma
methylnaphthalene, 1-	4.0E-03	methylnaphthalene, 2-
phenanthrene	3.0E-02	pyrene [a]
trichlorobenzene, 1,2,3-	1.0E-02	trichlorobenzene, 1,2,4-
trimethylbenzene, 1,2,3-	5.0E-02	trimethylbenzene, 1,2,4-

[a] For acenaphthylene, benzo(g,h,i)perylene, and phenanthrene, pyrene is chosen as a surrogate because its RfD is in the mid-range of RfDs for other non-carcinogenic PAHs. For all of the other contaminants in this table, the surrogate is chosen because it is the closest structurally-related compound with a RfD listed in IRIS.

c) Occupational Exposure Limits

Occupational exposure limits are often based on relatively extensive study in humans, which is an advantage. Because they are intended for healthy adults, an adjustment must be made in order for them to be considered protective for a broader range of exposed individuals that may include some with special sensitivity. By incorporating the appropriate "safety factor," toxicity values from occupational exposure limits can be, in general, conservative and health protective (Williams et al., 1994). There may be, however, some situations in which a chemical poses special toxicity to sensitive individuals not found in the workplace (e.g., lead in children), where extrapolation from occupational limits may not be appropriate. Extrapolation from occupational exposure limits was only used to develop CTLs for tert-butyl alcohol.

C. Comparing Site Contaminant Concentration Data with Cleanup Target Levels

Site concentration data shall be compared to the applicable CTLs, apportioned as appropriate (i.e., default CTLs set forth in Table 1 and Table 2 herein or alternative CTLs derived

in accordance with the guidelines presented herein). However, in accordance with Section 24-44(2)(f) of the Code, the CTLs shall not be more stringent than the practical quanitation limits or naturally occurring background concentrations determined in a natural background concentration study which has been approved by DERM.

III. Development of Groundwater Cleanup Target Levels

GCTLs provided in Table 1 are equivalent to the numerical standards set forth in Section 24-43.3(2)(h) of the Code. Where such standards do not exist, the GCTLs are equivalent to the numerical standards set forth in Chapter 62-550, F.A.C., Tables 1, 4, 5, and 6; these GCTLs are designated by the notations "Primary Standard" or "Secondary Standard." For chemicals not listed in Section 24-43.3(2)(h) of the Code or Chapter 62-550, F.A.C., GCTLs are based on the following factors, as applicable: 1) human health risk calculations using a lifetime excess cancer risk of one in a million (1×10^{-6}), or using a hazard quotient of one (1.0) or less [Note: these are designated by the notation "Miminum Criteria" followed, respectively, by "Carcinogen" or "Systemic Toxicant"], and 2) aesthetic considerations [Note: these are designated by the notation "Minimum Criteria, Organoleptic"]. Aesthetic considerations include altered taste, odor, or color of the water. While these factors do not pertain to health directly, they nonetheless degrade the quality of the water, and therefore its suitability as a drinking water source. The equation used to calculate risk-based GCTLs for carcinogens is shown in Figure 1. The equation for calculating GCTLs for non-carcinogens is shown in Figure 2.

GCTLs are based on consumption of 2 L of water per day and a body weight of 70 kg. Exposure is assumed to occur over a lifetime. For non-carcinogens, a Relative Source Contribution (RSC) factor is included. This represents the fraction of the total allowable daily intake that can come from groundwater. Consistent with USEPA methods, a default RSC of 0.2 (20%) is used.

A. Development of Groundwater Cleanup Target Levels for Class C Carcinogens

There are some chemicals designated as Class C carcinogens (i.e., possible human carcinogens) for which no CSF is available. Without a CSF, a groundwater CTL based on cancer risk could not be calculated. Consistent with the approach used by FDEP, GCTLs for these chemicals are developed by reducing the GCTL calculated for non-cancer health effects by an additional factor of 10. The equation used to calculate GCTLs for Class C carcinogens without defined slope factors is shown below.

$$\text{Groundwater CTL } (\mu g/L) = \frac{\frac{RfD_o}{10} \bullet 0.2\,RSC \bullet 70\,kg \bullet 1000\,\mu g/mg}{2\,L/day}$$

where,

RfD_o = Oral Reference Dose (mg/kg-day)

RSC = Relative Source Contribution (20% default)

The Class C carcinogens that have GCTLs based on non-cancer health effects, along with their RfD, are shown in Table 10 below.

Table 10
RfDs for Class C Carcinogens Based on Non-Cancer Health Effects

Contaminant	CAS#	Oral RfD (mg/kg-d)
acrolein	107-02-8	5.00E-04
allyl chloride	107-05-1	5.00E-02
benomyl	17804-35-2	5.00E-02
bromacil	314-40-9	1.00E-01
butyl benzyl phthalate	85-68-7	2.00E-01
chloral hydrate	302-17-0	1.00E-01
cypermethrin	52315-07-8	1.00E-02
dichloroacetonitrile	3018-12-0	8.00E-03
dichloroethane, 1,1-	75-34-3	1.00E-01
linuron	330-55-2	2.00E-03
mercuric chloride (as Mercury)	7487-94-7	3.00E-04
methidathion	950-37-8	1.00E-03
methylmercury [or Mercury, methyl]	22967-92-6	1.00E-04
methylphenol, 2- [or Cresol, o-]	95-48-7	5.00E-02
methylphenol, 3- [or Cresol, m-]	108-39-4	5.00E-02
methylphenol, 4- [or Cresol, p-]	106-44-5	5.00E-03
metolachlor	51218-45-2	1.50E-01
naphthalene	91-20-3	2.00E-02
oryzalin	19044-88-3	5.00E-02
paraquat	1910-42-5	4.50E-03
parathion	56-38-2	6.00E-03
pronamide	23950-58-5	7.50E-02
propazine	139-40-2	2.00E-02
thiocyanomethylthio-benzothiazole, 2- [or TCMTB]	21564-17-0	4.00E-03
trichloroacetic acid	76-03-9	1.30E-02

B. Comparing Site Contaminant Concentration Data with Groundwater Cleanup Target Levels

Concentrations of contaminants in each monitoring well shall be compared to the applicable GCTLs. As explained in the section on SCTLs, in some situations the average contaminant concentration in soil can be calculated over an exposure area for comparison with the cleanup target. This averaging is based on the concept that an individual will be exposed, over time, to contaminants in soil over an area of the site rather than a single location. For groundwater, on the other hand, an individual will be exposed generally to the water where a potable well is placed. Thus, the rationale for averaging concentrations over a broad area is absent, and the goal is to achieve GCTLs at all locations where individuals might become exposed.

IV. Development of Surface Water Cleanup Target Levels

Freshwater and marine surface water CTLs (SWCTLs) listed in Table 1 are equivalent to the numerical standards set forth in Section 24-42(4) of Code and, where such standards do not exist, Chapter 62-302, F.A.C. and are designated by the notations "24-42(4)" or "62-302". For those contaminant that do not have numerical standards, SWCTLs are based on protection of aquatic organisms and protection of human health [using a lifetime excess cancer risk of one in a million (1×10^{-6}) or a hazard quotient of one (1.0)], whichever is lower, and are designated, respectively, by the notations "Toxicity Criteria" or "Human Health". CTLs based on nuisance considerations are calculated considering factors that do not affect risks to health and the environment, but nonetheless degrade the usability of the water.

A. Suface Water Cleanup Targets Based on Human Health

The equations used to derive SWCTLs based on human health risk are shown in Figure 3A. There are separate equations for carcinogens and non-carcinogens. Both equations are based on the partitioning of the contaminant from surface water to fish, and ingestion of the contaminated fish by humans. Critical exposure inputs in the equation include a fish ingestion rate of 17.5 g/day, a body weight of 76.1 kg, and a chemical-specific bioconcentration factor (BCF). The fish ingestion rate of 17.5 g/day corresponds to the recommendation presented in a recent USEPA document (USEPA, 2000a). The BCF represents the ratio of the concentration of the contaminant in fish to its concentration in surface water. BCF values used to calculate SWCTLs based on human health risks are presented in Table 11.

Table 11
Bioconcentration Factors (BCF) and Resultant
Surface Water Cleanup Target Levels (SWCTLs)

Contaminant	BCF (L/kg)	Source of BCF Value	SWCTL (mcg/L)
Acrylamide	3.16	EPIWin [a]	0.3
Acrylonitrile	30	AWQC [b]	0.3
Alachlor	102	EPIWin	0.5
Atrazine	9.77	EPIWin	2
Azobenzene	10.0	EPIWin	4
Benzidine	87.5	AWQC	0.0002
Benzotrichloride	200	EPIWin	0.002
Benzyl chloride	11.8	EPIWin	2.2
Bis(2-chloroethyl)ether	6.9	AWQC	0.6
Bis(2-chloroisopropyl)ether [or Bis(2-chloro-1-methylethyl)ether]	2.47	AWQC	25
Bis(2-ethylhexyl)phthalate [or DEHP]	130	AWQC	2.4
Chlorobenzilate	891	EPIWin	0.02
Chloronaphthalene, beta-	202	AWQC	1700
Cyhalothrin [or Karate]	1100	EPIWin	20
Dibromobenzene, 1,4-	165	EPIWin	260
Dichlorobenzene, 1,4-	55.6	AWQC	3.3
Dichlorobenzidine, 3,3'-	312	AWQC	0.03
Dichlorodiphenyldichloroethane, p,p'- [or DDD, 4,4'-]	53600	AWQC	0.0003
Dichlorodiphenyldichloroethylene, p,p'- [or DDE, 4,4'-]	53600	AWQC	0.0002
Dichloroethane, 1,2- [or EDC]	1.2	AWQC	40
Dichloropropane, 1,2-	4.1	AWQC	16
Dicofol [or Kelthane]	1460	EPIWin	0.007
Dimethylphenol, 3,4-	10.4	EPIWin	420
Dinitrotoluene, 2,6-	8.26	EPIWin	0.8
Dioxane, 1,4-	3.16	EPIWin	130
Dioxins, as total 2,3,7,8-TCDD equivalents	5000	AWQC	6E-09
Diphenylhydrazine, 1,2-	24.9	AWQC	0.2
Epichlorohydrin	3.16	EPIWin	140
Heptachlor epoxide	11200	AWQC	0.00004
Hexachlorobenzene	8690	AWQC	0.0003
Hexachlorocyclohexane, alpha- [or BHC, alpha-]	130	AWQC	0.005
Hexachloroethane	86.9	AWQC	3.6
Hexazinone	5.30	EPIWin	27000
Nitroso-diethylamine, N-	3.16	EPIWin	0.009
Nitroso-dimethylamine, N-	0.026	AWQC	3.3
Nitroso-di-n-butylamine, N-	21.1	EPIWin	0.04
Nitroso-di-n-propylamine, N-	1.13	AWQC	0.5
Nitroso-diphenylamine, N-	136	AWQC	6.5
Nitroso-N-methylethylamine, N-	3.16	EPIWin	0.06
Pentachlorobenzene	1910	EPIWin	1.8
Pentachloronitrobenzene	746	EPIWin	0.02
Simazine	4.56	EPIWin	8
Tetrachlorobenzene, 1,2,4,5-	746	EPIWin	1.7

Contaminant	BCF (L/kg)	Source of BCF Value	SWCTL (mcg/L)
Trichloroethane, 1,1,2-	4.5	AWQC	17
Trichloropropane, 1,2,3-	11.2	EPIWin	0.2
Trifluralin	2580	EPIWin	0.2
Vinyl chloride	1.17	AWQC	2.7

[a] Value estimated from K_{ow} data using USEPA's Estimation Program Interface Suite (EPIWIN).
[b] Value obtained from USEPA (2002c).

B. Aquatic Toxicity Criteria

The method for deriving standards from aquatic toxicity information is borrowed from Chapter 62- 777, F.A.C. and is presented in Figure 3B. Generally, toxicity information from aquatic animals is used to calculate SWCTLs. In some circumstances, data from aquatic plants can also be used. Basically, the procedure involves identifying the most sensitive relevant species and the median lethal concentration (LC_{50}) of the chemical in that species. The LC_{50} is then divided by 20 to obtain the SWCTL.

C. Comparing Site Contaminant Concentration Data with Surface Water Cleanup Target Levels

Concentrations of contaminants in each surface water sample shall be compared to the applicable (fresh or marine) surface water CTLs. Note that dilution to achieve surface water CTLs is not acceptable; consequently, the surfacewater CTLs must be met at all locations. When contaminated groundwater is discharging to surface water, surface water CLTs must be met in groundwater samples collected from monitioring wells located as close to the groundwater/surface water interface as is physically possible.

V. Development of Soil Cleanup Target Levels

Default SCTLs based on direct exposure and on leachability to groundwater or surface water are presented in Table 2. These are expressed in mg/kg dry weight and, therefore, the user must convert any wet weight concentrations to a dry weight basis before they are compared with the respective SCTL. The SCTLs presented here include several updates, including:

1) Default assumptions regarding gastrointestinal absorption have been changed to be consistent with new USEPA guidance. When chemical-specific values for gastrointestinal absorption are unavailable, a default gastrointestinal absorption value of 100% is used.
2) Several toxicity values provided by the USEPA have changed since the last report. These have been updated.
3) CTLs for new chemicals have been added.
4) The USEPA Technical Working Group for Lead has calculated new background blood lead concentrations for adult women. Calculation of the industrial SCTL for lead has been revised using these new data, following the Group's recommendations.

As with SCTLs previously developed, there are a number of limitations that are important to acknowledge:

1) The SCTL methods for direct human exposure presented in this report are based on protection of human health only. Soil contamination guidance concentrations to protect non-human species and ecosystems are very much dependent upon the site characteristics and species present and are therefore difficult to generalize. Under some circumstances, the SCTLs based on human health may not be protective of other species. For example, SCTLs for some metals (cadmium, mercury, nickel, selenium, and zinc) exceed concentrations shown to produce phytotoxicity (USEPA, 1996b).
2) The SCTL methodology described here is based on direct exposure and leachability only, and does not consider intake and human health risks that may occur via indirect pathways such as uptake into plants and animals that are used as a food source[1].
3) The SCTL methodology does not address odors or staining in soil. As such, depending upon the setting and the management of a site, the SCTLs described here may not address all of the potential issues of concern.

A. Development of Default SCTLs

1. Direct Contact SCTLs Based on Chronic Exposure

a) Equations for Calculating Direct Contact SCTLs

[1] Intake via food uptake is not regarded as a major exposure pathway for most contaminated sites. For special circumstances where individuals may make extensive use of crops or animals on contaminated soils, these SCTLs may not be appropriate.

The equations for calculating SCTLs based on direct contact are shown in Figures 4 and 5. These equations are functionally equivalent to those used by USEPA Region 9 in developing their preliminary remediation goals (USEPA, 2002b). One equation is provided for calculating SCTLs based on non-cancer health effects and another for calculating SCTLs based on cancer risk, if appropriate (i.e., if the chemical is regarded as a potential carcinogen). For chemicals with both cancer and non-cancer health effects, the SCTL is based on the most sensitive endpoint. Both equations consider intake from ingestion of contaminated soil, dermal contact with the soil, and inhalation of contaminants present in soil that have volatilized or have adhered to soil-derived particulates [dust]. The combined impact of exposure from all three routes[1] simultaneously is used to calculate the SCTL. For purposes of discussion, this is termed the multi-route approach.

In their *Soil Screening Guidance: Technical Background Document* (SSG, USEPA, 1996b) the USEPA has employed a somewhat different approach from the one used here. In the SSG, SSLs[2] for a chemical are calculated separately for ingestion and inhalation exposure, in what could be called a route-specific approach. In determining an SSL based on direct contact, the lower of the two values for a chemical would be selected. As a general rule, dermal intake is ignored unless there is evidence in the literature of substantial dermal absorption of the chemical (e.g., pentachlorophenol). In such instances, some adjustment of the SSL is made to account for this uptake.

The principal advantage of the multi-route approach is that it is easier to defend on conceptual grounds. An individual will be exposed to contaminated soil by all three routes simultaneously in the vast majority of cases. The multi-route approach considers the risk or hazard from a chemical to an individual to be the sum of the risks or hazards from each of these exposure routes. The route-specific approach, in contrast, considers the risk or hazard posed by each route of exposure in isolation and makes the implicit assumption that risks or hazards from exposure to a chemical by multiple routes are unrelated, even if they involve the same target organ. Such an argument could be made if the toxicity posed by the chemical is route-dependent (i.e., is associated specifically and exclusively with a particular route of exposure). This situation is seldom the case. For the vast majority of chemicals, the toxicity upon which the SSL/SCTL is based is systemic in nature. That is, the reference doses and slope factors used to calculate the soil values are based on systemic toxicity endpoints, and a chemical reaching the target organ from any and all routes is

[1] In this context, *route* refers to route of entry into the body, such as through dermal contact or inhalation. Pathway refers to the means by which chemicals in soil (or other environmental media) reach the body, such as volatilization into the air, direct contact with the skin, migration to groundwater that is used as a drinking water source, etc.

[2] The USEPA Soil Screening Guidance soil concentrations are termed Soil Screening Levels (SSLs). The Miami-Dade County soil values are termed Soil Cleanup Target Levels (SCTLs).

likely to contribute to toxicity[1]. Under these circumstances it is difficult to consider the risks from the various routes of exposure to be less than additive.

From a practical standpoint, the difference between the values derived for a given chemical by the multi-route and route-specific approaches is relatively small, provided both ingestion and inhalation toxicity values are available and the risk from dermal exposure is small. In basing an SSL on only one route of exposure, and ignoring other routes, the route-specific approach will tend to underestimate exposure and risk. Assuming that risks from dermal exposure are negligible and that the lower of the ingestion and inhalation SSLs is selected, the maximum underestimation of risk would be by a factor of two. This maximum underestimation would occur when ingestion and inhalation risks from a chemical in soil are equal. Under these circumstances, choosing either the ingestion or inhalation SSL as the value for that chemical will capture only 50% of the total risk. In situations where risk from soil contamination is dominated by one exposure route — ingestion, for example — ignoring other routes has little effect on risk, and the error introduced into health-based soil target level development by the route-specific approach is minimal. In this situation, the multi-route and route-specific approaches should yield comparable health-based soil target levels.

Although the difference between soil target levels calculated using the multi-route approach and those calculated using the route-specific approach may in theory be small, the latter approach may yield results not wholly compatible with baseline risk assessments. In baseline risk assessments, the hazard index for a chemical is calculated from the sum of the hazard quotients for each of the exposure routes. When a soil target level is based on exposure from only one of those routes, it can provide a different indication of hazard potential. To illustrate the potential problem, suppose a site has Chemical A in the soil at a concentration just below a soil target level developed using a route-specific approach. Because the concentration of Chemical A is below the target level, the risk assessor for the site might choose to drop it from the baseline risk assessment. If it is retained, however, its hazard index could be as high as 2.0 (based on the discussion in the preceding paragraph). Any value greater than 1.0 signals a possible non-cancer health problem. In this example, the use of a route-specific soil target level can allow the elimination from a baseline risk assessment of a chemical that would otherwise be flagged as posing a potentially unacceptable health risk. This inconsistency cannot occur for soil target levels developed using the multi-route approach since, like baseline risk assessments, they are based on risks summed from all relevant routes.

[1] The amount of chemical reaching the target organ can be affected by the route of entry through physiological processes such as extent of local vascularization, diffusional barriers, presence or absence of transport mechanisms, pre-systemic elimination, and distribution. Such differences can be taken into account through estimation of relative systemic bioavailability from different routes.

The multi-route approach does not preclude the development of soil target levels based on route-specific toxicity. For chemicals with toxicities unique and specific to certain routes of administration, the analysis may default to a route-specific approach. Perhaps the best example of this situation is toxicity resulting strictly from local effects at the site of contact (e.g., skin, gastrointestinal tract, or lungs). In this case, chemical exposure by other routes would probably not contribute to this toxicity, and risks for individual routes arguably should not be summed. In these instances, while the multi-route approach forces all routes to be considered, it results in a route-specifically determined soil target level.

In many cases it can be difficult to determine whether or not a toxicity value is route-specific. In the absence of definitive information, one approach is to infer route specificity when the target organ is the portal of entry for the administered dose (i.e., the GI tract in the case of ingestion and the pulmonary tract in the case of inhalation) in the study providing the toxicity information. While no doubt imperfect, this approach allows route specificity to be addressed in SCTL development for a broad range of chemicals.

Unlike the SSG, the approach presented here explicitly includes dermal exposure as a contributor to risk and a component of the SCTL for direct contact with soil. For most chemicals, the use of default assumptions regarding absorption through the skin demonstrates that contribution of this route to risk, and therefore SCTLs, is very small. This observation is consistent with the generally held notion that dermal absorption of chemicals present in soil is a minor exposure route for all but a few chemicals. Despite the typically small contribution of dermal exposure, it is nevertheless included in the SCTL equations for two reasons: 1) to make the equations complete with respect to potential exposure routes; and 2) to provide a mechanism to address those chemicals for which dermal absorption truly represents a significant exposure route.

The inhalation component of the equations presented in Figures 4 and 5 includes intake from airborne concentrations of chemicals resulting from volatilization as well as airborne dusts derived from contaminated soils. As noted in the SSG, inhalation of soil-derived particulates is a significant contributor to risk in only a few instances, such as the risk of cancer from hexavalent chromium. Volatilization is an issue only for chemicals with the appropriate physical/chemical properties. Consequently, when developing SSLs, the SSG evaluates separately particulate inhalation of non-volatile inorganics from surface soil, and volatilization of contaminants from subsurface soil. This approach requires the use of different equations for different chemicals, depending upon their classification or grouping. Rather than develop multiple equations, the approach taken in this report is to use a single equation each for cancer and non-cancer health effects, with the influence of physical/chemical properties on inhalation exposure considered through the input values selected for use in the equation rather than through changes in the equation

itself. The inhalation component for volatilization does not take into account volatilization from subsurface soil into structures through cracks in building foundations. If the possibility exists for this route of exposure, then potential volatilization into buildings should be assessed using models such as those developed by Johnson and Ettinger (1991).

b) Input Values for Direct Exposure

As can be seen in Figures 4 and 5, the calculation of direct contact SCTLs requires the selection of toxicity values, exposure variables, and several physical/chemical parameters for each chemical. The selection and development of toxicity values was discussed above in section II B. The following discussions present the approaches used for selecting exposure parameters for residential and industrial/commercial scenarios, and the selection or calculation of physical/chemical parameters for the contaminants considered herein.

Exposure parameters. Most sites can be evaluated using SCTLs based on either of two basic land uses — residential and industrial/commercial. In the case of residential land use, potentially exposed individuals include both children and adults. Only adult exposure to contaminated soil is assumed to exist for industrial/commercial land use[1].

Children are assumed to experience the greatest daily exposure to soil under residential land use scenarios. When risk is a function of the daily intake rate of a chemical (as in the evaluation of non-cancer health effects), SCTLs must be based on childhood exposure assumptions in order to be protective. When risk is a function of cumulative exposure (as in the evaluation of cancer risk), the exposure period for the residential scenario may cover time spent both as a child and as an adult. Of course, many physiological parameters such as body weight, surface area, and inhalation rate change with age. Other exposure parameters such as soil ingestion rate are also age-dependent. In this situation, time-weighted average values reflecting both childhood and adult exposures must be used in calculating SCTLs for residential land use. In this report, the individual exposed both as a child and as an adult is termed the *aggregate resident.*

For generic SCTLs (i.e., SCTLs applicable and protective for a broad range of sites), default exposure assumptions are available from the USEPA for both residential and commercial/industrial land uses. These are listed in Table 3. Some input parameters for the aggregate resident, such as inhalation rate and exposed dermal surface area, are not readily available from the USEPA and were developed from USEPA data sources. The values calculated

[1] For commercial uses involving significant regular contact by children, such as a school or daycare, residential rather than industrial/commercial SCTLs would be applicable.

for these parameters are also listed in Table 3, and the method of derivation is described in Appendix A.

In the case of soil ingestion rate for the aggregate resident, the USEPA calculates an age-adjusted soil ingestion rate based on a 30-year exposure period being divided into 6 years of consumption of 200 mg of soil per day at a body weight of 15 kg, followed by 24 years of consumption of 100 mg of soil per day at a body weight of 70 kg (see USEPA, 1996b, for more information on the calculation of this value). Although there is logic in this method of calculation, the use of this approach along with cancer slope factors to develop carcinogenicity-based SCTLs may be problematic. Specifically, the problem involves the way the body weight is used in the averaging process. When cancer slope factors are developed, the typical approach in determining dose is to use an average intake rate of the chemical divided by an average body weight over the exposure period, usually a lifetime in the case of rodent bioassays. To be strictly comparable, a similar approach should be used in the development of the aggregate resident (time-weighted average) soil ingestion rate for use in calculating SCTLs. That is, a time-weighted average soil ingestion rate is calculated and is then divided by a time-weighted annual average body to yield a time-weighted average soil ingestion rate, in mg soil/kg body weight/day. Aggregate resident values derived using this approach are employed in the calculation of residential SCTLs based on carcinogenicity. These values are listed in Table 3. The practical implications of this difference in time-weighted averaging is that, all other factors being equal, the SCTLs derived based on carcinogenicity are about two-fold higher than those calculated using the SSG approach.

The adherence factor (AF) represents the amount of soil that adheres to the skin per unit of surface area. Previously, the AF assumptions for residents and workers were taken from a range of values presented in the 1992 USEPA's document *Dermal Exposure Assessment: Principles and Applications* (USEPA, 1992). For the SCTLs presented here, a different method of selecting the AF is used, consistent with more recent USEPA guidance (RAGS Part E, USEPA, 2000b). The newer approach is based on studies demonstrating that the amount of soil adhering to the skin is different for different areas of the body. Data are now available regarding the soil loading that occurs on different regions of the skin associated with different activities. This information was used to derive weighted AF values for residents and workers, based on their anticipated activities and the areas of the body assumed to be exposed and available for soil contact. For example, as explained in Appendix A, the skin surface area assumed to be exposed for a child includes the head, forearms, hands, lower legs, and feet. Soil adherence data for these surfaces were averaged, weighting the contribution of the soil adherence for each part by its relative surface area. [Note: Soil adherence data were available for the face only, rather than the entire head. In weighting the soil adherence data, adherence data for the face were conservatively assumed to be applicable to

the entire head.] Adherence data were taken from the 95th percentile of observations of children playing at a daycare center, regarded as a typical (or central tendency) activity. The resulting weighted AF for a child resident (1 to 7 years of age) is 0.2 mg/cm^2. The same weighted AF is obtained if soil adherence data from the 50th percentile is used for a high-contact activity (i.e., children playing in wet soil). For older children and adult residents, calculation of SCTLs assumes that the head, forearms, hand, and lower legs are exposed. A different weighted AF is derived for these individuals, based both on different weighting from somewhat different surface areas exposed, as well as soil adherence data from different activities. In this case, soil adherence data from the 50th percentile of a high contact activity (gardening) was used to derive an AF of 0.07 mg/cm^2. For workers, the head, forearms, hands, and lower legs are assumed to be exposed. Soil adherence data for these surfaces from utility workers along with their respective surface areas were used to derive a weighted AF of 0.2 mg/cm^2 for the industrial/commercial worker scenario. Since the utility worker data were regarded as a high-end soil contact activity, 50th percentile values were used. For the aggregate resident, the AF for the child (0.2 mg/cm^2) and the adult (0.07 mg/cm^2) were time-weighted to derive an average ([(6 years x 0.2)+(24 years x 0.07)]/30 years) of 0.1 mg/cm^2.

One of the exposure variables, the particulate emission factor (PEF), is used to address intake from inhalation of contaminated soil-derived particulates. This value is a function both of site and local climatic conditions. The formula for calculating a PEF value is taken from the SSG (USEPA, 1996b) and appears in Figure 6. In calculating a PEF for Miami-Dade County, default parameters from the SSG were used except for the soil particulate dispersion coefficient (Q/C) term. The SSG selected as default a Q/C for 0.5 acres of contaminated soil in Los Angeles, CA. In order to make the default PEF more relevant to Miami-Dade County climatic conditions, a Q/C for 0.5 acres in Miami[1] is used instead.

Another input parameter used to assess the soil-to-air pathway of exposure is the volatilization factor (VF). This term is used to define the relationship between the concentration of the chemical in soil and the flux of the volatilized chemical to air. The VF is calculated using an equation from the SSG as shown in Figure 7. Parameters related to characteristics of both the chemical and the soil are used in the calculation of a VF. For the purposes of establishing default SCTLs, default soil characteristics specified in the SSG have been adopted, although it is recognized that the relevant characteristics can vary widely among soils. As discussed above, a Q/C for Miami is used rather than the default Q/C from the SSG, which is based on meteorological conditions in Southern California.

[1] The only city in Florida for which a modeled Q/C value is presented in the SSG.

The default exposure assumptions identified in Table 3 are intended to be health protective under circumstances of chronic exposure. Site-specific conditions may restrict exposure to such an extent that the default assumptions are not valid, and the acceptable risk levels (i.e., excess lifetime cancer risk of 1E-06 or less, and a hazard index of 1 or less) can be achieved with higher SCTLs. On the other hand, there may be situations in which exposure exceeds the default assumptions employed in developing generic SCTLs, e.g., workers with extensive soil contact and opportunity for exposure, such as construction workers involved in excavation, or children with soil pica. For these sites, the SCTLs may not be sufficiently protective. Whenever generic SCTLs are used for site evaluation, it is important to verify, to the extent possible, that the default assumptions upon which they are based are neither greatly above nor below actual present and predicted future exposure conditions.

Physical/chemical parameters. The equations for the calculation of SCTLs for direct contact require the input of several chemical-specific values. These values, which include the organic carbon normalized soil-water partition coefficient for organic compounds (K_{oc}), Henry's Law constant (HLC), diffusivity in air (D_i), and diffusivity in water (D_w), are a function of the physical/chemical properties of each chemical. In some cases, it may be necessary to calculate these values when published values do not exist. In these cases, additional physical/chemical values such as density (d), water solubility (S), vapor pressure (VP), or adsorption coefficient (K) are needed. In addition, the physical state of a chemical at ambient soil temperatures is an important parameter when determining the soil saturation limit (C_{sat}) for that chemical. The melting point (MP) is needed for this purpose. There are many sources for physical/chemical parameter values, but unfortunately the values listed in various sources can sometimes differ. In order to foster consistency in the development of SCTLs, it is important to have a designated hierarchy of sources for the selection of physical/chemical values.

In agreement with the SSG, chemical-specific values for MP[1], d, S, HLC, and K_{oc} are preferentially selected from the Superfund Chemical Data Matrix (SCDM) (EPA/540/R-96/028). The SCDM is a database that can be accessed and downloaded via the Internet. The SCDM database is composed of information selected from specified literature sources or other databases, and calculated values. The SCDM then ranks those values that reasonably apply to a hazardous substance and reports a single value for each of the physical/chemical parameters.

[1] MP was not available for all chemicals. If a specific MP could not be found in any of the reference sources, but a source listed it as a liquid, a default MP of –9.99 °C was assigned.

When data for these parameters are unavailable from the SCDM, the Hazardous Substance Data Bank (HSDB)[1] and the Estimation Program Interface Suite (EPIWIN) are used. EPIWIN is a recently developed Windows-based suite of physical/chemical property and environmental fate estimation models created by the USEPA's Office of Pollution Prevention Toxics and Syracuse Research Corporation. EPIWIN uses a single input to run estimation models predictive of MP, BP, S, HLC, and K_{oc}. Most useful is the fact that this suite also includes a database containing physical-chemical parameters for more than 25,000 chemicals. If these sources are exhausted, then K_{oc} values are calculated from K_d values in the SCDM according to equation (1) below, or by obtaining the geometric mean of values presented in the HSDB. Additionally, ATSDR Toxicological Profiles or other reference texts are used. If data for d are not available from any of these sources, these values can be calculated using equation (2) below.

The primary source of diffusivity values is the CHEM9 database. Some values have changed from the previous version (CHEMDAT8) and some chemicals have been added. If diffusivity values are not provided in the CHEM9 database, they can be calculated using equations 3, 4 and 5 below taken from the literature accompanying this database.

To summarize, the following is the list of sources (in order of preference) for the chemical/physical parameters used in the development of the SCTLs.

For HLC, d, S, and MP

1. The Superfund Chemical Data Matrix (SCDM)
2. The Hazardous Substances Data Bank (HSDB)
3. The Estimation Program Interface for Windows (EPIWIN)
4. The Agency for Toxic Substances and Disease Registry's Toxicological Profiles (ATSDR)
5. Reference texts [e.g., *CRC Handbook of Chemistry and Physics* (Lide and Frederikse, 1994); CRC *Groundwater Chemicals Desk Reference* (Montgomery, 2000); *Handbook of Environmental Data on Organic Chemicals* (Verschueren, 1996); *Handbook of Environmental Fate and Exposure Data for Organic Chemicals*, Volumes I-V (Howard, 1989, 1990, 1991, 1993, 1997); *Handbook of Physical Properties of Organic Chemicals* (Howard and Meylan, 1997); *Illustrated Handbook of Physical Chemical Properties and Environmental Fate for Organic Chemicals*, Volumes I-V (Mackay et al., 1992a,b, 1993, 1995, 1997)]
6. Values calculated using equations from reference texts [e.g., *Chemical Property Estimation* (Baum, 1998)].

[1] For some chemicals, the HSDB reports several values for one or more of the physical/chemical parameters (e.g., S, K_{oc}, MP). Rather than choosing a single value from the range of reported values, a geometric mean was calculated from all the values. This is noted in Table 4 (Chemical-Specific Values) with the notation "HSDB-GeoMean."

For Koc[1]:

1. Superfund Chemical Data Matrix (SCDM)
2. Calculated from the K_d published in SCDM using the following equation:

$$K_{oc}=K_d/0.002 \quad (1)$$

3. The Estimation Program Interface Suite for Windows (EPIWIN)
4. The Hazardous Substances Data Bank (HSDB)
5. The Agency for Toxic Substances and Disease Registry's Toxicological Profiles (ATSDR)
6. Reference texts (see reference texts listed above)

For density (d):

1. The Hazardous Substances Data Bank (HSDB)
2. Calculated using the following equation:

$$d = \frac{MW}{5\sum_i n_i \times v_{a,i}} \quad (2)$$

where,

MW = molecular weight of chemical (g/mol)
n_i = number of atoms i in a molecule
$v_{a,i}$ = relative volume of atom i (cm3/mol)

source: Baum (1998)

For D_i and D_w:

1. The CHEM9 database
2. Calculated using equations identified in the CHEM9 database support document and shown below:

For diffusivity in air (D_i):

For compounds with a MW ≤ 100

$$D_i = 0.0067\ T^{1.5} \times (0.034 + MW^{-1})^{0.5} \times MW^{-0.17} \times [(MW / 2.5\ d)^{0.33} + 1.81]^{-2} \quad (3)$$

For compounds with a MW > 100

$$D_i = 0.0067\ T^{1.5} \times (0.034 + MW^{-1})^{0.5} \times MW^{-1.7} \times [(MW / 2.5\ d)^{0.33} + 1.81]^{2} \quad (4)$$

[1] The K_{oc} and Kd parameters are used in the development of SCTLs based on leaching to groundwater. In the case of some inorganic chemicals, the SSG developed Kd's using the MINTEQ model and used them to generate soil screening levels for leaching to groundwater. For those chemicals, the SSG leachability value was cited in the Code, rather than a value based on the Kd from the SCDM.

where,

T = temperature, degrees Kelvin
MW = molecular weight of chemical (g/mol)
d = density of liquid chemical (g/cm^3)

For diffusivity in water (D_w):

$$D_w = 1.518 \times (10^{-4}) \times V_{cm}^{-0.6} \quad (5)$$

where,

V_{cm} = molar volume of chemical (cm^3/mol)

The precision with which the values from the various reference sources are reported can vary. In order to foster consistency in the development of SCTLs, it is important to have a designated rounding policy for the physical/chemical values. The precision to which values from reference sources were used in calculating the SCTLs are listed in Table 12.

Table 12
Input Precision for Physical/Chemical Parameters

Parameter	Numerical Precision
MW	2 decimal places
d	4 decimal places
HLC	3 significant figures
S	2 significant figures
MP	1 decimal place
K_{oc}	2 decimal places
D_i	3 significant figures
D_w	3 significant figures

The physical/chemical parameters for chemicals specifically listed in this report are provided in Table 4.

For a limited number of contaminants, the hierarchy of sources of physical/chemical values listed above was exhausted without finding a value for one or more of the required parameters. As noted previously, some density values were calculated using equations available in reference texts. Table 13 lists the calculated density values (d) for some chemicals.

Table 13
Calculated Density Values for Some Chemicals

Contaminant	Calculated Density
ammonium sulfamate	1.2945
benomyl	1.2582
benzo(g,h,i)perylene	1.2830
bromoxynil	1.7406
p-chloro-m-cresol	1.2674
dimethoxybenzidine, 3,3'-	1.2215
dimethylaniline, N,N-	1.9193
dimethylbenzidine, 3,3'-	1.1500
diuron	1.3320
ethylene thiourea	1.0215
ethylphthalyl ethylglycolate	1.1010
fluoridone	1.3810
heptachlor epoxide	1.5219
hexachlorophene	1.7633
linuron	1.3588
propionic acid, 2-(2-methyl-4-chlorophenoxy)	1.5082

There were also nine chemicals for which surrogate density values were used. Surrogate density values were considered appropriate only when the density of an isomer of the chemical in question was available in the hierarchy of physical/chemical sources. Table 14 lists the chemicals for which surrogate density values were used, the value, and the source of the surrogate value.

Table 14
Surrogate Density Values for Some Chemicals

Contaminant	Surrogate Density Value	Surrogate Contaminant
benzo(b)fluoranthene	1.3510	benzo(a)pyrene
benzo(k)fluoranthene	1.3510	benzo(a)pyrene
dichlorophenol, 2,3-	1.3830	dichlorophenol, 2,4-
dichlorophenol, 2,5-	1.3830	dichlorophenol, 2,4-
dichlorophenol, 2,6-	1.3830	dichlorophenol, 2,4-
dichlorophenol, 3,4-	1.3830	dichlorophenol, 2,4-
hexachlorocyclohexane, delta	1.8900	hexachlorocyclohexane, beta
indeno(1,2,3-cd)pyrene	1.3510	benzo(a)pyrene
phenylenediamine, p-	1.0096	phenylenediamine, m-

2. Development of Acute Toxicity SCTLs for Some Chemicals in Chapter 24, Miami-Dade County Code

Default residential direct exposure SCTLs for non-carcinogenic chemicals are typically developed based on assumptions of chronic exposure, and are intended to be health protective for both children and adults. While it is generally assumed that these contaminant concentration limits

are protective for acute as well as chronic exposure, there may be circumstances where acute exposure is significantly larger than time-averaged chronic exposure. This larger exposure could result in acute toxicity.

A striking example of this situation can be seen with soil ingestion rates in children. While most children may ingest up to 200 mg of soil per day on average (the standard USEPA default assumption), in some instances episodic ingestion can be 250 times that amount or more. Wong et al. (1988) measured soil ingestion in children of normal mental capacity one day per month for four months. They found that five of the 24 children ingested > 1 g of soil on at least one of the four observation days, ranging from 3.8 to 60.7 g. Stanek and Calabrese (1995) used data from soil ingestion studies to develop a model to predict soil ingestion patterns in children. The results of this model indicated that "the majority (62%) of children will ingest > 1 g soil on 1-2 days/year, while 42% and 33% of children were estimated to ingest > 5 and > 10 g soil on 1-2 days/year, respectively." Although a soil ingestion rate of 5 g soil/day has been proposed by the USEPA (USEPA, 1986) to address the possibility that some children may exhibit soil pica (ingestion) in quantities far greater than the 200 mg/day value, this approach is regularly disregarded in practice. To prevent this oversight when assessing a site whose current or future uses may include contact with soil by small children, the potential for acute toxicity must be adequately addressed in the development of SCTLs.

Calabrese et al. (1997) evaluated the potential for acute toxicity from a pica episode involving soil with contaminant concentrations regarded by the USEPA as conservative (i.e., at or below the USEPA Soil Screening Levels and USEPA Region 3 Risk-Based Soil Concentrations). Contaminant doses expected to result from a one-time soil pica episode of 5 to 50 g of soil were estimated and compared with acute doses demonstrated to produce toxicity in humans in poisoning episodes. The findings indicated that some residential soil cleanup target levels could result, following a single large soil ingestion event, in doses in the range reported to produce acute toxicity, and even death. Of the thirteen chemicals included in the analysis, ingestion of soil containing cyanide, fluoride, phenol, or vanadium was found to result in a contaminant dose exceeding a reported acute human lethal dose. Ingestion of barium, cadmium, copper, lead, or nickel from soil was found to produce doses associated with acute toxicity other than death.

Although the selective use of human data contributes greater confidence in the relevance and implications of these findings, it is important to acknowledge the limitations associated with this analysis. Estimates of the acute toxic and lethal doses were primarily extrapolated from reports on accidental ingestion, and exact dose estimation was difficult. In addition, most incidents of exposure were limited to adults; doses were then modified to approximate equivalent doses for children. Doses reported to produce toxicity in humans indicate only that the dose needed to cause

the effect was met or exceeded; that is, they can only be used to approximate a Lowest Observed Adverse Effect Level (LOAEL). For most of the effects of interest, data were insufficient to establish a No Observed Adverse Effect Level (NOAEL). Some case reports in the literature may represent sensitive individuals and therefore the extent to which dose-response information from these cases applies to the general population is uncertain. Also, the doses in this analysis were ingested doses rather than absorbed doses, and in many cases involved solutions from which absorption may be extensive. The presence of these contaminants in soil may reduce their bioavailability, and therefore their toxicity. Despite these limitations, the serious nature of acute toxicity potentially associated with consumption of contaminated soil during a soil pica episode requires that attention be paid to this issue when developing residential soil cleanup target levels. The USEPA has acknowledged in the *Soil Screening Guidance: Technical Background Document* (USEPA, 1996b) that their residential screening values for cyanide and phenol may not be protective of small children in the event of acute soil ingestion episodes, but provides no guidance on how to address this problem.

a) Equation for Calculating Acute Toxicity SCTLs

The chemicals identified by Calabrese et al. (1997) as having the potential to produce an acute toxicity problem were evaluated for Chapter 24 to determine whether an adjustment in the residential SCTL was required. Because the intake under these circumstances would be driven almost exclusively by ingestion, the SCTL equation was altered to remove the dermal contact and inhalation components. Also, because the value is based on a single exposure event, terms related to averaging time and exposure frequency were deleted to produce the following equation:

$$SCTL = \frac{BW}{\frac{1}{RfD_{acute}} \times SI \times CF}$$

where,

BW = body weight (kg)

RfD_{acute} = safe dose for acute exposure (mg/kg)

SI = amount of soil ingested (g)

CF = conversion factor for units (kg/g) (10^{-3})

Consistent with other SCTLs based on exposure of a child, a body weight of 16.8 kg was assumed. The amount of soil ingested per event (SI) was assumed to be 10 g, in order to make the derivation of acute toxicity SCTLs not excessively conservative. This value is well within the range of observations reported by Calabrese and others for single soil pica events. In addition, a

recent USEPA external review draft document also recommends 10 g as a reasonable value for use in acute exposure assessments (USEPA, 2000c).

b) Development of Acute Toxicity Values

Unfortunately, safe doses intended specifically for acute exposures are not provided by the USEPA. An analysis was therefore required in order to develop RfD_{acute} values for each of the eight chemicals of interest — barium, cadmium, copper, cyanide, fluoride, nickel, phenol, and vanadium. The analysis focused primarily on studies and reports of poisonings in humans. For most of these chemicals, there is little in the way of acute toxicity studies in animals, and the studies that exist tend to focus on severe endpoints (e.g., death) and are of limited value in identifying lesser effects that still may be of concern. In addition, the use of human data avoids the uncertainty inherent to extrapolating observations across species.

The principal objective of the literature analysis was to identify the acute LOAEL or NOAEL for each chemical. Initially, this dose was then divided by an uncertainty factor (UF) and/or modifying factor (MF) to produce a tentative acute toxicity reference dose (RfD_{acute}), analogous to the procedure used by the USEPA to develop chronic RfDs. UFs are intended to offer a safety margin in the face of uncertainty regarding extrapolation of doses (e.g., from animals to humans, from healthy subjects to sensitive subjects, etc.) and MFs can be applied to extend the safety margin when the database available for assessment is limited or weak. The calculated RfD_{acute} was then compared with the USEPA chronic oral RfD for that chemical or, in the case of copper, with dietary allowance guidelines. For many of the chemicals (e.g., cyanide), the calculated RfD_{acute} was lower than the USEPA chronic RfD for that chemical. This result represents an apparent conflict, since a dose that is safe to receive every day for a lifetime (i.e., the chronic RfD) should also be safe to receive on a single occasion. To avoid this conflict, the USEPA chronic RfD was adopted as the RfD_{acute} in these situations. Similarly, in the case of copper, application of any UF or MF other than 1.0 to an acute LOAEL resulted in a calculated RfD_{acute} lower than dietary allowance recommendations. As explained below (under "Copper"), the RfD_{acute} for copper was set at its upper limit for dietary intake in small children.

The appropriate doses representing the NOAEL or LOAEL for each chemical, as well as the appropriate UF and MF to be applied, were discussed by the FDEP-sponsored Methodology Focus Group of the Contaminated Soils Forum, and in some cases modifications were recommended from values used in the previous, May 1999 FDEP technical support document. The values presented in this report reflect the recommendations of the Methodology Focus Group. As before, a distinction was made in the application of "safety factors" depending upon the toxic endpoint. Specifically, if the RfD_{acute} was based on transient gastrointestinal distress, a lower factor

(UF and/or MF) was applied as compared with more serious toxic endpoints. This procedure reflects FDEP's risk management position, which DERM will maintain for consistency, that for acute soil ingestion, some risk of transient gastrointestinal distress is acceptable, but the SCTLs should be fully protective against more serious toxicity (including more serious gastrointestinal effects).

A brief summary of the analysis for each of the eight chemicals appears below:

Barium. The toxicity of barium is very much dependent upon the solubility of the barium salt being considered. Barium sulfate, for example, is insoluble in water, is poorly absorbed, and is used safely in medicine as a radiocontrast medium. Soluble barium salts, however, are quite toxic and have been used as rodenticides. Numerous poisonings with soluble forms of barium have been reported in the medical literature. Some have resulted from accidental ingestion, suicide attempts, or mistaken use of a soluble form of barium for medical procedures (e.g., barium sulfide instead of barium sulfate). Perhaps the most significant reported incident of accidental poisoning with barium occurred when 144 persons ingested barium carbonate that was mistakenly substituted for potato starch in the preparation of sausage (Lewi et al., 1964; Ogen et al., 1967). Among the individuals poisoned, 19 were hospitalized and one died. Vomiting, abdominal pain and spasms, diarrhea, weakness, hypokalemia (decreased blood potassium levels), cardiac arrhythmias, paresthesias (abnormal sensation such as tingling), and muscle paralysis are typical signs and symptoms of barium poisoning (Ellenhorn et al., 1997). For barium carbonate, the lowest reported acute lethal dose is 57 mg/kg, and the lowest reported toxic dose is 29 mg/kg (Ellenhorn et al., 1997). Effects at this lowest toxic dose include flaccid paralysis, weakness, and paresthesia. Barium chloride appears to be somewhat more toxic. The lowest lethal dose is reported to be 11 mg/kg (Ellenhorn et al., 1997). McNally (1925) stated, "Kobert believes that under certain conditions, 2 g (barium) would be fatal. The toxic dose he believes to be 0.2 g." The latter value, which corresponds to about 3 mg/kg in a 70 kg adult, is similar to the threshold toxic dose of soluble barium compounds of 200-500 mg (i.e., 3-7 mg/kg), reported by the World Health Organization (WHO, 1991). Unfortunately, the symptoms that constitute this reported threshold for toxic effects are unclear, and there is no clear distinction in the literature between doses that cause gastrointestinal symptoms and those producing more serious effects like paresthesia, muscle paralysis, and cardiac arrhythmia. The principal action of barium contributing to neuromuscular and cardiac toxicity is dysregulation of potassium. Experiments in dogs have found that an intravenous dose of 0.022 to 0.154 mg/kg produces significant decreases in serum potassium and the appearance of abnormal electrocardiograms (Roza and Berman, 1971). This result suggests that the 3 mg/kg threshold dose applies equally to neuromuscular and cardiotoxicity, as well as to gastrointestinal effects.

Application of a UF of 100 (10 for sensitive subjects and 10 for extrapolation from a LOAEL to a NOAEL) to a LOAEL of 3.0 mg/kg yields a dose of 0.03 mg/kg. This value is lower than the current USEPA chronic oral RfD of 0.07 mg/kg-day. The value for the chronic oral RfD was therefore selected as the RfD_{acute}, resulting in an acute toxicity SCTL for barium of 120 mg/kg.

Cadmium. With chronic exposure, the health effects of primary concern are renal toxicity and lung cancer. Both require long-term exposure, and neither is an issue with acute (one time) ingestion of cadmium. The health effects occurring at the lowest acute dosages are primarily gastrointestinal — nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea (ATSDR, 1997a). Several cases of acute cadmium poisoning occurred during the 1940s and 1950s when cadmium was substituted for scarce chromium in plating cooking utensils and containers. In one report, two adults and four children experienced vomiting and cramps after drinking tea from a pitcher plated on the inside with cadmium (Frant and Kleeman, 1941). From information provided in their report, doses ranging from 0.2 to 1 mg/kg can be calculated. Other studies have reported that doses as low as 0.04 to 0.07 mg/kg cadmium are capable of inducing vomiting (Nordberg et al., 1973; and Lauwerys, 1979; as cited in ATSDR, 1997a). In all cases of cadmium ingestion within this dose range, recovery was rapid and complete, usually within 24 hours.

From these studies, it appears that the LOAEL for vomiting is about 0.05 mg/kg. Because the endpoint was gastrointestinal distress and the effect temporary, a UF and MF of 1 were applied. Using this value as the RfD_{acute}, a SCTL of 84 mg/kg is calculated. This value is slightly higher than the residential SCTL for cadmium based on chronic exposure (82 mg/kg), which was adopted as the residential SCTL for cadmium to protect against toxicity from both acute and chronic exposure.

Copper. Several studies have reported that ingestion of drinking water or beverages with elevated copper concentrations results in gastrointestinal effects including nausea, vomiting, diarrhea, and abdominal pain (Knobeloch et al., 1994; Sidhu et al., 1995; ATSDR, 1990a). In fact, copper sulfate was used historically in medicine to induce vomiting (Goodman and Gillman, 1941). Three separate reports provide relatively consistent information regarding the doses of copper required to produce these effects. In one report, military nurses experienced nausea, vomiting, and diarrhea within 30 minutes to one hour after consuming cocktails from a copper-lined shaker (Wyllie, 1957). All but five of the 15 nurses experienced weakness, abdominal cramps, dizziness, and headache the next day. Reconstruction of the cocktail mixture and measurement of copper concentrations, coupled with consumption estimates for each of the nurses, can be used to derive copper dose estimates. The lowest dose (received by three of the nurses who became sick), was 0.09 mg/kg. Nicholas (1968) reported an incident in which 20 workmen became sick after drinking tea at work that contained 30 mg/L copper. All experienced nausea and several had diarrhea, with

or without vomiting. The estimated dose of copper was 0.07 mg/kg. Spitalny et al. (1984) reported recurrent, acute gastrointestinal symptoms including nausea, vomiting, and abdominal pain in a family associated with drinking copper-contaminated well water, or beverages (juice or coffee) made with the water. Based on the concentration of copper in the water (7.8 mg/L), a copper dose of 0.06 mg/kg is estimated. It is not clear whether children have increased sensitivity to gastrointestinal irritation from copper. One study of gastrointestinal complaints from copper in drinking water in two communities in Wisconsin found a greater prevalence of symptoms in children, but this difference could have resulted from higher exposures than adults (Knobeloch et al., 1994).

The acute gastrointestinal effects of copper in drinking water were investigated in a well-controlled prospective study (Pizarro et al., 1999). Sixty healthy adult women were randomly assigned drinking water containing 0, 1, 3, or 5 mg Cu/L for one-week intervals. During the study, the participants were reassigned into a different consumption group so that each individual received one week of water at each of the exposure levels. At 3 mg/L Cu in water, a significant increase in gastrointestinal symptoms (nausea, abdominal pain, and vomiting) was reported. Using the mean water consumption (1.64 L/d) and body weight (63.6 kg) reported in the study, this concentration corresponds to a gastrointestinal effects dose of 0.077 mg/kg.

Copper is considered to be an essential element, and various recommendations for daily copper intake are only slightly below values shown to produce gastrointestinal distress. A WHO expert committee has recommended intake of 0.08 mg/kg-day for infants and children (as cited in NRC, 1989), and the American Academy of Pediatrics has recommended inclusion of copper in infant formulas that could result in approximately 0.4 mg copper per day (as cited in NRC, 1989). However, even while recognizing the nutritional importance of copper, health agencies caution against too much intake. A WHO/FAO guidance document - *Trace Elements in Human Nutrition and Health* (WHO, 1996) - discusses nutritional copper requirements in children and sets an upper limit of the safe range of copper intakes for children ages 1 to 6 years old of 0.09 mg/kg.

The best dose-response data for gastrointestinal distress from copper come from the study by Pizarro et al. (1999), and indicate a LOAEL of about 0.08 mg/kg. Application of a UF and MF of 1 (based on transient gastrointestinal distress as the endpoint) would yield a calculated RfD_{acute} of 0.08 mg/kg. Since this value is within dietary allowance limits for copper, the WHO-recommended copper intake limit of 0.09 mg/kg-day for small children was selected instead as the RfD_{acute}. This intake limit results in an acute toxicity residential SCTL for copper of 150 mg/kg.

Cyanide. Cyanide is a potent and rapid-acting toxicant that has been involved in numerous intentional and accidental poisonings. The ATSDR reviewed the medical literature and determined that the average fatal dose of cyanide is 1.52 mg/kg (ATSDR, 1997b). The lowest human lethal

dose reported in the medical literature is 0.56 mg/kg (Gettler and Baine, 1938). Comparisons of acute oral toxicity data (with lethality as the endpoint) indicate that the toxicity of potassium cyanide, sodium cyanide, and hydrogen cyanide are similar on a molar basis. Symptoms of cyanide poisoning include anxiety, confusion, vertigo, and giddiness. Severe cases can result in loss of consciousness followed by convulsions, involuntary defecation, and death from respiratory failure (Gosselin et al., 1984). While clinical experience with cyanide is extensive, an upper-bound no-effect level has not been identified in humans. Any dose of cyanide capable of producing symptoms is potentially serious and medical attention will be required.

Clearly the best dose-toxicity information for cyanide exists for death as an endpoint, and when deriving an acute toxicity SCTL for cyanide, the exceptional toxicity and steep dose-response curve of this chemical must be taken into consideration. There is no standard set of uncertainty factors to develop a safe dose based on a lethal dose, particularly one established in humans. Extrapolating from the average human lethal dose (approx. 1.5 mg/kg) places the safe acute dose below the USEPA chronic reference dose (0.02 mg/kg-day), even if a UF as small as 100 is used. There is little logic in placing the safe acute dose lower than the safe chronic dose used for risk calculations, and so the RfD_{acute} for cyanide was placed at a value equal to the USEPA chronic RfD. This procedure results in an acute toxicity SCTL for cyanide of 34 mg/kg.

Fluoride. Because of the widespread use of fluoride compounds as supplements to municipal water supplies for the prevention of dental caries, there is substantial information available regarding the effects of fluoride in humans. Malfunctioning fluoridation equipment is often the cause of fluoride intoxications. In an elementary school, 34 children became ill from ingestion of over-fluorinated water (Hoffman et al., 1980). The intakes were estimated to range from 1.4 to 90 mg fluoride (based on a 20 kg body weight, which would result in an upper-end dose of 4.5 mg/kg). In another case, 22 adults became ill after ingesting water containing 1,041 mg/L fluoride (Vogt et al., 1982). Doses producing nausea alone were estimated at 1.2 mg/kg. More severe gastrointestinal symptoms were reported in those individuals who received doses of 2-3 mg/kg.

Fluoride supplements are often recommended for children who do not live in an area served by a fluorinated water supply. These tablets are typically flavored to aid in compliance and represent an important cause of accidental poisonings in the home. Spoerke et al. (1980) reviewed 150 reported cases of accidental poisonings with fluoride and found that a dose below 5 mg (absolute dose, not mg/kg) produced no gastrointestinal symptoms. These authors also found that a dose of 5-9 mg produced gastrointestinal symptoms in 10% of individuals, while 10-19, 20-29, and 30-39 mg caused symptoms in 21%, 50%, and 100% of individuals, respectively. Augenstein et al. (1991) reviewed the medical records of children referred to the Rocky Mountain Poison Control

Center for accidental fluoride ingestion. Of the 87 children included in the study, 70 had intake estimates sufficient to construct a dose response. Gastrointestinal symptoms predominated and included nausea, vomiting, diarrhea, abdominal pain, and lethargy. Percentages of symptomatic patients, as a function of dose, were: < 1 mg/kg fluoride, 8%; 1-2 mg/kg fluoride, 17%; 2-3 mg/kg fluoride, 27%; 3-4 mg/kg fluoride, 50%; and 4-8.4 mg/kg fluoride, 100%.

Gastrointestinal symptoms from acute fluoride ingestion arise because fluoride is corrosive to the gastrointestinal tract. At higher doses, more severe toxicity can occur, including hypocalcemia, hyperkalemia, cardiac arrhythmias, muscle spasm, tetany, and convulsions (Spoerke et al., 1980; Augenstein et al., 1991).

Emergency medicine and toxicology texts often make recommendations about treatment options and dosages expected to produce serious adverse effects. Ellenhorn et al. (1997) suggested seeking immediate medical treatment for doses of fluoride exceeding 5 mg/kg. This is the same fluoride dose for which the CDC recommends prompt medical treatment (CDC, 1995). Estimates of the lethal dose of fluoride in adults vary widely in the literature ranging from approximately 32 to 64 mg/kg. However, a 3-year-old weighing 12.5 kg died after ingesting 200 mg fluoride (16 mg/kg). The lowest reported fatality from fluoride was in a boy of 27 months who died after ingestion of 50 mg of fluoride (Anonymous, 1979). Based on the mean body weight for his age (12 kg) the fatal dose was only 4 mg/kg. Two factors may have contributed to the severity of his reaction — the mother had been taking fluoride tablets during pregnancy and the child had received daily fluoride supplements (0.5 mg) for the 15 months prior to his death.

In developing a RfD_{acute} for fluoride, a 5 mg/kg dose was selected as the starting point. This is the dose above which clinical texts recommend seeking medical attention. Even though this guidance value is intended to be applicable to the general population, it was divided by a UF of 10 (for sensitive individuals) to yield a RfD_{acute} of 0.5 mg/kg. The acute toxicity SCTL corresponding to this dose is 840 mg/kg. According to the study by Augenstein et al. (1991), the dose of fluoride in 10 g of soil at this concentration (0.5 mg/kg) would be expected to produce gastrointestinal symptoms in only a small percentage of children.

Nickel. There is only one report of death from acute ingestion of nickel. A 2-year old child ingested nickel sulfate crystals (570 mg/kg) and died from cardiac arrest eight hours later (Daldrup et al., 1986). Sunderman et al. (1988) reported an incident in which 32 individuals drank from a water fountain contaminated with nickel sulfate and nickel chloride. It was estimated that the ingested doses ranged between 0.5 to 2.5 g of nickel. Twenty workers promptly developed symptoms of gastrointestinal distress including nausea, vomiting and abdominal cramps. Systemic effects included episodes of giddiness, lassitude, headache and cough. The lower end of the dose associated with adverse side effects was 7 mg/kg (assuming a 70 kg body weight).

The acute toxicity SCTL for nickel is based on a LOAEL of 7 mg/kg from the Sunderman study. As with cadmium and copper, the toxic endpoint for the LOAEL is gastrointestinal effects. However, unlike the gastrointestinal effects associated with the LOAEL for these other chemicals, the LOAEL for nickel came from a study in which 10 out of 20 of the poisoned individuals were hospitalized. Given this information, the LOAEL for nickel (unlike cadmium and copper) was divided by a UF of 10. It was also divided by an additional MF of 3, given the limited data upon which the LOAEL is based. This approach results in an RfD_{acute} of 0.2 mg/kg (0.23 rounded to one significant figure) for nickel. The corresponding SCTL for nickel is 340 mg/kg.

In discussing the development of risk-based criteria for nickel in soils, it is worth noting that gastrointestinal effects are not the most sensitive effects of nickel. Nickel ingestion has been shown to produce dermal hypersensitivity reactions in individuals with nickel sensitivity. Nickel sensitivity appears to exist in about 10% of women and 1% of men. Nickel exposure in these individuals via the inhalation, dermal, or oral route results in dermal responses characterized by eczema, erythema, and dermal eruptions. Several clinical studies document the exacerbation of eczema and dermal eruptions following ingestion of nickel. Cronin et al. (1980) observed worsening of hand eczema in nickel-sensitive women from a single oral dose of as little as 0.6 mg nickel in solution. A Study by Burrows et al. (1981) suggests that the NOAEL may be 0.5 mg nickel. Gawkrodger et al. (1986) reported that a single dose of nickel produced dermatitis, eczema, and measle-like eruptions on the limbs of women previously sensitized. All of the women responded to 5.6 mg, the dose they identified as the LOAEL from their study. Protection against dermal hypersensitivity reactions from nickel would require a RfD_{acute} lower than the current USEPA chronic oral RfD. In fact, the USEPA acknowledges in their IRIS record for nickel that the chronic oral RfD is probably adequate to prevent the development of nickel hypersensitivity, but may not protect nickel sensitive individuals from experiencing reactions at this dose.

Phenol. Acute ingestion of non-fatal doses of phenol results in burning mouth and gastrointestinal irritation and distress (Deichman, 1969). Bennett et al. (1950) reported an acute lethal dose of 230 mg/kg for an adult. Deichman (1969) reported the lethal range for adults to be between 14.3 mg/kg and 143 mg/kg. Interestingly, there is also a report of an ingestion of 14 mg/kg that caused only gastrointestinal effects (Cleland and Kingsbury, 1977). Intake of water contaminated with phenol for a period of several weeks resulted in diarrhea, burning mouth, and mouth sores (Baker et al., 1978). The dose calculated to have been ingested in these cases ranged from 0.14 to 3.4 mg/kg-day. Phenol is another chemical for which the USEPA acknowledges that their residential soil screening level based on chronic exposure may not be protective of children under acute exposure circumstances.

Application of a UF of 100 (10 for sensitive individuals and 10 for extrapolation from a LOAEL to a NOAEL) to the LOAEL for mouth lesions, 0.14 mg/kg-day, would yield a calculated RfD_{acute} of 0.0014 mg/kg, well below the USEPA chronic oral RfD of 0.3 mg/kg-day. The chronic oral RfD was therefore used as the RfD_{acute} value, resulting in a residential SCTL of 500 mg/kg.

Vanadium. Vanadium toxicity in humans primarily occurs following respiratory exposure in occupational settings, and data regarding toxicity following oral ingestion are lacking. However, vanadium has been examined for its therapeutic applications, including the treatment of syphilis, as a cholesterol-lowering agent (Dimond et al., 1963), and its ability to lower blood glucose in diabetic patients (Boden et al., 1996; Goldfine et al., 1985). Recently, vanadium supplements have been introduced to the consumer market for enhancing athletic performance (Fawcett et al., 1997).

From clinical studies, information is available regarding adverse side effects following oral ingestion of vanadium compounds. In several cases it was reported that patients experienced some form of gastrointestinal distress following oral ingestion of vanadium. Dimond et al. (1963) administered vanadium (ammonium vanadyl tartrate) to six patients for a period of six weeks. The subjects received 25, 50, 75 or 100 mg of the compound per day (0.36, 0.71, 1.1, and 1.4 mg/kg-day, assuming a 70 kg body weight). It is stated in the manuscript that all patients experienced gastrointestinal difficulties manifested by diarrhea and cramps. Two patients reported greater fatigue and lethargy. The oral dosage for each patient was limited by cramping and diarrhea, and on a daily dosage of 50 mg or more, a purple-green tint developed on the tongue. Doses had to be lowered to 25 mg to reduce symptoms to tolerable levels.

In the study by Fawcett et al. (1996), two subjects receiving a 35 mg dose of vanadyl sulfate had to withdraw from the study due to health complaints. These studies collectively suggest that the threshold dose for gastrointestinal toxicity is probably close to 25 mg of these vanadium compounds. [Note: This value is very similar to the 30-mg/day dose of vanadyl sulfate commonly recommended as a dietary supplement.] Using the molecular composition of vanadyl sulfate, where vanadium comprises 31% of the total molecular weight, a 25 mg dose contains 7.8 mg vanadium. Assuming a 70 kg body weight for adults in these studies, this dose per unit body weight is 0.11 mg/kg. Since this endpoint is based on transient distress, a UF of 1 was applied. However, the LOAEL was divided by a modifying factor of 3 given the weakness in the data set available to assess toxicity, resulting in a RfD_{acute} of 0.04 mg/kg (rounded to one significant figure), corresponding to an acute toxicity SCTL of 67 mg/kg vanadium in soil.

c) Summary of Residential SCTLs Based on Acute Toxicity

Table 15 summarizes the RfD_{acute} values developed for each of the eight chemicals and the corresponding acute toxicity-based SCTL. For comparison, the residential SCTL for a child based

on chronic exposure is also provided. The acute toxicity SCTL is lower for each of the chemicals except cadmium. In all cases, the lower of the acute and chronic exposure-based SCTL was adopted as the residential SCTL. These values apply in situations where small children at play might come in contact with soils (e.g., residential areas, schools, daycare facilities, etc). They are not applicable for industrial sites.

Table 15
Provisional Acute Oral Reference Doses and Corresponding Acute Toxicity SCTLs for Eight Chemicals

Chemical	Acute Oral Reference Dose (mg/kg)	Residential SCTL	
		Based on Acute Toxicity (mg/kg)	Based on Chronic Exposure (mg/kg)
barium	7E-02	120	5800
cadmium	5E-02	84	82
copper	9E-02	150	3300
cyanide	2E-02	34	1700
fluoride	5E-01	840	5200
nickel	2E-01	340	1600
phenol	3E-01	500	18500
vanadium	4E-02	67	550

d) Caveats in the Acute Toxicity Analysis

There are several caveats in the acute toxicity analysis that should be acknowledged. These include the following:

1) The focus of the analysis was intentionally on data relevant to acute (single dose) exposure in humans. In our opinion, these data are most pertinent in assessing potential human health risks from acute ingestion of soils. These data are limited, however, and there are several uncertainties inherent in human studies. Principal among these is the fact that doses must nearly always be estimated. The only alternative to this approach would be to use animal data. While dose estimation is more precise, studies of acute toxicity in animals are usually restricted to death as the endpoint, and extrapolation of safe human doses from lethal doses in animals is an extremely uncertain process.
2) Despite efforts to update the analysis, the possibility remains that some poisoning reports or other relevant data were missed. In particular, studies appearing in the scientific literature during the first half of the century may be informative, but are very difficult to

access because they cannot be identified through computerized search vehicles such as Medline and Toxline.

3) The chemicals selected for this analysis were those identified by Calabrese et al. (1997) as representing a potential acute toxicity problem for children. While these are regarded as the most likely to pose an acute toxicity hazard, it is possible that there are other chemicals for which a similar concern is warranted. Should evidence arise that a chemical might pose an acute toxicity hazard for small children, the residential SCTL for that chemical should be reconsidered.

4) None of the studies in the analysis involved exposure to the chemical in soil. In most of the cases reported, the chemical was ingested in a soluble form, and the dose from soil required to produce equivalent toxicity may be much different. Presence of the chemical in soil in an insoluble form, or interactions between the chemical and soil that reduce its absorption from the gut could significantly reduce toxicity.

5) A related issue deals with the form of the chemical. In some cases, the chemical can exist in more than one form, with substantial differences in toxic potential. Differences in bioavailability can contribute to these differences, but there can be other factors that influence the toxicity of different forms. Since default SCTLs are intended to be applicable and protective, regardless of the form of the chemical, the choice in developing SCTLs (including acute toxicity-based SCTLs) has consistently been to use data from the most toxic form. It is recognized that this approach will overestimate risk in situations where a less toxic form is present.

3. Development of Default SCTLs Based on Migration to Groundwater (Leaching)

a) Equation for Calculating SCTLs Based on Leachability

The migration to groundwater pathway was developed to identify chemical concentrations in soil that have the potential to contaminate groundwater. The migration of chemicals from soil to groundwater can be envisioned as a two-stage process: the release of chemicals in soil into leachate, and the transport of the dissolved chemicals through the soil to and within an underlying aquifer. The method for calculating a leachability-based SCTL is taken from the SSG and incorporates a standard linear equilibrium soil/water partition equation to estimate release of chemicals in soil leachate and a dilution factor to account for dilution of soil leachates above and in an aquifer. The SCTLs are then back-calculated from applicable groundwater cleanup target levels (GCTLs). In circumstances where contaminated soil is adjacent to surface water bodies, GCTLs based on protection of the surface water body can also be employed. The GCTL is multiplied by a

dilution attenuation factor (DAF) to derive a target leachate concentration. The equation for calculating SCTLs based on migration of chemicals from soil to groundwater is shown in Figure 8.

b) Input Values for Leachability

The equation for the calculation of SCTLs based on leachability requires the input of several chemical-specific factors. These values include the organic carbon normalized soil-water partition coefficient for organic compounds (K_{oc}) and the Henry's Law constant (HLC). Because soil sorption for inorganics is not as dependent on soil organic carbon content as it is for organic chemicals, the development of leachability-based SCTLs for inorganics requires the use of K_d values (soil-water partition coefficient). It is sometimes necessary to calculate values such as K_{oc} or HLC when they are not otherwise available. In these cases, additional physical/chemical values such as the density (d), water solubility (S), vapor pressure (VP), or the adsorption coefficient (K) are needed. Different references for physical/chemical parameters can cite very different values and, as discussed in Section IV A 2 c above, a hierarchy of sources for these values is recommended. Chemical-specific values for d, S, and HLC are preferentially selected from the *Superfund Chemical Data Matrix* (SCDM) (EPA/540/R-96/028). The primary source for K_{oc} values is the SCDM. Secondarily, K_{oc} values are calculated from K_d values in the SCDM according to the equation $K_{oc} = K_d/0.002$. When data are unavailable from the SCDM, the *Hazardous Substance Database* (HSDB), ATSDR Toxicological Profiles, or other reference texts (in that order of preference) are used.

Because of the complex nature of the interaction between inorganic contaminants and the soil matrix, generating K_d values for inorganics can be problematic. For this reason, the USEPA suggests using an equilibrium geochemical speciation model (MINTEQ) for estimating these values. However, modeled values may not accurately represent the potential for leachability because, unlike organic compounds, K_d values for inorganics are significantly affected by a variety of soil conditions. Iron oxide content, soil organic matter content, cation exchange capacity, pH, oxidation-reduction conditions, and major ion chemistry, are significant parameters that can affect the soil/water partition of metals and hence the leachability values. The number of significant influencing parameters and their variability among sites within Florida may contribute to differences in K_d values of several orders of magnitude with similar variability in the resulting leachability SCTLs based on groundwater criteria. Therefore, for some inorganics (including arsenic), it was decided not to develop SCTLs based on leachability, but to require that leaching potential be assessed through a leaching test such as the Synthetic Precipitation Leaching Procedure (SPLP).

B. Development of Site-Specific SCTLs

While default SCTLs are useful tools in site evaluation and when formulating remediation strategies for a broad range of sites, there will be some sites for which default SCTL values are overly conservative or not conservative enough. That is, there will be some sites for which present and future site use and exposure characteristics are so different from the assumptions used to calculate default SCTLs that these SCTLs do not accurately correspond to the acceptable risk levels for that site.

1. Direct Contact SCTLs

This section identifies variables in the SCTL equations for which site-specific information can be substituted in order to obtain a more accurate SCTL, as well as some considerations in making site-specific modifications.

a) Exposure variables

When evaluating whether to use alternative assumptions for exposure frequency and exposure duration, responsible risk management requires consideration of not only the present use of the site, but also the range of plausible future uses. If site use is unrestricted, or only broadly restricted (e.g., to residential or commercial use), this range will almost always include some uses or site conditions in which exposure to soil can be substantial. In these situations, the default assumptions will represent the best choice. If site management includes engineering and/or institutional controls, then exposure assumptions should be based on the upper limit of exposures possible within those controls. Deviation from the default assumptions should occur only in circumstances where it can be shown that the engineering and/or institutional controls proposed for the site would reliably restrict exposure frequency and duration. In addition, caution must be exercised in proposing limited exposure frequencies and/or durations even if the effectiveness of engineering and institutional controls can be assured. The SCTL methodology described here is based on chronic exposure. When exposure is of short duration or intermittent, the SCTLs calculated with these exposure assumptions are <u>not</u> valid. This type of exposure is most commonly associated with construction worker scenarios. For these situations, the policy of the DERM, like FDEP, is to rely primarily on requirements from the Occupational Safety and Health Administration (OSHA) and any other applicable worker safety procedures.

Under extraordinary circumstances, the exposed dermal surface area and inhalation rates could be modified (e.g., if protective clothing and/or a respirator is required while on site). There will be very few, if any, sites where the long-term management involves such restrictions, however.

The adherence factor (the amount of soil which adheres to the skin, per unit of surface area) might conceivably be influenced by local soil conditions, but empirical data to support an alternative value would probably be required.

b) Site soil and weather characteristics

Site soil characteristics can influence the rate of volatilization of organic chemicals into air, and thus the level of the chemical in soil that may be acceptable. Measuring appropriate soil characteristics in order to develop a site-specific VF may be useful, particularly if risks from soil at a site are thought to be dominated by inhalation of volatile chemicals from soil. Parameters necessary for the determination of the VF include the average soil moisture content (ω), the dry soil bulk density (ρ_b), fraction of organic carbon (f_{oc}), and soil pH (used to select pH-specific K_{oc} and K_d values). Methods for determining these site-specific measured values for the derivation of the VF are listed in Table 16 and outlined in the SSG (USEPA, 1996a).

Table 16
Methods for Determining Site-Specific Measured Values for the Derivation of the Volatilization Factor

Soil Characteristic	Data Source	Method
Soil moisture content (ω)	Lab measurement	ASTM D 2216
Dry soil bulk density (ρ_b)	Field measurement	All soils: ASTM D 2937; shallow soils: ASTM D 1556, ASTM D 2167, ASTM D 2922
Soil organic carbon (f_{oc})	Lab measurement	Nelson & Sommers (1982)
Soil texture	Lab measurement	Particle size analysis (Gee & Bauder, 1986) and USDA classification; used to estimate θ_w
Soil pH	Field measurement	McLean (1982)

It is important to note that many site-specific values require data collected over a one-year period and that testing for all the soil properties is requried. Thus, while site-specific SCTLs may be desirable, the use of generic SCTLs may in fact be more cost-effective and less time-consuming. In addition to the time needed for the collection of site-specific data, the investigator must be in strict accordance with the approved methods. This condition is particularly important because the collected data are also used for the derivation of other site-specific parameters. Values derived from site-specific data include θ_w (water-filled soil porosity), θ_a (air-filled soil porosity), η (total soil porosity), and K_d (soil-water organic partition coefficient for organics). Therefore, errors in the collection of data would result not only in one incorrect value, but in several other incorrectly

derived values as well. For example θ_w and θ_a are derived from the soil moisture content (ω). To generate an unbiased value for ω, the soil moisture content must represent the *annual* average. The use of moisture content data from discrete soil samples which may be affected by preceding rainfall events would incorrectly represent the moisture content and therefore result in the incorrect derivation of θw and θa. Correctly deriving values such as θ_a is of great significance, because other than the initial soil concentration, air-filled soil porosity (θ_a) is the most significant soil parameter affecting the volatilization of chemicals from soil. The higher the θ_a, the greater the potential for emission of volatile chemicals. The equations, sources, and methods for deriving soil characteristics using site-specific data are provided in Table 17 on the following page.

VF is also a function of local climatic conditions and the size of contaminated area as expressed in the Q/C term. The USEPA (1996b) has tabulated Q/C values for contaminated areas ranging from 0.5 to 30 acres in size for selected cities, including Miami, around the U.S. These values are based on a modeling exercise that incorporated, among other things, meteorological data for these cities. The default Q/C recommended in Figure 7 is based on Miami data and a 0.5 acre contaminated area. A site-specific Q/C term should be considered if the area of contaminated soil is significantly greater than 0.5 acres and inhalation exposure is a significant concern.

Table 17
Equations, Sources, and Methods for Deriving Soil Characteristics Using Site-Specific Data

Soil Characteristic	Data Source	Method
Water-filled soil porosity (θ_w)	$\theta_w = \eta \cdot (I/K_s)1/(2b+3)$ or $\theta_w = \omega \cdot \rho_b$	η = total soil porosity (L_{pore}/L_{soil}) I = infiltration rate (m/yr) K_s = saturated hydraulic conductivity (m/yr) b = soil-specific exponential parameter (unitless) ω = soil moisture content (g_{water}/g_{soil}) ρb = dry soil bulk density (g/cm3)
Total soil porosity (η)	$\eta = 1 - (\rho b/\rho s)$	ρ_b = dry soil bulk density (g/cm^3) ρ_s = soil particle density = 2.65 kg/L
Infiltration rate (I)	HELP model; Regional estimates	HELP (Schroeder et al., 1984); may be used for site-specific infiltration estimates; used to calculate θ_w
Soil-specific exponential parameter (b) (Moisture retention component)	Look-up	Attachment A (USEPA, 1996a); used to calculate θ_w
Saturated hydraulic conductivity (K_s)	Look-up	Attachment A (USEPA, 1996a); used to calculate θ_w
Air-filled soil porosity (θ_a)	$\theta a = \eta - (\omega \cdot \rho b)$ or $\theta a = \eta - \theta w$	η = total soil porosity (L_{pore}/L_{soil}) ω = soil moisture content (g_{water}/g_{soil}) ρ_b = dry soil bulk density (g/cm^3) θ_w = water-filled soil porosity (L_{water}/L_{soil})
Soil-water organic partition coefficient (organics) (K_d)	$K_d = K_{oc} \cdot f_{oc}$	K_{oc} = soil-organic carbon partition coefficient (cm^3/g) f_{oc} = organic carbon content of soil (g/g)

The PEF term is also influenced by local meteorological conditions, as well as site characteristics (Figure 6). An important site characteristic influencing the PEF is the percent of vegetative cover over the contaminated soil. The default assumption is that 50% of the contaminated area has vegetative cover. This value can be adjusted for a specific site, but if a higher value is used, some mechanism must be in place to ensure that the vegetative cover remains in place in the future. Local wind conditions can also influence the PEF and could conceivably be used to adjust the PEF in the development of site-specific SCTLs. However, a preliminary analysis of annual average meteorological data from cities around Florida found average wind speeds only slightly different from the default value (unpublished observations). Because the PEF is a quantitatively important factor in the SCTL of only a very few chemicals, there is generally little incentive for developing site-specific PEF values. It is important to note that the PEF is applicable only for undisturbed soil. If there is significant soil disturbance at a site, such as from vehicular traffic, site-specific estimates of dust levels may have to be substituted for the PEF in deriving an SCTL.

While the VF model used in the calculations of SCTLs for Chapter 24 of the Code is capable of adjusting the VF for different durations of exposure, the model is limited to exposures that begin immediately. The model assumes that the rate of flux of a volatile chemical from soil to air is highest when the concentration in surface soil is highest and declines over time. As the flux declines over time, so too does the air concentration. For a chemical at a given initial concentration in soil, the average concentration in air will depend on the averaging period (or exposure duration) such that longer periods have lower average concentrations. This is because as the concentration in soil declines over time, lower concentrations are included in the averaging process. For example, the model predicts that, for a given concentration of xylenes in soil, the average concentration over the first six years will be approximately twice the average concentration over the first 25 years because the air concentrations in later years are quite low.

The assumption in developing default SCTLs is that exposure begins immediately and continues for the number of years associated with the given exposure scenario. It is possible that in some site-specific situations other exposure periods may be relevant, including exposures that do not begin immediately. An alternative approach under these circumstances is the use of the computer software EMSOFT, developed by the USEPA National Center for Environmental Assessment. VFs calculated by EMSOFT do not differ from those calculated with the current VF model for exposure durations that begin immediately. However, EMSOFT will compute average soil VFs for exposure intervals beginning and ending at any time in the future. Therefore, EMSOFT may be of value in deriving site-specific volatilization factors for exposure scenarios that differ from default assumptions.

c) Mass limits

The VF equation is based in part on the assumption of an infinite source. When the contaminant's soil concentration and the volume of contaminated soil (i.e., the area and depth) are known, the VF equation can be modified to take mass of the volatile chemicals into consideration. An alternative VF equation incorporating estimates of volume of contaminated soil is described in the SSG (USEPA, 1996a,b). However, it should be noted this mass-limit VF model is only based on assuming that the whole mass of contaminant will volatilize during the exposure period considered, without regard to the actual volatilization potential of the contaminant.

d) Soil Saturation Limit

The inhalation component of the SCTL for residential and industrial exposure to volatile contaminants is calculated using a VF. The equation for the VF (Figure 7), which defines the relationship between the concentration of the chemical in soil and its flux to air, assumes an infinite source of the chemical and only one mechanism of transport, vapor phase diffusion. As emission flux increases, the air concentration increases, along with risks from inhalation exposure. The VF model assumes that this relationship holds throughout the possible range of chemical concentrations in soil, although at high concentrations this is not the case. At a sufficiently high concentration, the soil pore air and pore water are saturated and the adsorptive limit of the soil particles is reached. Any increase in concentration above this point does not result in greater flux — the rate of flux reaches a plateau and volatile emissions (and air concentrations) can go no higher no matter how much additional chemical is present in soil. This concentration is termed the soil saturation limit (C_{sat}).

The C_{sat} value for a chemical depends upon a variety of factors, including chemical-specific physical/chemical properties, as well as characteristics of the soil. As such, the C_{sat} value for different chemicals at a site will vary, and C_{sat} values for a given chemical can be different from site to site. A formula for estimating C_{sat}, using chemical-specific inputs and default soil assumptions, is shown in Figure 9.

Whenever the concentration of a chemical in soil exceeds its C_{sat} value, the standard formula for estimating volatilization and inhalation exposure will yield inaccurate results. Specifically, the formula will overestimate flux and inhalation exposure. This is because it fails to recognize that flux reaches a maximum at or around the C_{sat} value, and assumes instead that it continues to increase with concentration. This is an issue in SCTL development because for some chemicals (primarily volatile chemicals of low toxic potency) the calculated SCTL for the chemical

is greater than its C_{sat} value. This situation exists for about 40 of the chemicals for which SCTLs were developed..

It is possible to correct for the influence of C_{sat} on the inhalation component of the SCTL, but this requires that the C_{sat} value be estimated with some confidence. Alternatively, the SCTLs can be uncorrected, recognizing that this adds some extra measure of conservatism to the value. Given the uncertainties in developing accurate C_{sat} values applicable to a wide variety of sites, the latter approach was chosen.

C_{sat} can also potentially influence the development of SCTLs for leachability. However, among the chemicals listed in Table 2, only di-n-octylphthalate and 1,1,2-trichloro-1,2,2-trifluoroethane have a leachability SCTL > C_{sat}. This information indicates that, for practical purposes, C_{sat} is not an issue of concern in developing leachability goals.

C_{sat} values may be useful in identifying situations in which free product may be present. Soil concentrations of a chemical above the saturation limit could result in their presence as free product, which may be undesirable at the site for a number of reasons. It should be emphasized that the C_{sat} value does not signify the concentration at which free product is present, but rather that concentrations greater than C_{sat} could serve as a "red flag" for the possibility of free product being present at the site. As a site management tool for this purpose, C_{sat} values have been tabulated for chemicals that can exist as liquids at room temperature. These are presented in Table 8. Actual determination of whether free product exists in soils should be made by other means.

e) Values that do not change from site to site

It is worth stating explicitly that there are some variables and assumptions that are unrelated to site conditions and circumstances and therefore should not be modified in deriving a site-specific SCTL. These parameters include toxicity values, fundamental physical/chemical properties of chemicals, and the averaging time for carcinogenic effects. [Note: The averaging time for non-carcinogenic effects is a function of the exposure duration, which could be modified at a particular site.] Also, it is generally impractical to consider body weight as a site-specific variable (except as it relates to the age of the exposed individuals, e.g., adults versus children).

2. SCTLs Based on Leachability

In Florida, soil types vary significantly across the state, from quartz sand to muck, and leaching potential covers an extreme range. The default soil characteristics used to develop generic leachability-based SCTLs lie somewhere in the middle of the range of values possible in Florida. Development of site-specific leachability-based SCTLs can be justified because characteristics at a given site may bear little resemblance to the default assumptions. Although the use of default soil

parameters may equally lead to under or over prediction of leaching potential, the complexities associated with deriving site-specific estimates suggest it is preferable to use default values, unless a protocol has been approved by DERM to derive the required site-specific information. To develop a site-specific SCTL, default values of soil characteristics can be replaced by values measured at the site, including f_{oc}, θ_w, θ_a, η, and ρ_b.

Another parameter that is important in calculating leachability-based SCTLs is the dilution attenuation factor (DAF). The USEPA arrived at a default DAF using results from OSW's EPACMTP Model. This model utilized a Monte Carlo analysis with input parameters obtained from nationwide surveys of waste sites and from applying the SSL dilution model to 300 groundwater sites across the country. The model distributions were repeated 15,000 times for each scenario and a cumulative frequency distribution of DAF values was generated. The results of the accompanying sensitivity analysis indicated that climate, soil type, and size of the contaminated area have the greatest effect on the DAF. To gain further information on the national range and distribution of DAF values, the dilution model was applied to two large surveys of hydrogeologic site investigations. These were the American Petroleum Institute's hydrogeologic database (HGDB) and USEPA's database of conditions at DNAPL sites. DAF modeling information from a combination of 300 sites indicated that the geometric mean DAF of all sites combined was 20 for a source area of 0.5 acre. This value was carefully selected using a "weight of evidence" approach which best represents a nationwide average and is therefore regarded as an acceptable default for use at most sites. In special circumstances, such as very complex sites, a site-specific DAF can be calculated, but the aquifer hydraulic conductivity, the hydraulic gradient, the mixing zone depth, the infiltration rate, and the source length parallel to groundwater flow must be determined (USEPA, 1996a).

It has been demonstrated that the leachability-based SCTL partition equation can be used to derive leachibility-based SCTLs for organic compounds. However, inorganics present at cleanup sites can also pose risks to an underlying aquifer. To derive leachability-based values for most metals is more complicated, however. Unlike organic compounds, K_d values (soil/water partition coefficient) for metals are significantly affected by a variety of soil conditions, so derivation of a site-specific value may be a rather involved process. In these circumstances, a leaching test may be more useful than the partitioning method. Therefore, DERM, like FDEP, recommends the use of a leaching test instead of the soil/water partition equation. However, site-specific leachability values for inorganics derived using K_d values estimated with the MINTEQA2 model are considered acceptable leachability SCTLs, if oily wastes are not present. If the decision is made to determine site-specific leachate values, the Synthetic Precipitation Leaching Procedure (SPLP), developed to model an acid rain leaching environment, can be used when there

are no oily wastes[1]. When oily wastes are present, DERM, like FDEP, specifically requires the use of the Toxicity Characteristic Leaching Procedure (TCLP) for cleanup of these sites. While this procedure was developed to model leaching from the bottom of a landfill, it may be used to estimate leaching potential when the SPLP method is not appropriate (i.e., when soil is contaminated with oily constituents, such as used oil or similar petroleum products).

C. Comparing Site Contaminant Concentration Data with Soil Cleanup Target Levels

There are distinct issues associated with the comparison of soil concentrations to direct contact versus leachability-based SCTLs. Consequently, the two types of comparisons are discussed separately.

1. Comparison with Direct Contact SCTLs

There are two approaches for comparing site concentrations to the respective SCTLs, apportioned (as appropriate) in accordance with Section VI of this report. Responsible parties may choose to compare the site maximum concentration of each contaminant with the respective default SCTL listed in Chapter 24. Alternatively, the responsible party may choose to calculate a 95% Upper Confidence Limit (UCL) of the mean for the site concentrations to compare with chronic toxicity-based SCTLs. [Note: For SCTLs based on acute toxicity, comparison shall always be made with the maximum concentration, as explained below.]

a) Comparison Using the Maximum Concentration

For this approach, the maximum concentration for each chemical is compared with its appropriate direct contact SCTL. If the maximum concentrations for all chemicals are equal to or below their SCTLs, the site is considered to meet the County's acceptable risk levels for direct contact. [Note: A chemical might still pose a concern with respect to leaching to groundwater or surface water, and must also be evaluated for leaching separately.]

b) Comparison Using the 95% UCL Concentration

Most risks from contaminated soils are evaluated based on chronic exposure. It is assumed that an individual will be exposed over time to an area of contaminated soils, rather than to soils at one specific location. If the individual's contact with the contaminated area is random, the best representation of the concentration to which he/she is exposed is the mean concentration over that

[1] Direct leachability testing should include a minimum of three representative soil samples, pursuant to USEPA Test Method 1312 (SPLP). Leachate concentrations from SPLP should not exceed the applicable GCTLs. SPLP should not be used for chemicals derived from used oil or similar petroleum products.

area. This assumption provides the basis for using a mean chemical concentration in determining whether an SCTL has been met.

The ability to accurately determine the mean concentration over an area is dependent upon a number of factors, including sampling locations and the number of samples. Because there is always some uncertainty as to whether the average of any given set of samples in fact represents the true mean over the area of interest, DERM requires the use of a 95% upper confidence limit (UCL) estimate of the mean. Specifically, in circumstances in which the use of a mean concentration is appropriate, the 95% UCL of the mean must be used. The 95% UCL has been defined by the USEPA as the upper bound of a confidence interval around an average. The 95% confidence interval for an average is the range of values that will contain the true average (i.e., the average of the full statistical population of all possible data) 95% of the time.

Exposure Units. Implicit in using a 95% UCL approach is the concept that the site consists of one or more "exposure units" — areas over which receptors will have equal and random contact. Exposure units must be clearly delineated and justified based on current and future activity patterns. An exposure concentration must be calculated for each exposure unit, and delineation of exposure units determines which concentrations should be included in the 95% UCL calculations.

A site can have more than one exposure unit, as well as different exposure units for different receptors. For example, operations at a commercial facility may require some employees to spend most of their time in one area, while maintenance workers divide their time equally across the site. In this example, the 95% UCL values would likely be different for the operations and maintenance workers, and both would need to be calculated for comparison with SCTLs. Recreational parks are another example, where some areas are expected to provide little opportunity for contact with soil (e.g., paved areas), whereas others may prove to be very attractive to receptors (e.g., playgrounds). In these situations, areas expected to differ in their potential for exposure should be evaluated separately.

Future changes in exposure units also need to be considered. Different commercial use of a property, for example, might lead to different activity patterns and different exposure units. If acceptability of contaminants at a site is based on a particular set of activity patterns and exposure units, institutional controls are required to insure that either: a) future site use retains those activity patterns, or b) the site is re-evaluated if activity patterns change. If exposure units cannot be properly delineated for future [or for current] land use, or institutional controls are undesirable, the approach of comparing the maximum concentration to the residential SCTL should be used instead.

For residential land use involving single-family dwellings, the exposure unit is typically the residential lot. If land is not currently used for residential purposes, but could be developed as

such in the future, assessment of the site must consider potential residential exposure units. DERM considers the default residential lot to be 0.25 acres in size. This means that for unrestricted sites where future residential use is possible, DERM requires a demonstration that contaminants in each potential 0.25-acre residential lot meet the acceptable risk level. It is not necessarily acceptable to develop a single 95% UCL for the entire residential development area.

Different exposure units can be managed with different approaches for comparing site concentrations with cleanup targets. For example, the comparison-with-maximum approach could be selected for one or more exposure units within a site, while the 95% UCL approach is used for others.

Calculation of the 95% UCL. Several methods are available for calculating a 95% UCL on the mean for a set of data. However, the performance of these methods varies dramatically and is dependent on the nature of the data set (e.g., number of values, their distribution and variability, the extent of censoring). The method chosen to calculate a 95% UCL should give a true 95% UCL value, while at the same time not be overly conservative. For calculating 95% UCL values, DERM recommends using the FLUCL tool. This program calculates a 95% UCL using the optimal method, given the characteristics of the data. FLUCL is particularly useful for data sets that include censored values (i.e., with "non-detects"). DERM also considers the USEPA's ProUCL, version 3 to be acceptable when used in accordance with the guidelines provided in Appendix D. Other computational tools for calculating 95% UCL can be used, if approved by DERM.

If the site concentrations of a chemical vary substantially, the 95% UCL can sometimes exceed the highest concentration observed on site. In this situation, the SCTL shall be compared with the maximum detected concentration rather than the 95% UCL.

Data and Sampling Requirements. Sufficient data for a reasonably accurate calculation of the 95% UCL must be available regardless of the calculation tool employed. At a minimum, 10 samples are needed within an exposure unit to calculate a 95% UCL (unless an alternate number of samples is appropriate for another computational tool which has been approved by DERM).

Concentration data from most sites reflect biased sampling, given that sampling focuses primarily on areas where contamination is suspected. Data sets with concentrated sampling in one or a few areas and sparse sampling in others may satisfy the need to characterize the nature and extent of contamination, but they are not well suited for calculating a representative 95% UCL. Biased sampling where contaminated areas are over-represented likely overestimates the true average, but because it is conservative and health protective, this approach is acceptable to DERM. Biased sampling in which contaminated areas are under-represented spatially is not acceptable. This situation could arise, for example, during "virtual remediation," where data values from intensively sampled contaminated areas are replaced with nondetects or background concentrations

to examine the effect of cleaning specific areas based on the 95% UCL. In this situation, additional effort may be required, in consultation with DERM, to achieve a spatially representative data set. However, formal geostatistical approaches are seldom needed, and have their own set of requirements, such as larger sample sizes and spatially representative sampling.

DERM requires that direct contact SCTLs be met throughout the entire unsaturated zone unless institutional controls are applied. With or without institutional controls, the 0 to 24'' soil horizon below land surface (bls) must meet the respective SCTLs in order to avoid remediation or installation of engineering controls. Thus, vertical sampling of soils is required at most sites. Vertical compositing of soil samples results in loss of information regarding the depth at which contamination is located. In general, soils must be sampled at sufficient intervals such that exposure concentrations are not underestimated. The 95% UCL should be calculated using soil concentrations from the same depth interval.

2. Comparison with Leachability-based SCTLs

The potential for leaching can be addressed either through comparison with SCTLs or through empirical means, such as leaching tests or evaluation of site history and contamination data for evidence of leaching. Unlike direct contact SCTLs, which are based primarily on long-term exposure covering a specified area, leachability-based default SCTLs are intended to protect water resources at all locations. Consequently, maximum rather than average (or 95% UCL) concentrations must be compared with leaching criteria. Under most circumstances, soil concentrations throughout the unsaturated zone should be compared with leachability criteria. It may be impossible for technical and economic reasons to develop soil concentration data for numerous discrete vertical intervals. However, as with assessment of risks from direct contact, it is important not to collect samples using large vertical spacing because pockets of contamination may be overlooked. The selection of appropriate sampling intervals will be a matter of professional judgment, but should at a minimum take into account soil profile characteristics that would be expected to influence the retention or concentration of contaminants.

Leachability-based SCTLs can be influenced by site-specific soil properties. Consequently, site-specific soil properties can be used to develop leachability-based SCTLs using methods described in Section V. A. 3. of this document. Sampling to determine soil properties must meet two criteria: 1) samples must be taken such that chemical contamination does not influence soil property measurement, and 2) the soil samples should be representative of the depth intervals over which contamination exists.

D. Special Cases

1. Development of SCTLs for Ammonia

Ammonia is an inorganic compound that exists in a state of equilibrium between un-ionized ammonia (NH_3) and ammonium ion (NH_4^+). The state of ionization, and thus the percentages present as NH_3 and NH_4^+, are generally dependent upon the pH of the medium (i.e., soil or water), and to a lesser degree upon temperature. Higher pH results in a greater percentage as NH_3, whereas lower pH favors the formation of NH_4^+.

Some environmental criteria are intended to be applied to NH_3 specifically, while others are applied to total ammonia (NH_3 plus NH_4^+). For example, the GCTL for ammonia of 2800 µg/L and the fresh and marine surface water CTL of 500 ug/l are applicable to the sum of the NH_3 and NH_4^+ concentrations. Alternatively, the freshwater SWCTL for ammonia of 20 µg/L is applicable to NH_3 only, and compliance must be determined based on estimated NH_3 levels. Since standard analytical methods only provide information on total ammonia concentration, the concentration of NH_3 in samples must be estimated based on the total ammonia concentration and the pH of the water.

Site-specific soil characteristics may greatly affect the ionization of ammonia and therefore the potential for leaching. Leachability is based, in part, on the partitioning of a compound between soil and water. For organic contaminants, the partitioning is dependent on the organic carbon normalized partitioning coefficient (K_{oc}). However, the simple relationship between soil organic carbon and sorption observed for organic compounds does not apply to inorganic contaminants such as ammonia. The soil-water partition coefficient (K_d) for inorganic compounds is affected by numerous geochemical parameters and processes, including pH, sorption to clays, organic matter, iron oxides, other soil constituents, oxidation/reduction conditions, major ion chemistry, and the chemical form of the inorganic present. For sites where ammonia leachability is a concern, leachability SCTLs based on groundwater criteria may require site-specific adjustments.

Direct exposure SCTLs for total ammonia are derived using the default equation for non-carcinogens (see Figure 5) and an oral reference dose of 0.4 mg/kg-day, based on a minimal risk level (MRL) derived by ATSDR (ATSDR, 1990a)[1]. For the inhalation route of exposure, an inhalation reference dose of 0.03 mg/kg-day is used. This dose is derived from the inhalation reference concentration of 0.1 mg/m^3 presented in IRIS. Given that the percentage of total

[1] The oral MRL for ammonia currently listed in the ATSDR Toxicological Profile for Ammonia is 0.3 mg/kg-day. This value was derived by adjusting the NOAEL of 40 mg/kg-day by an uncertainty factor of 100 and an adjustment factor for intermittent exposure. Per discussion with John Wheeler at ATSDR it was indicated that the use of an intermittent exposure factor in the extrapolation of the NOAEL to the MRL is no longer recommended. As such, the ATSDR recommended oral MRL for ammonia has been modified to 0.4 mg/kg-day and the drinking water MRL is 14,000 µg/L. Although an MRL of 14 mg/L exists for ammonia in drinking water, a value of 2800 µg/L was used here since it incorporates a relative source contribution factor of 20%.

ammonia present as NH_3 depends on soil pH, direct exposure SCTLs are conservatively developed by assuming that all of the ammonia in soil is in the NH_3 form. This is because, while ammonia as NH_3 has a significant capacity to volatilize, NH_4^+ does not and it will be fully dissolved in water within the soil matrix. Consequently, for ammonia in soil, ingestion exposure is not as important as inhalation because once ingested the potential toxicity of NH_3 and NH_4^+ will be similar due to equilibrium between the two forms in the presence of gastric acids. When volatilization is minimal (i.e., low soil pH, see Table 18 below), the direct exposure SCTL will be driven primarily by the oral component. The ammonia SCTLs that are based on oral and dermal exposure pathways only are 35,000 mg/kg and 870,000 mg/kg for residential and industrial scenarios, respectively. Alternatively, at higher soil pH, the SCTL for ammonia is predominantly driven by the inhalation component of the equation, and therefore reflects the capacity of these compounds to volatilize. In these cases, the inhalation component of the SCTL equation must be adjusted to account for the proportion of ammonia available for volatilization. Thus, to select accurately a direct exposure SCTL for ammonia on a site-specific basis, the soil pH must be known. In Miami-Dade County, soil is limestone-based and soil pH ranges from approximately 7.4 to 8.4 (Li 2001). To be protective, a pH of 8.5 was estimated to represent Miami-Dade County soil. Table 18 below provides SCTLs for ammonia based on soil pH at an ambient soil temperature of 25°C.

Table 18
SCTLs for Ammonia as a Function of Soil pH at an Ambient Temperature of 25°C

Soil pH*	Percent Un-Ionized Ammonia (NH_3)**	Residential (mg/kg)‡	Industrial (mg/kg)‡
	100%	750	4000
9.50	64.3%	1200	6200
❖8.50	15.2%	4400	26000
7.50	1.77%	19000	180000
6.50	0.18%	32000	630000
6.00	0.0568%	34000	780000
5.50	0.0180%	35000	840000
***5.04	0.00624%	35000	860000
5.00	0.00569%	35000	870000

*Increasing ammonia concentrations will tend to increase soil pH. Situations of low soil pH and high ammonia concentrations, while theoretically possible, are unlikely to exist at contaminated sites.
**USEPA: Aqueous Ammonia Equilibrium-Tabulation of Percent Un-Ionized Ammonia, EPA/600/3-79/091.
***Average pH of soils in Florida.
‡Calculated by adjusting inhalation contribution in the SCTL equation by the percent NH3 corresponding to the selected pH, but limited by the oral and dermal contribution.
❖Estimatted soil pH in Miami-Dade County.

2. Development of the Direct Exposrue SCTLs for Arsenic

Direct exposure SCTLs for arsenic were previously calculated using the default assumption of a relative oral bioavailability of 100%. This means in effect that the absorption of arsenic from ingested soil was assumed to be equivalent to the absorption of arsenic from water. [Note: The absorption of arsenic in water is the appropriate point of comparison because the oral cancer slope factor for arsenic was developed from studies of populations exposed to arsenic in drinking water.] Several studies in animals have shown consistently that the absorption of arsenic from soils is less than its absorption from water (see Ruby et al., 1999 for a review). Based on a review of the studies of arsenic bioavailability from soils, and in particular on results of a study conducted in non-human primates measuring bioavailability of arsenic from contaminated soils from Florida sites (Roberts et al., 2002), FDEP determined, and DERM concurred, that a decrease in arsenic risks from soil ingestion by a factor of 3 is warranted. This has been incorporated into the calculation of the direct exposure SCTLs for arsenic.

3. Development of CTLs for Chloroform

The USEPA has recently updated the IRIS record for chloroform. In it, the Agency states that, under the Proposed Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1996; U.S. EPA, 1999), chloroform is likely to be carcinogenic to humans (Group B2), but only under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia. Under low exposure conditions chloroform is not likely to be carcinogenic to humans by any route of exposure.

The USEPA has concluded that chloroform carcinogenesis occurs only after some exposure level is exceeded based on studies showing that cytotoxicity is always present at doses equal or lower to those associated with an increased incidence of tumors. Studies in animals reveal that chloroform can cause an increased incidence of kidney tumors in male rats and an increased incidence of liver tumors in male and female mice. Current data show there are three steps in the sequence of events leading to liver and kidney cancer in rodents due to chloroform exposure. The first step involves oxidative metabolism of chloroform in the target organs, kidney and liver. The second step is cytotoxicity and cell death caused by oxidative metabolites, primarly phosgene. The third and final step is regenerative cell proliferation, which is thought to be responsible for the increased probability of cancer.

Given that cytotoxicity appears to be a prerequisite for tumor formation, the USEPA has concluded that a RfD protective of this effect will also protect against cancer. Both the NOAEL/LOAEL and benchmark dose approaches produce the same oral RfD of 0.01 mg/kg-d.

The USEPA has stated that cytotoxicity is likely to be a requirement for chloroform carcinogenesis for all routes of exposure, and that the current cancer potency factor for the

inhalation route presented in IRIS is under review. Although it is expected that the same approach presented for the oral route of setting a Reference Concentration will finally be selected, the current SCTLs are calculated using the Inhalation Unit Risk (IUR) for chloroform presented in IRIS.

4. Development of the Direct Exposure SCTLs for Lead

a) Residential

The residential direct exposure SCTL for lead is based on OSWER Directive #9355.4–12, *Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities* (USEPA, 1994a). The guidance level for lead in soils described in this directive was calculated with the USEPA's *Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children* (USEPA, 1994b). This model takes into account the multimedia nature of lead exposure in children and calculates distributions of exposure and risk likely to occur at a site using default assumptions. Research indicates that young children are particularly sensitive to the effects of lead and require specific attention in the development of an SCTL for lead. Thus, an SCTL that is protective for young children is expected to be protective for older persons as well. The 400 mg/kg guidance level for lead in residential soils cited in the 1994 OSWER directive was calculated such that a hypothetical child would have no more than 5% risk of exceeding 10 µg/dL blood lead concentration. This target blood lead level is based on research conducted by the Centers for Disease Control and by the USEPA that associates blood lead levels exceeding 10 µg/dL with health effects in children.

b) Industrial

To calculate the industrial direct exposure SCTL for lead, the approach outlined in *Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil* (or TRW; USEPA, 1996c) was followed. This guidance document provides methodology for assessing risks associated with non-residential adult exposures to lead in soil based on the potentially most sensitive workers – women of child-bearing age. The methodology focuses on estimating fetal blood lead concentrations in pregnant women exposed to lead contaminated soil. That is, the model is designed to estimate an acceptable soil lead concentration to which women could be exposed, while pregnant, without the risk of producing unacceptable blood lead concentrations in the developing fetus (i.e., levels above 10 µg/dL).

This method is based, in part, on a simplified representation of lead biokinetics assumed to predict quasi-steady state blood lead concentrations among adults (women of child-bearing age) who are relatively consistently exposed to a site. A constant of proportionality between fetal blood

lead concentration at birth and maternal blood lead concentration is also employed. As such, this model provides a means for consistency in calculating acceptable industrial soil lead levels.

A series of equations, discussed in detail in the TRW document, are used to derive an acceptable lead concentration in soil. The limit for lead concentration in maternal blood ($PbB_{a,c,g}$) is derived first. This value represents the risk-based goal for the central estimate of blood lead concentrations in adult women that ensures the fetal blood lead concentration goal of 10 µg/dL is not exceeded. This value is derived from the equation:

$$PbB_{a,c,g} = \frac{PbB_{fetal,0.95,goal}}{GSD_{i,adult}^{1.645} \times R_{fetal/maternal}}$$

In this equation, $PbB_{fetal,0.95,goal}$ = 10 µg/dL represents the goal for the 95th percentile blood lead concentration among fetuses born to women having exposures to the specified site soil concentration. This value is divided by the product of R and GSD. R (0.9) is the constant of proportionality between fetal blood lead concentration at birth and maternal blood lead concentration. GSD is the geometric standard deviation for blood lead concentrations among adult females having exposures to similar on-site lead concentrations but having varying responses to site lead (intake, biokinetics) and non-uniform off-site lead exposures. Ideally, the GSD used in the model is estimated from the population of concern at the site, although site-specific data are rarely available. The TRW has recently published estimates of GSD and mean baseline blood lead concentration ($PbB_{a,0}$, see below) derived for 17-45 year old women from data collected during the Third National Health and Nutrition Evaluation Survey (NHANES III). This document recommends using Region-specific values for both parameters when calculating SCTLs for lead (USEPA 2002a). The GSD recommended by the TRW for the South Region is 2.07 µg/dL, resulting in a $PbB_{a,c,g}$ = 3.357 µg/dL.

Next, the target blood lead concentration ($PbB_{a,c,g}$) is employed along with several other variables to calculate lead in soil (Pb_S), the SCTL.

$$Pb_S = \frac{(PbB_{a,c,g} - PbB_{a,0}) \times AT}{BKSF \times IRsoil \times AFsoil \times EFsoil}$$

where,

$PbB_{a,c,g}$ (target blood lead concentration) = 3.357 µg/dL

$PbB_{a,0}$ (baseline blood lead concentration) = 1.39 µg/dL

AT (averaging time) = 365 days/year

BKSF (biokinetic slope factor) = 0.4 µg/dL per µg/day

IR_{soil} (ingestion rate) = 0.05 g/day

AF_{soil} (absorption factor) = 0.12 [unitless]

EF_{soil} (exposure frequency) = 219 days/year

In this equation, the baseline blood lead concentration, $PbB_{a,0}$, represents the adult blood lead concentration (µg/dL) in the absence of site exposures. It is intended to be a best estimate of a reasonable central value of blood lead concentrations in women of child-bearing age that are not exposed to lead-contaminated soil or dust at the site. This value was also derived using data for 17-45 year old women collected during NHANES III. The TRW recommends the mean value of 1.39 for the South Region (USEPA 2002a).

In the TRW model, the baseline $PbB_{a,0}$ is subtracted from the target $PbB_{a,c,g}$ to obtain a value representative of the allowable increase in blood lead level that will not result in exceeding the target blood lead level. Using the default values selected for Chapter 24 of the Code, this value equals 1.967 µg/dL (3.357 µg/dL minus 1.39 µg/dL). Additionally, the model uses an averaging time of 365 days/year and an exposure frequency of 219 days/year. The exposure frequency is based on USEPA recommendations provided as part of the lead guidance for average time spent at work by both full-time and part-time workers. Even though this exposure frequency is different from the standard worker default, it was used here to be consistent with USEPA calculation of soil lead limits. Exposure duration was assumed to be one year (not shown in the denominator of the equation because it is 1). The other variables are defined as follows:

BKSF — Biokinetic slope factor relating increase in the typical adult blood lead concentration to average daily lead uptake. The recommended value is 0.4 µg/dL blood lead increase per µg/day lead uptake.

AF_{soil} — Fraction of lead in soil ingested daily that is absorbed from the gastrointestinal tract. TRW recommends a default value of 0.12 based on the assumption that the absorption factor for soluble lead is 0.2 and that the relative bioavailability of lead in soil compared to soluble lead is 0.6; thus 0.2 x 0.6 = 0.12.

IR_{soil} — Intake rate of soil. The recommended value is 0.05 g/day. Although the 0.05 g/day default value addresses all occupational soil intake by an individual, whether directly from soil or indirectly through contact with dust, risks associated with more intensive soil contact activities such as construction and excavation are not included. Site-specific data on soil contact intensity should be considered

when evaluating the applicability of the default industrial direct exposure SCTL. Depending on the duration of exposure and type of exposure scenario being evaluated, larger ingestion rates may be more appropriate.

Using these standard equations with the recommended defaults and values selected to represent a contaminated site, a value of 1400 mg/kg lead is calculated as the industrial direct exposure SCTL.

$$PbB_{a,c,g} = \frac{10\ \mu g/L}{2.07^{1.645} \times 0.9} = 3.357\ \mu g/dL$$

$$SCTL\ Pb = \frac{3.357 - 1.39\ \mu g/dL}{0.4\ \mu g/dL\ per\ \mu g/d \times 0.05\ g/d \times 0.12 \times 219\ d/yr}$$

SCTL Pb = 1366 or 1400 mg/kg

The TRW recognizes that other models with more detailed blood lead kinetics could provide better estimates regarding brief acute exposures or intermittent exposure patterns. However, pending further development and evaluation of other biokinetic models, the methodology provided by the TRW is the recommended approach.

5. Development of SCTLs for Methylmercury

Most USEPA-approved analytical methods for determining methylmercury concentrations in soil are based on measurement of total organic mercury. As such, soil concentrations reported as methylmercury may, in fact, include or consist of other organic mercury species. Recognizing this, the default SCTL for methylmercury was developed in a way that would be protective for organic mercury species in general. Data regarding the comparative toxicity of organic mercurial compounds are limited. Only methylmercury has an RfD from the USEPA, and this value was tentatively assumed to be applicable to all forms of organic mercury. The physical/chemical properties of organic mercury compounds can vary significantly, however. Dimethylmercury has much greater volatility than methylmercury, and the dose received from a given concentration in soil would be much higher. In order to develop an SCTL protective under circumstances of dimethylmercury exposure, the physical/chemical properties of this compound were used to derive the default methylmercury SCTL. Under site-specific circumstances where analytical methodology capable of reliably speciating organic mercury is employed, alternative SCTLs directed to specific forms (including methylmercury) could be utilized. Measuring organic forms of mercury

specifically would be desirable, for example, in situations where mercury has been introduced into the environment in an organic form.

6. Development of SCTLs for Total Recoverable Petroleum Hydrocarbons (TRPHs)

The TRPH SCTLs were developed to be used in a two-tiered approach with a default TRPH SCTL as the starting value. Default TRPH SCTLs for direct exposure and leachability included in Table 2 are to be compared with site-specific results obtained using the Florida Petroleum Residual Organic (FL-PRO) analytical method. Currently, the FL-PRO method is limited to measuring the concentration of mixed petroleum hydrocarbons in the range of C_8-C_{40}. While FL-PRO does not measure hydrocarbons in the C_5-C_7 range, the most toxic and prevalent chemicals within this range are quantified by other analyses and have individual SCTLs. Therefore, the default TRPH SCTL is based on the most conservative and health protective carbon range that can be detected by FL-PRO, the >C_8-C_{10} carbon range (Table C-5, Appendix C).

In the event that any of these default SCTLs is exceeded, the assessment should enter a second tier where TRPH site concentrations for individual fraction ranges are compared with their respective SCTLs. There are currently two analytical methods that provide satisfactory concentration information for specific fractions, although the fractions measured by the two methods are not identical. DERM has approved using the TPHCWG (Total Petroleum Hydrocarbon Criteria Working Group) method and the method developed by the Massachusetts Department of Environmental Protection (MADEP, 1997). TRPH SCTLs for fractions evaluated using the TPHCWG and MADEP methods are derived from chemical/physical parameters and toxicity values assigned to the carbon range for each fraction as described in Appendix C. Because the carbon fractions measured by the two methods are slightly different, sets of carbon fraction SCTLs specific to each method are provided (Table C-9, Appendix C)."

7. Development of SCTLs for Polychlorinated Dibenzodioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs)

Polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PDCFs) are typically found in the environment as mixtures of PCDD or PCDF congeners. The individual PCDD and PCDF congeners can vary widely in terms of toxic potency, and therefore the same total concentration can pose different risks. Most analyses of PCDDs and PCDFs in environmental samples provide information on the congeners present. The current approach to assessing the toxicity of these mixtures involves the use of toxic equivalency factors (or TEFs), which are discussed in the *Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of*

Chlorinated Dibenzo-p-Dioxins and –Dibenzofurans (CDDs and CDFs) and 1989 Update (USEPA, 1989c). In a 1997 workshop in Stockholm, the World Health Organization (WHO) working group on TEFs agreed on TEFs to be used to assess risks posed by dioxin-like compounds to humans and other mammals. The USEPA has endorsed the use of these TEFs for evaluating contaminated sites in the U.S. TEFs proposed by the WHO and presented in Table 19 below.

For dioxin-contaminated sites, 2,3,7,8-TCDD equivalent concentrations are calculated as the sum of each dioxin congener concentration times its TEF. This concentration, in 2,3,7,8-TCDD equivalents, should then be compared with the dioxin SCTL. The dioxin SCTL is also applicable to PCDFs, given the similarity in the toxicity of these two classes of chemicals.

Detection of any PCDD congener signifies that PCDD contamination occurs in the sample, while detection of any PCDF congener in the sample signifies that PCDF contamination is present. In these cases, PCDD or PCDF concentrations should be converted to 2,3,7,7-TCDD equivalents using the recommended TEFs presented in Table 19a and compared to the CTL for 2,3,7,8-TCDD. For non-detected congeners, equivalent concentrations should be calculated using a proxy value for the concentration. Specifically, for data with "U", "M", or "EDL" qualifiers, use one-half the reported limit as the proxy value for calculating 2,3,7,8-TCDD equivalents. Estimated concentrations with a "J", "I", "T", or "EMPC" qualifier should be used without modification for calculating TEQs. For samples with both PCDF and PCDD contamination, the sum of the 2,3,7,8-TCDD equivalent concentrations for both classes of compounds should be compared to the CTL for 2,3,7,8-TCDD. Selection of the laboratory method should pay attention to the method detection limit achievable for each congener in order to avoid calculated equivalent concentrations that are artificially high due to elevated detection limits. In this respect, EPA method 8290 seems to provide adequate sensitivity.

Reports from analytical laboratories often include total concentrations for dioxin or furan congeners with the same degree of chlorination (e.g., total tetrachloro congeners). These data cannot be used to estimate a 2,3,7,8-TCDD equivalent concentration. On the other hand, these concentrations represent the maximum possible total concentration of certain congeners of concern. Concentrations for individual congeners estimated as half the detection limit can be checked against these totals to minimize possible overestimation of the 2,3,7,8-TCDD equivalent concentration for the sample.

TEFs are also available to convert certain PCB congeners to 2,3,7,8-TCDD equivalents (see Table 19, Source Van den Berg et. al., 1998). Therefore, if concentrations of these individual congeners are known, it is possible to use the toxic equivalency approach to assess cumulative risks posed by these contaminants. Calculating total TEQs from PCB congeners would be warranted, for example, in circumstances where there is both PCB and dioxin/furan contamination. In this

situation, the sum of TEQs for PCBs would be added to the dioxin and/or furan TEQs in the sample to obtain the total 2,3,7,8-TCDD equivalents. This value would then be compared with the cleanup target for 2,3,7,8-TCDD. For most sites in Dade County, PCBs are not found with dioxins or furans, and congener analysis is not required. For these sites, risks from PCB contamination are evaluated by comparison with cleanup targets developed using toxicity values for PCB mixtures (i.e., Aroclors). Toxicity values are available for both cancer and non-cancer effects, although in most situations cleanup targets based on carcinogenicity (and a target excess cancer risk of 1 E-06) are lower than those based on non-cancer effects. Toxicity values developed for PCB mixtures include contributions to toxicity of both dioxin-like and non-dioxin-like congeners.

Table 19

Toxic Equivalency Factors (TEFs) Used to Express PCDD PCDF, and PCB Congener Concentrations as 2,3,7,8-TCDD Equivalents

Congener	Toxic Equivalency Factor
Polychlorinated dibenzodioxins	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	.1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
Polychlorinated dibenzofurans	
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001
Polychlorinated biphenyls	
Congener 77	0.0001
Congener 81	0.0001
Congener 105	0.0001
Congener 114	0.0005
Congener 118	0.0001
Congener 123	0.0001
Congener 126	0.1
Congener 156	0.0005
Congener 157	0.0005
Congener 167	0.00001
Congener 169	0.01
Congener 189	0.0001

8. Development of SCTLs for Carcinogenic Policyclic Aromatic Hydrocarbons

As in the case of dioxins and furans, carcinogenic polycyclic aromatic hydrocarbons (PAHs) are found as mixtures in contaminated media. Given that carcinogenic PAHs have a common toxicity mechanism, but display difference toxic potencies, the TEF approach can be used to convert individual PAH site concentrations into a single concentration of the index chemical, benzo(a)pyrene. This approach should be followed to evaluate risks from direct exposure. Consequently, direct exposure SCTLs were derived only for benzo(a)pyrene. SCTLs based on leachibility were developed for individual carcinogenic PAHs because of their varying leaching potential. Table 20 below presents the TEFs that should be used to calculate site concentrations before comparison with the direct toxicity SCTLs for benzo(a)pyrene. When one or more carcinogenic PAHs is present in a sample, proxy values of one-half the detection limit should be used to calculate benzo(a)pyrene equivalents for non-detect data (i.e., data with a "U" or "M" qualifier).

Table 20
Toxic Equivalency Factors for Carcinogenic PAHs

Contaminant	TEF
benzo(a)pyrene	1.0
benzo(a)anthracene	0.1
benzo(b)fluoranthene	0.1
benzo(k)fluoranthene	0.01
Chrysene	0.001
dibenz(a,h)anthracene	1.0
indeno(1,2,3-cd)pyrene	0.1

9. Development of CTLs for Vinyl Chloride

The IRIS record for vinyl chloride lists two sets of cancer potency values for the oral and inhalation routes; one for continuous lifetime exposure during adulthood and the other for continuous lifetime exposure from birth. The supporting documentation (USEPA, 2000d) discusses laboratory animal data showing that exposures during early life produce a cancer response that is qualitatively different from that observed from lifetime exposure of mature animals, but that produce similar cancer incidences. Therefore, the cancer potency estimates developed using data from mature animals should be doubled when evaluating cancer risks from continuous lifetime exposures commencing at birth. This approach was followed for developing SCTLs to be protective of lifetime exposure from birth. The industrial/commercial SCTL was

calculated using the cancer potency estimates appropriate for continuous lifetime exposure during adulthood and prorating the cumulative dose over the averaging time of 70 years. According to USEPA guidance (USEPA, 2000d), calculation of cancer risks for exposures less than a lifetime but that start at birth should add risks from continous lifetime exposure not prorated over a lifetime and exposures over the entire exposure period but prorated over a lifetime (i.e., averaged over 70 years). This procedure was followed to develop SCTLs for the residential scenario. Non-prorated risks were calculated by assuming a lifetime resident is exposed continuously for 70 yr. Lifetime resident exposure assumptions are presented in Table 21. These risks were then added to those calculated for the aggregate resident using the equation shown in Figure 4. The SCTL was calculated through an iterative process until the total risk equaled 1.0E-6.

Table 21
Exposure Assumptions Used for the Lifetime Resident

Assumption	Value	Source
Body weight, kg	64.05	Average of NHANES III body weight data for 0-70 year olds
Soil ingestion rate, mg/d	108.6	Time weighted average of 6 yr ingesting 200 mg/d and 64 yr ingesting 100 mg/d
Skin surface exposed, cm^2	5920	Product of total surface area exposed for lifetime resident (17,496 cm^2) calculated using Burmaster's equation (1998) and time weighted average of 6 yr exposing 43.1 % of skin surface and 64 yr exposing 33.3% (See Table A-5)
Dermal adherence factor, mg/cm^2	0.08	Time weighted average of 6 yr with child dermal adherence of 0.2 mg/ cm^2 and 64 yr with adult dermal adherence of 0.07 mg/ cm^2
Inhalation rate, m^2/d	12.56	Time weighted average of male and femal inhalation rates presented in Table 5-12 of the EFH (USEPA, 1997)

VI. Chemical Interactions

When selecting the target risk or hazard for CTL development, it must be kept in mind that this is the accepted incremental excess risk per chemical, and not necessarily the accepted increase in risk to the individual. For most sites, exposure is to more than one chemical, and the overall risk to the individual posed by contamination at the site will be some composite of the individual chemical risks. CTLs for generic application cannot be developed based on total target risk to the exposed individual, since this risk will vary depending upon the number and type of chemicals (i.e., carcinogenic versus non-carcinogenic) present at specific sites.

Exposure to combinations of chemicals may result in interactions leading to a significant increase or decrease in the overall toxicity of the mixture compared to the summation of the toxicities of the individual chemicals. Toxic interactions may occur as a result of an alteration in

the absorption, distribution, metabolism, and excretion of one chemical by another, modifying its toxicity. Studies in animals have reported the occurrence of such interactions among gaseous pollutants, pesticides, metals, and solvents. Interactions may also occur when one chemical alters the responsiveness of cells and target organs to the effects of other chemicals, such as through receptor up-regulation or altered cell-signaling pathways. Very little information exists on toxic interactions in humans, and inferences must be made from studies of toxicant effects in laboratory animals. Even in circumstances where significant interactions have been observed in these studies, 1) the dosages at which the interaction occurs are usually not well characterized; 2) there is often uncertainty as to whether the mechanism for the interaction is relevant to humans, particularly at the comparatively low exposures typically encountered from contaminated environmental media; and 3) most such studies involve exposure to two chemicals, whereas exposure at contaminated sites can involve several toxicants. For these reasons, the utility of these observations in evaluating the human health implications of multiple chemical exposures is limited, and it is extremely difficult to address chemical interactions in quantitative risk assessment other than on a rather simplistic level.

The standard approach taken in baseline risk assessments for contaminated sites is to assume that risks to the individual from multiple chemicals are additive. The incremental excess cancer risk to the exposed individual is the sum of the cancer risks from individual carcinogens. It is recognized that the cancer risks from individual chemicals are not truly independent (e.g., death from cancer from one contaminant reduces the risk of cancer from other contaminants to zero; also, there is evidence suggesting that developing one cancer may increase the risk of developing a second cancer), and therefore, some error will be introduced in calculating total cancer risk from the sum of the individual cancer risks. However, since the probability of developing cancer from environmental exposure to contaminants is usually small, the error in summing them will also be small and of little consequence in estimating total cancer risk.

For non-carcinogens, hazard quotients for individual chemicals are summed when there is evidence that the chemicals may have additive effects. The same mechanisms of action or the same target organ for toxicity are usually taken as evidence for potential additivity. Tables 1, 2, 5b, 6, and 7 provide information regarding non-cancer human health effects that may result from exposure to chemicals for which CTLs have been developed. The non-cancer effect(s) listed are from the critical study [or studies] used to develop the toxicity value for that chemical. The table does not provide an exhaustive list of potential target organs/systems and effects for each chemical. Rather, the table is intended to list target organs/systems most likely to be affected at or near the lowest observable adverse effect level (LOAEL). These effects are most relevant in determining when additive toxicity might occur under circumstances of environmental exposure. In general, the

target organs/systems and effects were identified from narratives accompanying presentation of toxicity values in the sources identified in Table 5b (e.g., IRIS).

In some situations, a very specific type of effect was identified for a chemical in the toxicity value narrative. When this was encountered, a more general listing of target organ/system was made so that circumstances of potential additive toxicity would not be missed. For example, the critical effect for chlorothalonil is listed in IRIS as "renal tubular epithelial vacuolation," an effect on a specific region of the kidney. Other chemicals, affecting other regions of the kidney, could produce cumulative renal toxicity with chlorothalonil. For this reason, the target for chlorothalonil was generalized to "kidney" so insure adequate consideration of potential additive toxicity. In other situations, the type of effect identified for a chemical was very non-specific, such as "decreased body weight." Decreased body weight suggests that an adverse effect is occurring, but provides no indication of the target organ/system. When decreased body weight is found along with another, more specific effect (such as liver toxicity), it is assumed to be secondary to the more specific toxicity listed. It is not, therefore, listed among the target organs/systems and effects. It is, however, listed when it is the only critical effect identified. Chemicals that share non-specific effects such as decreased body weight should be considered additive, unless there is convincing evidence that they do not produce cumulative toxicity.

While, in principle, interactions can occur among chemicals that result in greater-than-additive effects, at present there are no specific examples that indicate that the additive approach described above is not sufficiently conservative for initial site evaluation purposes. If evidence arises in the future for interactions between specific chemicals that would render this approach less than health-protective, the approach should be modified to take these interactions into consideration.

Although simple additivity is the most commonly recommended approach for risk assessment, the incorporation of quantitative information on toxic interactions as a means to more specifically evaluate the potential for additivity is an alternative for more detailed, site-specific risk assessments. Additivity may result from *dose addition*, which occurs when chemicals act on similar biological systems and elicit a common response, whereas *response addition* occurs when chemicals act by independent mechanisms to produce toxicity to the same organ or tissue (Hertzberg et al., 1997). With *dose addition*, the chemicals are assumed to be functional clones and thereby follow similar pathways of uptake, metabolism, distribution and elimination, and elicit the same toxic effect. Thus, although the dose of one chemical may be too small to elicit an effect, the addition of a second chemical may be enough so as to increase the total dose to a level that results in an adverse effect. Under *response addition*, different physiologic pathways are followed and the response to one chemical occurs whether or not the second chemical is present. For example, the

liver may be the common target organ, but the mechanism of injury can differ (e.g., peroxisomal proliferation, induction of oxidant stress, protein adduction). However, it is the sum of the responses at the common target organ that is measured as the additive effect, regardless of the differences in mechanism of action. *Dose addition* should always be treated as a summation of hazard quotients. *Response addition*, however, may not always be accurately characterized by a simple summation of hazard quotients, depending upon the toxic mechanisms involved. In cases of *response addition*, approaches other than simple addition can be used to derive site-specific CTLs, but must be carefully justified by the mechanism(s) of action of the chemicals and supported by empirical observations.

In the context of a detailed, site-specific risk assessment, chemical interactions other than addition need to be considered, such as antagonism, inhibition, masking, synergism, and potentiation. As with *response addition*, manipulation of CTLs based on these interactions should be soundly and carefully based on mechanistic principles supported by empirical observations from the peer-reviewed scientific literature.

VII. Sources of Variability and Uncertainty

Development of CTLs requires the inclusion of several different inputs, each associated with some degree of variability and uncertainty. Variability and uncertainty exist in inputs related both to toxic potency (i.e., the toxicity values) and to exposure.

A. Variability and Uncertainty in Toxic Potency Estimates

Variability and uncertainty are important considerations in the development and use of toxicity values. Toxicity values are numerical expressions of the toxic potency of a chemical. They are developed based on information collected from epidemiological studies of human populations or from studies involving controlled exposure of laboratory animals. While epidemiological lines of evidence might apply more directly to the assessment of risks to human health, the lack of control of the exposure level almost always introduces significant uncertainty in the dose-response information gained from this type of study. In addition, many of these studies rely on occupational cohorts, where exposure occurs almost exclusively to healthy adults. As such, potentially sensitive populations such as pregnant women, the elderly, and children are usually not represented. Studies using animal models allow for precise control of exposure, but require extrapolation of results from animals to humans. There is always uncertainty associated with inter-species extrapolation due to a variety of factors, including possible differences in uptake and

metabolism of the chemical, sensitivity of the target organ or tissue to the effects of the chemical, and issues related to scaling of doses from laboratory animals to much larger humans. Data from animal or even human studies may also not match environmental exposures well in other ways, leading to the need for other types of extrapolation, including extrapolation of information obtained from one route of exposure to another (e.g., using data from an inhalation exposure study to assess toxicity from oral exposure), from one length of exposure to another (e.g., using data from a subchronic study to determine safe doses for chronic exposure), and from one dose range to another (typically, from high doses used in toxicity studies to much lower doses associated with environmental exposures). Each of these types of extrapolation contributes uncertainty to the risk assessment process.

Yet another type of uncertainty involves the extent to which the toxicity of a chemical has been well characterized. The use of toxicity values to derive safe doses for chemicals relies upon the assumption that all possible adverse effects have been documented, and therefore complete protection against a chemical's toxic effects is afforded by basing the toxicity value on the most sensitive effect (i.e., that which occurs at the lowest dose). Some chemicals have been extensively studied, leading to confidence that both the most sensitive effect and the doses at which it occurs are well understood. For other chemicals, however, toxicity data are limited, and the extent to which available information has adequately characterized sensitive effects is uncertain.

When developing safe dose values for chemicals, such as the USEPA's reference doses (RfDs), the regulatory response to the existence of these uncertainties is the use of safety or uncertainty factors. The process begins with a no-effect dose or concentration for the most sensitive effect as identified from existing studies. Depending upon the nature of the data available for the chemical, one or more uncertainty factors may be applied. For example, a factor of up to 10 is applied when extrapolating from studies in animals to humans, and a factor of up to 10 is applied when using data from less than chronic exposure. If a no-effect dose is not available (i.e., all of the doses tested have produced an effect), the lowest dose tested is used and an additional factor of 10 is applied. Variability is also addressed in this process. Individuals may vary in their sensitivity to a chemical due to a variety of factors. Since most toxicity data are derived from studies of healthy test subjects, these data may not adequately represent responses in sensitive subjects. In view of this, an uncertainty factor of 10 is applied for protection of sensitive individuals (except in unusual cases in which the toxicity data are derived from sensitive subjects). Finally, a "modifying factor" may be used based on professional judgment. This modifying factor may range from 1 to 10, the magnitude depending on factors such as the completeness of the overall database and the number of species tested. The uncertainty factors and modifying factor are multiplicative rather than additive, and the overall reduction in dose can range up to 10,000-fold, depending upon the

component uncertainties. In the case of oral RfDs presented in IRIS, the median total adjustment factor is 300, while the average is 887 and the maximum value is 12,000. The uncertainty factors incorporated into the RfDs used to develop CTLs are not listed in this technical background document, but can usually be obtained from the source of the RfD (e.g., IRIS, etc.; see Table 5b).

As with non-cancer health effects, there are a number of uncertainties associated with development of CTLs based on carcinogenicity. A major source of uncertainty is the shape of the dose-response relationship below the observation range. The target cancer probability for CTLs (10^{-6} excess cancer incidence) is several orders of magnitude lower than what can be reliably measured in cancer studies, requiring that assumptions be made about cancer responses at low, environmentally-relevant doses. Several models for estimating low-dose responses to carcinogens have been proposed, and can yield very different estimates of cancer risks at low doses. The USEPA has chosen to use the multi-stage model for developing estimates of cancer potency for most carcinogens, in part because it has a biological basis and in part because it tends to give higher estimates of risks than other models, and is therefore unlikely to underestimate the true cancer risk. The linearized multistage procedure is typically used, in which an upper confidence limit fit of the cancer data is used rather than the best fit. Because data sets from cancer studies are usually very limited in terms of the numbers of doses tested, use of an upper confidence limit value on the slope helps to ensure that the result from a particular data set does not underestimate the true slope. This approach also contributes to the conservatism of the cancer potency estimates.

An additional source of uncertainty is whether a chemical is in fact capable of producing cancer in humans. The USEPA uses a weight-of-evidence scheme to characterize chemicals as to the certainty with which they are, or are not, carcinogenic in humans, based on evidence from animal and human studies. Traditionally, chemicals have been classified using letter designations for weight-of-evidence: Group A chemicals are "known to produce cancer in humans," Group B are "probable human carcinogens" either based on limited evidence from epidemiological studies and sufficient evidence from animal studies (Group B1), or based only on sufficient evidence from animal studies (Group B2). Group C are "possible human carcinogens," Group D chemicals are "not classifiable as to human carcinogenicity," and Group E are those with "no evidence of carcinogenicity for humans." More recently, the USEPA has chosen to characterize the weigh-of-evidence in narrative form. For chemicals still characterized under the older classification scheme, the designation (A, B2, etc.) is listed in Table 5a. Many chemicals have not been tested for carcinogenicity. For those chemicals, some degree of uncertainty exists as to whether they pose a potential cancer risk.

B. Variability and Uncertainty in Exposure Parameters

Variability exists in exposure because of inherent differences among individuals within an exposed population. Among individuals exposed to contaminated soils or drinking water in a residential setting, for example, there will be differences in virtually all of the variables used in the risk equations (body weight, drinking water or soil ingestion rates, exposure frequency, etc.). As a result, when calculating doses of contaminants resulting from exposure, there is no single dose that corresponds to a given concentration of chemical in soil or water, but rather a distribution of doses within an exposed population of interest. As a practical matter, a single dose must be selected upon which to base a CTL. From a regulatory perspective, if the goal is to protect most or all of an exposed population, that dose should reflect the upper end of the range of plausible exposures. This approach was used for the development of CTLs for Chapter 24. That is, from a range of possible values for each exposure variable, values were selected to produce dose estimates near the upper end of the likely range of doses for an exposed population. These dose estimates are intended to correspond to what the USEPA terms "reasonable maximum exposure" or "high-end exposure" — exposure at about the 90^{th} or 95^{th} percentile.

There are several potential sources of uncertainty in the exposure component of the CTL formula. For example, CTL development requires several inputs regarding physical/chemical properties of the contaminant. Several of these physical/chemical properties are hard to measure directly with reasonable accuracy, and consequently a range of values for a given parameter can often be found in the literature. When measured values are unavailable, they can be predicted from the chemical's structure or other properties, although the accuracy of these predictions is also a source of uncertainty. In general, uncertainty in the selection of physical/chemical inputs is minimized by a preference for measured over estimated values, and by choosing values from sources which utilize some form of data quality assessment (e.g., peer review). Another example is uncertainty regarding the way in which chemical concentrations in soil might change in the future. Chemical concentrations in soil may decline due to a variety of processes such as volatilization to air, leaching to groundwater, or biodegradation. The rate of change in concentration, however, can seldom be predicted with certainty. [Note: There is an element of variability in this as well, in that the rate of disappearance of a chemical will depend in part on factors that may change from site to site.] Loss of chemical over time is potentially an important issue since all but a few soil CTLs, and all groundwater CTLs, are based on chronic exposure. For the purpose of creating default CTLs, this particular uncertainty is addressed by assuming that there is no loss of chemical over time; that is, that the concentration of chemical presently found at a site will persist indefinitely. For persistent chemicals (such as inorganics), this assumption is fairly accurate, while for other

chemicals it is conservative. In the context of developing an alternative CTL for a specific site, this uncertainty could conceivably be reduced by obtaining site-specific information on the rate of loss of the chemical from soil, and using this information in the development of the CTL.

The conservatism of the CTLs is a function of the combined conservatism of the individual assumptions and inputs used to create them. Not all individual inputs are high-end values -- using all high end values would produce CTLs based on extreme, unrealistic exposure assumptions. Rather, the intent is to combine high-end and central tendency assumptions such that the outcome is a CTL that reflects a reasonable, high-end exposure. Table 3 shows the specific values chosen for each exposure variable. The section below discusses the individual inputs and provides information as to whether each is considered a high-end, central tendency, or (in a few cases), a less-than-central tendency value.

1. Soil Ingestion Rate

Default soil ingestion rates of 200 mg/day for a child (1-6 years), 100 mg/day for an older resident (7-31 years) and 50 mg/day for an adult worker (age not specified) were obtained from USEPA (1996b). The USEPA (USEPA, 1997) reviewed several studies to derive estimates of the amount of soil ingested by children and adults in its Exposure Factors Handbook (EFH) document. There is a wide range in mean soil ingestion rates due to differences in study design and methods used to determine soil ingestion (USEPA, 1997). The mean soil ingestion rate values for children from the studies reviewed in the EFH ranged from 39 mg/day to 271 mg/day, with an average of 146 mg/day. Therefore, a value of 200 mg/day is considered to be a conservative estimate of the mean. Upper (95^{th}) percentile values ranged from 106 mg/day to 1432 mg/day, with an average of 383 mg/day. Rounding to one significant figure, the upper percentile soil ingestion rate for children is 400 mg/day. A default, mean soil ingestion rate of 50 mg/day for workers was derived in the EFH based on a study conducted by Calabrese and collaborators on six adults. The mean soil ingestion rates for the six individuals ranged from 30 mg/day to 100 mg/day. The soil ingestion rate assumption for adult residents (100 mg/day) corresponds to the upper end of this range, whereas the soil ingestion rate assumed for workers is approximately in the middle. It should be noted that uncertainties are associated with the soil ingestion defaults because of the short time frame of these studies, which lasted from several days to a couple of weeks. The soil ingestion rate for an aggregate resident (120 mg/day) is a time-weighed average of a 6-year exposure of a child to 200 mg/day soil and 24 years of exposure of an individual aged 7-31 years to 100 mg/day soil. Consequently, it is a combination of a conservative estimate of the mean soil ingestion rate for a child and a high-end ingestion rate assumption for an adult resident.

2. Groundwater Ingestion Rate

The groundwater ingestion rate of 2 L/day (adult) is the value commonly used by the USEPA to derive reference water concentrations. The Exposure Factors Handbook (USEPA, 1997) recommends 1.3 and 2.3 L/day to represent mean and upper percentile (90^{th}) water intake rates, respectively. These values are based on data from two national surveys. According to the EFH, the customary value of 2 L/day represents the 84^{th} percentile of the national dataset used to derive the water intake values.

3. Body Weight

Appendix A of this Technical Report includes a detailed description of the derivation of body weights, exposed surface areas, and inhalation rates. The default body weights for child (16.8 kg), aggregate resident (51.9 kg) and adult (76.1 kg) receptors were derived from the Third National Health and Nutritional Examination Survey (NHANES III). These default body weights are calculated as the weighted means of individuals aged 1 to 7 years (child), 1 to 31 years (aggregate resident) and 18 to 65 years (adult). As such, they are central tendency measures.

4. Exposed Skin Surface Area

The exposed skin skin surface (SA) for each of the receptors (child, aggregate resident and worker) was calculated by multiplying the total skin SA estimate and the percentage of the total skin SA assumed to be exposed. The default values for total SA for the child, aggregate resident and worker were central tendency values because they were calculated from the weighted mean body weights using a "best fit" allometric equation proposed by Burmaster (1998). The percentage exposed was based on assumptions regarding clothing patterns and the fraction of total area represented by each body part area. The fractions represented by each body part are average, or central tendency values as listed in the EFH (USEPA, 1997). For a child, the head, hands, feet, lower legs and forearms were the exposed body parts assuming that the child is wearing short pants, short-sleeved shirt and no shoes. This assumption is reasonably conservative in that it is doubtful that a child would have a larger skin area exposed on a long-term basis. For the aggregate resident, the exposed SA was a time-weighted average of the exposed SA of a child and an individual through ages 1-31 years (see Table A-6 in Appendix A). Exposed portions of the body for the individual from ages 7-31 years were similar to the child with the exception that shoes were assumed to be worn. Thus, exposed skin SA for the aggregate resident was also reasonably conservative. The exposed portions of the body for the worker were the head, hands and forearms, assuming that the worker wore long pants, shoes and short sleeved shirt. The exposed skin SA

might be viewed as central tendency, since this pattern of clothing probably applies to most workers in Florida.

5. Inhalation Rate

The default inhalation rate of 8.1 m^3/day for a child 1-7 years of age was derived as the time-weighted average of age-specific inhalation rates presented in Table 5-23 of the EFH (USEPA, 1997). The inhalation rate of 12.2 m^3/day for the aggregate resident (1-31 years of age) was also calculated from data presented in Table 5-23 of the EFH. These values are based on energy requirements to sustain basal metabolism and normal ativity and therefore should be considered to represent central tendency values. The inhalation rate of an adult worker (20 m^3/day) was originally proposed by the USEPA (1991) and is said to represent a "reasonable upper-bound" value for adults.

6. Relative Source Contribution

The USEPA Office of Drinking Water uses a Relative source Contribution (RSC) term in the derivation of reference concentrations for drinking water. The RSC is an estimate of drinking water's contribution to total exposure to the contaminant. The 20% RSC represents a default value to be replaced with a chemical-specific value when data are available. For chemicals not commonly found in other sources such as food, nutritional supplements, and other consumer products, the 20% RSC would be conservative. Conversely, for chemicals that are either part of the diet or nutritional supplements, there may be little or no conservatism associated with this assumption.

7. Averaging Time

The Averaging Time (AT) values for all receptors were obtained from RAGS- Part A (USEPA, 1989a). For carcinogens, the default AT value is 25,550 days (70 years, aggregate resident and worker receptors) because cancer effects are considered cumulative over a lifetime, and cancer potency values (cancer slope factors) are standardized for lifetime exposure. Unlike groundwater CTLs, all soil CTLs are based on less-than-lifetime contact. Averaging doses received from this contact over a lifetime may be appropriate in estimating cancer risks for some chemicals, but not others (Halmes et al., 2000). Consequently, lifetime averaging of doses from soils may be less than conservative for some chemicals. For non-carcinogens, the AT default values are equal to exposure duration, and are not in themselves either conservative or un-conservative.

8. Exposure Frequency and Exposure Duration

Exposure Frequency (EF) and Exposure Duration (ED) are used to estimate the total time of exposure of a receptor to contaminants. Default values for EF and ED were obtained from RAGS-Part A. Default EF values are 350 days (child and aggregate resident) and 250 days (worker) whereas default values for ED are 6 years (child), 30 years (aggregate resident) and 25 years (worker). These values are considered to be "upper-bound" values of exposure by the USEPA. With respect to exposure duration, data presented in the EFH (USEPA, 1997) show that 83.5% of U.S. householders reside in the same place for 25 years or less and 92% reside at the same location for 35 years or less. Thus, the 30-year ED is a high-end assumption. For workers, a 25-year ED represents the 95th percentile for number of years at a specific job based on 1987 Bureau of Labor Statistics. It too is a high-end assumption. The EF assumptions are not based on specific percentiles, but are selected to represent minimal time away from home or the workplace.

9. Adherence Factor

In order to estimate the intake of contaminants from dermal contact with soil one needs to know the soil adherence factor (mg/cm^2), i.e., the amount of soil that comes in contact with a specified area of skin. The default AF values for a child (0.2 mg/cm^2) and worker (0.2 mg/cm^2) were derived from RAGS-Part E (USEPA, 2000b). AF varies among different areas of the skin, so a weighted average was computed to derive an overall AF representative of exposed areas of the skin. The recommended AF for a child corresponds to the 95th percentile weighted AF for children playing at a day care center (central tendency soil contact activity), and to the 50th percentile for children playing in wet soil (high-end soil contact activity). The recommended AF for a worker corresponds to the 50th percentile weighted AF for utility workers (the activity determined to represent a high-end contact activity). The USEPA recommends an AF of 0.07 mg/cm^2 for adult residents, which is a central tendency (50th percentile) AF for gardeners (a high-end activity). In order to obtain an AF for the aggregate resident (0.1 mg/cm^2) a time-weighted average of the AF for the child and adult resident was derived. Since all values are based on either an upper percentile adherence or a high-end contact activity, the AF assumptions are considered high-end exposure values.

10. Dermal Absorption Factor

The fraction of a dose that is absorbed through the skin is known as the dermal absorption factor (DA). The default DA assumptions are based on USEPA Region 4 (2000e) guidance recommending a value of 0.01 for organics and 0.001 for inorganics. The technical basis for these values is not explained in the guidance other than to state that they include consideration of reduced

dermal absorption of chemicals from a soil matrix. There is evidence to indicate that the dermal absorption of some chemicals may exceed these defaults, and specific examples are provided in the USEPA Dermal Assessment guidance (USEPA, 2000b).

11. Particulate Emission Factor (PEF)

The particulate emission factor (PEF) relates the concentration of contaminant in soil with the concentration of dust particles in air. The default value used in Chapter 24 of the Code is 1.24×10^9 m^3/kg. The variables that are used to calculate the PEF are inverse of mean concentration at the center of a 0.5 acre-square source also known as air dispersion factor (Q/C), mean annual windspeed (Um), equivalent threshold value of windspeed at a height of 7 m (Ut) and function dependent on Um/Ut (F(x)). The default values for these parameters are listed in the Soil Screening Guidance (SSG) (USEPA, 1996b). The Q/C default value is an upper-end estimate because it best approximates the 90^{th} percentile Q/C term for conditions in Miami. The mean annual windspeed (Um) value, although based on national data, is similar to the annual average windspeed measured in Florida. It can be considered a "best estimate" or central tendency value. Another default assumption for calculating the PEFs is that 50% of the contaminated area has vegetative cover. This value might be considered central tendency, since individual sites can have more or less vegetative cover.

12. Physical/chemical parameters

In order to calculate an SCTL, one needs to know certain physical/chemical parameters of the contaminant such as melting point (mp), density (d), solubility (S), Henry's Law Constant (HLC), diffusivity in air (D_i), diffusivity in water (D_w), soil-water partition coefficient (K_d) and soil-water partition coefficients for organic compounds (K_{oc}) (see Table 4 for values). Measured values are preferred over estimated values. Some of these parameters that depend solely on the characteristics of the chemical, such as melting point and density, are well established for most chemicals, and therefore not much uncertainty is associated with them. For some parameters, the reported values can vary among different sources. In those cases, a central tendency estimate is used, such as the geometric mean of reported values. Other parameters are calculated using formulas intended to provide best estimates. Overall, the physical/chemical parameters used to derive CTLs are based on central tendency or best estimate values.

13. Volatilization Factor

Volatilization Factors (VF) are receptor- and chemical- specific values calculated using several inputs. One is an air dispersion factor (Q/C), which is an upper end assumption (as

explained above). Chemical-specific factors that affect volatility are all central tendency values (also as discussed above). Soil characteristics are based on loamy soil. These characteristics could be considered central tendency — some soil conditions in Florida may favor more volatilization and others lesser volatilization than would occur from loamy soil. The VF term assumes that exposure begins immediately and occurs over the entire exposure duration. Flux of volatile contaminants to air declines over time, and the VF is used to reflect the average air concentration over the exposure interval. Depending upon the actual exposure circumstances (when exposure starts and how long it persists), this assumption may over- or underestimate the actual intake rate. Finally, the VF model is based on the assumption of an infinite source (i.e., that the concentration present at the soil surface extends below surface to infinity). This is a highly conservative assumption because it allows volatilization rates to be calculated over time that could not possibly occur because of contaminant mass limitations.

14. Dilution Attenuation Factor

DAF is defined as the ratio of contaminant concentration in soil leachate to the concentration in groundwater at the receptor point. The DAF used to calculate leachability-based soil CTLs is based on a recommendation in the SSG for sites with a contaminated area of 0.5 acres. The USEPA selected a default DAF of 20 using a "weight of evidence" approach that considered results from the EPA Composite Model for Leachate Migration with Transformation Products (EPACMTP), as well as results from applying the SSL dilution model described in Section 2.5.5 of the SSG to 300 groundwater sites across the country. The DAF value of 20 lies between the 90^{th} and 95^{th} percentile for 0.5 acre contamination using the EPACMTP model, but was found to be the geometric mean DAF for all 0.5 acre groundwater sites included in their analysis. Consequently, it should be viewed as a central tendency value.

C. Overall Conservatism of the Exposure Parameters

The intent of selecting a combination of central tendency and upper bound exposure inputs is to create an overall exposure estimate that represents high-end exposure. This is typically defined as an upper, but not extreme, percentile of the contaminant intake anticipated to occur within an exposed population. An estimate of the percentile of intake corresponding to a set of exposure assumptions, such as those employed for CTL development, can be obtained through a probabilistic analysis (e.g., Monte Carlo simulation using distributions as inputs for exposure variables rather than single values). This type of analysis can be resource-intensive, however, and

has not been conducted for the CTL exposure assumptions. Consequently, a quantitative estimate of the degree of conservatism afforded by the input values selected cannot be made.

From a qualitative standpoint, groundwater intake is calculated from only two variables, drinking water consumption rate and body weight. The water intake value selected is at the 84th percentile (see section VII B 2, above) and the body weight is at the 50th percentile (see section VII B 3). These two variables are likely to be positively correlated, i.e., heavier individuals ingest more water. If so, the combined water ingestion per unit body weight value used for CTL development may be higher than the 84th percentile.

An additional aspect of groundwater CTL development is the use of the relative source contribution (RSC) term. The RSC is not part of the exposure estimate, but it affects how the exposure estimate is used. The default RSC of 0.2 allots only 20% of an acceptable daily intake of a non-carcinogen to drinking water. The intent is to insure that the total intake of the contaminant, from both drinking water and non-drinking water sources, does not exceed risk based limits. For most chemicals for which GCTLs have been developed, it is unlikely that there will be substantial non-drinking water intake, and the default 20% RSC restriction is therefore conservative.

Assessing the conservatism of the exposure estimates used to derive SCTLs is more complex because of the larger number of input values. SCTLs for most chemicals (i.e., all but highly volatile chemicals) are dominated by incidental soil ingestion; this component of the equation is the risk driver. Soil ingestion rate assumptions range from central tendency to high end, depending upon the scenario and receptor. Body weight assumptions are central tendency, and the other critical inputs, exposure duration and exposure frequency, are upper percentile values. Overall intakes derived using these sets of assumptions will be upper percentile values (i.e., greater than central tendency), but the magnitude of the conservatism cannot be established without formal probabilistic analysis.

Under some circumstances, combined risk when there is exposure to more than one chemical at a site is addressed explicitly through apportionment of default CTLs. There are several limitations to simple and weighted apportionment approaches that should be acknowledged. These arise from combining upper bound estimates inherent in the toxicity and exposure portions of the risk calculations for different chemicals. For carcinogens, most slope factors are derived from an upper 95th percentile estimate of potency, and because upper 95th percentiles of probability distributions are not strictly additive, the total cancer risk estimate might become artificially more conservative as risks from a number of different contaminants are summed. If one or two carcinogens drive the risks, however, this problem is not of concern.

Similarly, exposure point concentrations are often based on 95% UCLs or maximums of sample data. Because these values are not strictly additive, summing the risks from multiple

chemicals will inflate the resulting estimates and lead to more conservative apportioned CTLs. The degree of this additional conservatism is related to the degree of collocation of chemical contaminants across the site. In cases where high levels of one chemical are collocated with high levels of a second chemical, this problem is less of a concern.

Although technically more complicated, alternative apportionment methods could be developed, and if validated, employed to calculate apportioned CTLs that more precisely meet the acceptable risk levels. Depending upon the number of chemicals apportioned, the degree of collocation of the contaminants, and the associated costs of analysis and remediation, the effort to derive more precise risk estimates may or may not be warranted.

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http://www.epa.gov/opptintr/exposure/docs/episuite.htm

HALs: Drinking Water Regulations and Health Advisories (EPA/822/B-96/002)
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MINTEQA2: Metal Speciation Equilibrium for Surface and Groundwater
http://www.epa.gov/ceampubl/mmedia/minteq/

REG 3: USEPA Region 3 Risk Based Concentration Tables
http://www.epa.gov/reg3hwmd/risk/index.htm

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XI. List of Acronyms and Definitions

Acute Exposure:	A single, brief exposure, usually less than 24 hours in duration.
Acute Toxicity:	The ability of a substance to cause adverse health effects as a result of an acute exposure.
Additivity:	The interaction of chemicals within the body that results in a toxic response to the combined exposure comparable to adding the toxic effects elicited by each chemical separately.
Aliphatic Hydrocarbon:	A chemical composed of hydrogen and carbon in which the carbon atoms form a chain.
Antagonism:	Type of chemical interaction that exists when toxic effects from exposure to a combination of chemicals are less than what is expected based on their individual toxicities.
Aromatic Hydrocarbon:	A chemical composed of hydrogen and carbon that contains one or more aromatic (benzene) rings.
ATSDR:	Agency for Toxic Substances and Disease Registry.
BCF:	Bioconcentration Factor. The ratio of the concentration of a contaminant in a given organism to its concentration in the surrounding medium (water, soil, etc.).
Bioavailability:	The rate and extent of systemic absorption of a chemical.
BP:	Boiling Point. The temperature at which a component's vapor pressure equals atmospheric pressure. Boiling point is a relative indicator of volatility and generally increases with increasing molecular weight.
CAS number:	A unique identification number assigned to a chemical by the Chemical Abstract Service.
CERCLA:	Comprehensive Environmental Response, Compensation, and Liability Act.
Chronic Exposure:	Repeated or continuous exposure occurring over an extended period.
Chronic Toxicity:	The ability of a substance to cause adverse health effects as a result of chronic exposure.
Cleanup:	Actions taken to deal with a release or threat of release of a hazardous substance that could affect human and environmental health. The term "cleanup" is sometimes used interchangeably with the terms remedial action, removal action, response action, or corrective action.
Contaminant:	Any undesired physical, chemical, biological, or radiological substance that is present in the air, water, soil, or sediment.
C_{sat}:	Soil saturation limit. The concentration in soil at which the absorptive limits of the soil particles, the solubility limits of the soil pore water, and saturation of soil pore air have been reached.
CSF:	Cancer Slope Factor. A dose-response metric derived from human or animal studies that is used to calculate cancer risk.

d:	Density. A measure of how heavy a specific volume of a solid, liquid, or gas is in comparison to water.
DAF:	Dilution Attenuation Factor. The numerical factor by which a contaminant concentration is diminished as the contaminant moves through soil and groundwater from its source to the point of contact. As chemicals leach from soil and move through groundwater, attenuating effects include adsorption of the contaminant onto soil and aquifer media, chemical transformation, biological degradation, and dilution from mixing of the leachate with ambient groundwater.
DERM:	The Department of Environmental Resources Management.
Dermal Absorption:	The process by which a chemical penetrates the skin and enters the body.
Dermal Exposure:	Contact between a chemical and the skin.
Dermal Toxicity:	Adverse effects of a toxicant on the skin.
Detection Limit:	The lowest concentration of a chemical that can be distinguished from zero or background.
D_i:	Diffusivity in air. The ability of a substance to diffuse in air, the process by which molecules in a single phase equilibrate to a zero concentration gradient by random (Brownian) molecular motion.
Dose:	The quantity of a chemical administered to an organism or to which it is exposed. The absorbed dose is the amount that is absorbed and enters the body.
Dry weight:	Data reported as dry weight concentrations are intended to be corrected for moisture content contained in fractions dried at 103-105°C or in freeze dried fractions.
D_w:	Diffusivity in water. The ability of a substance to diffuse in water, the process by which molecules in a single phase equilibrate to a zero concentration gradient by random (Brownian) molecular motion.
EC:	Equivalent carbon number. An empirically-derived parameter for petroleum hydrocarbons related to the boiling point of the given chemical normalized to the boiling point of the n-alkanes, or its retention time in a boiling gas chromatographic column.
EFH:	Exposure Factors Handbook.
Exposure Route:	The route by which a toxicant enters the body — through the lungs (from inhalation), through the skin (from dermal contact), or through the gastrointestinal tract (from ingestion).
Exposure:	In the context of this report, exposure refers to contact with a toxicant.
F.A.C:	Florida Administrative Code.
FDEP:	Florida Department of Environmental Protection.
FL-PRO:	Florida Petroleum Residual Organic analytical method.
f_{oc}:	Fraction of organic carbon.

Free product:	In the context of this report, product refers to a contaminant present in environmental media in a pure or undissolved state, usually as a liquid.
GC:	Gas Chromatography. An analytical technique for detecting and quantitating chemicals. This technique uses an instrument called a gas chromatograph.
GI:	Gastrointestinal.
GSD:	Geometric Standard Deviation.
η:	Total soil porosity. The total amount of interconnected pore space in a soil or rock through which fluids can pass.
Hazard:	Potential for a chemical to produce adverse health effects.
HEAST:	USEPA Health Effects Assessment Summary Tables.
HGDB:	American Petroleum Institute's Hydrogeologic Database.
HI:	Hazard Index. The HI is the sum of the hazard quotients (HQs) and can be used to predict the non-cancer risk of simultaneous exposure of a receptor to several chemicals.
HLC:	Henry's Law constant. The ratio of the concentration of a compound in air (or vapor) to the concentration of the compound in water under equilibrium conditions.
HQ:	Hazard Quotient. The ratio of the projected dose of a chemical resulting from exposure divided by the appropriate reference dose for that chemical.
HSDB:	Hazardous Substances Data Bank.
IEUBK:	Integrated Exposure Uptake Biokinetic Model. A model developed by the USEPA to predict blood lead concentrations in children resulting from exposure to lead in soil and other sources.
Inhibition:	Type of chemical interaction that exists when the toxic effect of a chemical is reduced by the presence of a second substance that does not have that toxic effect.
IRIS:	Integrated Risk Information System. A USEPA electronic database containing toxicity values (e.g., reference doses and slope factors).
ISF:	Inhalation Slope Factor. A dose-response metric based on human or animal studies that is used to calculate cancer risk from inhalation exposure.
IUR:	Inhalation Unit Risk. A chemical-specific value that, when multiplied by the concentration of the chemical in air, yields the excess cancer risk associated with that concentration.
K_d:	Soil-water organic partition coefficient for organics.
K_{oc}:	Organic carbon normalized soil-water partition coefficient for organic compounds.

LC_{50}:	Median Lethal Concentration. The concentration of a toxicant that is lethal to 50 percent of the test organisms within a designated period of time.
LD_{50}:	Median Lethal Dose. The dose of a toxicant that is lethal to 50 percent of the test organisms within a designated period of time.
Leaching:	The process by which soluble constituents are dissolved from, and transported through, the soils by water.
LOAEL:	Lowest Observable Adverse Effect Level. The lowest dose of a chemical observed to cause an adverse effect.
Masking:	Type of chemical interaction that exists when concurrent toxic effects of two or more chemicals are opposite or functionally competing, reducing or obscuring their individual toxic effects.
MDL:	Method Detection Limit. The minimum concentration of a substance that can be measured and reported with a 99 percent confidence that the analyte concentration is greater than zero.
MP:	Melting Point. Temperature at which a change of the state of a substance from the solid phase to the liquid phase occurs.
MRL:	Minimal Risk Level. A safe dose (or dosing rate) for a chemical developed by the Agency for Toxic Substances and Disease Registry, U.S. Public Health Service.
MW:	Molecular Weight. The amount of mass in one mole of molecules of a substance as determined by summing the masses of the individual atoms that make up the molecule.
NCEA:	USEPA National Center for Environmental Assessment.
NCHS:	National Center for Health Statistics.
NHANES:	National Health and Nutrition Examination Survey.
NOAEL:	No Observable Adverse Effect Level. The highest dose of a chemical observed not to produce an adverse health effect.
NRC:	National Research Council.
OPP:	USEPA Office of Pesticide Programs.
Organoleptic:	Based on taste or odor.
OSHA	Occupational Safety and Health Administration.
OSWER:	USEPA Office of Solid Waste and Emergency Response.
PAH:	Polycyclic Aromatic Hydrocarbon.
PCB:	Polychlorinated Biphenyl.
PCDD:	Polychlorinated Dibenzodioxin.
PCDF:	Polychlorinated Dibenzofuran.
PEF:	Particulate Emission Factor. A term used to relate the concentration of a contaminant in soil with its concentration in air as dust particles. Factors that are used to determine the PEF include the extent of dust

	dispersion, the extent of vegetative cover, wind speed, and the extent to which the soil surface is erodible.
Porosity:	Degree to which soil, gravel, sediment, or rock is permeated with pores or cavities through which liquids or air can move.
Potentiation:	When the toxic effect of a substance is increased by the presence of a second chemical that does not have that toxic effect.
PQL:	Practical Quantitation Limit. A concentration below which quantitation is unreliable.
Primary Standard:	Enforceable groundwater standard based on health effects.
Q/C:	Technically, the inverse mean concentration at the center of a square source. When calculating the concentration of volatiles or dust in the air, it is the term that represents their dispersion in the atmosphere. Q/C values are derived from air modeling and can vary depending upon climatic conditions and the size of the contaminated area.
θ_a:	Air-filled soil porosity. The amount of total soil porosity (η) that is filled with air or other gas.
θ_w:	Water-filled soil porosity. The amount of total soil porosity (η) that is filled with water or other liquid.
ρ_b:	Dry soil bulk density. The density of a soil sample, including the volume of both particles and total porosity.
ρ_s:	Soil particle density. The density of a soil sample, not including the volume occupied by total porosity.
RCRA:	Resource Conservation and Recovery Act.
Remediation:	Cleanup or other methods used to remove or contain a toxic spill or hazardous materials from a contaminated site.
RfC:	Reference Concentration. An estimate of the concentration of a toxicant that is likely to be without appreciable risk of adverse effects during a lifetime of continuous exposure.
RfD:	Reference Dose. An estimate of the dose of a toxicant that, when given every day over a lifetime, is likely to be without appreciable risk of adverse effects. The RfD is specific for the route of exposure (i.e., ingestion versus dermal versus inhalation).
Risk:	A measure of the probability that an adverse effect will occur in exposed individuals or the environment as a result of a specified exposure.
Route of Exposure:	The route by which a chemical comes into contact with an organism (e.g., inhalation, ingestion, or dermal contact).
RSC:	Relative Source Contribution. The fraction of the total allowable intake of a chemical allocated to a particular source (such as intake of contaminated groundwater).
S:	Water solubility. The maximum concentration of a chemical that will dissolve in pure water at a reference temperature.

SCDM:	Superfund Chemical Data Matrix.
SCTL:	Soil Cleanup Target Level.
Secondary Standard:	Enforceable groundwater standard based on nuisance considerations.
Soil Pica:	Aberrant behavior, especially prevalent in children, characterized by intentional ingestion of soil.
SPLP:	Synthetic Precipitation Leaching Procedure. A method for predicting leaching of a chemical from soil to water under typical environmental conditions.
SSG:	Soil Screening Guidance. A USEPA document describing the development of soil screening levels (SSLs).
SSL:	Soil Screening Levels. Risk-based screening levels for chemicals in soil developed by the USEPA.
Surrogate:	A substance that shares similar chemical and/or physical properties with another substance. When toxicity or physical/chemical properties for a chemical are unavailable, values from another, surrogate chemical may be used in the development of its SCTL.
Synergism:	Type of chemical interaction that exists when the toxic effect from exposure to two or more chemicals is greater than what is expected based on their individual toxicities (i.e., the effects are greater than additive).
Systemic toxicant:	A contaminant whose health effects are widespread.
TCDD:	Tetrachlorodibenzo-*p*-dioxin. TCDD sometimes refers to 2,3,7,8-tetrachloro-*p*-dibenzodioxin, which is the most toxic congener.
TCLP:	Toxicity Characteristic Leaching Procedure. A method for predicting leaching of a chemical from soil to water under conditions that might exist in a landfill.
TEFs:	Toxic Equivalency Factors. Numerical expression of the potencies of a series of related compounds relative to the potency of a reference or index chemical.
TEQs:	Toxic equivalents. Toxic potency of a chemical mixture calculated by adding the product of the concentration of each individual compound in the mixture times its respective TEF.
Threshold:	The dose of a chemical just sufficient to produce an effect.
Toxicity:	The ability of a substance to cause adverse health effects.
TPHCWG:	Total Petroleum Hydrocarbon Criteria Working Group.
TRPHs:	Total Recoverable Petroleum Hydrocarbons. A means of expressing the total concentration of petroleum-related hydrocarbons in soil or water.
USEPA:	United States Environmental Protection Agency.
VF:	Volatilization Factor. A measure of the process of transfer of a chemical from the aqueous or liquid phase to the gas phase under specific environmental conditions and exposure durations.

VP:	Vapor Pressure. The force per unit area exerted by a vapor in an equilibrium state with its pure solid, liquid, or solution at a given temperature.
ω:	Soil moisture content. The total amount of water contained in a given volume of bulk soil.
WHO:	World Health Organization.

XII. Appendix A. Derivation of Body Weight, Dermal Surface Area, and Inhalation Rate Estimates for Calculating the Direct Exposure SCTLs

A. Introduction

With the exception of inhalation rate in workers, standard USEPA defaults for body weight, surface area, and inhalation rate have been replaced with values derived directly from health statistics for the purpose of calculating the direct exposure SCTLs. The 1997 *Exposure Factors Handbook*, which relies on data from the Second National Health and Nutrition Examination Survey (NHANES II), was not used as the primary source of information for body weight and surface area. Instead, data from the newer NHANES III were analyzed to develop assumptions for these parameters. This change was warranted because the more recent NHANES III survey indicates that body weights have changed nationally since the NHANES II survey in the mid-1980s. Increases in body weights mean that surface areas have changed as well. Use of the more recent data provides a more accurate and contemporary view of these body parameters that affect risk.

Another refinement is the manner in which body weight, surface area, and inhalation rates are developed. All three of these parameters change dramatically as an individual matures from age 1 to age 31, and time averaging of each is required to derive an accurate exposure estimate, particularly for carcinogens where exposure is assumed to occur for long periods. Previously, averaging for the aggregate resident was accomplished by dividing the 30-year exposure period into two intervals — one exposure interval as a child, with fixed body weight, surface area, and inhalation rate assumptions, and the second interval as an adult with a different set of fixed assumptions for these variables. These two sets of assumptions (child and adult) were then time-weighted to derive an average.

Body weight and surface area values are developed for each age, in annual increments from ages 1 to 65 years. These values are then used to develop averages for each interval of interest. This procedure includes not only the aggregate resident (ages 1 to 31 years), but also the child resident (ages 1 to 7 years) and the adult worker (ages 18 to 65 years). This method of averaging, made possible by the more comprehensive data set available directly from NHANES III, offers more precise estimates of these exposure parameters. Age-specific inhalation rates, available from the *Exposure Factors Handbook*, were also averaged in an analogous fashion to derive inhalation rate assumptions for each scenario. Although inhalation rate data are only available for children for 2 to 3 year age intervals, and a single value is presented for adults (ages 19 to 65+ years), this averaging procedure nonetheless represents an improvement over the method of inhalation rate estimation used previously.

The values derived for these parameters are summarized in Table A-1 below.

Table A-1
Summary of Body Weight, Surface Area, and Inhalation Rate Assumptions

Parameter	Exposure Scenario		
	Child	**Aggregate Resident**	**Worker**
Body Weight (kg)	16.8	51.9	76.1
Surface Area (cm^2)	2960	4810	3500
Inhalation Rate (m^3/day)	8.1	12.2	20*

* Unchanged from the previous Chapter 62-777, F.A.C. default.

B. Description of NHANES III

The National Center for Health Statistics (NCHS) collected vital and health statistics on 33,994 non-institutionalized individuals aged two months to 90 years old, living in the United States during 1988-1994, as part of the NHANES III. To obtain reliable estimates of characteristics of Black Americans, Mexican Americans, infants and young children (1 to 5 years), and older persons (60+ years), individuals in these groups were sampled at a higher rate. While this approach assisted in developing statistically valid data for these limited-size groups of special interest, it created an overall data set in which responses from these groups were over-represented relative to the U.S. population as a whole.

In order to develop data suitable for SCTL development, raw data from NHANES III were adjusted to account for non-responses and stratified to reflect the composition of the entire U.S. population by age, sex, and race, using a weighting factor provided by the NCHS. NHANES III data on body weights, including clothing (estimated as ranging from 0.09 to 0.28 kg), age, sex, and race, were downloaded from the NCHS using the FERRETS data extraction tools, and converted into a Statistical Analysis System (SAS) dataset. A total of 31,311 records were available from the NHANES III data set. Those records with complete information applicable to the analysis of interest were included in the data set. Missing data accounted for the loss of 1,244 records for the body weight calculations. Mean body weights were calculated for each age grouping. Age groups were defined traditionally as starting with the birth month and including the next 11 months. For example, age group 2 includes individuals who were 24 to 35 months old at the time of the NHANES III exam.

1. Body weights

Previous studies have shown that body weights tend to follow a lognormal distribution (Brainard and Burmaster, 1992; Burmaster and Crouch, 1997). To confirm this observation with the NHANES III data, goodness-of-fit tests were performed for each age group. These tests indicated that the lognormal assumption provides a reasonable fit for these data (results not shown). Given that the body weight data are lognormally distributed implies that:

$$\ln[\mathrm{BW}] \sim \mathrm{Normal}(\mu, \sigma)$$

where [BW] represents body weight in kg, and the natural logarithm transformation of the body weight (ln[BW]) is approximately normally distributed with parameters μ (mean) and σ (standard deviation).

A simple method for deriving an estimate of the mean and variance for two-parameter lognormal distributions such as this is given by:

$$\mu = \exp\left(y + \frac{s_y^2}{2}\right) \qquad \sigma^2 = \mu^2[\exp(s_y^2) - 1]$$

This method produces estimates of the population mean and variance that may be somewhat biased. However, because of the rather large sample sizes for each age group, any bias in the resulting estimates will be small. The bias introduced into the analysis using these techniques can be estimated directly from the data by the following equation (Gilbert, 1987):

$$\mathrm{Bias} = \left(1 - \frac{\sigma_y^2}{n}\right)^{-(n-1)/2} \exp\left(-\frac{n-1}{2n}\sigma_y^2\right)$$

Given that the maximum variance of the log-transformed data is generally less than 0.1 and the sample sizes are generally greater than 50, then the maximum bias introduced using this procedure will be less than 0.05%. Because the mean body weights are rounded to three significant figures, the error introduced through this method is inconsequential.

Mean and standard deviations of the body weight data for males, females, and both genders combined ("composite" body weight) for ages 1 through 31 years are given in Table A-2. It should be noted that the results for the composite body weights are not simply the average of the male and female body weights for each age group. Means for the composite body weights were generated from the raw data using the specified weighting factors that account for sample demographics including expected proportions of each sex in the population. Aggregate resident (ages 1 to 31 years) body weight for combined males and females is **51.9 kg**. The child (ages 1 to 7 years) body weight for male and female children combined is **16.8 kg**.

Workers were assumed to include, with equal probability, adults aged 18 to 65 years. The assumption that all ages in this range are equally represented in a worker population may not be correct, but the error introduced by this assumption is likely to be small. Yearly body weight estimates for male, female, and both genders combined ("composite" body weight) workers are given in Table A-3. Again, means for the composite body weights were generated from the raw data using the specified weighing factors that account for sample demographics that included expected proportions of each sex in the population. The average body weight for male and female workers aged 18 to 65 years is **76.1 kg**.

2. Surface area

Limited empirical data exist for surface area measurements in adults and children. In an attempt to extend the utility of the considerable body weight data available, a number of authors have described allometric relationships between body weight and surface area (e.g., Burmaster, 1998; Dubois and Dubois, 1916). Both univariate (based on weight only) and bivariate (based on both height and weight) models have been employed. Based on our analysis of surface areas predicted from the NHANES III dataset, these models performed equally well in predicting surface areas across a wide range of body weights (data not shown). Therefore, the univariate model proposed by Burmaster (1998) was chosen to calculate total body surface area from body weights. The advantages of this model are its inherent simplicity and the ability to extend the results to produce distributional parameters without complications resulting from confounded variables. The model is given below,

$$SA = BW^{0.6821} * 1025$$

where SA is the total skin surface area (cm^2) and BW is the body weight (kg). Total body surface areas for males and females by age are listed in Table A-4.

Exposed surface area is based, in part, on guidance specified in RAGS-Part E (USEPA, 2000b). Specifically, estimates of exposed surface area depend upon assumptions about the types of clothing a particular receptor population is likely to wear, and are computed by summing the area of the body parts not covered by the clothes. The percentage that each body part contributes to the total surface area is required to calculate the sum of exposed body surface area for each exposure scenario. Data on body part percentages of total surface area derived from empirical measurements of children and adults, as presented in the *Exposure Factors Handbook* (USEPA, 1997), were used for these calculations. The number of individuals sampled to derive these data was extremely limited; sometimes as few as a single individual constitutes the sample size for an entire age group. However, no alternative source with better data was identified for this report.

The percentage of total body surface area, by part, for children and adults is shown in Table A-4. No specific age group data are presented in the *Exposure Factors Handbook* for children at ages 1, 5, 7, 8, 10, 11, 14, and 15 years. Therefore, the surface area information for these ages was linearly interpolated from the adjacent age groups. Based on the relationships in RAGS-Part E (USEPA, 2000b), surface area percentage for the forearms and lower legs were assumed to equal 0.45 and 0.40 of the arm and leg, respectively.

Child surface area exposed was calculated based on a child wearing short pants, a short-sleeved shirt, and no shoes. The exposed area considered was, therefore, the head, hands, feet, lower legs and forearms. The surface area represented by each body part was calculated by multiplying the composite male/female total surface area for each age group by the percentage surface area for each body part.

$$SA_{body\ part} = (\text{Percentage Body Part for Age}) * (\text{Total Surface Area for Age})$$

The surface areas for each of the exposed body parts (head, hands, feet, lower legs, and forearms) were summed to derive a total exposed surface area for each age, as shown in Table A-6. Total surface area exposed values for each age were then averaged over the age range of interest, e.g., for a child resident, from ages 1 to 7 years. Based on this approach, the exposed surface area for a child resident is **2960 cm^2**.

Aggregate resident surface area exposed was calculated in a manner similar to that for a child resident, with the exception that shoes are assumed to be worn from ages 7 to 31 years. Therefore, the exposed area considered is the head, hands, feet, lower legs and forearms for the first six years, and the head, hands, lower legs and forearms for the remaining 24 years. As above, the skin surface area for each exposed body part was calculated by multiplying its percentage relative of total body surface area by the male/female total surface area. This calculation was performed for each age group, and age-specific exposed surface areas for ages 1 to 31 years were averaged to derive the exposed surface area for the aggregate resident of **4810 cm^2**.

Worker surface area exposed was calculated based on a worker wearing long pants, shoes and a short-sleeved shirt. Therefore, the exposed area considered was the head, hands, and forearms. Surface areas for each of these exposed parts of the body, as well as the total exposed surface area, were calculated for each age in a manner identical to the procedures described above (see Table A-7). Age-specific exposed surface areas for the workers were averaged for ages 18 to 65 to derive an exposed surface area for workers of **3500 cm^2**.

C. Inhalation Rates

Inhalation rates for children and aggregate residents are based on the average daily inhalation required to support metabolism as presented in the *Exposure Factors Handbook* (Table 5-23 of USEPA, 1997). Inhalation rates are given in Table A-8 for each age group. Averaging the inhalation rate for the ages 1 to 31 years produced a mean aggregate resident inhalation rate of **12.2 m^3/day**. Averaging the inhalation rates for ages 1 to 7 years produced a mean child inhalation rate of **8.1 m^3/day**. The worker inhalation rate was unchanged from the previously used value of **20 m^3/day**.

Table A-2

Mean Body Weight Estimates for Males and Females Ages 1 to 31 Years

Age	Mean Body Weights (kg)		
	Males	Females	Composite
1-2	11.6	10.9	11.2
2-3	13.6	13.2	13.4
3-4	15.8	15.4	15.6
4-5	17.6	17.8	17.7
5-6	20.1	20.1	20.1
6-7	23.2	22.5	22.9
7-8	26.3	26.4	26.3
8-9	30.1	29.8	30.0
9-10	34.4	34.3	34.3
10-11	37.3	37.9	37.6
11-12	42.4	44.1	43.3
12-13	49.1	49.0	49.0
13-14	54.0	55.8	54.8
14-15	63.8	58.4	61.1
15-16	66.8	58.2	62.0
16-17	68.6	61.6	65.3
17-18	72.8	62.3	67.8
18-19	71.2	61.4	66.2
19-20	73.0	63.7	68.2
20-21	72.5	61.7	66.2
21-22	72.9	64.9	69.0
22-23	76.6	64.0	69.8
23-24	77.8	66.8	72.6
24-25	78.5	62.7	70.6
25-26	80.2	66.2	74.4
26-27	75.8	64.7	69.6
27-28	81.2	65.0	73.6
28-29	80.8	67.0	73.7
29-30	81.8	66.0	74.0
30-31	83.4	67.6	75.2
Average Aggregate Resident (1 to 31 years) Body Weight			**51.9**
Average Child Resident (1 to 7 years) Body Weight			**16.8**

Table A-3 (page 1 of 2)

Mean Body Weight Estimates for Males and Females Ages 18 to 65 Years

Age	Mean Male Body Weight (kg)	Mean Female Body Weight (kg)	Composite Body Weight (kg)
18-19	71.2	61.4	66.2
19-20	73.0	63.7	68.2
20-21	72.5	61.7	66.2
21-22	72.9	64.9	69.0
22-23	76.6	64.0	69.8
23-24	77.8	66.8	72.6
24-25	78.5	62.7	70.6
25-26	80.2	66.2	74.4
26-27	75.8	64.7	69.6
27-28	81.2	65.0	73.6
28-29	80.8	67.0	73.7
29-30	81.8	66.0	74.0
30-31	83.4	67.6	75.2
31-32	79.5	72.6	76.4
32-33	81.6	67.5	74.3
33-34	83.9	68.3	75.2
34-35	83.1	67.4	76.8
35-36	81.5	71.4	76.0
36-37	87.5	65.9	78.3
37-38	83.2	72.0	76.4
38-39	82.4	71.6	76.6
39-40	82.6	74.6	78.7
40-41	85.8	68.5	75.7
41-42	86.3	70.0	79.0
42-43	85.1	72.6	78.9
43-44	86.4	68.8	78.1
44-45	90.6	72.5	79.4
45-46	83.6	71.7	78.0
46-47	80.8	72.0	76.2
47-48	85.5	72.0	79.4
48-49	82.3	75.8	79.0
49-50	82.1	73.3	77.6
50-51	81.7	73.8	76.9
51-52	85.6	79.5	83.1

Table A-3 (page 2 of 2)
Mean Body Weight Estimates for Males and Females Ages 18 to 65 Years

Age	Mean Male Body Weight (kg)	Mean Female Body Weight (kg)	Composite Body Weight (kg)
52-53	87.1	72.0	79.8
53-54	89.3	73.8	81.7
54-55	86.0	74.5	79.6
55-56	83.0	72.6	76.7
56-57	87.1	77.6	82.9
57-58	86.3	75.6	81.7
58-59	83.4	72.2	76.8
59-60	87.9	73.9	80.5
60-61	83.5	68.9	76.0
61-62	81.8	72.1	76.2
62-63	82.0	72.8	76.7
63-64	84.4	71.3	76.9
64-65	84.3	74.5	78.7
Average Worker (18 to 65 years) Body Weight			**76.1**

Table A-4 (page 1 of 2)

Surface Area for Males and Females Based on Body Weight Estimates

Age	Total Surface Area (cm^2)		
	Male	Female	Composite
1-2	5390	5170	5280
2-3	6020	5890	5960
3-4	6660	6550	6610
4-5	7190	7230	7210
5-6	7840	7860	7850
6-7	8640	8470	8560
7-8	9410	9410	9410
8-9	10320	10240	10290
9-10	11280	11240	11260
10-11	11930	12040	11980
11-12	13010	13370	13190
12-13	14380	14350	14360
13-14	15330	15680	15500
14-15	17150	16200	16690
15-16	17750	16180	16880
16-17	18060	16790	17470
17-18	18850	16940	17940
18-19	18550	16740	17630
19-20	18880	17170	17990
20-21	18790	16810	17640
21-22	18880	17380	18130
22-23	19490	17250	18280
23-24	19720	17740	18770
24-25	19820	17010	18420
25-26	20100	17610	19060
26-27	19380	17360	18240
27-28	20300	17410	18940
28-29	20190	17780	18940
29-30	20380	17610	19000
30-31	20660	17870	19200
31-32	20010	18740	19440
32-33	20360	17840	19060
33-34	20750	18000	19210
34-35	20610	17870	19510
35-36	20330	18540	19350
36-37	21310	17590	19720

Table A-4 (page 2 of 2)
Surface Area for Males and Females Based on Body Weight Estimates

Age	Surface Area (cm^2)		
	Male	Female	Composite
37-38	20620	18650	19420
38-39	20500	18570	19460
39-40	20560	19100	19830
40-41	21080	18050	19300
41-42	21120	18330	19870
42-43	20940	18730	19850
43-44	21160	18110	19720
44-45	21830	18740	19930
45-46	20720	18620	19730
46-47	20250	18680	19420
47-48	21010	18680	19950
48-49	20490	19340	19920
49-50	20450	18870	19640
50-51	20390	18980	19520
51-52	21040	19960	20590
52-53	21310	18660	20030
53-54	21680	18980	20340
54-55	21100	19070	19960
55-56	20610	18810	19520
56-57	21310	19650	20570
57-58	21160	19280	20350
58-59	20670	18700	19510
59-60	21420	19020	20150
60-61	20700	18140	19380
61-62	20400	18700	19410
62-63	20430	18800	19490
63-64	20850	18560	19530
64-65	20820	19100	19830

Table A-5

Percentage Surface Area by Body Part

Age	Surface Area (%)						
	Head	**Arms**	**Hands**	**Legs**	**Feet**	***Forearms***	***Lower legs***
0-1	**18.20**	**13.70**	**5.30**	**20.60**	**6.54**	*6.17*	*8.24*
1-2	**16.50**	**13.00**	**5.68**	**23.10**	**6.27**	*5.85*	*9.24*
2-3	**14.20**	**11.80**	**5.30**	**23.20**	**7.07**	*5.31*	*9.28*
3-4	**13.60**	**14.40**	**6.07**	**26.80**	**7.21**	*6.48*	*10.72*
4-5	**13.80**	**14.00**	**5.70**	**27.80**	**7.29**	*6.30*	*11.12*
5-6	*13.45*	*13.55*	*5.21*	*27.45*	*7.10*	*6.10*	*10.98*
6-7	**13.10**	**13.10**	**4.71**	**27.10**	**6.90**	*5.90*	*10.84*
7-8	*12.73*	*12.83*	*4.91*	*27.63*	*7.13*	*5.78*	*11.05*
8-9	*12.37*	*12.57*	*5.10*	*28.17*	*7.35*	*5.66*	*11.27*
9-10	**12.00**	**12.30**	**5.30**	**28.70**	**7.58**	*5.54*	*11.48*
10-11	*10.91*	*12.77*	*5.33*	*29.30*	*7.40*	*5.75*	*11.72*
11-12	*9.83*	*13.23*	*5.36*	*29.90*	*7.21*	*5.96*	*11.96*
12-13	**8.74**	**13.70**	**5.39**	**30.50**	**7.03**	*6.17*	*12.20*
13-14	**9.97**	**12.10**	**5.11**	**32.00**	**8.02**	*5.45*	*12.80*
14-15	*9.30*	*12.43*	*5.30*	*32.53*	*7.66*	*5.60*	*13.01*
15-16	*8.63*	*12.77*	*5.49*	*33.07*	*7.29*	*5.75*	*13.23*
16-17	**7.96**	**13.10**	**5.68**	**33.60**	**6.93**	*5.90*	*13.44*
17-18	**7.58**	**17.50**	**5.13**	**30.80**	**7.28**	*7.88*	*12.32*
18-65	**6.64**	**14.35**	**4.98**	**32.67**	**6.75**	*6.46*	*13.07*

* Values in **bold** are taken directly from the EFH, values in *italics* are derived as specified in the text.

Table A-6

Exposed Surface Areas for Child and Aggregate Residents

Age	Body Part Surface Area (cm^2)					Surface Area (cm^2)
	Head	Hands	Feet	Forearms	Lower Legs	Total Exposed
1-2	871.2	299.9	331.1	308.9	487.9	2299
2-3	846.3	315.9	421.4	316.5	553.1	2453
3-4	899.0	401.2	476.6	428.3	708.6	2914
4-5	995.0	411.0	525.6	454.2	801.8	3188
5-6	1055.8	408.6	557.0	478.7	861.9	3362
6-7	1121.4	403.2	590.6	504.6	927.9	3548
7-8	1198.2	461.7		543.4	1040.1	3244
8-9	1272.5	525.1		581.9	1159.3	3539
9-10	1351.2	596.8		623.2	1292.6	3864
10-11	1307.4	638.5		688.3	1404.1	4038
11-12	1296.1	707.0		785.5	1577.5	4366
12-13	1255.1	774.0		885.3	1751.9	4666
13-14	1545.4	792.1		844.0	1984.0	5165
14-15	1552.2	884.6		933.8	2171.9	5543
15-16	1456.7	926.7		969.8	2232.7	5586
16-17	1390.6	992.3		1029.9	2348.0	5761
17-18	1359.9	920.3		1412.8	2210.2	5903
18-19	1170.6	878.0		1138.5	2303.9	5491
19-20	1194.5	895.9		1161.7	2350.9	5603
20-21	1171.3	878.5		1139.1	2305.2	5494
21-22	1203.8	902.9		1170.7	2369.2	5647
22-23	1213.8	910.3		1180.4	2388.8	5693
23-24	1246.3	934.7		1212.1	2452.9	5846
24-25	1223.1	917.3		1189.5	2407.1	5737
25-26	1265.6	949.2		1230.8	2490.8	5936
26-27	1211.1	908.4		1177.8	2383.6	5681
27-28	1257.6	943.2		1223.1	2475.1	5899
28-29	1257.6	943.2		1223.1	2475.1	5899
29-30	1261.6	946.2		1226.9	2482.9	5917
30-31	1274.9	956.2		1239.8	2509.1	5980
Average Child Resident (1 to 7 years)* Surface Area						**2960**
Average Aggregate Resident (1 to 31 years)* Surface Area						**4810**

* Final surface area rounded to three significant figures.

Table A-7 (page 1 of 2)
Exposed Surface Areas for Workers

Age	Surface Area for Body Part (cm^2)			Surface Area (cm^2) Total Exposed
	Head	Hands	Forearms	
18-19	1170.6	878.0	1138.5	3187
19-20	1194.5	895.9	1161.7	3252
20-21	1171.3	878.5	1139.1	3189
21-22	1203.8	902.9	1170.7	3277
22-23	1213.8	910.3	1180.4	3305
23-24	1246.3	934.7	1212.1	3393
24-25	1223.1	917.3	1189.5	3330
25-26	1265.6	949.2	1230.8	3446
26-27	1211.1	908.4	1177.8	3297
27-28	1257.6	943.2	1223.1	3424
28-29	1257.6	943.2	1223.1	3424
29-30	1261.6	946.2	1226.9	3435
30-31	1274.9	956.2	1239.8	3470
31-32	1290.8	968.1	1255.3	3514
32-33	1265.6	949.2	1230.8	3446
33-34	1275.5	956.7	1240.5	3473
34-35	1295.5	971.6	1259.9	3527
35-36	1284.8	963.6	1249.5	3498
36-37	1309.4	982.1	1273.4	3565
37-38	1289.5	967.1	1254.0	3511
38-39	1292.1	969.1	1256.6	3518
39-40	1316.7	987.5	1280.5	3585
40-41	1281.5	961.1	1246.3	3489
41-42	1319.4	989.5	1283.1	3592
42-43	1318.0	988.5	1281.8	3588
43-44	1309.4	982.1	1273.4	3565
44-45	1323.4	992.5	1287.0	3603
45-46	1310.1	982.6	1274.1	3567
46-47	1289.5	967.1	1254.0	3511
47-48	1324.7	993.5	1288.3	3606
48-49	1322.7	992.0	1286.3	3601
49-50	1304.1	978.1	1268.3	3550
50-51	1296.1	972.1	1260.5	3529
51-52	1367.2	1025.4	1329.6	3722
52-53	1330.0	997.5	1293.4	3621
53-54	1350.6	1012.9	1313.5	3677

Table A-7 (page 2 of 2)
Exposed Surface Areas for Workers

Age	Surface Area for Body Part (cm^2)			Surface Area (cm^2) Total Exposed
	Head	Hands	Forearms	
54-55	1325.3	994.0	1288.9	3608
55-56	1296.1	972.1	1260.5	3529
56-57	1365.8	1024.4	1328.3	3719
57-58	1351.2	1013.4	1314.1	3679
58-59	1295.5	971.6	1259.9	3527
59-60	1338.0	1003.5	1301.2	3643
60-61	1286.8	965.1	1251.5	3503
61-62	1288.8	966.6	1253.4	3509
62-63	1294.1	970.6	1258.6	3523
63-64	1296.8	972.6	1261.1	3531
64-65	1316.7	987.5	1280.5	3585
Average Worker (18 to 65 years) Surface Area*				**3500**

* Final surface area rounded to three significant figures.

Table A-8

Inhalation Rates for Child and Adult Residents Ages 1 to 31 Years

Age	Inhalation Rate (m^3/day)		
	Male	Female	Average Male and Female
1-2	6.8	6.8	6.8
2-3	6.8	6.8	6.8
3-4	8.3	8.3	8.3
4-5	8.3	8.3	8.3
5-6	8.3	8.3	8.3
6-7	10	10	10
7-8	10	10	10
8-9	10	10	10
9-10	14	13	13.5
10-11	14	13	13.5
11-12	14	13	13.5
12-13	15	12	13.5
13-14	15	12	13.5
14-15	15	12	13.5
15-16	17	12	14.5
16-17	17	12	14.5
17-18	17	12	14.5
18-19	17	12	14.5
19-20	15.2	11.3	13.25
20-21	15.2	11.3	13.25
21-22	15.2	11.3	13.25
22-23	15.2	11.3	13.25
23-24	15.2	11.3	13.25
24-25	15.2	11.3	13.25
25-26	15.2	11.3	13.25
26-27	15.2	11.3	13.25
27-28	15.2	11.3	13.25
28-29	15.2	11.3	13.25
29-30	15.2	11.3	13.25
30-31	15.2	11.3	13.25
Aggregate Resident (1 to 31 years) Inhalation Rate)*			**12.2**
Child Resident (1 to 7 years) Inhalation Rate*			**8.1**

* Final inhalation rate rounded to 0.1 m^3/day.

XIII. Appendix B: Derivation of Inhalation and Dermal Toxicity Values

A. Inhalation Toxicity Values

For evaluating hazard from the inhalation of a chemical, the USEPA develops toxicity values in the form of a reference dose (RfD) or reference concentration (RfC). While the USEPA has recently shown preference for RfC, the equations for the methods described in this report use RfD exclusively. The reason for this decision is that it is well recognized that children have much higher ventilation rates relative to body weight than adults. Consequently, they will receive a higher dosage of a chemical from air than an adult at the same air concentration. The use of RfDs allows this difference to be taken into consideration, whereas the use of RfCs involves the implicit assumption that adults and children are equally sensitive to contamination in air. For the same reason, the equation for carcinogenicity utilizes Inhalation Cancer Slope Factors (CSF_i) rather than Inhalation Unit Risk (IUR) values (which are expressed as reciprocal concentrations in air).

In situations where the USEPA lists both an inhalation RfD and an inhalation RfC for a non-carcinogen or, alternatively, a CSF_i and an IUR for a carcinogen, the RfD or CSF_i in question has been converted from the RfC or IUR, respectively. The USEPA reports these converted toxicity values to one significant figure for inhalation RfD and two significant figures for CSF_i. In the development of the SCTLs, inhalation RfD and CSF_i converted from RfC and IUR without rounding of the final value were used in preference to the rounded USEPA inhalation RfD or CSF_i.

1. Reference Dose (RfD)

When an inhalation RfC was available, it was converted to an inhalation RfD for the calculation of a soil target level. The conversion from RfC to inhalation RfD assumed a 70 kg individual breathing 20 m^3/day. Thus, the RfC was multiplied by 20 m^3/day and divided by 70 kg to obtain a value with the units mg/kg-day. The final value was not rounded:

e.g., methyl tert-butyl ether: Inhalation RfC = 3 mg/m^3

Thus, (3 mg/m^3 x 20 m^3/day) / 70 kg = 8.57 x 10^{-1} mg/kg-day = RfD_i

When an RfC was not available, the second choice was to develop an inhalation RfD from the oral RfD using route-to-route extrapolation. Such extrapolation was only done when the toxic endpoint being addressed was systemic in nature. Oral RfDs that were known or likely to be route-specific (e.g., where the toxic endpoint involved the gastrointestinal tract) were not extrapolated.

The formula for the conversion of an oral RfD to an inhalation RfD was as follows:

$$RfDi = RfDo \times GI\ absorption$$

e.g., bromodichloromethane: $RfD_o = 2.0 \times 10^{-2}$ mg/kg-day
Chemical-specific GI absorption = 0.98
Thus, $(2.0 \times 10^{-2}$ mg/kg-day) x (0.98) =
RfDo = 1.96×10^{-2} mg/kg-day

The GI absorption term represents the bioavailability of the chemical following exposure through the oral route. Formerly, the GI absorption inputs were chemical-specific values taken from the literature or default values specified by Region IV. Current USEPA guidance (USEPA, 1989a) recommends assuming 100% GI absorption for all chemicals that do not have chemical-specific values. Previously used chemical-specific values were retained, and the new USEPA default assumption of 100% was substituted for the Region IV defaults.

2. Cancer Slope Factor (CSF)

When a carcinogen had an inhalation unit risk (IUR), the IUR was converted to a CSF_i for the calculation of a soil target level. The conversion assumes a 70 kg individual breathing 20 m^3/day. Thus, the IUR (per $\mu g/m^3$) is divided by 20 m^3/day and multiplied by 70 kg and a conversion factor of 1000 µg/mg to obtain a value with the units $(mg/kg\text{-}day)^{-1}$. The final value was not rounded.

e.g., 1,2-diphenylhydrazine: IUR = 2.2×10^{-4} $(\mu g/m^3)^{-1}$
Thus, [$(2.2 \times 10^{-4}$ $(\mu g/m^3)^{-1}$ / 20 m^3/day) x 70 kg x 1000 µg/mg] =
$CSF_i = 7.70 \times 10^{-1}$ $(mg/kg\text{-}day)^{-1}$

If an IUR was not available and the chemical was regarded as likely producing carcinogenicity via a systemic effect, a CSF_i was derived from the oral slope factor (CSF_o), if available. This route-to-route extrapolation was accomplished by using the following formula:

$$CSFo = CSF_i / GI\ absorption$$

In general, route-to-route extrapolation from the CSF_o was not performed if the CSF_o was known or presumed to reflect route-specific toxicity. When a chemical exhibits route-specific

toxicity, it exerts its toxic effect (i.e., cancer) only by a specific exposure route. For example, chromium only causes lung cancer if it is inhaled, thus the toxic effect (lung cancer) is route-specific and target organ-specific. No other exposure route for chromium has been shown to cause cancer.

B. Dermal Toxicity Values

1. Reference Dose (RfD)

Dermal RfDs were derived from either the oral or inhalation RfD (if both were available and suitable, preference was given to the oral RfD). The following formula was used:

$$RfD_d = RfD_o \times GI\ absorption$$

If an RfD (either oral or inhalation) was known or presumed to be route-specific, it was not regarded as suitable for route-to-route extrapolation.

2. Cancer Slope Factor (CSF)

Dermal slope factors (CSF_d) were derived from CSF_o using route-to-route extrapolation:

$$CSF_d = CSF_o / GI\ absorption$$

e.g., carbon tetrachloride: $CSF_o = 1.3 \times 10^{-1}\ (mg/kg\text{-}day)^{-1}$

Chemical-specific GI absorption = 0.85

Thus, $(1.3 \times 10^{-1}\ (mg/kg\text{-}day)^{-1}) / (0.85) =$

$CSF_d = 1.5 \times 10^{-1}\ (mg/kg\text{-}day)^{-1}$

In general, a CSF_o was not extrapolated to produce a CSF_d if it was thought to reflect route-specific toxicity.

In the case of carcinogenic PAHs, the toxic endpoint (cancer) occurs regardless of the route of exposure. The CSF_o for benzo(a)pyrene is based on data in which oral dosing resulted in GI tract tumors in rodents, arguably a route-specific cancer. However, benzo(a)pyrene has also been observed to produce other types of cancer in several species when administered by a variety of routes, including inhalation and dermal contact. Although no cancer slope factor has yet been derived for these routes, the rather strong evidence that benzo(a)pyrene (and, by implication, other carcinogenic PAHs) is carcinogenic by a variety of routes, indicates that PAH-induced cancer is

not wholly route-specific. Because of this property, route-to-route extrapolation was performed to derive both inhalation and dermal slope factors from the CSF_o for this group of chemicals.

XIV. Appendix C: Technical Basis for the TRPH SCTLs

A. Development of SCTLs for Hydrocarbon Fractions Developed by the Total Petroleum Hydrocarbon Criteria Working Group

The following calculations for total recoverable petroleum hydrocarbon (TRPH) values were adopted essentially as described by the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG, 1997a,b,c). The application of a general standard for TRPHs is difficult because of the variation in mobility and toxicity of the chemicals included. To overcome this problem, the TPHCWG (1997a) suggested a sub-classification methodology in which aromatics and aliphatics are considered separately because these groups vary considerably in their environmental behavior. Each of these groups was then further subdivided on the basis of equivalent carbon number index (EC). The EC is a function of the molecular weight (MW) and boiling point (BP) of a chemical normalized to the BP of the n-alkanes, or its retention time in a BP gas chromatographic column. This approach is used since it is consistent with methods routinely used in the petroleum industry for separating complex mixtures and is a more appropriate differentiation technique than the actual carbon number of the chemical.

Table C-1
Hydrocarbon Fractions Defined by the Total Petroleum Hydrocarbon Criteria Working Group

Range of Equivalent Carbon Number (EC)	Avg EC	Classification
C_5-C_7	6.5	Aromatic
>C_7-C_8	7.5	Aromatic
>C_8-C_{10}	9.0	Aromatic
>C_{10}-C_{12}	11	Aromatic
>C_{12}-C_{16}	14	Aromatic
>C_{16}-C_{21}	18.5	Aromatic
>C_{21}-C_{35}	28.5	Aromatic
C_5-C_6	5.5	Aliphatic
>C_6-C_8	7.0	Aliphatic
>C_8-C_{10}	9.0	Aliphatic
>C_{10}- C_{12}	11	Aliphatic
>C_{12}- C_{16}	14	Aliphatic
>C_{16}- C_{21}	18.5	Aliphatic

1. Calculation of TRPH Fraction-Specific Physical Properties

Several alternatives for estimating representative physical/chemical properties for each fraction were reviewed by the TPHCWG. They included simple averaging of all available property data, composition-based averaging in which a weighted average of the available property data was computed based on the relative mass of each component in gasoline, and correlation to relative boiling point index in which the properties were developed based on EC values. While all of the approaches had similar results, it was determined that the correlations approach was most useful, because if the definitions of the fractions change, new properties can be easily computed for each fraction.

Utilizing the values correlations approach, the TRPHs are grouped into EC fractions, a method which allows for the calculation of the fate and transport characteristics of solubility (S), organic carbon partition coefficient (K_{oc}) and vapor pressure (VP). While Henry's Law constant (HLC) could also be estimated from a similar type of equation, the TPHCWG determined that using the estimated molecular weights, solubilities and vapor pressures to calculate HLC allowed for internal consistency with the other estimated values. The formulas provided by the TPHCWG (1997a) are as follows:

Aromatics:

$\text{Log } S = (-0.21 \times EC) + 3.7$

$\text{Log } K_{oc} = (0.10 \times EC) + 2.3$

Aliphatics:

$\text{Log } S = (-0.55 \times EC) + 4.58$

$\text{Log } K_{oc} = (0.45 \times EC) + 0.43$

Aliphatics and Aromatics

$\text{Log VP} = (-0.50 \times EC) + 2.30 \text{ [for EC} \leq 12]$

$\text{Log VP} = (-0.36 \times EC) + 0.72 \text{ [for EC} > 12]$

$$H' \text{ (unitless)} = \frac{\text{Vapor Pressure (atm)} \times \text{ Molecular Weight (g/mol)}}{\text{Solubility (mg/L)} \times 8.2\text{x}10^{-5} \text{ (atm - m}^3\text{/mol - K)} \times 293\text{K}}$$

Henry's Law constant (atm-m^3/mol) = H' (unitless)/41

When diffusivity in air or water was plotted as a function of equivalent carbon number, the TPHCWG found that the values did not vary significantly from compound to compound. Thus, a conservative, reasonable assumption was to set $D_{air} = 10^{-1}$ cm^2/sec and $D_{water} = 10^{-5}$ cm^2/sec for all fractions.

Using the models above, the following chemical values for the TRPH fractions have been assigned:

Table C-2

Assigned Chemical Properties of TRPH Fractions Based on an Equivalent Carbon Number (EC)

TRPH Fraction	Avg. EC	Proposed Value				
		HLC (atm-m^3/mol)[a]	MW (g/mol)	K_{oc} (mL/g)[b]	S (mg/L)[b]	VP (atm)[b]
C_5-C_7 Aromatic	6.5	5.61 E-3	NC	NC	NC	NC
>C_7-C_8 Aromatic	7.5	6.64 E-3	NC	NC	NC	NC
>C_8-C_{10} Aromatic	9.0	1.17 E-2	1.2 E+2	1.58 E+3	6.5 E+1	6.3 E-3
>C_{10}-C_{12} Aromatic	11	3.41 E-3	1.3 E+2	2.51 E+3	2.5 E+1	6.3 E-4
>C_{12}-C_{16} Aromatic	14	1.29 E-3	1.5 E+2	5.01 E+3	5.8 E+0	4.8 E-5
>C_{16} -C_{21} Aromatic	18.5	3.17 E-4	1.9 E+2	1.58 E+4	6.5 E-1	1.1 E-6
>C_{21} -C_{35} Aromatic	28.5	1.63 E-5	2.4 E+2	1.26 E+5	6.6 E-3	4.4 E-10
C_5-C_6 Aliphatic	5.5	8.05 E-1	8.1 E+1	7.94 E+2	3.6 E+1	3.5 E-1
>C_6-C_8 Aliphatic	7.0	1.22 E+0	1.0 E+2	3.98 E+3	5.4 E+0	6.3 E-2
>C_8-C_{10} Aliphatic	9.0	1.93 E+0	1.3 E+2	3.16 E+4	4.3 E-1	6.3 E-3
>C_{10}-C_{12} Aliphatic	11	2.93 E+0	1.6 E+2	2.51 E+5	3.4 E-2	6.3 E-4
>C_{12}-C_{16} Aliphatic	14	1.29 E+1	2.0 E+2	5.01 E+6	7.6 E-4	4.8 E-5
>C_{16}-C_{21} Aliphatic	18.5	1.20 E+2	2.7 E+2	6.30 E+8	2.5 E-6	1.1 E-6

NC: Values for the C_5-C_7 and >C_7-C_8 aromatics, were made to correspond to benzene and toluene, respectively per TPHCWG guidance. Chemical-specific values for these fractions were assumed to be equal to those of benzene and toluene.

[a] Henry's Law constant (HLC) calculated using methods described above. Final values rounded to two significant figures.

[b] Organic carbon normalized soil-water partition coefficient (K_{oc}), Solubility (S), and Vapor Pressure (VP) values calculated according to formulas in Tables 7, 9, and 12 of TPHCWG 1997a.

Table C-3

Calculated Chemical Properties of TRPH Fractions

TRPH Fraction	Calculated Fraction-Specific Values*		
	D_a(cm^2/sec)	Volatilization Factor** (m^3/kg)	
		Residential	Industrial
C_5-C_7 Aromatic	2.439E-03	1.408E+03	2.875E+03
>C_7-C_8 Aromatic	1.166E-03	2.037E+03	4.157E+03
>C_8-C_{10} Aromatic	2.635E-04	4.285E+03	8.748E+03
>C_{10}-C_{12} Aromatic	4.901E-05	9.935E+03	2.028E+04
>C_{12}-C_{16} Aromatic	9.338E-06	2.276E+04	4.646E+04
>C_{16} -C_{21} Aromatic	7.280E-07	8.152E+04	1.664E+05
>C_{21} -C_{35} Aromatic	4.797E-09	1.004E+06	2.050E+06
C_5-C_6 Aliphatic	1.582E-02	5.530E+02	1.129E+03
>C_6-C_8 Aliphatic	7.966E-03	7.794E+02	1.591E+03
>C_8-C_{10} Aliphatic	2.060E-03	1.533E+03	3.129E+03
>C_{10}-C_{12} Aliphatic	4.186E-04	3.400E+03	6.939E+03
>C_{12}-C_{16} Aliphatic	9.343E-05	7.196E+03	1.469E+04
>C_{16}-C_{21} Aliphatic	6.933E-06	2.642E+04	5.392E+04

*All calculations carried out to 18 decimal places. Values provided have been rounded for presentation in this table.

**For residential exposure to non-carcinogens, VFs are based on exposure duration of six years. Industrial exposure duration is 25 years.

2. Derivation of TRPH Fraction Toxicological Values

The toxicity values for the various TRPH fractions (Table C-4) were obtained from the TPHCWG (1997b) or were derived from route-to-route extrapolation.

Table C-4
Toxicity Values of TRPH Classes[a]

TRPH Fraction	GI absorption	RfD_o	RfD_d [c]	RfD_i [d]	Target Organs/ Systems or Effects
	(%)[b]	(mg/kg-day)			
C_5-C_7 Aromatic	90%	0.2	0.180	0.1143	Liver, neurological
>C_7-C_8 Aromatic	80%	0.2	0.160	0.1143	
>C_8-C_{10} Aromatic	50%	0.04	0.020	0.05714	Body weight
>C_{10}-C_{12} Aromatic	50%	0.04	0.020	0.05714	
>C_{12}-C_{16} Aromatic	50%	0.04	0.020	0.05714	
>C_{16}-C_{21} Aromatic	50%	0.03	0.015	0.015[e]	Kidney
>C_{21}-C_{35} Aromatic	50%	0.03	0.015	0.015[e]	
C_5-C_6 Aliphatic	50%	5.0	2.5	5.257	Neurological
>C_6-C_8 Aliphatic	50%	5.0	2.5	5.257	
>C_8-C_{10} Aliphatic	50%	0.1	0.05	0.2857	Liver, blood
>C_{10}-C_{12} Aliphatic	50%	0.1	0.05	0.2857	
>C_{12}-C_{16} Aliphatic	50%	0.1	0.05	0.2857	
>C_{16}-C_{35} Aliphatic	50%	2.0	1.0	1.0[e]	Liver

[a] Toxicity Values from TPHCWG 1997b.
[b] Developed using professional judgment based on ATSDR Toxicological Profile for TPH (ATSDR, 1999).
[c] RfD_d values extrapolated from RfD_o, using fraction-specific GI absorption (see Appendix B).
[d] RfD_i values extrapolated from RfC_i values when available (see Appendix B).
[e] RfD_i values extrapolated from RfD_o, using fraction-specific GI absorption (see Appendix B).

3. Derivation of TRPH SCTLs

The TRPH SCTLs are based on a 2-tiered approach. The first tier consists of comparing site total TRPH concentrations to a default TRPH SCTL developed using the toxicity values and other inputs developed for the >C_8-C_{10} aromatic range. If the default SCTL is exceeded, then the TRPHs may be sub-classified so that each fraction can be compared to its respective fraction-specific SCTL. Given the potential for the sub-classification methodology to yield relatively high SCTLs, it is possible that the human health SCTL for some constituents, particularly those with relatively low toxicity and low mobility potential could result in staining, odor and/or nuisance conditions.

The default TRPH SCTL is based on the >C_8-C_{10} carbon range as a result of two factors. First, the analytical method identified by the FDEP for the purpose of measuring petroleum hydrocarbons in water and soil is limited to the detection of products within a carbon chain range of C_8-C_{40}. This method, the Florida Petroleum Residual Organic (FL-PRO) — Alternative Method to Total Petroleum Hydrocarbons, 418.1 or 9073 — combines several of the commonly used

methods so that the targeted range of petroleum hydrocarbons can be analyzed in a single step. However, because of its limitations, the smallest detectable C-range using the FL-PRO method is the $>C_8$-C_{10} grouping. Secondly, the TRPH SCTL value was selected based on the identification of the most conservative values. The calculation of the SCTLs (listed below) using standard FDEP and USEPA protocols results in the most conservative values for the C_5-C_7 aromatics. However, due to the limitations of the TRPH method of analysis, and since the most toxic and prevalent chemicals within this range are addressed by other analyses and individual SCTLs, the values in this group are not used as TRPH SCTLs. The most conservative values for residential and industrial direct exposure that occur within a carbon range that can be analyzed by FL-PRO are found in the $>C_8$-C_{10} aromatics grouping. Therefore, the default TRPH SCTL values are based on this group of total petroleum hydrocarbons.

With the assignment of the above chemical and toxicological values, the determination of risk-based SCTLs follows the same methodology as that used for individual compounds.

Table C-5
Calculated SCTLs for TRPH Fractions

TRPH Fraction	SCTL (mg/kg)			Target Organs/ Systems or Effects
	Residential	Industrial	Leachability [a]	
C_5-C_7 Aromatic	340	1800	34	Liver, neurological
$>C_7$-C_8 Aromatic	490	3700	59	
$>C_8$-C_{10} Aromatic	**460**	**2700**	**340**	Body weight
$>C_{10}$-C_{12} Aromatic	900	5900	520	
$>C_{12}$-C_{16} Aromatic	1500	12000	1000	
$>C_{16}$-C_{21} Aromatic	1300	11000	3200	Kidney
$>C_{21}$-C_{35} Aromatic	2300	40000	25000	
C_5-C_6 Aliphatic	6200	33000	470	Neurological
$>C_6$-C_8 Aliphatic	8700	46000	1300	
$>C_8$-C_{10} Aliphatic	850	4800	7000	Liver, blood
$>C_{10}$-C_{12} Aliphatic	1700	10000	51000	
$>C_{12}$-C_{16} Aliphatic	2900	21000	*	
$>C_{16}$-C_{35} Aliphatic	42000	280000	*	Liver

[a] Based on the acceptable concentration of 5000 µg/L for groundwater and surface waters.
* Not a health concern for this exposure scenario.

B. Development of SCTLs for Hydrocarbon Fractions Identified Using the MADEP Approach

As mentioned earlier, the two main advantages of the MADEP approach over the FL-PRO analytical method are that it can quantify petroleum hydrocarbons in the C_5-C_8 range, and it can distinguish between aliphatics and aromatics. Like FL-PRO, the MADEP approach provides an alternative to the determination of TRPHs, which is not particularly useful in health risk assessment.

1. Analytical Methodology

MADEP developed the Volatile Petroleum Hydrocarbons (VPH) and Extractable Petroleum Hydrocarbons (EPH) methods based on USEPA analytical approaches that have traditionally used the purge and trap method for the analysis of volatile organics, and solvent extraction for the semi-volatile/extractable organics. The use of two approaches to determine petroleum hydrocarbons is needed because neither approach alone is capable of measuring petroleum compounds in all of the hydrocarbon ranges of interest. The MADEP approach breaks up the C_9-C_{18} aliphatic range (despite the fact that compounds in this range are considered to be relatively consistent in terms of toxicity) to enable detection of all gasoline-range hydrocarbons by the VPH method. As a result, the aliphatic and aromatic hydrocarbons are divided into six separate ranges, three detected by the VPH method, and three by the EPH method, as follows:

Table C-6

Hydrocarbon Fractions Identified Using the MADEP Methodology

Toxicologically Defined Hydrocarbon Fractions	Analytically Defined Hydrocarbon Fractions	Analytical Method
C_9-C_{22} Aromatics	C_9-C_{10} Aromatics	VPH
	C_{11}-C_{22} Aromatics	EPH
C_5-C_8 Aliphatics	C_5-C_8 Aliphatics	VPH
C_9-C_{18} Aliphatics	C_9-C_{12} Aliphatics	VPH
	C_9-C_{18} Aliphatics	EPH
C_{19}-C_{36} Aliphatics	C_{19}-C_{36} Aliphatics	EPH

The MADEP VPH method is a purge and trap procedure. The collective concentrations of hydrocarbon fractions can be quantified in solid and aqueous matrices. This method is comparable

to the Gasoline Range Organics (GRO) method, because both detect hydrocarbons in the C_5-C_{12} range. The VPH goes one step further and separates the GRO fraction into 3 subfractions (see Table C-6 above) and also provides specific measurements of six target compounds: benzene, toluene, ethylbenzene, xylenes (BTEX), methyl tert-butyl ether (MTBE), and naphthalene. Detection is achieved by a photoionization detector (PID) and flame ionization detector (FID) working in series. The PID chromatogram is used to determine the collective fractional concentration of aromatic hydrocarbons in the C_9-C_{10} range. Because the PID can detect sample analytes without destroying them, compounds can then pass through the FID where they are combusted in a hydrogen flame. In theory, the FID will detect the total concentrations of all petroleum hydrocarbons in the sample, and the PID will detect only aromatic compounds. Aliphatic compounds can then be quantified by subtracting the PID response from the FID response.

Two potential problems have been identified for the use of the VPH method:

1) Given that the PID detects both *Pi* and double carbon bonds, alkenes will be falsely quantitated as aromatics. This is not considered a major methodological limitation because alkenes are not typically found in high concentration in most petroleum products, and because they are more toxicologically similar to aromatics than to aliphatics.
2) Some aliphatic compounds, especially heavier molecular weight branched and cyclic alkenes will produce some response on the PID detector. This response can lead to significant over-quantitation of the aromatic fraction when dealing with products such as kerosene and Jet A fuel, which contain predominantly aliphatic compounds within this range.

The MADEP EPH method is a solvent extraction/fractionation gas chromatography (GC) / FID procedure. The EPH method can be viewed as directly comparable to the TPH USEPA Method 418.1. Like the TPH, the EPH method quantitates hydrocarbons > C_9 in solid and aqueous samples. In addition, the EPH method separates the TPH fraction into three subfractions (see Table C-6 above) and measures 17 targeted PAH compounds. Samples are extracted with methylene chloride, exchanged into hexane, and loaded onto silica gel. The silica gel is first rinsed with hexane to strip aliphatic compounds, and then with methylene chloride to strip aromatic compounds. Both extracts are then analyzed separately by direct injection into a temperature-programmed GC/FID.

Two methodological elements should be considered when evaluating EPH data:

1) Small errors during the fractionation between aromatic and aliphatic compounds can result in significant over- or underestimation of aromatic and aliphatic ranges. For this reason, the method specifies the use of a *Fractionation Check Solution* to verify proper separation of the aliphatic and aromatic fractions.
2) Laboratories using the EPH method must use a *forced projected baseline* when integrating chromatographic areas of fractional ranges. This means that, when quantifying peak areas by internal or external calibration, the collective peak area integration for the fractional ranges must be from baseline. This is necessary because, like any GC/FID procedure, the EPH method may produce an Unresolved Complex Mixture (UCM), particularly when analyzing weathered products. This UCM is produced when many individual hydrocarbons are eluting from the capillary column at the same time, preventing the detector signal from returning to baseline. If a forced projected baseline is not used, resultant fractional range data may significantly under-report true hydrocarbon concentrations.

2. Development of Cleanup Target levels

This section describes the procedures used to develop Cleanup Target Levels (CTLs) for the petroleum hydrocarbon fractions identified using the MADEP methodology. Although MADEP has developed CTLs for residential and industrial scenarios (S1 and S2 standards), the different climatic conditions between Massachusetts and Florida preclude their direct use. In addition, MADEP has decided to use ceiling criteria based on professional judgment and, as a result, most of their CTLs are not health-based.

All exposure assumptions used in these calculations are consistent with Chapter 62-777, F.A.C. GI absorption was estimated as 50% for all fractions using professional judgment based on the 1999 ATSDR toxicological profile for TPH (1999).

a) Toxicity values

Reference Doses (RfDs) used were those developed by the TPHCWG for fractions that encompass similar ranges of hydrocarbons to those identified by the MADEP methodology. This approach for developing RfDs is consistent with SCTLs based in TPHCWG fractions, and is based on a combination of approaches including the assessment of toxicity of mixtures and the use of surrogate chemicals representative of the fractions under study. It must be noted that MADEP has developed RfDs for use with the fractions defined by their method using surrogate compounds for each fraction. Oral reference doses (RfD_o) used by MADEP are for the most part either similar or higher than the RfDs developed by the TPHCWG (1997b). Inhalation RfDs (RfD_i) were calculated

from Reference Concentrations (RfC) when available, or extrapolated from RfD_os, assuming that GI absorption is 50%. Dermal RfDs (RfD_d) were extrapolated from RfD_o using also a GI absorption value of 50%.

Table C-7
Reference Doses Used for Developing CTLs for Hydrocarbons
Identified Using the MADEP Approach

MADEP Fraction	Comparable TPHCWG Fraction	RfD_o	RfD_d	RfD_i	Target Organs/ Systems of Effects
		(mg/kg-day)			
Aromatics					
C_9-C_{10}	>C_8-C_{10}	0.04	0.02	0.05714	Body weight
C_{11}-C_{22}	>C_{12}-C_{16}	0.04	0.02	0.05714	
Aliphatics					
C_5-C_8	>C_6-C_8	5.0	2.5	5.257	Neurological
C_9-C_{12}	>C_{10}-C_{12}	0.1	0.05	0.2857	Liver, blood
C_9-C_{18}	>C_{12}-C_{16}	0.1	0.05	0.2857	
C_{19}-C_{36}	>C_{16}-C_{35}	2.0	1.0	1.0	Liver

b) Physical-Chemical Properties

To conduct fate and transport evaluations/modeling, we used the approaches and procedures set forth in the document *Volume 3: Selection of Representative TPH Fractions Based on Fate and Transport Considerations* (TPHCWG, 1997a). Chemical-physical properties for each fraction were calculated using the correlation approach using the average Equivalent Carbon Number (EC) as the independent variable. The fraction-specific chemical-physical properties presented in the table below were obtained from MADEP (1997), except for the aliphatic C_{19}-C_{36} fraction, for which data for the C_{16}-C_{21} fraction from the TPHCWG were used. MADEP has assumed that this fraction is immobile. However, this assumption may not be valid for compounds at the lighter end of this fraction, and therefore the more conservative approach of using data for the C_{16}-C_{21} fraction provided by the TPHCWG has been adopted.

Table C-8
Physical-Chemical Properties Assigned to MADEP Fractions
Based on Equivalent Carbon Number (EC)

Range of Carbons	Avg. EC	MW (g/mol)	VP (atm)	S (mg/L)	Henry's Law Constant	Koc (mL/g)	D (cm^2/s)
C_9-C_{10} Aromatics	9.5	120	2.9 E-3	51	0.33	1778	0.07
C_{11}-C_{22} Aromatics	14	150	3.2 E-5	5.8	0.03	5000	0.06
C_5-C_8 Aliphatics	6.5	94	1.0 E-1	11	54	2265	0.08
C_9-C_{12} Aliphatics	10.5	149	8.7 E-4	0.07	65	1.5 E+5	0.07
C_9-C_{18} Aliphatics	12	170	1.4 E-4	0.01	69	6.8 E+5	0.07
C_{19}-C_{36} Aliphatics	18.5*	270	1.1E-6	2.5E-6	4900	6.3E+8	6.9E-6

*EC and derived physical / chemical properties correspond to those of the surrogate TPHCWG C16-C21 fraction (see text above).

3. SCTLs for Petroleum Hydrocarbon Fractions Identified Using the MADEP Approach

The following table presents the CTLs developed to evaluate laboratory results that use the MADEP approach for fractionation of TRPHs. In some instances, MADEP laboratory results may include benzene,toluene, ethylbenzene, xylene, MTBE, and individual PAH concentrations. However, this method has not been approved for quantification of these compounds in Florida. CTLs for the comparable fractions identified using the TPHCWG methodology are also provided. Leachability values were calculated using 5000 µg/L as the groundwater and surface water acceptable concentration.

Table C-9
Direct Exposure and Leachability Soil CTLs for TRPH Fractions
Identified Using the MADEP and the TPHCWG Methodologies

MADEP Fraction	Comparable TPHCWG Fraction	Residential (mg/kg)		Industrial (mg/kg)		Leachability (mg/kg)		Target Organs / Systems or Effects
		MADEP	TPHCWG	MADEP	TPHCWG	MADEP	TPHCWG	
Aromatics								
C_9-C_{10}	>C_8-C_{10}	560	460	3400	2700	380	340	Body weight, kidney
C_{11}-C_{22}	>C_{12}-C_{16}	1800	1500	15000	12000	1000	1000	
Aliphatics								
C_5-C_8	>C_6-C_8	7100	8700	38000	46000	960	1300	Neurological
C_9-C_{12}	>C_{10}-C_{12}	1700	1700	11000	10000	31000	51000	Liver, blood
C_9-C_{18}	>C_{12}-C_{16}	2900	2900	21000	21000	1.4E+5	1E+6	
C_{19}-C_{36}	>C_{16}-C_{21}	42000	42000	2.8E-5	2.8E-5	1E+6	1E+6	Liver

XV. Appendix D: ProUCL Memo

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June 21, 2005

Ligia Mora-Applegate
Bureau of Waste Cleanup
Florida Department of Environmental Protection
2600 Blair Stone Road
Tallahassee, FL 32399

Re: Status of ProUCL as an Approved Statistical Method

Dear Ms. Mora-Applegate:

In this letter, we would like to clarify our recommendation regarding the status of ProUCL (Version 3) as an "approved statistical method" for calculating 95% UCL values, as provided in Chapter 62-780-610(2), F.A.C. As you know, ProUCL is a software tool developed by a U.S. EPA contractor and has been approved by the Agency for use in calculating exposure point concentrations. It is publicly available, and we have been in contact with one of its principal architects, Dr. Anita Singh, during the development of FLUCL. We coded a previous version of ProUCL into FLUCL during beta testing, and are familiar with how ProUCL works, including the current version. Although the validation studies used to create ProUCL Version 3 have not been published, we know how they were performed and have an understanding of the strengths and limitations of this tool.

We recommend that the Florida Department of Environmental Protection (FDEP) allow the use of ProUCL Version 3, provided that it is used within its limitations. This means specifically that it should be used only for data sets with:

1. A total of 10 or more samples, and at least 7 measured concentrations (either unqualified or carrying a "J", "I", or "T" qualifier). This condition applies regardless the software tool used to calculate the 95% UCL. The reliability of 95% UCL estimates from ProUCL, like FLUCL, is dependent upon being able to identify the underlying distribution of the data. This is impossible with only a few samples. While ProUCL Version 3 will operate when only a few data values are entered, the output is unreliable. In other words, just because the software will run with less than 10 samples doesn't mean that the output should be accepted.

2. No more than 15% of the data set is "non-detect". ProUCL Version 3 has not been validated to perform reliably when more 15% of the data are censored (i.e., non-detects). FLUCL should be used for data sets with more than 15% censoring, up to a limit of 70%. For very highly censored data sets, the bounding method can be used to estimate the 95% UCL. This method has been programmed into FLUCL, and can be used provided there are at least three measured concentrations.

It is our understanding that ProUCL Version 4 is under development, and that one of the planned improvements is better ability to handle censored data sets. When this version is released, we will evaluate it and give you our recommendation, including any limitations in its use, if warranted.

Sincerely,

Stephen M. Roberts, Ph..D.

Kenneth M. Portier, Ph.D.