New York State Brownfield Cleanup Program

Development of Soil Cleanup Objectives

Technical Support Document

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APPENDICES

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ABBREVIATIONS

AF	absorption fraction
ALAD	aminolevulinic acid dehydratase
ALM	Adult Lead Methodology
ASP	Analytical Services Protocol
ATSDR	Agency for Toxic Substances and Disease Registry
b.g.s.	below ground surface
BAF	bioaccumulation factor
BaP	benzo[a]pyrene
BKSF	biokinetic slope factor
BMCL	benchmark concentration level
BMDL	benchmark dose level
BW	body weight
CAS RN	Chemical Abstracts Service Registry Number
CDC	Centers for Disease Control and Prevention
CPF	cancer potency factor
CRQL	contract required quantitation level
C_s	soil concentration
C_{sat}	soil saturation concentration
C_w	groundwater/drinking water standard
DAF	dilution attenuation factor
DDT	dichlorodiphenyltrichloroethane
DEP	Department of Environmental Protection
DW	dry weight
Eco-SSL	ecological soil screening levels
EF	exposure frequency
ERP	Environmental Restoration Program
ESCO	ecological soil cleanup objective
FIR	food ingestion rate
f_{oc}	fraction of organic carbon
GSD	geometric standard deviation
HPDB	Household Products Database
HQ	hazard quotient
IARC	International Agency for Research on Cancer
ICD	irritant contact dermatitis
IEUBK	Integrated Exposure Uptake Biokinetic
IR	ingestion rate
K_d	sorption partition coefficient / soil-water distribuition coefficient
K_H	Henry's Law constant
K_{oc}	organic-carbon partition coefficient
K_{ow}	octanol-water partition coefficient
LC_{50}	50% lethal concentration
LEC	lower bound on estimated air concentration
LED	lower bound on estimated dose
LOEC/LOEL	lowest observed effects concentration or level

MCP	Massachusetts Contingency Plan
MDL	method detection limit
NAPL	non-aqueous phase liquids
NHANES III	Third National Health and Nutrition Examination Survey
NOEC/NOEL	no observed effects concentration or level
NRCC	National Resource Conservation Commission
NTP	National Toxicology Program
NYCRR	New York Code of Rules and Regulations
NYS DEC	New York State Department of Environmental Conservation
NYS DOH	New York State Department of Health
PAH	polycyclic aromatic hydrocarbon
PbB	blood lead level
PB-PK	physiologically-based pharmacokinetic
PCA	Pollution Control Agency
PCBs	polychlorinated biphenyls
PCDDs	polychlorinated dibenzo-*p*-dioxins
PCDFs	polychlorinated dibenzofurans
PEF	particulate emission factor
RAGS	Risk Assessment Guidance for Superfund
RfC	reference concentration
RfD	reference dose
RPF	relative potency factor
RSBC	rural soil background concentration
RSBC-ER	rural soil background concentration for ecological resources
RSC	relative source contribution
S	solubility
SCO	soil cleanup objective
SD	standard deviation
SSL	soil screening level
SVOC	semi-volatile organic compound
TAGM	Technical and Administrative Guidance Memorandum
TAL	Target Analyte List
TCDD	tetrachlorodibenzo-*p*-dioxin
TCL	Target Compound List
TEF	toxic equivalency factor
TRV	toxic reference value
TSCA	Toxic Substances Control Act
UR	unit risk
US EPA	United States Environmental Protection Agency
US FDA	United States Food and Drug Administration
USGS	United States Geological Survey
VF	volatilization factor
VOC	volatile organic compound
WHO	World Health Organization
WW	wet weight

1.0 Executive Summary

Article 27, Title 14 of the Environmental Conservation Law establishes the Brownfield Cleanup Program for New York State. Section 27-1415.4 of the Legislation directs the Commissioner of Environmental Conservation, in consultation with the Commissioner of Health, to promulgate regulations that create a multi-track approach for the remediation of contamination at brownfield sites. Section 27-1415.6 of the Legislation describes the requirements for soil cleanup objectives (SCOs), which are contaminant-specific remedial action objectives for soil based on a site's current, intended, or reasonably anticipated future use.

SCOs are included in the Brownfield Cleanup Program regulation (Title 6, New York Codes Rules and Regulations, Part 375-6). This Technical Support Document explains the technical basis of the methods used to develop the SCOs. A proposed process for developing SCOs was described in summary documents that were posted on the New York State Department of Environmental Conservation (NYS DEC) web site and provided to the public at three public meetings in 2004. Public input was solicited, and written comments were considered in the development of the SCOs.

Separate sets of SCOs were developed in consideration of public health, groundwater, and ecological resources. Background concentrations of contaminants in rural soils were also considered, and maximum acceptable levels of chemicals in soil (i.e., "caps") were identified. The final SCOs presented in the Regulation reflect all of these considerations.

The Legislation required that SCOs be specific to land use categories, including sites where no restrictions would be placed on use (unrestricted), as well as for sites where land use restrictions or engineering controls may limit possible exposures (commercial and industrial). SCOs were developed for these three land use categories, as well as two additional categories - residential and restricted residential. These additional categories were developed for Track 2, in place of unrestricted use as referenced in Article 27-1415(7) of the Environmental Conservation Law, to avoid confusion with Track 1 and better describe the remedial scenario (see the Brownfield Cleanup Program regulation for a description of each of the four cleanup tracks). The residential

category is intended for sites that could be developed for single family housing, but with restrictions that prohibit raising livestock or producing animal products for human consumption. The restricted residential category is intended for sites that could be developed for residential uses, specifically, multi-family residential housing and other uses with potentially higher exposures than commercial and industrial uses, but with restrictions that prohibit single family housing and vegetable gardens (although community vegetable gardens may be considered with NYS DEC approval).

The Legislation did not specify the contaminants for which SCOs were to be developed. With a goal of including frequently encountered contaminants, a broad list of potential SCO contaminants was developed based on contaminant lists previously developed by NYS DEC and the United States Environmental Protection Agency (US EPA). From this broad list of contaminants, a priority list was developed based on contaminants commonly found at New York State waste sites. Several contaminants were added to the list in response to public comments. SCOs were developed for this subset - termed the "Soil Cleanup Objectives Priority List."

In order to develop the health-based SCOs, contaminant-specific information was needed on levels of exposure (e.g., intake amounts or environmental levels) associated with certain categories of health effects. This contaminant-specific information is referred to as "toxicity values." The categories of health effects for which toxicity values were identified include long-term (chronic) effects (including both cancer and non-cancer effects), short-term (acute) effects, and irritant contact dermatitis (i.e., non-allergic skin irritation). This information was used to derive SCOs.

For chronic health-based SCOs, the toxicity values included reference doses and reference air concentrations (for non-cancer endpoints), and cancer potency factors, and inhalation unit risks (for cancer). These toxicity values were selected from those published by various governmental and/or health organizations according to established methods. Each chemical was evaluated for whether or not it could cause health effects only at the site (e.g., the lungs) of exposure (known as "local" health effects), or whether it could cause effects in other parts of the body ("systemic"

effects). This evaluation provided the basis for decisions regarding the appropriateness of combining exposure pathways in the calculation of SCOs.

The potential for short-term (acute) exposure to cause health effects also was considered in developing health-based SCOs. Acute toxicity reference doses were derived for seven Priority List contaminants (arsenic, barium, cadmium, copper, cyanide, nickel, naphthalene, pentachlorophenol, and phenol) that had been identified in the scientific literature as being of particular concern for acute soil exposure.

The potential for non-allergic skin irritation also was considered, and skin reference doses were derived for three contaminants (chromium, nickel, and phenol) on the Priority List. A default skin reference dose was also derived for application to Priority List semivolatile organic chemicals (SVOCs), including pesticides.

Some of the Priority List chemicals occur as components of commercial products (chlordane, endosulfan, endrin, and xylene). The polycyclic aromatic hydrocarbons (PAHs) on the Priority List (e.g., benzo[a]pyrene, benz[a]anthracene) are usually present at contaminated sites as a mixture of related chemicals. Approaches were developed to evaluate the toxicity of these products and mixtures.

Developing the health-based SCOs required a number of exposure considerations including who might be exposed to soil contaminants, in what ways they might be exposed, and for how long the exposure might occur. Since these considerations can vary with the use of a site, health-based SCOs differ depending upon site use. For example, the health-based SCOs for an industrial facility, where the exposed population is primarily limited to adult workers, differ from SCOs for a residential setting where children may be present and vegetable gardening activities may occur.

In developing the chronic health-based SCOs for each land use category, exposure scenarios were developed for potentially exposed individuals with assumed patterns of exposure-related activity. For unrestricted, residential and restricted residential land uses, residential scenarios

were developed, with both an adult and a child selected as potentially exposed individuals. For the commercial land use category, an adult worker and a child "visitor" were chosen as representative of potentially exposed individuals. The industrial land use category included a potentially exposed adult worker and an adolescent "trespasser."

The exposure pathways evaluated were soil ingestion, inhalation, dermal absorption, homegrown vegetable consumption and home produced animal product consumption (e.g., meat, milk, eggs). Evaluating the soil ingestion pathway required information on how much soil people may ingest. Estimated soil ingestion rates have been reported in a number of studies, and information from these studies was used in developing the SCOs. People may also be exposed to contaminants in soil through inhalation of vapors or suspended soil particles (called particulates). Models have been developed and used by the US EPA to account for this exposure pathway. These models were used in the development of SCOs. The SCOs account for inhalation exposure to vapor from volatile organic compounds (VOCs) and elemental mercury in soil, and particulates for all other Priority List contaminants. People can also be exposed to soil contaminants through skin contact. The US EPA has established a method to estimate dermal exposure to some soil contaminants. This approach was used to develop the SCOs.

Consumption of homegrown garden vegetables can also contribute to exposure to soil contaminants at sites where there are gardens (unrestricted and residential land use categories), and consumption of home produced animal products can contribute to exposure to soil contaminants at sites where there may be farms (unrestricted land use category). A review of the scientific literature suggested that exposure to soil contaminants by these pathways can be significant, but also that methods for quantifying the exposures are very uncertain. Because of the uncertainty in exposure quantification, a quantitative estimate of such exposures was not included in the SCOs. Instead, unrestricted and residential land use SCOs that were calculated for soil ingestion were adjusted downwards to account for these additional exposure pathways.

Various contaminant sources and pathways of exposure can contribute to overall exposure to a site-related contaminant. These include site-related exposures as well as those not associated with the site, such as exposures that could result from the presence of contaminants in drinking

water, air, food, or consumer products. Therefore, an approach was used to account for exposures unrelated to brownfield sites. Data upon which to base contaminant-specific estimates of non-site exposures are limited. Therefore, the approach assumes that non-site exposures account for 80% of the estimated overall (site-related and non-site) exposure. This percentage, and the application of this approach to non-cancer health effects but not to cancer health effects, is consistent with US EPA approaches for developing some environmental standards and guidelines (such as those for drinking water).

The toxicity and exposure information described above was used to calculate chronic (non-cancer and cancer) human health-based SCOs for the soil ingestion, dermal exposure, and inhalation exposure pathways for each land use category. SCOs were calculated for children (unrestricted, residential, restricted residential and commercial land uses), adults (all land uses) and adolescent trespassers (industrial land use). Where appropriate (based on toxicity information), SCOs for individual exposure pathways (soil ingestion, dermal exposure, inhalation exposure) were combined to yield a "combined pathway" SCO. For each land use category, the final chronic human health-based SCO was the lowest of these values. SCOs were also calculated for acute soil ingestion exposure and irritant contact dermatitis. For each land use category, the lowest health-based SCO (chronic, acute, or irritant contact dermatitis) was chosen as the final health-based SCO.

Section 27-1415.1 of the Legislation requires that all remedies be protective of groundwater according to its classification pursuant to Section 17-0301 of the Environmental Conservation Law. A number of different approaches were considered for developing SCOs that would not result in a violation of groundwater and/or drinking water standards or guidelines due to impacts of contaminants in the soil dissolving in water (e.g., from rain) - and moving downward through the soil column ("leaching") to the groundwater. Based on NYS DEC's experience with estimating impacts on groundwater from soils at inactive hazardous waste sites, an approach was selected which estimates the amount of contamination that may be present in water when it is in direct contact with soil for a long time, and the amount of contaminant that may leach out of contaminated soil as water travels down through the soil column. The approach also accounts for the reduction in water contaminant concentrations as the water in the soil travels to

groundwater. This approach was used to calculate SCOs for protection of groundwater, by setting them at the maximum soil contaminant concentration that were estimated not to result in a violation of groundwater and/or drinking water standards or guidelines.

Ecological resources also were considered in developing the SCOs. In order to adopt suitable SCOs for protection of terrestrial ecological resources in a timely manner, the NYS DEC reviewed existing soil criteria available in the literature along with the corresponding derivation methodologies. After an extensive review, the NYS DEC chose to adopt many of the procedures and methods developed by the US EPA Ecological Soil Screening Levels (Eco-SSL) program. The Eco-SSL derivation process represents the efforts of a multi-stakeholder workgroup consisting of federal, state, consulting, industry, and academic participants lead by US EPA's Office of Superfund Remediation and Technology Innovation .

The NYS DEC adopted the Eco-SSLs model for calculating hazard quotients, plant bioaccumulation models, and earthworm bioaccumulation models. Eco-SSL methodologies were modified somewhat because Eco-SSLs were specifically designed to be used as screening values and not cleanup levels. The US EPA emphasizes that it is inappropriate to adopt Eco-SSL values as cleanup standards. Eco-SSL methodologies (not Eco-SSL values themselves) were adopted for use in deriving ecological SCOs because they represent the best, most current, accepted scientific methods for assessing the uptake and bioaccumulation of soil-borne contaminants by plants and soil invertebrates and for estimating food chain risks to birds and terrestrial wildlife. These methodologies can easily be modified to develop cleanup objectives rather than screening concentrations by changing some of the variables and parameters that tend to be more conservative; for example, using lowest observed effects concentrations in the calculation of hazard quotients rather than no observed effects concentrations.

In addition to protection of health, groundwater, and ecological resources, two other considerations contributed to the basis of the final SCOs in the Regulation. These considerations were the levels of Priority List contaminants in rural soils of New York State, and maximum acceptable soil contaminant concentrations.

A statewide rural surface soil survey was undertaken to examine the concentrations of selected analytes in rural New York State soils. Information from this survey and other data sources was used to identify rural soil background concentrations for Priority List contaminants. For some of these contaminants, ecological and health-based SCOs are lower than rural soil background concentrations. For those contaminants, SCOs were set at the rural soil background concentrations.

The calculated SCOs (health-based, groundwater, or ecological) for certain contaminants could allow extremely high levels of those contaminants in soil. Therefore it was decided that the SCOs would be limited to a maximum value, above which the soil would be considered to be unacceptably contaminated. These maximum values are referred to as "caps." Since the law provides for land use-specific soil cleanup objectives, the cap levels also vary by land use. Factors that were considered in the determination of these maximum acceptable levels include visual considerations (appearance), olfactory impacts (odor), and saturation levels (C_{sat}), among other considerations.

In addition to all the considerations discussed above, four other considerations were incorporated into the development of SCOs. These include intrusion of contaminant vapors into indoor air, protection of adjacent residential land uses, exposure to residual mixtures of site contaminants, and soil cleanup levels historically achieved at other sites.

Potential exposures to site-related contaminants through the migration of vapors from soil or groundwater into the indoor air of buildings ("vapor intrusion") were considered. The vapor intrusion pathway is complex and depends on numerous site-specific factors that may vary considerably from site-to-site. NYS Department of Health (NYS DOH) guidance on identifying and addressing current and potential human exposures associated with vapor intrusion will be used to address this pathway.

Potential exposures to site-related contaminants at adjacent residential properties (due to contaminant transport, e.g., via wind or surface water runoff) were considered. To address these potential exposures, the remedial (cleanup) program for each site will include measures to

minimize the transport of soil contaminants from brownfield sites to adjacent residential properties, during and after implementation of the remedial program.

SCOs for individual chemicals reflect risk levels that do not exceed one-in-one million for carcinogenic endpoints and a hazard index of one for non-cancer endpoints. Remedial actions taken to address elevated levels of individual chemicals typically reduce the concentrations of those chemicals at the site to levels that are substantially lower than the SCO values. Therefore, the risk associated with exposure to residual contaminants at brownfield sites is expected to be below the target risk levels identified in the Legislation.

Remedial objectives historically achieved at contaminated sites under existing state remedial programs were considered in the development of SCOs. Cleanup objectives put forth in the 1992 *Technical and Administrative Guidance Memorandum* (TAGM 4046) were identified as the most stringent objectives achieved under the State Superfund, Environmental Restoration, or Voluntary Cleanup Programs. However, in many cases, cleanup objectives have been higher than those put forth in TAGM 4046 based upon site-specific considerations. For some sites, the cleanup number may have been lower to provide protection for ecological resources.

2.0 Introduction

Article 27, Title 14 of the Environmental Conservation Law establishes the Brownfield Cleanup Program for New York State. Section 27-1415.4 of the Legislation directs the Commissioner of Environmental Conservation, in consultation with the Commissioner of Health, to promulgate regulations which create a multi-track approach for the remediation of contamination at brownfield sites. Section 27-1415.6 of the Legislation describes the requirements for soil cleanup objectives (SCOs), which are contaminant-specific remedial action objectives for soil based on a site's current, intended, or reasonably anticipated future use.

Soil cleanup objectives are included in the Brownfield Cleanup Program regulation (Title 6, New York Codes Rules and Regulations, Part 375). This Technical Support Document explains the derivation of the SCOs. The proposed process for developing SCOs was described in summary documents that were posted on the New York State Department of Environmental Conservation (NYS DEC) web site and provided to the public at three public meetings in 2004. Public input into the proposed process was solicited, and written comments were considered in the development of the SCOs. The proposed regulation and a public review draft of this Technical Support Document were published for public comment in November 2005. A series of public informational meetings and three legislative public hearings were held during the public comment period, which closed in March 2006. The comments have been reviewed and the regulation and Technical Support Document have been revised in response to the comments.

Throughout this document, "SCO" may refer to either the final soil cleanup objective presented in the Regulation or soil cleanup objectives that are based on limited considerations, depending upon preceding descriptors (e.g., chronic soil ingestion SCOs, irritant contact dermatitis SCOs, groundwater SCOs, etc.). Tables, figures, and references associated with each section of the document are presented at the end of the section. Subsequent sections of this document describe the chemicals and land use categories for which SCOs were developed, as well as the methods and data used to derive health-based SCOs, groundwater SCOs, and ecological SCOs. Other considerations, including background concentrations of chemicals in surface soils of rural New York State and maximum acceptable concentrations of chemicals in soil (i.e., "caps"), also are

described. The final SCOs, which reflect consideration of all of the above, are presented at the end of the document.

3.0 Land Use Descriptions

SCOs were developed for five land-use categories: unrestricted use, residential use, restricted residential use, commercial use, and industrial use. Following are descriptions of these categories, as presented in the proposed regulation:

(1) "Unrestricted use" which is a use without imposed restrictions, such as environmental easements or other land use controls; or

(2) "Restricted use" which is a use with imposed restrictions, such as environmental easements, which as part of the remedy selected for the site require a site management plan which relies on institutional controls or engineering controls to manage exposure to contamination remaining at a site.

(3) Restricted uses include:

(i) "Residential use" which is a land use category which allows a site to be used for any use other than raising livestock or producing animal products for human consumption. Restrictions on the use of ground water are allowed, but no other institutional or engineering control relative to the Track 2 residential soil cleanup objectives, such as a site management plan, would be allowed. This is the land use category which will be considered for single family housing;

(ii) "Restricted-residential use" which is a land use category which shall only be considered when there is common ownership or a single owner/managing entity of the site. Restricted-residential use:

(a) shall, at a minimum, include restrictions which prohibit:

(1) any vegetable gardens on a site, although community vegetable gardens may be considered with Department approval; and

(2) single family housing; and

(b) includes active recreational uses, which are public uses with a reasonable potential for soil contact;

(iii) “Commercial use” which is a land use for the primary purpose of buying, selling or trading of merchandise or services. Commercial use includes passive recreational uses, which are public uses with limited potential for soil contact; and

(iv) “Industrial use” which is a land use for the primary purpose of manufacturing, production, fabrication or assembly process and ancillary services. Industrial uses do not include any recreational component.

4.0 Target Chemicals

Legislation establishing New York State's Brownfield Cleanup Program (Article 27, Title 14 of the Environmental Conservation Law) requires the NYS DEC, in consultation with the New York State Department of Health (NYS DOH), to develop regulations which create an approach for the remediation of contamination at brownfield sites. The regulations will include tables of contaminant-specific SCOs that are protective of public health and the environment. The legislation does not specify the contaminants for which SCOs are to be developed; therefore, an initial list of proposed target contaminants was developed.

4.1 Identification of Target Chemicals

The NYS DEC has established a Target Compound List (TCL) and a Target Analyte List (TAL). The TCL and TAL are expanded lists of chemicals originally on the US Environmental Protection Agency's (US EPA) Priority Pollutant list. The TCL/TAL lists are used by project managers at hazardous waste sites, Brownfield Cleanup Program sites, and Environmental Restoration Program (ERP) sites.

Use of the TCL/TAL, with the addition of some commonly found petroleum chemicals ("TCL/TAL Plus"), was proposed as an initial list of contaminants for which SCOs might be developed (see Table 4.1-1, "Target Compound List/Target Analyte List Plus"). The TCL/TAL Plus list of proposed target contaminants includes chemicals for which media at contaminated sites are routinely analyzed and are in the following categories: volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs) including polycyclic aromatic hydrocarbons (PAHs), pesticides, polychlorinated biphenyls (PCBs), and inorganic chemicals. A subset of the chemicals in Table 4.1-1 was prioritized for developing SCOs. The subset list (see Table 4.1-2), called "Initial Soil Cleanup Objectives Priority List," consists of contaminants commonly found at sites based on staff experience and was developed in consideration of US EPA's "Common Chemicals Found at Superfund Sites" (www.epa.gov/superfund/resources/chemicals.htm). SCOs for chemicals in Table 4.1-1 that are not in Table 4.1-2 will be developed on an as needed basis.

The proposed lists of compounds (along with other aspects of the process for developing SCOs) were provided for public comment in spring 2004. In addition, three public meetings were held at different locations within the state. Comments were received suggesting that a number of individual contaminants or groups of contaminants (e.g., total volatile organic compounds) be added to the SCO Target Chemical List and/or the SCO Priority List.

The comments on the Target Chemical List focused on certain requested additions as summarized below:

- several individual compounds, including additional pesticides, additional PAHs, additional inorganic compounds, organic forms of inorganics, radionuclides and others;
- other isomers of chemicals listed (i.e., isomers of chlordane other than alpha & gamma);
- some groups of chemicals such as total VOCs, total semi-VOCs, total petroleum hydrocarbons.

We considered the comments and modified the initial list of chemicals for the development of SCOs using the following criteria:

- the chemical is listed on typical analytical scans,
- the chemical is typically found at sites, and
- the chemical is typically found in soils.

Compounds added to the priority list include acenaphthene, acenaphthylene, barium, beryllium, 2-methylphenol, 3-methylphenol, 4-methylphenol, pyrene, selenium, silver, and 2-(2,4,5 – trichlorophenoxy) propionic acid (also known as Silvex). We deleted 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and equivalents from the priority list because, in the State's experience, dioxins are typically not found at sites. The US EPA's National Center for Environmental Assessment recently released its revised draft human health assessment for 2,3,7,8-TCDD (US EPA, 2003). This document is a draft intended for review by the National Academy of Sciences and does not represent final agency positions. If dioxins are identified as contaminants of concern at brownfield sites, the information in the US EPA

document and other relevant information will be considered in the selection of remedial programs. For the reasons described in Section 6.0 (Polychlorinated Biphenyls), Aroclors were deleted from the priority list and polychlorinated biphenyls (PCBs) were added. Table 4.1-3 is the updated priority list for which SCOs are developed in this document. Two compounds were added to the to the TCL/TAL Plus list (Table 4.1-1), in addition to the priority list, they were; 3-methylphenol, and 2-(2,4,5–trichlorophenoxy) propionic acid. The other compounds added to the priority list were already included on to the TCL/TAL Plus list (Table 4.1-1).

Some of the recommendations were not included explicitly in the priority target list but were considered in other aspects of the development of SCOs. For instance, chlordane, endosulfan and endrin are addressed by an approach described in the section on systemic health effects from exposure to mixtures (Section 5.1.5).

References

US EPA (United States Environmental Protection Agency). 2003. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds. Part III: Integrated Summary and Risk Characterization for 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds. Draft. Washington, DC: Office of Research and Development. Available:
www.epa.gov/ncea/dioxin.

Table 4.1-1. Target Compound List/Target Analyte List Plus.

Volatile Organic Compounds	CAS Number	Volatile Organic Compounds	CAS Number
Acetone	67-64-1	1,3-Dichloropropene (trans)	10061-02-6
Benzene	71-43-2	Dibromochloromethane	124-48-1
Bromodichloromethane	75-27-4	Dichlorodifluoromethane	75-71-8
Bromoform	75-25-2	1,4-Dioxane (1)	123-91-1
Bromomethane	74-83-9	Ethylbenzene	100-41-4
2-Butanone (methyl ethyl ketone)	78-93-3	2-Hexanone	591-78-6
n-Butylbenzene (1)	104-51-8	Isopropylbenzene (cumene)	98-82-8
sec-Butylbenzene (1)	135-98-8	n-Propylbenzene(1)	103-65-1
tert-Butylbenzene (1)	98-06-6	Methyl acetate	79-20-9
Carbon Disulfide	75-15-0	Methyl tert-butyl ether	1634-04-4
Carbon Tetrachloride	56-23-5	Methylcyclohexane	108-87-2
Chlorobenzene	108-90-7	4-Methyl-2-pentanone	108-10-1
Chloroethane	75-00-3	Methylene chloride	75-09-2
Chloroform	67-66-3	Styrene	100-42-5
Chloromethane	74-87-3	1,1,2,2-Tetrachloroethane	79-34-5
Cyclohexane	110-82-7	Tetrachloroethene	127-18-4
1,2-Dibromo-3-chloropropane	96-12-8	Toluene	108-88-3
1,2-Dibromoethane	106-93-4	1,2,4-Trichlorobenzene	120-82-1
1,2-Dichlorobenzene	95-50-1	1,1,1-Trichloroethane	71-55-6
1,3-Dichlorobenzene	541-73-1	1,1,2-Trichloroethane	79-00-5
1,4-Dichlorobenzene	106-46-7	Trichloroethene	79-01-6
1,1-Dichloroethane	75-34-3	Trichlorofluoromethane	75-69-4
1,2-Dichloroethane	107-06-2	1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1
1,1-Dichloroethene	75-35-4	1,2,4-Trimethylbenzene (1)	95-63-6
1,2-Dichloroethene (cis)	156-59-2	1,3,5-Trimethylbenzene (1)	108-67-8
1,2-Dichloroethene (trans)	156-60-5	Vinyl chloride	75-01-4
1,2-Dichloropropane	78-87-5	Xylenes	1330-20-7
1,3-Dichloropropene (cis)	10061-01-5		

Table 4.1-1. Target Compound List/Target Analyte List Plus (continued).

Semivolatile Organic Compounds	CAS Number	Semivolatile Organic Compounds	CAS Number
Acenaphthene	83-32-9	2,4-Dinitrophenol	51-28-5
Acenaphthylene	208-96-8	2,4-Dinitrotoluene	121-14-2
Anthracene	120-12-7	2,6-Dinitrotoluene	606-20-2
Benz[a]anthracene	56-55-3	Di-n-octyl phthalate	117-84-0
Benzo[a]pyrene	50-32-8	Fluoranthene	206-44-0
Benzo[b]fluoranthene	205-99-2	Fluorene	86-73-7
Benzo[g,h,i]perylene	191-24-2	Hexachloroethane	67-72-1
Benzo[k]fluoranthene	207-08-9	Hexachlorobenzene	118-74-1
Bis(2-Chloroethoxy) methane	111-91-1	Hexachlorobutadiene	87-68-3
Bis(2-Chloroethyl) ether	111-44-4	Hexachlorocyclopentadiene	77-47-4
Bis(2-Ethylhexyl)phthalate	117-81-7	Indeno[1,2,3-cd]pyrene	193-39-5
4-Bromophenyl phenyl ether	101-55-3	Isophorone	78-59-1
Butyl benzyl phthalate	85-68-7	2-Methylnaphthalene	91-57-6
Carbazole	86-74-8	2-Methylphenol	95-48-7
4-Chloroaniline	106-47-8	4-Methylphenol	106-44-5
4-Chloro-3-methylphenol	59-50-7	Naphthalene	91-20-3
2-Chloronaphthalene	91-58-7	2-Nitroaniline	88-74-4
2-Chlorophenol	95-57-8	3-Nitroaniline	99-09-2
4-Chlorophenyl phenyl ether	7005-72-3	4-Nitroaniline	100-01-6
Chrysene	218-01-9	Nitrobenzene	98-95-3
Dibenz[a,h]anthracene	53-70-3	2-Nitrophenol	88-75-5
Dibenzofuran	132-64-9	4-Nitrophenol	100-02-7
1,2-Dichlorobenzene	95-50-1	n-Nitroso-di-n-propylamine	621-64-7
1,3-Dichlorobenzene	541-73-1	n-Nitrosodiphenylamine	86-30-6
1,4-Dichlorobenzene	106-46-7	2,2’-Oxybis(1-chloropropane)	108-60-1
3,3’-Dichlorobenzidine	91-94-1	Pentachlorophenol	87-86-5
2,4-Dichlorophenol	120-83-2	Phenol	108-95-2
Diethylphthalate	84-66-2	Phenanthrene	85-01-8
2,4-Dimethylphenol	105-67-9	Pyrene	129-00-0
Dimethyl phthalate	131-11-3	1,2,4-Trichlorobenzene	120-82-1
Di-n-butyl phthalate	84-74-2	2,4,5-Trichlorophenol	95-95-4
4,6-Dinitro-2-methylphenol	534-52-1	2,4,6-Trichlorophenol	88-06-2

Table 4.1-1. Target Compound List/Target Analyte List Plus (continued).

Pesticides/Aroclors	CAS Number	Pesticides/Aroclors	CAS Number
Aldrin	309-00-2	Delta-hexachlorocyclohexane	319-86-8
Alpha-hexachlorocyclohexane	319-84-6	Dieldrin	60-57-1
Alpha-chlordane	5103-71-9	Endosulfan I	959-98-8
Aroclor 1016	12674-11-2	Endosulfan II	33213-65-9
Aroclor 1221	11104-28-2	Endosulfan sulfate	1031-07-8
Aroclor 1232	11141-16-5	Endrin	72-20-8
Aroclor 1242	53469-21-9	Endrin aldehyde	7421-93-4
Aroclor 1248	12672-29-6	Endrin ketone	53494-70-5
Aroclor 1254	11097-69-1	Gamma-hexachlorocyclohexane (lindane)	58-89-9
Aroclor 1260	11096-82-5	Gamma-chlordane	57-74-9
Beta-hexachlorocyclohexane	319-85-7	Heptachlor	76-44-8
4,4'-DDD	72-54-8	Heptachlor epoxide	1024-57-3
4,4'-DDE	72-55-9	Methoxychlor	72-43-5
4,4'-DDT	50-29-3	Toxaphene	8001-35-2

Other	CAS Number		
2,3,7,8-TCDD (dioxin) equivalents [(1)]	1746-01-06		

Inorganics	CAS Number	Inorganics	CAS Number
Aluminum	7429-90-5	Lead	7439-92-1
Antimony	7440-36-0	Magnesium	7439-95-4
Arsenic	7440-38-2	Manganese	7439-96-5
Barium	7440-39-3	Mercury	7439-97-6
Beryllium	7440-41-7	Nickel	7440-02-0
Cadmium	7440-43-9	Potassium	7440-09-7
Calcium	7440-70-2	Selenium	7782-49-2
Chromium	7440-47-3	Silver	7440-22-4
Cobalt	7440-48-4	Sodium	7440-23-5
Copper	7440-50-8	Thallium	7440-28-0
Cyanide		Vanadium	7440-62-2
Iron	7439-89-6	Zinc	7440-66-6

[(1)] Not included on TCL or TAL

Table 4.1-2. Initial Soil Cleanup Objectives Priority List.

Volatile Organic Compounds	CAS Number	Volatile Organic Compounds	CAS Number
Acetone	67-64-1	1,2-Dichloroethene (cis)	156-59-2
Benzene	71-43-2	1,2-Dichloroethene (trans)	156-60-5
2-Butanone (methyl ethyl ketone)	78-93-3	1,4-Dioxane [(1)]	123-91-1
n-Butylbenzene	104-51-8	Ethylbenzene	100-41-4
sec-Butylbenzene	135-98-8	Methylene chloride	75-09-2
tert-Butylbenzene	98-06-6	Methyl tert-butyl ether	1634-04-4
Carbon tetrachloride	56-23-5	n-Propylbenzene	103-65-1
Chlorobenzene	108-90-7	Tetrachloroethene	127-18-4
Chloroform	67-66-3	Toluene	108-88-3
1,2-Dichlorobenzene	95-50-1	1,1,1-Trichloroethane	71-55-6
1,3-Dichlorobenzene	541-73-1	Trichloroethene	79-01-6
1,4-Dichlorobenzene	106-46-7	1,2,4-Trimethylbenzene [(1)]	95-63-6
1,1-Dichloroethane	75-34-3	1,3,5-Trimethylbenzene [(1)]	108-67-8
1,2-Dichloroethane	107-06-2	Vinyl chloride	75-01-4
1,1-Dichloroethene	75-35-4	Xylenes	1330-20-7

Semi Volatile Organic Compounds	CAS Number	Semi Volatile Organic Compounds	CAS Number
Anthracene	120-12-7	Dibenz[a,h]anthracene	53-70-3
Benz[a]anthracene	56-55-3	Fluoranthene	206-44-0
Benzo[b]fluoranthene	205-99-2	Fluorene	86-73-7
Benzo[k]fluoranthene	207-08-9	Hexachlorobenzene	118-74-1
Benzo[g,h,i]perylene	191-24-2	Indeno[1,2,3-cd]pyrene	193-39-5
Benzo[a]pyrene	50-32-8	Naphthalene	91-20-3
Chrysene	218-01-9	Pentachlorophenol	87-86-5
Dibenzofuran	132-64-9	Phenol	108-95-2

Table 4.1-2. Initial Soil Cleanup Objectives Priority List. (continued).

Pesticides	CAS Number	Pesticides	CAS Number
Aldrin	309-00-2	Dieldrin	60-57-1
Alpha-hexachlorocyclohexane	319-84-6	Endosulfan I	959-98-8
Beta- hexachlorocyclohexane	319-85-7	Endosulfan II	33213-65-9
Chlordane (alpha)	5103-71-9	Endosulfan sulfate	1031-07-8
4,4'-DDD	72-54-8	Endrin	72-20-8
4,4'-DDE	72-55-9	Heptachlor	76-44-8
4,4'-DDT	50-29-3	Gamma-hexachlorocyclohexane (lindane)	58-89-9
Delta-hexachlorocyclohexane	319-86-8		

PCBs	CAS Number	PCBs	CAS Number
Aroclor 1016	12674-11-2	Aroclor 1248	12672-29-6
Aroclor 1221	11104-28-2	Aroclor 1254	11097-69-1
Aroclor 1232	11141-16-5	Aroclor 1260	11096-82-5
Aroclor 1242	53469-21-9		

Other	CAS Number		
2,3,7,8-TCDD (dioxin) & equivalents [(1)]	1746-01-6		

Inorganics	CAS Number	Inorganics	CAS Number
Arsenic	7440-38-2	Lead	7439-92-1
Cadmium	7440-43-9	Manganese	7439-96-5
Chromium III	16065-83-1	Mercury	7439-97-6
Chromium VI	18540-29-9	Nickel	7440-02-0
Copper	7440-50-8	Zinc	7440-66-6
Cyanide			

[(1)] Not included on TCL or TAL

Table 4.1-3. Soil Cleanup Objectives Priority List.

Volatile Organic Compounds	CAS Number	Volatile Organic Compounds	CAS Number
Acetone	67-64-1	1,2-Dichloroethene (cis)	156-59-2
Benzene	71-43-2	1,2-Dichloroethene (trans)	156-60-5
2-Butanone (methyl ethyl ketone)	78-93-3	1,4-Dioxane	123-91-1
n-Butylbenzene	104-51-8	Ethylbenzene	100-41-4
sec-Butylbenzene	135-98-8	Methylene chloride	75-09-2
tert-Butylbenzene	98-06-6	Methyl tert-butyl ether	1634-04-4
Carbon tetrachloride	56-23-5	n-Propylbenzene	103-65-1
Chlorobenzene	108-90-7	Tetrachloroethene	127-18-4
Chloroform	67-66-3	Toluene	108-88-3
1,2-Dichlorobenzene	95-50-1	1,1,1-Trichloroethane	71-55-6
1,3-Dichlorobenzene	541-73-1	Trichloroethene	79-01-6
1,4-Dichlorobenzene	106-46-7	1,2,4-Trimethylbenzene	95-63-6
1,1-Dichloroethane	75-34-3	1,3,5-Trimethylbenzene	108-67-8
1,2-Dichloroethane	107-06-2	Vinyl chloride	75-01-4
1,1-Dichloroethene	75-35-4	Xylenes	1330-20-7

Semi Volatile Organic Compounds	CAS Number	Semi Volatile Organic Compounds	CAS Number
Acenaphthene	83-32-9	Fluorene	86-73-7
Acenaphthylene	208-96-8	Hexachlorobenzene	118-74-1
Anthracene	120-12-7	Indeno[1,2,3-cd]pyrene	193-39-5
Benz[a]anthracene	56-55-3	2-Methylphenol	95-48-7
Benzo[b]fluoranthene	205-99-2	3-Methylphenol	108-39-4
Benzo[k]fluoranthene	207-08-9	4-Methylphenol	106-44-5
Benzo[g,h,i]perylene	191-24-2	Naphthalene	91-20-3
Benzo[a]pyrene	50-32-8	Pentachlorophenol	87-86-5
Chrysene	218-01-9	Phenanthrene	85-01-8
Dibenzofuran	132-64-9	Phenol	108-95-2
Dibenz[a,h]anthracene	53-70-3	Pyrene	129-00-0
Fluoranthene	206-44-0		

Table 4.1-3. Soil Cleanup Objectives Priority List (continued).

Pesticides/PCBs	CAS Number	Pesticides/PCBs	CAS Number
Aldrin	309-00-2	Dieldrin	60-57-1
Alpha-chlordane	5103-71-9	Endosulfan I	959-98-8
Alpha-hexachlorocyclohexane	319-84-6	Endosulfan II	33213-65-9
Beta-hexachlorocyclohexane	319-85-7	Endosulfan sulfate	1031-07-8
4,4'-DDD	72-54-8	Endrin	72-20-8
4,4'-DDE	72-55-9	Heptachlor	76-44-8
4,4'-DDT	50-29-3	Gamma-hexachlorocyclohexane (lindane)	58-89-9
Delta-hexachlorocyclohexane	319-86-8	2-(2,4,5–trichlorophenoxy) propionic acid	93-72-1
		Polychlorinated biphenyls	1336-36-3

Inorganics	CAS Number[(1)]	Inorganics	CAS Number
Arsenic		Lead	
Barium		Manganese	
Beryllium		Mercury (elemental)	7439-97-6
Cadmium		Mercury (inorganic salts)	
Chromium III		Nickel	
Chromium VI		Selenium	
Copper		Silver	
Cyanide		Zinc	

[(1)] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

5.0 Protection of Human Health

5.1 Toxicity Assessment

5.1.1 Toxicity Values for Systemic Health Effects (Non-Cancer and Cancer Human Health Effects)

Non-cancer and cancer toxicity values are necessary to calculate contaminant-specific SCOs. The toxicity values used for non-cancer effects are the reference dose (to evaluate the oral, dermal, and in certain circumstances inhalation exposures) and reference concentration (to evaluate inhalation exposures). Two toxicity values were used to calculate the excess lifetime cancer risk from contaminant exposures. A cancer potency factor was used to estimate the lifetime cancer risk from oral and dermal exposures (and on occasion, from inhalation exposures). An air unit risk was used to estimate the lifetime cancer risk from inhalation exposures.

Toxicity values for many of the contaminants have been derived by state, national, or international regulatory or advisory public health organizations ("authoritative bodies"). The available toxicity values from these authoritative bodies were evaluated, and a reference dose, and when necessary and available, a reference concentration, cancer potency factor, and air unit risk were selected for calculating SCOs based on chronic toxicity data for non-cancer and cancer health effects and chronic exposure scenarios (see Section 5.3 Calculation of Chronic Human Health-based Soil Cleanup Objectives).

5.1.1.1 Established Methods for the Derivation of Contaminant-Specific Toxicity Values

At high enough doses, all chemicals cause non-cancer effects. Headaches, liver, or kidney damage, skin or eye irritation, allergic reactions, birth defects, infertility, and nerve damage are examples of non-cancer effects. Only some chemicals cause cancer effects, including the formation of malignant tumors. Once the non-cancer effects and cancer effects (if present) of a chemical are identified, the next step in the risk assessment process is dose-response assessment

to identify a toxicity value for use in evaluating chemical exposures. Traditionally, the risk assessment methods used to derive toxicity values for non-cancer health risks (e.g., reference doses or reference concentrations) have differed from the methods used to derive toxicity values for cancer risks (e.g., cancer potency factors and air unit risks).

5.1.1.2 General Methods for the Derivation of Non-Cancer Toxicity Values

The reference dose is the toxicity value used to evaluate non-cancer systemic health risks from chronic exposure to contaminants by the oral and dermal routes. The reference dose is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups such as children, the sick, and the elderly) that is likely to be without an appreciable risk of deleterious effects during a lifetime (US EPA, 2002, 2004a; ATSDR, 1996). A reference concentration is the toxicity value used to evaluate the potential for non-cancer systemic health risks from chronic inhalation exposure to contaminants. The reference concentration is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups such as children, the sick and the elderly) that is likely to be without an appreciable risk of deleterious effects during a lifetime (US EPA, 1994, 2002, 2004a).

Under certain circumstances, a reference dose can be used to evaluate the exposures from the oral, dermal, and inhalation routes. Most typically, this is done when the effects of inhalation do not differ (or are not expected to differ) from the effects of ingestion, and a reference concentration is not available.

Reference doses and reference concentrations are derived from a point-of-departure, which is a point on a dose-response curve for an effect of a chemical that is within or near the range of experimental or observational data for the effect. The point-of-departure is the starting point for the extrapolation from the range-of-observation in human or animal studies to the human doses that are likely to be without appreciable risk of non-cancer health effects. Typically, the point-of-departure is based on the critical effect (i.e., the first adverse effect of exposures, or its known precursor, that occurs in the most sensitive species) from the critical study (i.e., the study that

contributes most significantly to the qualitative and quantitative assessment of risk). The point-of-departure can be a no-observed-effect level (NOEL), lowest-observed-effect level (LOEL), or the lower confidence level on a dose or concentration that is associated with an estimated level of excess risk for an effect (i.e., benchmark dose (BMDL) or concentration (BMCL)).

In a typical derivation, scientists review the animal and human data on a chemical, choose an appropriate point-of-departure, and then divide the selected point-of-departure by uncertainty factors to estimate the reference dose or reference concentration. Each uncertainty factor generally has a value of 3 or 10, and compensates for variation or areas of uncertainty in the toxicity data for the chemical. Typically, several uncertainty factors are used in the derivation of a reference dose or reference concentration, and are intended to account for:

- the variation in sensitivity among the members of the human population (intra-species uncertainty);
- the uncertainty in extrapolating animal data to humans (inter-species uncertainty);
- the uncertainty in extrapolating from data obtained in a study with less-than-chronic exposure to chronic exposure (subchronic uncertainty);
- the uncertainty in extrapolating from a LOEL rather than from a NOEL;
- the uncertainty associated with extrapolation of results from adult humans or animals to children; and
- the uncertainty about the completeness of the database on the toxicity of the chemical.

This general approach for deriving non-cancer toxicity values has been used by the National Academy of Sciences and federal agencies such as the US EPA, the United States Food and Drug Administration (US FDA), and the Agency for Toxic Substances and Disease Registry (ATSDR). The basic procedures are described in US EPA documents (1994, 2000b,c, 2002) and ATSDR (1996).

In deriving non-cancer toxicity values, an uncertainty factor of 10 is generally used to account for variation in sensitivity within the human population. Recently, US EPA workgroups have indicated that a default uncertainty factor of 10 for human variation, including the variation

among adults and children, is adequate in most cases (US EPA, 1999a, 2002). They based their conclusion on an analysis of the data on differences among adults and children, and on the realization that some of the uncertainty associated with human variation can be addressed by the use of an uncertainty factor for database deficiency (or a modifying factor). These factors are used when there are concerns about the adequacy of the database to identify effects in sensitive populations.

5.1.1.3 Selection of Toxicity Values for Non-Cancer Effects

Several authoritative health and environmental agencies (e.g., US EPA, ATSDR, World Health Organization (WHO), Health Canada, NYS DOH, NYS DEC, and California Environmental Protection Agency (CA EPA)) have derived reference doses or concentrations (or equivalent values with different names) for use in evaluating non-cancer human health risks associated with chronic exposure to chemical contaminants, using the same general approach outlined above. In many cases, the derivations have been reviewed by scientists from within and perhaps, outside the agency. Table 5.1.1-1 provides a list of the authoritative bodies (and Internet addresses) that were the primary sources used to identify toxicity values for the priority contaminants. In some cases, if toxicity values were available from other authoritative bodies, these may have been considered as well. Each chemical factsheet (see Appendix A) identifies all of the specific authoritative bodies checked for each specific compound.

The selection of toxicity values for use in deriving contaminant-specific SCOs protective of non-cancer health effects involved two steps: (1) information on the toxicity value and its derivation was collected and summarized in a fact sheet (see Appendix A); and (2) the values were compared and evaluated using a variety of criteria to select a value deemed the most appropriate for use in deriving SCOs for the contaminant. These criteria focus on the scientific soundness of the derivation. The primary criteria (i.e., those that substantially affect the level of confidence in the toxicity value) that were used in the evaluation included:

- scientific quality of the critical study or studies (study on which the toxicity value is based); examples of factors considered include number of dose groups, appropriate controls,

appropriate dose range, sample size, study duration, excess mortality, appropriate interpretation of dose-response information, whether or not adequate human epidemiological data were available, and if so, were exposure data adequate, and were bias and confounding adequately controlled;

- choice of the critical health effect (the effect in the critical study that was used to derive the toxicity value);
- route of exposure in the critical study;
- method used to adjust experimental or observational exposures to the daily (oral) or continuous (inhalation) exposures assumed in the derivation of reference dose or concentration;
- magnitude of the total uncertainty factor in relation to data gaps, uncertainties, and variability; and
- confidence in the toxicity value in view of data collected after its derivation.

Additional secondary criteria that were used in the evaluation included:

- method used to extrapolate from high doses to low doses; and
- data on mode-of-action for the toxic effect.

The selected non-cancer toxicity values are found in Table 5.1.1-2, and brief rationales for each selection are provided in the fact sheets in Appendix A.

5.1.1.4 General Method for the Derivation of Cancer Toxicity Values

The toxicity values most commonly used to evaluate the potential for cancer health risks from chronic exposure to contaminants are cancer potency factors for oral and dermal exposures and air unit risks for inhalation exposures. Both cancer potency factors and air unit risks are based on the relationship between exposure (lifetime average daily dose as milligram per kilogram of body weight per day (mg/kg/day) or air concentration as microgram per cubic meter of air (mcg/m^3)) and response (excess lifetime cancer risk). The cancer potency factor is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to a contaminant

at a dose of 1 mg/kg/day. The air unit risk is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to a contaminant at a concentration of 1 mcg/m^3.

At low exposures, the relationship between exposure and risk is assumed to be linear and described by a straight line that starts at zero excess risk and zero dose (i.e., the origin). The cancer potency factor or the unit risk is an upper-bound estimate of excess risk per increment of exposure that is used to estimate excess risks for different exposure levels. The excess cancer risk associated with any lifetime average daily dose or air concentration is estimated by multiplying the dose by the cancer potency factor (risk = slope x dose) or the air concentration by the air unit risk (risk = slope x concentration). The cancer potency factor and the unit risk are also used to calculate the human lifetime average daily dose or air concentration that is associated with a selected excess lifetime human cancer risk.

Most regulatory agencies, including the US EPA (2005a), CA EPA (1999, 2002), and the NYS DEC ambient water (NYS DEC, 1999) and air programs (NYS DEC, 1997) follow the same general methods for deriving cancer potency factors and air unit risks. The methods were first described in the carcinogen risk assessment guidelines published by the US EPA in 1986 (US EPA, 1986). More recently, the US EPA (1996, 1999b, and 2005a) has published a series of updated carcinogen risk assessment guidelines, which provide default recommendations when estimating human risks from human or animal studies. In most cases, human data are inadequate for use in dose-response assessment and most cancer potency factors and air unit risks are based on results from animal studies. Thus, the default recommendations for use with animal studies are briefly described below. Generally, the method for estimating human cancer risks from the results of animal studies contains four important elements.

1. Data Used in Extrapolation

The dose-response data in animal studies are used as the basis for the cancer potency factor if human dose-response data are inadequate or not available. Factors considered in evaluating and choosing among various animal dose-response datasets for the purpose of deriving cancer potency factors or air unit risks include route, duration and timing of exposure, species, strain,

tumor types and sites, nature and severity of effects, pharmacokinetics, mode of action, study quality, and statistical significance.

2. Method for High to Low Dose Extrapolation

The recommended starting point for the high-to-low dose extrapolation is the point-of-departure for cancer effects (US EPA, 2005a). The point-of-departure is frequently the LED_{10} (lower bound on estimated dose expressed as mg/kg/day) or LEC_{10} (lower bound on estimated air concentration), which is the 95% lower confidence limit on the daily dose or air concentration, respectively, associated with a 10% excess risk for cancer effects in animals adjusted for background risk. This or a similar value (e.g., LED_{05} or LEC_{05}) is frequently close to the range of doses or air concentrations used in animal studies, and is typically estimated using appropriate dose-response models. However, prior to the 1996 revision of the 1986 guidelines for carcinogen risk assessment, many of the derivations completed by federal and state agencies did not calculate the point-of-departure, but directly calculated the lifetime average daily dose or air concentration associated with an excess lifetime cancer risk of one-in-one million. This dose or air concentration was typically estimated using a mathematical model (i.e., the linearized multistage model) that was recommended by the US EPA (1986) as both biologically plausible and health-protective.

3. Method for Animal to Human Extrapolation

The extrapolation of results from animal studies to humans involves two adjustments. First, a dosimetric adjustment compensates for differences in pharmacokinetics between animals and humans. The preferred method for making this adjustment uses physiologically-based pharmacokinetic (PB-PK) models (US EPA, 2005a). An animal PB-PK model is used to determine the critical animal internal dose at the point-of-departure (e.g., an LED_{10} or LEC_{10}), and a human PB-PK model is then used to back-calculate the dose or concentration at which humans have the same critical internal dose that was estimated for animals at the point-of-departure. This exposure level is the human equivalent dose or concentration. The second adjustment in extrapolating from animals to humans accounts for differences between the two

species in their biological response to the same internal dose (i.e., sensitivity). This is a pharmacodynamic adjustment, and the nature of this adjustment is determined on a case-by-case basis or the use of pharmacodynamic models (US EPA, 2005a). For almost all chemicals, however, pharmacokinetic and pharmacodynamic data are unavailable, and default methods to compensate for pharmacokinetic and pharmacodynamic differences between animals and humans have been recommended for animal to human extrapolation.

The recommended default method for converting an oral animal dose to a equipotent oral human doses (i.e., the dose that has the same lifetime excess cancer risk) is calculated by multiplying the animal dose by the animal-to-human body-weight ratio raised to the 0.25 power (US EPA, 1992, 2005a). The equation can be used to calculate the human dose associated with the excess risk at the point-of-departure (recent derivations) or the human dose associated with an excess risk of one-in-one million (older derivations). This method of interspecies extrapolation has replaced an older default method based on body surface area (US EPA, 1986, 1992), where the human equipotent oral dose is estimated by multiplying the animal dose by the animal-to-human body-weight ratio raised to the 0.33 power.

The recommended default method for estimating an air concentration at which the human lifetime cancer risk is equal to the animal lifetime cancer risk at the point-of-departure (e.g., LEC_{10}) involves separate adjustments for pharmacokinetics and pharmacodynamics (US EPA, 2005a). Almost all of the priority contaminants that pose an inhalation cancer risk are volatile organic chemicals, and if they cause cancer, it is at sites other than the respiratory tract. For these chemicals, the recommended method for making a dosimetric adjustment is the same as the US EPA's (1994) method for making dosimetric adjustment when deriving reference concentrations. In this step, the human equivalent air concentration is estimated by multiplying the point-of-departure (e.g., LEC_{10}) derived from an animal study by the ratio of animal to human blood/air partition coefficients only if the ratio is less than one. If the ratio is greater than one, or if data are unavailable, then a default ratio of one is used. The default ratio is typically used, and thus, in such cases, the human equivalent concentration equals the concentration at point-of-departure (e.g., LEC_{10}) derived from an animal study (i.e., the adjustment factor is one). In the second step, the pharmacodynamic adjustment factor is applied to the human equivalent

concentration. An adjustment factor of one assumes that tissues of different species with similar internal doses for a lifetime should be assumed to have the same lifetime risk of cancer (US EPA, 1992).

4. Calculation of the Cancer Potency Factor or Unit Risk

The recommended method for calculating the cancer potency factor or air unit risk is to divide the level of excess risk associated with the point-of-departure by the human dose at the point-of-departure or by the human air concentration at the point-of-departure (US EPA, 2005a). In older cancer risk assessments, the cancer potency factor or unit risk was estimated by using the multistage linearized model to estimate the human dose or the air concentration associated with a lifetime excess risk of one-in-one million, and then dividing the excess risk of one-in-one million by the human dose or the air concentration. This method typically provides a similar estimate of the cancer potency factor or the air unit risk as the currently recommended methods using a point-of-departure (e.g., US EPA, 2004b).

5.1.1.5 Selection of Toxicity Values for Cancer Effects

Several authoritative health and environmental agencies (e.g., US EPA, NYS DOH, NYS DEC, and CA EPA) have derived cancer potency factors and air unit risks for use in evaluating the potential human cancer risks associated with chronic exposure to chemical contaminants using the same general approach outlined above. In many cases, the derivations have been reviewed by scientists from within and perhaps, outside the agency. Table 5.1.1-1 provides a list of authoritative bodies (and Internet addresses) that were the primary sources used to identify toxicity values for the priority contaminants. In some cases, if toxicity values were available from other authoritative bodies, these may have been considered as well. The selection of cancer toxicity values (cancer potency factors and air unit risks) for deriving the SCOs based on cancer effects was performed using the same two-step process used to select non-cancer toxicity values (see Section 5.1.1.3). However, criteria to determine which values were selected for deriving the SCOs are slightly different than those used for non-cancer effects.

Primary criteria that were used in the evaluation include:

- scientific quality of the critical study or studies (study on which the toxicity value is based); examples of factors considered include number of dose groups, appropriate controls, appropriate dose range, sample size, study duration, excess mortality, appropriate interpretation of dose-response information, whether or not adequate human epidemiological data were available, and if so, were exposure data adequate, and were bias and confounding adequately controlled;
- choice of the critical health effect (the effect in the critical study that was used to derive the toxicity value);
- method used to adjust experimental or observational exposures to the daily (oral) or continuous (inhalation) exposures assumed in the derivation of a cancer potency factor or air unit risk;
- route of exposure in the critical study; and
- confidence in the toxicity value in view of data collected after its derivation.

Additional secondary criteria that were used in the evaluation include:

- method used to extrapolate from high doses to low doses and from animals to humans;
- method used to estimate the cancer potency factor; and
- data on mode-of-action for the toxic effect.

The cancer toxicity values selected for derivation of SCOs are in Table 5.1.1-2. As with the non-cancer effects, a fact sheet containing a summary of the available cancer potency factors and air unit risks, the selected value, and a brief rationale in support of its selection for each contaminant is found in Appendix A.

5.1.1.6 Adjustments to Toxicity Values for Cancer Effects Based on the Potentially Increased Sensitivity of Children to the Carcinogenic Effects of Early-Life Exposures

Introduction

Children may be at a greater carcinogenic risk from chemical exposures than are adults, and in 2005, the US EPA released its Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (US EPA, 2005b). This document provides guidance on how to adjust oral cancer potency factors or inhalation unit risks to compensate for potentially increased sensitivity of children to early-life exposures. The guidance recommends that the adjustments should be only applied to potency factors or unit risks for carcinogens acting through a mutagenic mode-of-action. When the mode-of-action cannot be established, the US EPA recommends the use of linear, low-dose extrapolation, without further adjustment. When a mode-of-action other than mutagenicity is established, US EPA recommends the use of either linear or non-linear low-dose extrapolation, dependent on the data, but without further adjustment.

SCOs based on the cancer effects of a chemical are derived using toxicity values (oral cancer potency factors or inhalation unit risk) obtained using linear, low-dose extrapolation. This choice did not depend on a conclusion by NYS or an authoritative body that the chemical caused cancer by a mutagenic mode-of-action. Thus, the first step in the application of the US EPA (2005b) cancer risk guidelines in the development of SCOs would be a determination of whether each carcinogen has a mutagenic mode-of-action.

The US EPA did not classify carcinogens into those that have a mutagenic mode-of-action and those that do not have a mutagenic mode-of-action, rather the Agency identified the kind of information that should be evaluated using a weight-of-evidence approach to determine if a carcinogen has a mutagenic mode-of-action. This information includes results of short-term test of genetic toxicity, metabolic profiles, physicochemical properties and structure-activity relationship (SAR). Important findings that would increase the weight of evidence that a carcinogen has a mutagenic mode-of-action include evidence that the carcinogen or a metabolite is DNA reactive and/or has the ability to bind to DNA, evidence that the carcinogen has the ability to produce effects in multiple test systems for different genetic endpoints, particularly gene mutations and structural chromosome aberrations *in vivo* and *in vitro*, and evidence that the

carcinogen has similar properties and SAR to carcinogens with an established mutagenic mode-of-action.

Selection of Chemicals for Adjustments

There are 38 carcinogens in the priority list of chemicals. One of these chemicals (vinyl chloride) has a mutagenic mode-of-action (US EPA, 2000c), and the oral cancer potency factor and inhalation unit risk for vinyl chloride (Table 5.1.1-2) are values that include the increased sensitivity of children to the carcinogenic effects of early-life exposures. In addition, the US EPA (2005b) document characterized a limited number of chemicals as acting primarily by a mutagenic mode-of-action or primarily by a non-mutagenic mode-of-action. Only one Priority List chemical (benzo[a]pyrene) was identified as acting by a mutagenic mode-of-action. Only two Priority List chemicals (DDT, dieldrin) were identified as acting by a non-mutagenic mode-of-action.

Thus, the oral cancer potency factor and inhalation unit risk for benzo[a]pyrene (Table 5.1.1-2) were adjusted to compensate for the increased sensitivity of children to the carcinogenic effects of early-life exposures. This adjustment is also applied to the oral cancer potency factors and inhalation unit risks for the six other carcinogenic PAHs (benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, chrysene and indeno[1,2,3-cd]pyrene) that are based on the benzo[a]pyrene toxicity values.

Consistent with the US EPA guidance, the oral cancer potency factors and inhalation unit risks DDT and dieldrin (Table 5.1.1-2) were not adjusted to compensate for the increased sensitivity of children to the carcinogenic effects of early-life exposures to these compounds because they are likely to act by a non-mutagenic mode-of-action.

The Department will consider making adjustments for the remaining carcinogenic priority chemicals, consistent with the time frame specified by the legislation (S 27-1415, 6(c)), if US EPA or other authoritative bodies issues further guidance of the mode-of-action of carcinogens or if the Department performs evaluations for additional carcinogens.

Adjustment to the Oral Cancer Potency Factors and Inhalation Unit Risks for Benzo[a]pyrene and Six Other Carcinogenic PAHs

Ideally, potency adjustments should be based on chemical-specific data that directly assess the differential cancer susceptibilities of childhood and adult exposures (e.g., US EPA (2000c) assessment of vinyl chloride). In the absence of such data, the US EPA (2005b) recommends a default approach using potency estimates (i.e., oral cancer potency factors or inhalation unit risks) and adjustments to those potency estimates (called age-dependent adjustment factors).

The age-dependent adjustment factors are:

- a 10–fold adjustment to the oral cancer potency factor or inhalation unit risk for exposures before 2 years of age (i.e., spanning a 2–year time interval from the first day of birth up until a child's second birthday);

- a 3–fold adjustment to the oral cancer potency factor or inhalation unit risk for exposures between 2 and <16 years of age (i.e., spanning a 14–year time interval from a child's second birthday up until the sixteenth birthday); and

- an adjustment factor of 1 to the oral cancer potency factor or inhalation unit risk for exposures after turning 16 years of age (i.e., no adjustment).

Chemical-specific information on the differential sensitivities of young organisms and adults to the carcinogenic effects of chronic exposures to benzo[a]pyrene was not found, and benzo[a]pyrene-specific adjustment factors could not be derived. Thus, the US EPA (2005b) recommended default age-dependent adjustment factors were used to calculate adjusted oral cancer potency factors and inhalation unit risks for benzo[a]pyrene and the six other carcinogenic PAHs (benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, chrysene and indeno[1,2,3-cd]pyrene) that are based on the toxicity values for benzo[a]pyrene (see Tables 5.1.1-2 and 5.1.5-2). The use of these age-adjusted

toxicity values in the calculation of SCOs is described in Section 5.3 (Calculation of Chronic Human Health-based Soil Cleanup Objectives).

5.1.1.7 Additional Considerations in the Selection of Toxicity Values

The criteria for selecting a toxicity value from those derived by the authoritative bodies were applied as uniformly as possible to all priority contaminants. However, chemicals vary substantially in the quality of their available toxicity data, and in some cases, the available toxicity data for a chemical was not sufficient to allow a toxicity value to be selected based on the identified criteria alone. In these cases, additional toxicity information was considered. This information could include toxicity data on a priority contaminant published in the scientific literature. It also could include data and methods to extrapolate results from one route of exposure to another route of exposure (e.g., oral to inhalation). It could include toxicity data on chemicals that might be used as a surrogate for a priority contaminant. If appropriate, a toxicity value was then selected based on this additional information.

For some contaminants, a route-specific toxicity value could not be derived because route-specific data were not available. For these contaminants, a route-to-route extrapolation was done when three conditions were met: (1) adequate toxicity data for one exposure route were available, but adequate data for another route (usually inhalation) were not available; (2) the toxicity data indicated that the critical effects were systemic; and (3) the structural or pharmacokinetic data (i.e., data on its absorption, distribution, metabolism, and excretion) suggested that it was likely to be absorbed by both exposure routes. The toxicity value already chosen as the basis of an SCO for a different exposure route (most often oral) was used as the basis of the route-to-route extrapolation.

Theoretically, route-to-route extrapolations convert an absorbed dose (in mg/kg/day) from an oral exposure into an equal absorbed dose from an inhalation exposure, or vice versa. For non-cancer effects, the dose of interest is the absorbed dose at the reference dose or at the reference concentration for non-cancer effects. For cancer effects, the dose of interest is the absorbed oral

dose associated with an excess cancer risk of one-in-one-million or the absorbed dose at the air concentration associated with an excess cancer risk of one-in-one-million.

When extrapolating between oral doses expressed in mg/kg/day and inhalation exposures expressed as an air concentration in mcg/m^3, default values for adult daily inhalation rate (20 m^3/day) and adult body weight (70 kg) are used to relate absorbed doses to air concentrations (US EPA, 1997). To convert an oral non-cancer toxicity value (i.e., the reference dose (RfD) in mg/kg/day) to an inhalation reference concentration (RfC) in mcg/m^3, the following relationship is applied:

$$RfC = \frac{RfD(70kg)(1000mcg/mg)}{(20m^3/d)}$$

A RfD (in mg/kg/day) can be obtained from a RfC (in mcg/m^3) by rearranging the equation above to yield:

$$RfD = \frac{RfC(20m^3/d)}{(1000mcg/mg)(70kg)}$$

To convert an oral cancer toxicity value (i.e., the cancer potency factor (CPF) in $(mg/kg/day)^{-1}$) to an air unit risk (UR) in $(mcg/m^3)^{-1}$, the following relationship is applied:

$$UR = \frac{CPF(20m^3/d)}{(70kg)(1000mcg/mg)}$$

The cancer potency factor (in $(mg/kg/d)^{-1}$) can be obtained from an air unit risk (in $(mcg/m^3)^{-1}$) by rearranging the equation above to yield:

$$CPF = \frac{UR(1000mcg/mg)(70kg)}{(20m^3/d)}$$

These default relationships assume the same absorption fraction by both exposure routes. In cases where the relative absorption fraction by the oral and inhalation routes is known, an additional factor is applied to account for absorption differences between the two routes.

In cases where adequate chemical-specific toxicity data and adequate data for a route-to-route extrapolation were both unavailable, toxicity data from structurally related chemicals were considered as the basis for a toxicity value. The structure of a chemical largely determines its pharmacokinetics in the body, and therefore is an important determinant of its toxicity. Chemicals with very similar structures often have similar toxic properties. In cases where toxicity information for a chemical was unavailable, but toxicity data from a structurally similar chemical was available and satisfied the general selection criteria described above, the surrogate toxicity data were considered for use as the toxicity value in lieu of chemical-specific data.

5.1.1.8 Toxicity Values for Inorganic Lead

Non-Cancer

Lead and inorganic lead compounds cause a variety of health effects in humans, and can damage the nervous, cardiovascular, gastrointestinal, hematopoietic, and reproductive systems. The database on lead toxicity is unusual because it contains a large amount of data on dose-response relationships in humans (ATSDR, 1999). Consequently, the degree of uncertainty about the non-cancer human health effects of lead is relatively low compared to almost all other contaminants (US EPA, 2005c). In most studies, however, the measure of dose is an internal one (most commonly, blood lead level or PbB). In addition, most studies cannot attribute blood lead levels to one single route, pathway, or source of exposures or exposures during a limited, defined time. This is because lead can accumulate in the human body, and blood lead at any given time is dependent on current and past exposures to lead. Current exposures (e.g., food, water, air, and soil) are important because absorbed lead goes into the blood before distributing to other parts of the body. Past exposures are important because the body stores absorbed accumulated lead in bones. The lead in bones can be released into the blood under certain circumstances. Thus,

blood lead is considered the most reliable measure of a person's risk of non-cancer health effects from lead.

Experimental studies of the toxicity of lead in animals provide support for observations in humans. Current knowledge of lead pharmacokinetics indicates that toxicity values derived by the application of default risk assessment procedures (e.g., using administered, ingested, or inhaled dose) to animal dose-response data might not accurately estimate the potential risk (US EPA, 2005c). This stems from concerns that an adequate animal model for lead toxicity in humans is not available and because of the difficulty in accounting for pre-existing body burdens of lead (US EPA, 2005c). Moreover, an animal-based analysis would overlook the significant body of toxicological literature on human toxicity and blood lead levels (ATSDR, 1999). Thus, animal data on lead toxicity have not been used by the ATSDR (1999), US EPA (2001, 2005c), or other public health agencies to evaluate the potential human non-cancer health effects of lead exposures. Neither ATSDR (1999), nor the US EPA (2005c), nor other authoritative bodies have proposed or developed a lead reference dose or reference concentration based on animal data.

Public health agencies recognize that the primary population, dose measure, and health concern associated with environmental exposures to lead are children, blood lead levels, and neurotoxicity, respectively (e.g., ATSDR, 1999; FL DEP, 2004; NJ DEP, 2004; MN PCA, 1999; US EPA, 2001; WHO, 1996). Young children are especially vulnerable to the toxic effects of lead for at least two reasons:

(1) Increased Exposures Relative to Adults. Children are likely to be exposed to environmental lead in many more ways than are adults (e.g., more hand-to-mouth activity, more contact with dirt, more mouthing/ingestion of non-food items). Children also have greater food, water, and inhalation rates per unit body weights than do adults. In addition, young children absorb a greater percentage of ingested lead than do adults, and might absorb a greater percentage of inhaled lead than do adults (ATSDR, 1999).

(2) Increased Sensitivity Relative to Adults. For many effects, the lead blood levels that cause toxicity in children are lower than the levels that cause effects in adults, and the effects may be

more severe than those in adults (ATSDR, 1999). This suggests that children are more sensitive to the toxic effects of absorbed lead than adults. The toxicological data on the effects of lead on young children support concern for the increased sensitivity of fetuses, neonates, and infants to the toxicological effects of elevated blood lead levels (ATSDR, 1999). Much of the concern over lead exposure in women of child-bearing age stems from concerns that the exposures could lead to elevated blood lead levels in the fetus (US EPA, 2003).

Many environmental guidelines or standards for lead are based on children as the sensitive population (e.g., CA EPA, 1997; Health Canada, 1992; RIVM, 2001; US EPA, 2000a, 2001; WHO, 1996). The derivations of these guidelines, however, are different from the derivation of guidelines for most contaminants. The guidelines are not based directly on a daily intake of lead from one route of exposure (for example, a reference dose for oral intake or a reference concentration for air intake), but are based on a blood lead level. The blood lead level is typically 10 mcg/dL (micrograms of lead per deciliter of blood), which is the Centers for Disease Control and Prevention (CDC) level of concern for blood lead in young children (ATSDR, 1999; CDC, 1991). In most cases, the guidelines are derived so that the blood levels of almost all children exposed at the guideline would be below 10 mcg/dL. This is the approach taken in the derivation of the SCOs for lead (see Section 5.3.4 Chronic Lead SCOs). Thus, toxicity values (reference dose or reference concentration) for the non-cancer effects of lead are not proposed.

Cancer

The National Toxicology Program (NTP, 2005) classifies lead and lead compounds as "reasonably anticipated to be human carcinogens" based on limited evidence from studies in humans and sufficient evidence from studies in experimental animals. Similarly, the International Agency for Research on Cancer (IARC, 2004) classifies inorganic lead compounds as "probably carcinogenic to humans (Group 2A)" based on limited evidence for the carcinogenicity to humans and sufficient evidence for the carcinogenicity to experimental animals.

According to the NTP (2003, 2005) reviews, lead exposure has been associated with increased risks of lung, stomach, and bladder cancer in human populations. The epidemiological evidence is strongest for lung and stomach cancer. The evidence is not conclusive because most of the studies have limitations. These include poor exposure assessment and failure to control for confounders (other factors that could increase the risk of cancer, including lifestyle factors and concurrent occupational exposure to other carcinogens). In addition, they did not demonstrate relationships between the amount of exposure (e.g., concentration or duration) and the magnitude of cancer risk. Thus, the epidemiological data on lead are inadequate to develop cancer toxicity values (i.e., oral cancer potency factor or inhalation unit risk) for lead.

Long-term exposures to soluble (lead acetate and lead subacetate) or insoluble (lead phosphate, lead chromate) inorganic lead compounds have caused cancer in laboratory animals (NTP, 2003, 2005). Kidney tumors were most frequently associated with lead exposure, but tumors of the brain, hematopoietic system, and lung were reported in some studies. However, only two lead compounds (lead acetate and lead subacetate) have caused cancer in animals after oral exposures. Other lead compounds have caused cancer in animals after subcutaneous injection (lead phosphate or lead chromate), subcutaneous injection followed by intraperitoneal injection (lead phosphate), or intramuscular injection (lead chromate). The possibility that the carcinogenicity of lead chromate is caused by exposure to hexavalent chromium (chromate), which is an animal carcinogen, cannot be excluded. Lead naphthenate (dermal exposures), lead carbonate (diet), lead arsenate (diet), lead nitrate (drinking water), and metallic lead, as lead powder) (intramuscular or gavage) did not significantly increase tumor incidences in experimental animals. Studies of the carcinogenicity of inhaled lead were not found.

Only one of the authoritative bodies reviewed, the CA EPA, has derived oral cancer potency factors and inhalation unit risks for inorganic lead compounds (CA EPA, 1992, 1997, 2002, 2004). Most recently, the oral potency factor for lead was restricted to lead acetate, one of the two lead compounds shown to cause cancer via the oral route (CA EPA, 2005). In contrast, the US EPA (2005c) lead database for risk assessment in the Integrated Risk Assessment System, which is the peer-reviewed source for US EPA toxicity values for chemicals, contains the following statement:

Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used.

Given the problems associated with extrapolating animal data on lead to humans, animal-based oral cancer potency factors and inhalation unit risks for lead are not proposed.

5.1.1.9 Summary

Toxicity values (i.e., reference dose, reference concentration, cancer potency factor, and air unit risk) for evaluating chronic exposures were selected for priority list contaminants (Table 5.1.1-2). These values will be used to derive contaminant-specific SCOs based on chronic toxicity data and chronic exposure scenarios (see Section 5.3 Calculation of Chronic Human Health-based Soil Cleanup Objectives).

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Table 5.1.1-1. List of Authoritative Bodies Evaluated for Toxicity Values.

Authoritative Body[1]	Internet Website
United States Environmental Protection Agency	
Integrated Risk Information System	http://www.epa.gov/iris/
National Center for Environmental Assessment	http://hhpprtv.ornl.gov/
Region 3 Risk-Based Concentrations	http://www.epa.gov/reg3hwmd/risk/human/index.htm
Office of Pesticides	http://npic.orst.edu/tracking.htm
Office of Drinking Water	http://www.epa.gov/waterscience/drinking/
Health Effects Assessment Summary Tables (HEAST, FY 1997 Update)	not available online, document obtained from Office of Research and Development. Office of Emergency and Remedial Response
New York State Department of Health	not available online, values obtained from internal documents
New York State Department of Environmental Conservation	
United States Agency for Toxic Substances and Disease Registry	http://www.atsdr.cdc.gov/toxpro2.html
California Environmental Protection Agency	
Public Health Goals	http://www.oehha.ca.gov/water/phg/allphgs.html
Toxicity Criteria Database	http://www.oehha.ca.gov/risk/ChemicalDB/index.asp
Action Levels	http://www.oehha.ca.gov/water/pals/index.html
Relative Exposure Levels	http://www.oehha.ca.gov/air/chronic_rels/index.html
Cancer Potency Values	http://www.oehha.ca.gov/air/cancer_guide/index.html
Health Canada	http://www.hc-sc.gc.ca/hecs-sesc/exsd/psap.htm
World Health Organization	http://www.who.int/docstore/water_sanitation_health/GDWQ/Summary_tables/Sumtab.htm
National Institute of Public Health & Environmental Protection, Netherlands (RIVM)	http://www.rivm.nl/en/ [2] http://www.rivm.nl/bibliotheek/rapporten/711701025.pdf [3]

[1] Not all bodies derive all types of toxicity value for all contaminants.
[2] RIVM Home Page, support document can be found by searching online publications.
[3] Direct link to RIVM support document in pdf format.

Table 5.1.1-2. Toxicity Values for Priority Contaminants.

Chemical	CAS RN [1]	Oral Toxicity Values		Inhalation Toxicity Values	
		Reference Dose [2] (mg/kg/day)	Cancer Potency Factor [3] $(mg/kg/day)^{-1}$	Reference Concentration [2] (mcg/m^3)	Unit Risk [3] $(mcg/m^3)^{-1}$
acenaphthene	83-32-9	0.06	-	210 [4]	-
acenaphthylene	208-96-8	0.06 [5]	-	210 [4,5]	-
acetone	67-64-1	0.9	-	30,000	-
aldrin	309-00-2	0.00003	17	0.10 [4]	0.0049 [6]
anthracene	120-12-7	0.3	-	1000 [4]	-
arsenic		0.0003	1.5	0.03	0.0015
barium		0.02	-	0.5	-
benz[a]anthracene	56-55-3	0.03 [7]	0.903 [8]	100 [4,7]	0.00011 [8]
benzene	71-43-2	0.004	0.055	30	0.0000078
benzo[a]pyrene	50-32-8	0.03 [7]	9.03	100 [4,7]	0.0011
benzo[b]fluoranthene	205-99-2	0.03 [7]	0.903 [8]	100 [4,7]	0.00011 [8]
benzo[g,h,i]perylene	191-24-2	0.03 [7]	-	100 [4,7]	-
benzo[k]fluoranthene	207-08-9	0.03 [7]	0.0903 [8]	100 [4,7]	0.000011 [8]
beryllium		0.002	-	0.007	0.0024
n-butylbenzene	104-51-8	0.1 [9]	-	400 [9]	-
sec-butylbenzene	135-98-8	0.1 [9]	-	400 [9]	-
tert-butylbenzene	98-06-6	0.1 [9]	-	400 [9]	-
cadmium		0.0007	0.38	0.02	0.0042
carbon tetrachloride	56-23-5	0.0007	0.13	2	0.000015
chlordane	12789-03-6	0.0005	0.35	0.7	0.0001
chlorobenzene	108-90-7	0.02	-	60	-
chloroform	67-66-3	0.01	0.031	50	0.000000068
chromium III (soluble salts)		0.005	-	-	-
chromium III (insoluble salts)		1.5	-	60	-
chromium VI		0.003	-	0.1	0.05
chrysene	218-01-9	0.03 [7]	0.0903 [8]	100 [4,7]	0.000011 [8]
copper		0.14	-	490 [4]	-
cyanide	57-12-5	0.02	-	25	-
DDD	72-54-8	0.0005	0.125	1.8 [4]	0.000036 [6]
DDE	72-55-9	0.012	0.185	42 [4]	0.000053 [6]
DDT	50-29-3	0.0005	0.189	1.8 [4]	0.000054 [6]
dibenz[a,h]anthracene	53-70-3	0.03 [7]	9.03 [8]	100 [4,7]	0.0011 [8]
dibenzofuran	132-64-9	0.002	-	7 [4]	-
1,2-dichlorobenzene	95-50-1	0.021	-	200	-
1,3-dichlorobenzene	541-73-1	0.003	-	10 [4]	-
1,4-dichlorobenzene	106-46-7	0.03	0.011	800	0.0000031 [6]
1,1-dichloroethane	75-34-3	-	0.0057	-	0.0000016

Table 5.1.1-2. Toxicity Values for Priority Contaminants (continued).

Chemical	CAS RN [1]	Oral Toxicity Values		Inhalation Toxicity Values	
		Reference Dose [2] (mg/kg/day)	Cancer Potency Factor[3] $(mg/kg/day)^{-1}$	Reference Concentration[2] (mcg/m^3)	Unit Risk [3] $(mcg/m^3)^{-1}$
1,1-dichloroethene	75-35-4	0.05	-	200	-
1,2-dichloroethane	107-06-2	0.045	0.047	400	0.000013 [6]
cis-1,2-dichloroethene	156-59-2	0.01	-	35 [4]	-
trans-1,2-dichloroethene	156-60-5	0.02	-	60	-
dieldrin	60-57-1	0.00005	8.32	0.18 [4]	0.0024 [6]
1,4-dioxane	123-91-1	0.1	0.011	3600	0.0000031[6]
endosulfan (technical)	115-29-7	0.00067	-	2.3 [4]	-
endrin	72-20-8	0.0003	-	1 [4]	-
ethyl benzene	100-41-4	0.1	0.0035 [10]	2000	0.000001
fluoranthene	206-44-0	0.04	-	140 [4]	-
fluorene	86-73-7	0.04	-	140 [4]	-
heptachlor	76-44-8	0.0015	0.79	5.2 [4]	0.00023 [6]
hexachlorobenzene	118-74-1	0.0008	1.0	2.8 [4]	0.00029 [6]
alpha-hexachlorocyclohexane	319-84-6	0.0005	3.4	1.8 [4]	0.00097 [6]
beta-hexachlorocyclohexane	319-85-7	0.00001	0.96	0.035 [4]	0.00027 [6]
delta-hexachlorocyclohexane	319-86-8	0.025	-	88 [4]	-
gamma-hexachlorocyclohexane	58-89-9	0.00004	0.71	0.14 [4]	0.0002 [6]
indeno[1,2,3-cd]pyrene	193-39-5	0.03 [7]	0.903 [8]	100 [4,7]	0.00011 [8]
lead		-	0.00568	-	0.000012
manganese		0.05	-	0.15	-
mercury (elemental)	7439-97-6	-	-	0.09	-
mercury (inorganic salts)		0.00016	-	-	-
methylene chloride	75-09-2	0.06	0.0062	400	0.000000037
methyl ethyl ketone	78-93-3	0.6	-	5000	-
2-methylphenol	95-48-7	0.05	-	180 [4]	-
3-methylphenol	108-39-4	0.05	-	180 [4]	-
4-methylphenol	106-44-5	0.005	-	18 [4]	-
methyl *tert*-butyl ether	1634-04-4	0.033	0.0034	8000	0.00000026
naphthalene	91-20-3	0.02	-	9	-
nickel		0.02	-	0.09	0.00048
pentachlorophenol	87-86-5	0.001	0.12	3.5 [4]	0.000034 [6]
phenanthrene	85-01-8	0.03 [7]	-	100 [4,7]	-
phenol	108-95-2	0.3	-	20	-
n-propylbenzene	103-65-1	0.1 [9]	-	400 [9]	-
pyrene	129-00-0	0.03	-	100 [4]	-
selenium		0.005	-	18 [4]	-
silver		0.005	-	18 [4]	-
tetrachloroethene	127-18-4	0.01	0.05	100	0.000001
toluene	108-88-3	0.2	-	300	-
1,1,1-trichloroethane	71-55-6	0.28	-	2200	-
trichloroethene	79-01-6	0.00146	0.00572	40	0.000002
2-(2,4,5-trichlorophenoxy)propionic acid	93-72-1	0.008	-	28 [4]	-
1,2,4-trimethylbenzene	95-63-6	0.05	-	6	-
1,3,5-trimethylbenzene	108-67-8	0.05	-	6	-
vinyl chloride (child and adult exposure)	75-01-4	0.003	1.5	100	0.0000088

Table 5.1.1-2. Toxicity Values for Priority Contaminants (continued).

Chemical	CAS RN [1]	Oral Toxicity Values		Inhalation Toxicity Values	
		Reference Dose [2] (mg/kg/day)	Cancer Potency Factor[3] $(mg/kg/day)^{-1}$	Reference Concentration[2] (mcg/m^3)	Unit Risk [3] $(mcg/m^3)^{-1}$
vinyl chloride (adult exposure)	75-01-4	0.003	0.75	100	0.0000044
xylenes	1330-20-7	0.2	-	100	-
zinc		0.3	-	1000 [4]	-

CAS RN: Chemical Abstracts Service Registry Number
mg/kg/day: milligrams per kilogram per day
mcg/m^3: micrograms per cubic meter

[1] CAS Registry Numbers are not included for metals except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.
[2] Entries having a "-" under "Reference Dose" or "Reference Concentration" indicate that the contaminant lacks non-cancer toxicity data sufficient for the derivation of these non-cancer toxicity values.
[3] Entries having a "-" under "Cancer Potency Factor" or "Unit Risk" indicate that a) the carcinogenic potency has not been studied, b) studies of their carcinogenic potency did not show a dose-related increased in cancer incidence, or c) some evidence of carcinogenic potency has been observed but the quality of the studies or the data does not allow quantitative estimation of carcinogenic potency. The dose associated with an increased cancer risk of one-in-one million may be calculated from the cancer potency factor (1×10^{-6} dose = 1×10^{-6} / cancer potency factor). The air concentration associated with an increased cancer risk of one-in-one million may be calculated from the unit risk (1×10^{-6} air concentration = 1×10^{-6} / unit risk).
[4] A reference concentration is calculated from the recommended reference dose for chemicals that are systemic toxicants, assuming a 70 kilogram individual inhales 20 cubic meters of air per day.
[5] Based on acenapthene.
[6] A unit risk is calculated from the recommended cancer potency factor for chemicals that are systemic carcinogens, assuming a 70 kilogram individual inhales 20 cubic meters of air per day.
[7] Based on pyrene.
[8] Based on benzo[a]pyrene and application of recommended relative potency factors.
[9] Based on isopropylbenzene (cumene).
[10] A cancer potency factor is calculated from the recommended unit risk assuming a 70 kilogram individual inhales 20 cubic meters of air per day.

5.1.2 Combining Toxicity Values for Systemic Effects

People can be exposed to contaminants in soil (or dust derived from soil) through ingestion, contact with the skin, or inhalation of vapors or particles that come from the soil (or dust derived from soil). They can also be exposed when they eat food grown on contaminated soil. SCOs based on systemic toxicity should evaluate the risk associated with the exposure from oral, dermal, and inhalation exposures, since all routes of exposure can contribute to the risk for health effects. The methods to determine and combine exposures from five pathways (soil ingestion, inhalation, dermal absorption, vegetable consumption, animal product consumption) are discussed in Sections 5.2 (Exposure Assessment) and 5.3.5 (Combined Pathway Chronic SCOs). This section provides methods to develop and combine toxicity values to evaluate the combined exposures from the five pathways.

5.1.2.1 Toxicity Values for Dermal Exposures

In general, toxicity data to determine route–specific toxicity values to evaluate the potential systemic health risks of dermal exposures are not available. The US EPA (US EPA, 2001) combines dermal with oral exposures and evaluates the combined exposure using oral toxicity values (i.e., reference doses or cancer potency factors). In most cases, scientists assume different values for the bioavailability (the amount of chemical absorbed into the body) of oral and dermal doses, but assume that the distribution, metabolism, and excretion of absorbed oral and dermal doses are similar. In practice, however, the relative contribution of dermal exposures to the combined oral and dermal absorbed dose is often substantially less than the oral contribution, which minimizes concerns about data gaps for the potential route-specific differences in the metabolism, distribution, and excretion of oral and dermal doses of a contaminant.

5.1.2.2 Combining Toxicity Values for Oral/Dermal Exposures and Inhalation Exposures

Methods to evaluate the potential health risks from combined ingestion/dermal exposures and inhalation exposures vary depending on the toxicological data. Many contaminants have the same target organs regardless of the route of exposure. Toxicity values for these contaminants

are based on systemic effects of exposures, and evaluating oral/dermal and inhalation exposures separately may underestimate the risk from the combined exposures. Evaluating multi-route exposures to those contaminants that have or are likely to have the same target organ regardless of the route of exposure can be done in two ways.

The preferred approach is to use route-specific doses and route-specific toxicity values. This compensates somewhat for route-specific differences in pharmacokinetics and potential route-specific differences in potency. For non-cancer effects of a contaminant, oral and dermal doses are compared to the reference dose and inhalation doses are compared to the reference concentration. The ratio is the hazard quotient (e.g., estimated oral dose/reference dose). The hazard index can be calculated by summing the hazard quotient for each route of exposure. For cancer effects, a cancer potency factor is applied to the oral and dermal doses and the air unit risk is applied to the air concentration. The total cancer risk can be calculated by summing the cancer risks from each route of exposure.

If a chemical does not have a toxicity value for oral/dermal or inhalation exposures (most frequently inhalation), the combined dose from oral, dermal, and inhalation exposures can be evaluated using a single toxicity value (e.g., an oral reference dose or cancer potency factor) to calculate a hazard index or a lifetime excess cancer risk. These contaminants would be expected to share a common target organ given the toxicological and pharmacokinetic database on the contaminant. Most of the chemicals are evaluated using one of the above two methods.

For a limited number of contaminants, the health effects of inhalation exposure are substantially different from the effects of oral exposures (US EPA, 1994, 2002). For example, the health risk for air exposures to a contaminant that irritates and damages the respiratory tract may be totally independent of the degree of exposures via dermal or oral exposures. Consequently, for chemicals that are known to cause health effects primarily at the site of contact (e.g., on the lungs for inhalation exposure and in the gastrointestinal tract for oral exposures), inhalation and oral/dermal exposures are evaluated separately. The following three chemicals fall into this category:

Nickel

The reference concentration for nickel is based on localized effects (fibrosis and inflammation) in the lungs of rats inhaling particles of a nickel compound. In contrast, the reference dose for nickel is based on systemic effects (decreased body and organ weights) in rats ingesting food containing nickel. Consequently, inhalation and oral/dermal exposures for nickel are evaluated separately.

Beryllium

The reference concentration for beryllium is based on effects in the lungs (beryllium sensitization and chronic beryllium disease in humans), while the oral reference is based on site of contact effects on the gastrointestinal tract (gastrointestinal lesions in dogs). Since these effects appear specific to the route of exposure, beryllium exposures by the oral/dermal route and the inhalation route are evaluated separately.

Chromium VI

The reference concentration and unit risk for chromium VI are based on direct effects on the lungs. The reference concentration for chromium VI dissolved mists and aerosols is derived from studies reporting nasal septum atrophy in humans, and the reference concentration for chromium VI particulates is based on lower respiratory effects in animals. The unit risk for chromium VI is based on lung cancer in chromium workers. In contrast, the available oral reference doses for chromium VI are based on no effects in a 1-year drinking water study in rats, and there are no data which demonstrate that chromium VI causes cancer by the oral route of exposure. Thus, since the toxic effects of chromium VI appear to be route-specific with respect to inhalation, inhalation exposures are evaluated separately from oral/dermal exposures.

If pathways are not combined for chemicals whose oral and inhalation toxicity values are based on systemic effects, defensible scientific evidence should be presented that indicates the effects are highly dependent on the route of exposure.

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5.1.3 Assessment of the Potential for Acute Toxicity in Children Who May Ingest A Large Amount of Soil

The primary set of contaminant-specific health-based SCOs are based on chronic toxicity values for non-cancer and cancer effects and chronic exposure scenarios (see Section 5.3 Calculation of Chronic Human Health-based SCOs). Comments in response to the public information meetings on the Brownfield Cleanup Program expressed concern that chronic SCOs might pose a risk of acute toxicity to children who deliberately ingest a large amount of soil during a single event. Toxicity values (i.e., an acute reference doses) to evaluate the potential for acute toxicity from soil ingestion exposures are derived in this section. These values will be used to derive contaminant-specific SCOs based on acute toxicity data and acute exposure scenarios (see Section 5.4 Calculation of Acute Soil Ingestion SCOs.

5.1.3.1 Selection of Contaminants for Analysis

Few articles have evaluated the potential risk of acute effects from a large single dose of a soil contaminant. Calabrese et al. (1997) evaluated the risk in children for 13 soil contaminants (antimony, arsenic, barium, cadmium, copper, cyanide, fluoride, lead, nickel, vanadium, naphthalene, pentachlorophenol, and phenol). Antimony, fluoride, and vanadium are not discussed further in this section because they are not on the list of priority contaminants for the Brownfield Cleanup Program (see Section 4.1 Identification of Target Chemicals, Table 4.1-3). The other ten contaminants (arsenic, barium, cadmium, copper, cyanide, lead, nickel, naphthalene, pentachlorophenol, and phenol) are on the list of priority contaminants (Table 4.1-3) and are discussed below.

Calabrese et al. (1997) assumed children ingested a large amount of soil (5, 25, or 50 grams) per event. They calculated acute doses at each ingestion rate using a soil guideline concentration based on chronic toxicity data and an adult chronic residential exposure scenario. They compared the calculated acute doses to estimated doses associated with acute health effects. Their analysis indicated that the acute doses of arsenic and naphthalene were less than the estimated doses associated with acute effects. Thus, acute effects after the ingestion of a large

amount of soil at the chronic soil guideline for either contaminant would be unlikely. The health-based chronic SCOs for arsenic and naphthalene (Table 5.1.3-1) are lower than or similar to the chronic soil guidelines cited by Calabrese et al. (1997), thus, no further analysis was done.

Calabrese et al. (1997) also showed that the calculated acute doses of the eight other contaminants (barium, cadmium, copper, cyanide, lead, nickel, pentachlorophenol, and phenol) were higher than the estimated doses associated with acute effects. Thus, their analysis indicated the potential for acute effects at the chronic soil guidelines for these eight contaminants. These contaminants are discussed below.

5.1.3.2 Derivation of Provisional Acute Reference Doses

The literature on the acute toxicity of eight contaminants was reviewed and evaluated for use in the derivation of a provisional acute reference dose (RfD_{acute}). An acute reference dose is an estimate of an oral exposure of 24 hours or less to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects over a lifetime (US EPA, 2002a). The derived values are provisional values because of the screening nature of the data collection and evaluation and the limited amount of toxicological data on the acute effects of these contaminants. Provisional values were derived for seven contaminants, the data on lead was inadequate for the derivation of a provisional value (see discussion below).

The literature cited by Calabrese et al. (1997) and other public health agencies (FL DEP, 2004; MA DEP, 1992; MN PCA, 1999) was obtained and evaluated. Other sources of evaluated information included publications by national or international regulatory or advisory public health organizations (e.g., ATSDR, US EPA, National Research Council, WHO) and articles in the open literature. Based upon a review of the available data, doses for use in the derivation of provisional $RfDs_{acute}$ were identified

Ideally, a RfD is derived from a point-of-departure dose, which is a point on a dose-response curve for an effect that is within or near the range of experimental or observational data for the effect (see additional discussion on reference doses in Section 5.1.1 Toxicity Values for

Systemic Health Effects). The point-of-departure can be a NOEL, LOEL, or the lower confidence level on a dose that is associated with an estimated level of excess risk for an effect (i.e., benchmark dose). The point-of-departure is the starting point for the extrapolation from the range-of-observation to the reference dose. It is generally based on the critical effect (i.e., the first adverse effect, or its known precursor, that occurs to the most sensitive species as the exposure to an agent increases) from the critical study (i.e., the study that contributes most significantly to the qualitative and quantitative assessment of risk).

In practice, however, the data on the acute toxicity of the seven contaminants are insufficient to select a point-of-departure based on principles of risk assessment. The primary selection factor is the availability of data on effects and dose. The point-of-departure is divided typically by uncertainty factors to obtain the reference dose. These uncertainty factors compensate for inadequate test data, missing information essential for understanding the toxicity of the chemical, differences between animals and humans, and for variation among humans. General guidelines on the application of uncertainty factors exist (e.g., US EPA, 2002a). Table 5.1.3-2 contains the provisional acute reference doses for the seven contaminants. Appendix B contains a summary of the information used to derive each provisional acute reference dose. Whenever possible, the doses used in the analysis are those associated with health effects in humans. For cyanide, pentachlorophenol, and phenol, human toxicity or exposure data are insufficient to identify a point-of-departure, and animal data are used in the analysis.

Lead

Very high lead exposures can cause acute effects on the gastrointestinal tract, nervous system, and kidneys (ATSDR, 1999). In children, the population most sensitive to lead exposures, many acute poisonings occurred after the ingestion of paint chips containing high concentrations of lead or objects (e.g., medallions or toys) made of lead. More recently, however, other unusual sources of lead (e.g., folk medicines or cosmetics) have been associated with acute poisonings (Jones et al., 1999).

Studies on acute poisoning typically do not provide precise estimates of the amount of lead ingested. Rather, the diagnosis of lead poisoning is based on the identification of an ingested object containing large amounts of lead (e.g., paint chips or other objects) and/or high levels of lead in the blood of the poisoned child (e.g., VanArsdale et al., 2004). Typically, blood lead levels associated with acute effects on the nervous system and gastrointestinal systems are at least six-times higher than the CDC level of concern, which is 10 micrograms of lead per deciliter of blood (ATSDR, 1999; VanArsdale et al., 2004). Although the current US EPA (2002b, 2003) pharmacokinetic models of lead are designed to estimate a child's blood levels from the ingestion of lead-contaminated soil, the models are restricted to estimating steady-state blood lead levels from repeated exposures over months or years. They are not designed to estimate short-term lead blood levels from acute exposures. Thus, the acute toxicity data on children was not used to develop a provisional acute reference dose for lead because estimates of an acute dose associated with acute effects are unavailable as are US EPA models to accurately convert an acute lead blood level into an acute lead dose.

Calabrese et al. (1997) evaluated the potential for acute lead toxicity in children using limited data on the effects of short-term lead ingestion on the enzyme levels of adults (Cools et al., 1976; Stuik, 1974). These are among the few studies in humans that actually reported ingested doses (ATSDR, 1999). The most sensitive effect was a decrease in the level of an enzyme (aminolevulinic acid dehydratase or ALAD) involved in the production of hemoglobin (the oxygen carrying protein in the blood). However, the changes did not produce a detectable effect on hemoglobin levels (Cools et al., 1976; Stuik, 1974). ATSDR (1999) noted that that reductions in ALAD in the absence of detectable effects on hemoglobin levels are of questionable biological significance. US EPA (2000) noted that short-term reduction in ALAD are reversible once exposure ceases, and would not lead to reduction in hemoglobin unless exposure was extended for a longer period. Consequently, a provisional acute reference dose for lead was not proposed because the observed effect linked with an estimate of acute dose was a temporary, non-adverse, biochemical change in the level of an enzyme.

5.1.3.3 Discussion

Although the use of human data on acute toxicity eliminates the uncertainties associated with extrapolating the results of animal studies to humans, there are substantial limitations and uncertainties associated with the use of available human data on barium, cadmium, and nickel (Calabrese et al., 1997; FL DEP, 2004). All the studies involved small numbers of people, and many of the reports provide little quantitative information on the extent and nature of the signs/symptoms of exposure. Confidence in the estimates of the doses from these studies is low because they contained very little data on intake. Although most of these reports provided some data on the levels of the contaminant in the contaminated liquid/food, estimates of the amount of liquid/food consumed were commonly missing; thus, assumptions about intake were necessary to estimate dose. In some cases, estimates of the contaminant concentration in the contaminated liquid/food were based on a single measurement or a few measurements and might not be representative of the liquids/foods that were actually consumed.

None of the studies used to identify a point-of-departure for use in the analysis involved exposure to the contaminant in soil (Table 5.1.3-3). This is an important data gap. The contaminant was dissolved in water (barium, cadmium, copper, and nickel), contained in food (cyanide), or dissolved in gavage doses of corn oil (pentachlorophenol) or water (phenol). The forms of barium, cadmium, copper, and nickel that caused toxic effects were obviously very soluble. The form of cyanide ingested by rats was hydrogen cyanide, which is readily reduced to free cyanide[1] in the presence of water. Whether the administered dose of these contaminants would cause similar effects if the dose were ingested in a soil matrix is uncertain, but evidence suggests it is unlikely.

The bioavailability (the percentage of the ingested dose that is absorbed into the body of animals or humans) of organic and inorganic contaminants ingested in a soil matrix is dependent on many factors and processes (NEPI, 2000a,b; NRC, 2003). The collective evidence suggests that the bioavailability of most persistent organic compounds in soil is likely to decrease over time (NEPI, 2000a; NRC, 2003) because the contaminant molecules interact strongly with the soil matrix. This can substantially reduce the absorption of the contaminant into the body during passage thought the gastrointestinal tract (NRC, 2003). The physical, chemical, and biological

[1] Free cyanide is the sum of molecular HCN and the anion CN^- and is responsible for cyanide toxicity.

processes that reduce the bioavailability of contaminants in soil are described collectively as weathering or aging. These factors most likely would reduce the bioavailability of pentachlorophenol and phenol in soil compared to the bioavailability of pentachlorophenol in corn oil and phenol in water.

Metal ions in soil cannot be degraded to other elements during weathering, but weathering can change the identity and the amounts of particular metal compounds found in soil (NRC, 2003). These changes are important because different compounds containing the same metal ion can differ greatly in solubility, which in turn, leads to differences in bioavailability. In general, soluble metal compounds are more bioavailable than less soluble compounds or insoluble compounds (Goyer and Golub, 2003; NEPI, 2000b) because the majority of gastrointestinal absorption of many metals occurs in the small intestine via passive diffusion and requires the contaminant to be in solution during passage through the gastrointestinal tract (NRC, 2003). Thus, the bioavailability of a metal in soil depends largely on the solubility of the metal compounds present in the soil (NRC, 2003; US EPA, 2004).

A metal's solubility or its potential to become soluble if conditions change depends on many factors associated with the metal form, particle size, weathering, and soil chemistry (NRC, 2003; Ruby et al., 1999). Another important factor is the likelihood of disturbances that would alter the soil conditions that determine solubility and bioavailability (Ruby et al., 1999). There are limited data on how these factors vary with metals and soils and how these changes affect solubility and bioavailability. The missing data preclude accurate estimates of bioavailability of metals ingested with soils. Consequently, it is typically assumed that the bioavailability of a metal ingested in a soil matrix is the same as the bioavailability of the metal ingested in the studies used to determine the toxicity value.

Weathering seems to decrease the solubility and thus the bioavailability of metals in a soil matrix (NEPI, 2000b; NRC, 2003; US EPA, 2004). The various interactions between the metal ions and the components of the soil matrix reduce bioavailability by making it more difficult for them to become solubilized in the gastrointestinal tract. Thus, it is likely that the bioavailability of the barium, cadmium, copper, and nickel in a weathered soil matrix is less than the bioavailability of

the soluble forms of the metal that actually caused gastrointestinal effects when ingested in water.

Cyanide is neither an organic compound nor a metal. Cyanides in soil are likely a mixture of free cyanide, soluble and insoluble cyanide compounds, and cyanide complexes. Only a fraction of the cyanides in soil is free cyanide, which is responsible for the toxicity observed in toxicological studies with animals or in human poisoning. Some data suggest that weathering reduces the amount of cyanide available to interact with biological systems and induce toxicity (Zagury et al., 2004). Thus, it is likely that the bioavailability of the cyanide in a weathered soil matrix is less than the bioavailability of the free cyanide used in toxicological studies to determine the reference dose for cyanide.

5.1.3.4 Summary

Acute reference doses were derived for seven contaminants (arsenic, barium, cadmium, copper, cyanide, nickel, naphthalene, pentachlorophenol, and phenol) on the priority list. These values will be used to derive contaminant-specific SCOs based on acute toxicity data and acute exposure scenarios (see Section 5.4 Calculation of Acute Soil Ingestion SCOs).

The uncertainties in the derivation of acute reference doses are similar, but perhaps of a greater magnitude, than those present in the derivation of toxicity values (e.g., chronic reference doses or cancer potency factor) used to derived contaminant-specific SCO based on chronic toxicity data and chronic exposure scenarios (see Section 5.3 Calculation of Chronic Human Health-based Soil Cleanup Objectives). In most cases, the acute reference dose was based on less toxicity data than the chronic reference dose or cancer potency factor. In addition, the estimates of doses associated with effects observed in human poisonings (barium, cadmium, and nickel) are more uncertain than are estimates of doses in studies used to derive chronic reference doses.

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Table 5.1.3-1. Evidence That Arsenic and Naphthalene Do Not Pose a Substantial Risk of Acute Toxicity to Children Who May Ingest Large Amount of Soil.

<table>
<tr><th rowspan="2">Contaminant</th><th rowspan="2">Chronic Soil Guideline [1] (mg/kg soil)</th><th colspan="3">Chronic SCO (mg/kg soil) [2]</th></tr>
<tr><th>Unrestricted</th><th>Restricted Residential</th><th>Commercial</th></tr>
<tr><td rowspan="2">Arsenic</td><td rowspan="2">0.4</td><td>0.21</td><td>1</td><td>5.9</td></tr>
<tr><td>SCO lower than soil screening value used by Calabrese et al. (1997), thus, acute toxicity unlikely at SCO</td><td colspan="2">SCOs are above soil guideline used by Calabrese et al. (1997) but not high enough to pose a substantial risk of acute toxicity because the calculated acute doses at 1 and 5.9 mg/kg soil are 1,300- and 220-timess lower than the dose (1 mg/kg) associated with acute health effects in humans (Calabrese et al., 1997)</td></tr>
<tr><td rowspan="2">Naphthalene</td><td rowspan="2">3,100</td><td>140</td><td>590</td><td>3,500</td></tr>
<tr><td colspan="2">SCOs are lower than soil screening value used by Calabrese et al. (1997); thus acute toxicity unlikely at these SCOs</td><td>SCO is above soil guideline used by Calabrese et al. (1997) but not high enough to pose a substantial risk of acute toxicity because the calculated acute doses at 3,500 mg/kg soil is about 40-times lower than the acute dose (109 mg/kg) associated with health effects (Calabrese et al., 1997, ATSDR, 2003) [3]</td></tr>
</table>

[1] As reported in Calabrese et al. (1997).

[2] These are the land use categories than include children (see Section 5.2.1 Exposure Scenarios and Exposure Pathways); chronic health-based chronic SCOs from Table 5.3.6-1).

[3] Contaminant dose (mg/kg) at SCO (mg/kg soil) = SCO (mg/kg soil) x soil ingestion rate (10 g soil/child) / 13.3 kg child x 1 g soil/1,000 mg soil (taken from Section 5.4 Calculation of Acute Soil Ingestion SCOs).

Table 5.1.3-2. Acute Reference Doses for Use in Calculation of Acute SCOs for Seven Contaminants. (see Appendix B for details on derivation of reference doses)

Contaminant	RfD (mg/kg)	Basis
Barium	0.3	provisional acute reference dose
Cadmium	0.007	provisional acute reference dose
Copper	0.2	acute child's dose at drinking water guideline
Cyanide	0.02	chronic reference dose
Nickel	0.23	provisional acute reference dose
Pentachlorophenol	0.005	provisional acute reference dose
Phenol	0.6	provisional acute reference dose

Table 5.1.3-3. Doses, Exposure Conditions and Health Effects from Studies Used to Identify Acute Reference Doses for Use in an Analysis of the Potential for Acute Toxicity in Children Who May Ingest Large Amounts of Soil.

Contaminant	Species	Exposure Conditions	Effects and Rapidity of Onset
Inorganic Contaminants			
Barium [(1)]	human	acute: ingestion of aqueous solutions containing soluble barium salts (e.g., barium chloride)	gastrointestinal effects, effects reported to appear rapidly, but specific data were not found
Cadmium [(1)]	human	ingestion of aqueous solutions containing soluble cadmium salts	gastrointestinal effects almost immediately after ingestion
Copper [(1)]	humans	ingestion of aqueous solutions containing copper sulfate (a highly soluble compound)	gastrointestinal effects within 1 hour or less of ingestion
Cyanide [(2)]	rat	food fumigated with HCN (CN^-)	no effects on growth rate, no gross signs of toxicity, and no histopathologic lesions
Nickel [(1)]	human	ingestion of drinking water contaminated with soluble nickel salts (nickel sulfate and nickel chloride) on a warm evening	gastrointestinal effects, within 15 minutes to 2 hours of ingestion
Semi-Volatile Organic Compounds			
Pentachloro-phenol [(1)]	rat	gavage dose (corn oil); gestation days 6 - 15	developmental delays in pup on gestation day 20
Phenol [(1)]	rat	gavage dose (water); gestation days 6 - 15	maternal effects and developmental effects (body weigh loss) on gestation day 20

[(1)] From Appendix B.
[(2)] From Oral Non-Cancer Toxicity Value Documentation, Appendix A.

5.1.4 Toxicity Values for Non-Allergic Skin Irritation

Assessments of the potential health effects from contaminated soils typically focus on the chronic systemic effects of inhalation, ingestion, and dermal exposures (see Toxicity Assessment, Section 5.1 and Exposure Assessment, Section 5.2). However, skin itself can become damaged after direct contact with soils contaminated with chemicals. Irritant contact dermatitis (ICD) is one of most common forms of skin damage. ICD is a non-immunologic, local inflammatory response at the site of contact following single, repeated, or continuous exposure to a chemical (English, 2004; Maibach and Patrick, 2001).

Under a typical residential exposure scenario, soil cleanup guidelines based on the toxicity data for systemic effects and the soil-associated ingestion, dermal, and inhalation exposure are thought to be lower than soil cleanup guidelines based on the toxicity data for ICD and direct soil contact with the skin. Some exposure scenarios (industrial, for example) have higher cleanup guidelines than residential scenarios because the soil-associated ingestion, dermal, and inhalation exposures are lower than those for residential scenarios. Thus, some soil cleanup guidelines based on ICD might be lower than soil cleanup guidelines based on systemic effects.

Toxicity values (i.e., skin reference dose) to evaluate the potential for ICD from direct skin contact with soil are derived in this section. A more technical presentation can be found in Appendix C-1. These values will be used to derive, where possible, contaminant-specific SCOs based on toxicity data for ICD and dermal exposure scenarios (see Section 5.5 Calculation of Irritant Contact Dermatitis SCOs).

5.1.4.1 Skin Structure

The skin is composed of the epidermis and the dermis (Monteiro-Riviere, 1996; US EPA, 1992). The stratum corneum is the outermost layer of the epidermis and is the major barrier to the absorption of chemicals placed on the skin (Monteiro-Riviere, 1996). Below the stratum corneum are the lower layers of the epidermis and the dermis. The dermis is largely collagen (fibrous or connective) tissue, and contains blood vessels, lymph vessels, nerves, sweat and oil

glands, hair follicles, hair-erecting muscles, and other structures. Once past the stratum corneum, the absorbed contaminant ions/molecules have the potential to cause local damage (e.g., ICD) to the surrounding skin cells or to enter systemic circulation within the body.

5.1.4.2 Derivation of Skin Reference Doses

Toxicity values based on irritant contact dermatitis (skin reference $dose_{ICD}$ or skin RfD_{ICD}) were derived using dose-response assessment methods similar to those used to derive other toxicity values (i.e., oral reference dose or reference concentration) based on non-cancer systemic effects and chronic exposure scenarios (see Toxicity Assessment, Section 5.1.1). The toxicological literature was reviewed and evaluated. NOELs for ICD were identified and uncertainty factors were applied to estimate a skin $RfDs_{ICD}$. The skin $RfDs_{ICD}$ are expressed as mg contaminant per centimeter square of skin (mg/cm^2 skin), which is the preferred measure of dose in skin irritation studies (Felter et al., 2002, 2003; Robinson et al., 2000).

The priority contaminants include chemicals that are classified as VOCs, SVOCs, pesticides, and inorganic chemicals. The likelihood of contaminant movement from soil onto and through the stratum corneum skin is dependent, on part, on the physical and chemical properties of the contaminant. These properties differ greatly among the VOCs, SVOCs, pesticides, and inorganic chemicals (mostly metals). They determine, at least in part, the likelihood that a chemical in each chemical class would actually penetrate the stratum corneum and cause ICD.

Skin $RfDs_{ICD}$ were not derived for VOCs in soil because VOCs are more likely to move from soil to air rather than from soil to skin (US EPA, 2004), and thus, likely pose little risk of ICD. The physical and chemicals properties of SVOCs, pesticides, and metals suggest they have the potential to move from soil and be absorbed through the stratum corneum. Thus, the literature on irritant dermatitis was reviewed for data on organic chemicals (SVOCs, pesticides) and inorganic chemicals (metals).

Much of the data from dermal toxicology studies done for regulatory purposes is not useful for the estimation of animal or human NOELs for ICD. Consequently, data collected as part of US

EPA approved test (the mouse ear swelling test, see Gad et al., 1986; US EPA, 1998) for another form of irritants dermatitis (i.e., allergic contact dermatitis) were used to estimate mouse NOELs for 90 substances. NOELs were available to derive skin $RfDs_{ICD}$ for only three priority contaminants (chromium, nickel, and phenol) (Table 5.1.4-1). In addition, the NOELs for some relatively potent organic chemicals were used to derive a surrogate skin RfD_{ICD} for priority contaminants that are SVOCs, including pesticides (Table 5.1.4-1).

5.1.4.3 Discussion

The toxicity data on ICD used to estimate NOELs were generated from studies of the irritant properties of chemicals in solution. Data on the irritate potency of chemicals in soil were not found. This is a data gap.

Confidence in the estimated for ICD is limited because the data were not collected to directly estimate NOELs, and plausible assumptions were necessary to generate NOELs. Moreover, much of the data were limited to chemicals that were studied because they were potent irritants, and/or chemicals known to induce another form of irritant dermatitis (i.e., allergic contact dermatitis). The organic chemicals used as surrogates for SVOC priority contaminants might be more potent irritants that contaminants likely to be found at brownfield sites. However, the methods and dose metric used to estimate mouse NOELs for ICD and to extrapolate those results to humans are consistent with recent developments in risk assessment methods for allergic contact dermatitis.

5.1.4.4 Summary

Skin reference doses were derived for three contaminants (chromium, nickel, and phenol) on the priority list. A surrogate skin reference dose was also derived for application to SVOCs, including pesticides on the priority list. These values will be used to derive contaminant-specific SCOs, and surrogate-SVOC SCOs, based on ICD data and dermal exposure scenarios (see Section 5.5 Calculation of Irritant Contact Dermatitis SCOs).

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Table 5.1.4-1. Skin Reference Doses for Use in Calculation of SCOs for Chromium, Nickel, Phenol and SVOCs (see Appendix C for details on derivation of reference doses).

Chemical	Skin Reference Dose (mg/cm^2 skin)
nickel as nickel sulfate	0.0023
chromium VI as potassium dichromate	0.0016
phenol	0.012
SVOC surrogate [(1)]	0.0013

[(1)] Applies to any semi-volatile organic chemical or pesticide on the list of priority contaminants.

5.1.5 Systemic Health Effects from Exposure to Mixtures

The legislation (§ 27-1415 (6)(b)) states that "the department shall consider… contaminants which act through similar toxicological mechanisms or have the potential for additive and/or synergistic effects,…" in the development of tables of contaminant-specific soil cleanup objectives (SCOs) based on the potential health effects of exposures.

Regarding synergy, Agency for Toxic Substances and Disease Registry guidance for evaluating the non-cancer and cancer health risks of mixtures states that it is unlikely that a chemical mixture will have any significant synergistic effects that will result in a health hazard when environmental levels of the components in the mixtures are low (ATSDR, 2004). For the non-cancer effects of mixtures, experimental evidence suggests that synergistic interactions among components of mixtures at soil concentrations near or below the individual SCOs is unlikely when such concentrations result in very low doses (e.g., at doses equal to or below the reference dose for the individual contaminants) (Cassee et al., 1998; Groten et al., 1997; Jonker et al., 1990, 1993a,b, 1996; Wade et al., 2002). For the cancer effects of mixtures, different carcinogens in the mixture might show synergistic interactions. However, the consensus is that such interactions are unlikely at low doses (see NRC, 1989), such as those associated with the SCOs for individual contaminants, which are set at an excess cancer risk level of one-in-one million.

In general, additive interactions between chemicals are most likely to occur when the chemicals cause the same effect on the same organ by the same toxicological mode of action (ATSDR, 2004; US EPA, 2000). This is most likely to be the case for mixtures of chemically related contaminants. For example, an assumption of additivity is reasonable for mixed xylenes (*ortho*-, *meta*-, and *para*-xylene) because these chemicals are very similar in chemical structure, do not differ much in their toxicity, and are reasonably likely to share a common mode of action. As described below, the Department assumed that additive interactions occur for five groups of related contaminants (three pesticide mixtures, xylenes and carcinogenic polycyclic aromatic hydrocarbons). For those five groups of related contaminants the approach used by the Department is consistent with the recommended

approach for the assessment of the non-cancer risks (ATSDR, 2004; US EPA, 1986, 2000, 2003a) and cancer risks from mixtures (ATSDR, 2004; NRC, 1989; US EPA, 1986, 1989, 2000, 2003a) when data are absent or limited.

5.1.5.1 Mixtures of Chemically-Related Contaminants

Exposures to contaminant mixtures may pose different risks than exposures to individual contaminants. There are different types of mixtures. Some mixtures are composed of many chemically related compounds that can be generated simultaneously by a source or process (e.g., PAHs). Others are mixtures of chemically related compounds that are produced as commercial products (e.g., pesticides such as chlordane, endosulfan, and endrin).

Historically, different approaches have been developed to evaluate the toxicity of different types of mixtures (ATSDR, 2001; US EPA, 1986, 2000, 2002a,b, 2003a). Generally, one of three types of data is used to evaluate the toxicity of a mixture: data on the mixture itself, data on similar mixtures, or data on the individual components of the mixture. For mixtures of related compounds, the type of data used in an analysis is typically the type of data that was first used to study and measure the mixture.

Mixture-specific methodologies for five mixtures of chemically related contaminants (i.e., carcinogenic PAHs, chlordane, endosulfan, endrin, and xylenes) are described below. Scientific consensus (US EPA, ATSDR, CA EPA, NYS DOH, WHO) exists on the methods to evaluate the toxicity of carcinogenic PAHs. These mixture specific methodologies will be used to generate health-based SCOs for each mixture as if it was a single contaminant.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are a group of over 100 chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat. They can also be found in natural substances such as crude oil, coal, coal tar pitch, creosote, and roofing tar.

There are potentially a large number of PAHs, but attention has been focused on only some of the PAHs (e.g., ATSDR, 1995a). Fifteen PAHs are priority contaminants (Table 4.1-3). Of these, seven are classified as carcinogenic (Table 5.1.5-1).

Generally, a component-based approach has been used to evaluate the health risks associated with mixtures of carcinogenic PAHs (CA EPA, 2002; MA DEP, 1996; MN DOH, 2004; NJ DEP, 2004; US EPA, 1993, 2002a). This approach is supported by the experimental data that PAHs likely share common toxicological mechanisms for cancer (Bostrom et al., 2002; US EPA, 1993). It is similar to the toxic equivalency factor (TEF) approach usually used for mixtures of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDDs and PCDFs) because it uses an index chemical and relative potencies. However, the adjustment factors are called relative potency factors (RPF) rather than toxic equivalency factors because they are based are more limited data than are TEFs (US EPA, 2000).

The index chemical for the RPF approach is benzo[a]pyrene or BaP. BaP is among the most studied and potent PAHs, and more importantly, there are sufficient cancer data from long-term studies to calculate an oral cancer potency factor for BaP (see Appendix A, Cancer Toxicity Value Documentation for Benzo[a]pyrene)). In addition, there are sufficient data on the six other carcinogenic PAHs to provide an estimate of their carcinogenic potency relative to BaP. The RPF for each of the six PAHs is derived from the results of carcinogenesis bioassays with BaP compared to the results with other PAHs (CA EPA, 2002; US EPA, 1993). BaP is assigned a RPF of 1, and the RPFs for all the other PAHs are limited to multiples of 1 because of the inherent uncertainties in the data. A RPF of 0.1 indicates that the compound is 1/10 as potent as BaP.

RPFs are used to "convert" the soil concentrations of other carcinogenic PAHs into equivalents of BaP, and can be used to convert the SCO for BaP into an SCO for each carcinogenic PAH. A PAH-specific SCO can be calculated by dividing the SCO for BaP by the RPF for that carcinogenic PAH. In addition, RPFs are used to convert the soil concentrations of a mixture of carcinogenic PAHs into to a soil concentration expressed in BaP equivalents. In the conversion,

the soil concentration of each carcinogenic PAH is multiplied by its RPF to obtain an equivalent BaP concentration and the BaP-equivalent concentrations are summed across all PAHs to give the total BaP-equivalents of the mixture:

Total Soil Concentration in BaP-Equivalents = $SC_1 \times RPF_1 + SC_2 \times RPF_2 + \ldots + SC_i \times RPF_i$

Where:

SC_i = soil concentration for PAH_1, PAH_2, … PAH_i

RPF_i = relative potency factor for carcinogenic PAH_1, PAH_2, … PAH_i (applicable only to the systemic cancer effects).

The total BaP-equivalent soil concentration of each mixture could be compared to the SCO based on the cancer effects of BaP.

Agreement has been reached on the RPFs for three of the seven carcinogenic PAHs that are on the target contaminant list (Table 5.1.5-2). The recommended value for benz[a]anthracene (0.1), benzo[b]fluoranthene (0.1) and indeno[1,2,3-cd]pyrene (0.1) reflects a consensus based on the available scientific information, and these RPFs are used to assess cancer risks for all routes of exposure.

Three PAHs (benzo[k]fluoranthene, chrysene and dibenz[a,h]anthracene) have different assigned RPFs (Table 5.1.5-2). Nisbet and LaGoy (1992) and CA EPA (2002) based their RPFs for these PAHs (except for CA EPA's RPF for dibenz[a,h]anthracene) primarily on the work of Clement (1988), who derived RPFs under contract for the US EPA. Documentation for Clement's calculations is not available in the open toxicological literature. Therefore, the basis for the RPFs derived by Nisbet and LaGoy (1992) for benzo[k]fluoranthene, chrysene, and dibenz[a,h]anthracene, and CA EPA (2002) for benzo[k]fluoranthene and chrysene cannot be adequately evaluated nor compared.

For benzo[k]fluoranthene, the US EPA (1993) recommended a RPF of 0.01 based on a skin carcinogenicity study in mice (Habs et al., 1980). A RPF of 0.01 was also recommended by Deutsch-Wenzel et al. (1983) based on their intrapleural implantation study in rats. Since identical RPFs were derived from studies using different routes of exposure and species, an RPF of 0.01 is a reasonable value to assess the benzo[k]fluoranthene cancer risks for all routes of exposure.

For chrysene, US EPA (1993) recommended a RPF of 0.001 based on a study of skin carcinogenesis in mice (Wynder and Hoffman, 1959). However, a RPF of 0.01 was recommended by Wenzel-Hartung et al. (1990) based on their intrapleural implantation study in rats. The skin carcinogenesis study used only one dose group containing a limited number of mice (n = 20). The intrapleural implantation study used two dose groups containing 35 animals each. Neither mode of administration appears to be more appropriate than the other for development of a RPF to assess cancer risks for all route of exposure. Based on the overall quality of the studies, the RPF (0.01) derived from intrapleural implantation study (Wenzel-Hartung et al., 1990) is used to assess the chrysene cancer risks for all routes of exposure.

For dibenz[a,h]anthracene, the US EPA (1993) recommended a RPF of 1 based on a skin carcinogenicity study in mice (Wynder and Hoffman, 1959). A RPF of 1 was also recommended based on the intrapleural implantation study of Wenzel-Hartung et al. (1990) in rats. However, CA EPA (2002) derived a RPF of 0.4 for dibenz[a,h]anthracene. The CA EPA RPF is based on a potency estimate based on increased alveolar carcinomas in mice exposed to dibenz[a,h]anthracene in drinking water (Snell and Stewart, 1962) compared to a potency estimate based on increased gastric tumors in mice exposed to benzo[a]pyrene in feed (Neal and Rigdon, 1967). The CA EPA estimate has uncertainties not present in other estimates, primarily because dibenz[a,h]anthracene and benzo[a]pyrene were tested in different studies under different experimental protocols and induced different tumor types. In contrast, the RPFs derived from the skin carcinogenesis study or the intrapleural implantation study are based on the testing of dibenz[a,h]anthracene and benzo[a]pyrene in the same experiment and under the same protocol. Thus, the RPF (1) derived from these studies is used to assess the dibenz[a,h]anthracene cancer risks for all routes of exposure.

At present, a RPF approach has not been used to evaluate the non-carcinogenic effects of PAH mixtures (CA EPA, 2002; MA DEP, 1996; MN DOH, 2004; NJ DEP, 2004; US EPA, 1993, 2002a). The non-cancer effects of individual PAHs will be assessed using the approach described in Section 5.1.

Chlordane

Technical chlordane (CAS RN 12789-03-6) is a manufactured pesticide and a mixture of about 140 related chemicals (ATSDR, 1994; US EPA, 1997). Most of these compounds are minor or trace components, but two chlordane isomers, cis-chlordane (also called alpha-chlordane, CAS RN 5103-71-9) and trans-chlordane (also called gamma-chlordane, CAS RN 57-74-9), comprise 60% to 85% of technical chlordane. Other chemicals in the mixture include heptachlor, cis-nonachlor, trans-nonachlor, alpha-chlordene, beta-chlordene, and gamma-chlordene.

Alpha-chlordane is on the list of priority contaminants (Table 4.1-3). Authoritative bodies have not derived chemical-specific toxicity values for alpha-chlordane. Almost all the toxicity data on chlordane comes from studies with technical chlordane (ATSDR, 1994; US EPA 1997), and thus, the toxicity values for alpha-chlordane are based on studies with the technical mixture (see Appendix A, Non-Cancer and Cancer Toxicity Value Documentation for Chlordane). The toxicity values for technical chlordane are reasonable surrogates for the toxicity values alpha-chlordane because it and an isomer (gamma-chlorodane) are the predominate compounds in technical chlordane. In practice, the soil concentrations for alpha-chlordane could be compared to the SCOs (non-cancer and cancer effects) for technical chlordane.

Endosulfan

Technical endosulfan (CAS RN 115-29-7) is a manufactured pesticide and a mixture of several related chemicals (ATSDR, 2000). Two endosulfan isomers, alpha-endosulfan (also called endosulfan I, CAS RN 959-98-8) and beta-endosulfan (also called endosulfan II, CAS RN 33213-65-9), comprise at least 94% of technical endosulfan. Technical endosulfan also contains

endosulfan sulfate (CAS RN 1031-07-8), endosulfan alcohol, and endosulfan ether as impurities or degradates. Endosulfan sulfate is also an environmental degradate and a mammalian metabolite of endosulfan.

Endosulfan I, endosulfan II, and endosulfan sulfate are on the list of priority contaminants (Table 4.1-3), but authoritative bodies have not derived chemical-specific toxicity values for these compounds. Almost all the toxicity data for endosulfan comes from studies of technical endosulfan (ATSDR, 2000; US EPA, 2004a), and thus, toxicity values for endosulfan I, endosulfan II, and endosulfan sulfate are based on studies with the technical mixture (see Appendix A, Non-Cancer Toxicity Value Documentation for Endosulfan). The toxicity values for technical endosulfan are reasonable surrogates for the toxicity values for mixtures of endosulfan I, endosulfan II, and endosulfan sulfate because the two isomers are the predominate compounds in technical endosulfan and endosulfan sulfate is a mammalian metabolite of endosulfan. In practice, the sum of the soil concentrations for endosulfan I, endosulfan II, and endosulfan sulfate could be compared to the SCO (non-cancer) for technical endosulfan.

Endrin

Technical endrin is a manufactured pesticide and is a mixture of several related compounds (ATSDR, 1996). Technical endrin is 95-98% pure endrin (CAS RN 72-20-8),, and contains other chemicals as impurities or degradates. These chemicals include endrin aldehyde (CAS RN 7421-93-4), endrin ketone (CAS RN 53494-70-5), aldrin, dieldrin, isodrin, heptachloronorbornadiene, and heptachloronorborene. Endrin aldehyde and endrin ketone are environmental degradates of endrin, although the amount of endrin broken down to endrin aldehyde or endrin ketone is usually small (less than 5%).

Endrin (CAS RN 72-20-8) is on the list of priority contaminants (Table 4.1-3), but authoritative bodies have not derived chemical-specific toxicity values for endrin. All the toxicity data for endrin comes from studies of technical endrin (ATSDR, 1996; US EPA, 2004b), and the toxicity values for endrin are based on studies with the technical mixture (see Appendix A, Non-Cancer Toxicity Value Documentation for Endrin). The toxicity values for technical endrin are

reasonable surrogates for the toxicity values for endrin because technical endrin is almost all endrin. In practice, the soil concentrations for endrin could be compared to the SCO (non-cancer) for technical endrin.

Xylenes

Mixed or commercial xylenes (CAS RN 1330-20-7) are listed as a single priority contaminant, but are a mixture of three xylene isomers: *meta*-xylene (m-xylene, CAS RN 108-38-3), *ortho*-xylene (o-xylene, CAS RN 95-47-6), and *para*-xylene (p-xylene, CAS RN 106-42-3). The predominate isomer in the mixture is typically m-xylene (44% - 70%), and the mixture may also contain other chemicals, including ethyl benzene (6% -- 15%), toluene, and aromatic hydrocarbons containing nine carbon atoms (ATSDR, 1995b).

The toxicity value (non-cancer effects) for xylenes is based on a study of mixed xylenes (see Appendix A, Non-Cancer Toxicity Value Documentation for Xylenes). The toxicity value for mixed xylenes is a reasonable surrogate for the toxicity value for other mixtures of three isomers under the assumption, supported by some data (US EPA, 2003b), that the toxic potencies of the three isomers do not differ greatly. This suggests the potency of mixtures would not vary greatly with isomer composition. Thus, the SCO (non-cancer) derived for mixed xylenes could be used to evaluate the toxicity of mixtures of the three isomers. Two approaches are possible. If analytical data are reported as total xylenes, the soil concentration of total xylenes could be compared to the SCO (non-cancer) for mixed xylenes. If analytical data are reported as individual isomers, the sum of the soil concentrations of all three isomers could be compared to the SCO (non-cancer) for mixed xylenes.

5.1.5.2 Other Mixtures

The previous section (Section 5.1.5.1) addresses several mixtures of chemically-related contaminants. Other mixtures of related and unrelated contaminants will be present at sites. The chemicals and chemical concentrations in those mixtures will vary and, therefore, accounting for such mixtures in calculating the SCOs is not feasible. However, methods to assess the health

risks of mixtures are available (e.g., US EPA, 1986; 2000). Therefore, after careful consideration, the Department has decided to address these mixtures in the context of the selection of the remedy for a site which is protective of public health, rather than by modification of the SCOs.

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Table 5.1.5-1. Evaluations of the Human Carcinogenic Potential of 15 PAHs on the Brownfield Cleanup List of Priority Contaminants by the National Toxicology Program (NTP), the US Environmental Protection Agency (US EPA), and the International Agency for Research on Cancer (IARC).

PAH	Cancer Classification		
	NTP List of Carcinogen [1]	US EPA IRIS Database [2]	IARC (2004) [3]
Carcinogenic PAHs			
benzo[a]pyrene	reasonably anticipated to be a human carcinogen	probable human carcinogen	probably carcinogenic to humans
benz[a]anthracene	reasonably anticipated to be a human carcinogen	probable human carcinogen	probably carcinogenic to humans
benzo[b]fluoranthene	reasonably anticipated to be a human carcinogen	probable human carcinogen	possibly carcinogenic to humans
benzo[k]fluoranthene	reasonably anticipated to be a human carcinogen	probable human carcinogen	possibly carcinogenic to humans
chrysene	not on list	probable human carcinogen	not classifiable
dibenz[a,h]anthracene	reasonably anticipated to be a human carcinogen	probable human carcinogen	probably carcinogenic to humans
indeno[1,2,3-c,d]-pyrene	reasonably anticipated to be a human carcinogen	probable human carcinogen	possibly carcinogenic to humans
PAHs Not Identified as Carcinogenic			
acenaphthene	not on list	not evaluated	not evaluated
acenaphthylene	not on list	not classifiable	not evaluated
anthracene	not on list	not classifiable	not classifiable
benzo[g,h,i]perylene	not on list	not classifiable	not classifiable
fluoranthene	not on list	not classifiable	not classifiable
fluorene	not on list	not classifiable	not classifiable
phenanthrene	not on list	not classifiable	not classifiable
pyrene	not on list	not classifiable	not classifiable

[1] Abstracted from NTP Annual List of Carcinogens. http://ehp.niehs.nih.gov/roc/toc10.html#toc.

[2] Abstracted from US EPA Integrated risk Information System. http://www.epa.gov/iris/index.html.

[3] Abstracted from IARC. IARC Monographs Programme on the Evaluation of Carcinogenic Risks to Humans. http://monographs.iarc.fr/monoeval/grlist.html.

Table 5.1.5-2. Relative Potency Factors (RPF) for Carcinogenic PAHs Derived by Various Groups and the RPF Selected for Use in the Brownfield Cleanup Program.

PAH	Relative Cancer Potency Factors				
	Derived By				Selected Value
	Deutsch-Wenzel et al. (1983); Wenzel-Hartung et al. (1990) [2]	Nisbet & LaGoy (1992) [1]	US EPA (1993) [2]	CA EPA (1994)	
benzo[a]pyrene	index chemical, set at 1				
benz[a]anthracene	not tested	0.1	0.1	0.1	0.1
benzo[b]fluoranthene	0.1	0.1	0.1	0.1	0.1
benzo[k]fluoranthene	0.01	0.1	0.01	0.1	0.01 [3]
chrysene	0.01	0.01	0.001	0.01	0.01 [3]
dibenz[a,h]anthracene	1	5	1	0.4	1 [3]
indeno[1,2,3-cd]pyrene	0.1	0.1	0.1	0.1	0.1

[1] For comparative purposes.

[2] Reported relative potency factors based on experimental results were converted to order of magnitude potency factors using the following rounding scheme:

Reported Value	Order of Magnitude Potency
0.00051 – 0.005	0.001
0.0051 – 0.05	0.01
0.051 – 0.5	0.1
0.51- 5.0	1.0

[3] See text for selection rationale.

5.2 Exposure Assessment

As previously described, SCOs were developed for five land-use categories: unrestricted, residential, restricted residential, commercial and industrial. The first section of this chapter describes the exposure scenarios that were evaluated in developing the SCOs for each of the land use categories. In developing these exposure scenarios, we considered the exposure scenarios commonly used by US EPA (e.g., the residential and worker scenarios in US EPA's Soil Screening Guidance (US EPA, 1996; 2002b)) and the scenarios developed by other state regulatory agencies. We also considered the information on activity factors contained in US EPA's Exposure Factors Handbook (US EPA, 1997). While this information was helpful, it was not sufficient to provide a definitive technical basis for exposure scenario development. Therefore, these exposure scenarios are largely based on assumed activity patterns and assumptions about typical activities and behaviors in different exposure settings. Subsequent sections of this chapter describe the specific exposure parameters and parameter values that were used to calculate the SCOs for the various exposure scenarios.

5.2.1 Exposure Scenarios and Exposure Pathways

5.2.1.1 Unrestricted Land Use

Properties in this land use category may be used for any purpose without imposed restrictions. The key elements of the child and adult exposure scenarios for the unrestricted use category are listed below:

<u>Child Resident</u>

- A young child (2-3 years old) is present at home weekdays and weekends. On some of these days, the child may spend some time away from home (e.g., with a parent who is shopping, at a library, visiting relatives or neighbors, etc.). However, since the amount of time the child may be away from home could be small (e.g., several hours per week) we did not account for time away from home in developing exposure estimates.
- The child plays outdoors and ingests outdoor soil five days/week during the warmer months of the year.

- The child ingests indoor dust derived from outdoor soil (e.g., soil that has been tracked into the home) every day throughout the year.
- The child has dermal contact with outdoor soil on the same days during which outdoor soil ingestion occurs.
- The child inhales volatile contaminants (at home, indoors and outdoors) and particulate-bound contaminants (at home, outdoors), released from outdoor soil to outdoor air. We assumed that during the cooler months of the year, the release of volatile and particulate-bound contaminants either does not occur or is negligible. (This assumption also applies to several other exposure scenarios described below). Therefore, we assumed that inhalation exposure occurs only during the warmer months of the year. We also assumed the ingestion of indoor dust derived from outdoor soil accounts for all indoor exposures to particulate-bound contaminants (inhalation, ingestion, and dermal absorption). This assumption is discussed in more detail in Section 5.2.2.2 (Inhalation Pathway).
- The child consumes vegetables from a home garden and home produced animal products such as meat, eggs and milk.

Adult Resident

- The adult resident is a parent of the child resident and is present at home weekdays and weekends. On some of these days, the adult may spend some time away from home (e.g., during shopping, visits to a library, visits to relatives or neighbors, etc.). However, since the amount of time the adult may be away from home could be small (e.g., several hours per week) we did not account for time away from home in developing exposure estimates.
- On weekends during the warmer months of the year, the adult performs yard work and gardening activities and through these activities ingests outdoor soil.
- The adult has dermal contact with soil on the same days during which outdoor soil ingestion occurs.
- The adult inhales volatile contaminants (indoors and outdoors) and particulate-bound contaminants (outdoors) released from outdoor soil to outdoor air. Inhalation exposure occurs during the warmer months of the year.

- The adult consumes vegetables from a home garden and home produced animal products such as meat, eggs and milk.

5.2.1.2 Residential Land Use

Properties in this land-use category may be used for single family housing. Vegetable gardens are allowed, but raising livestock or producing animal products for human consumption are prohibited. With the exception of consumption of home produced animal products, people (children and adults) in these settings may have opportunities for exposure to soil contaminants that are similar to those in the unrestricted category. Therefore, with the exception of the animal product consumption pathway, we evaluated the same exposure scenarios for the unrestricted and the residential land-use categories.

5.2.1.3 Restricted Residential Land Use

Properties in this land-use category may be used for residences when there is common control of the property (e.g., apartment complexes, townhouse developments, etc.); single-family housing is excluded from this category. Farms and vegetable gardens are prohibited in this category, although community gardens may be allowed with NYS DEC approval. With the exception of the homegrown vegetable consumption exposure pathway, people (children and adults) in these settings may have opportunities for contact with soil that are similar to those in the residential category. Therefore, with the exception of the vegetable consumption pathway, we evaluated the same exposure scenarios for the residential and the restricted residential land-use categories.

5.2.1.4 Commercial Land Use

Properties in this land-use category would primarily be used for the buying, selling or trading of merchandise or services. Children could be present at commercial facilities as visitors, with a parent or other caregiver. Adults could be present at commercial facilities as customers/patrons, visitors, or workers. We examined several potential exposure scenarios for this land-use category as described below.

Child Visitor

For some commercial land uses, there will probably be few or no opportunities for a child to be exposed to soil contaminants (e.g., a large urban mall with paved parking lots and little or no greenspace). Also, a child's exposure frequency for some land uses will probably be quite limited (e.g., service stations). There also are some commercial settings (e.g., restaurants with outdoor picnic areas, a parent's workplace, a recreational facility with walking paths) where a child could be exposed to soil contaminants. However, unlike residential and worker exposure scenarios, we were not able to identify commercial exposure scenarios that have been routinely used to estimate exposure to soil contaminants. We therefore needed to explore commercial exposure scenarios where there is a reasonable expectation of exposure to soil contaminants on at least a somewhat regular basis. Such scenarios could include spending time with a parent at work (e.g., a small retail facility such as a bookstore or tailor, an appliance repair shop, or a family-owned restaurant, each with some greenspace), visiting a restaurant/snack bar with an outdoor eating area, visiting a recreational park, and probably others. Some specific examples of these kinds of exposure scenarios are described below:

Scenario #1

The commercial facility is a small restaurant with take-out service. The restaurant has an outdoor eating area consisting of several picnic tables in a grassy location. A young child (2-3 year-old) visits this restaurant with a parent or caregiver. The child spends 30 minutes indoors while food is ordered and prepared, and spends 60 minutes at an outdoor picnic area eating and playing. While indoors, the child ingests indoor dust derived from outdoor soil. While outdoors, the child ingests soil from a bare soil area near the picnic tables. The child also has dermal contact with bare soil and the child inhales volatile and particulate-bound contaminants, released from soil to outdoor air. The child and parent/caregiver visit this restaurant twice per week, during the warmer months of the year.

Scenario #2

The commercial facility is a snack-bar/ice cream stand with take-out service. The facility has an outdoor eating area consisting of several picnic tables in a grassy location. The facility also has a small playground. A young child (2-3 year old) visits this facility with a parent or caregiver. The child spends two hours (120 minutes) outdoors while food is ordered, prepared and eaten. While outdoors, the child ingests soil from bare soil areas near the picnic tables and near the play equipment. The child also has dermal contact with bare soil and the child inhales volatile and particulate-bound contaminants, released from soil to outdoor air. The child and parent/caregiver visit this snack-bar/ice cream stand twice per week, during the warmer months of the year.

Scenario #3

The commercial facility is an urban waterfront park consisting mostly of greenspace, with paved walking paths and several park benches overlooking the waterfront. Some bare soil is present in the vicinity of the park benches. An adult (babysitter, nanny, or parent) brings a young child (2-3 year-old) to the park. During a two-hour visit to the park, the child ingests soil from the bare soil areas near the park benches. The child also has dermal contact with bare soil and the child inhales volatile and particulate-bound contaminants, released from soil to outdoor air. The adult and child visit the park twice a week during the warmer months of the year.

Scenario #4

A parent's workplace is a commercial facility at a former brownfield site. The workplace is a small retail facility, such as a bookstore, with a small backyard area. A young child who attends school (e.g., a 5-6 year-old) spends the last two hours of each Monday through Friday at the parent's workplace. The child spends 30 minutes outdoors playing each day during the warmer months of the year (bare soil is present), and 90 minutes indoors each day (where some outdoor soil has been tracked in). During the cooler months of the year, the child spends the entire time indoors. While outdoors, the child ingests soil from a bare soil area and has dermal contact with the soil. While indoors, the child ingests indoor dust derived from outdoor soil. The child also inhales volatile and particulate-bound contaminants, released from soil to outdoor air.

Scenario #5

This scenario is the same as Scenario #4, except the retail facility has no backyard/outdoors play area. The child spends the entire two hours playing indoors.

These exposure scenarios illustrate that it is not unreasonable to assume that a child could be exposed to soil contaminants (by ingestion, dermal contact, and inhalation) at a commercial facility. While each of the above exposure scenarios is plausible, other scenarios may be plausible as well. Therefore, rather than selecting one particular scenario as the basis of SCOs for a child in a commercial setting, we evaluated a generalized exposure scenario. The elements of this exposure scenario are as follows:

- An adult brings a young child (2-3 years old) to a commercial facility twice per week. During the warmer months of the year, there are opportunities for the child to be exposed to soil contaminants. The duration of each visit is two hours.
- While at the commercial facility, the child ingests outdoor soil and indoor dust derived from outdoor soil.
- The child has dermal contact with outdoor soil.
- The child inhales volatile and particulate-bound contaminants, released from soil to outdoor air.

Adult Worker

While the adults who may be present at commercial facilities could be customers, visitors, or workers, some workers would have a greater opportunity for contact with soil contaminants and a greater frequency of exposure than customers or visitors. Therefore, we evaluated an adult worker scenario. Workers at commercial facilities could spend the majority of time indoors (e.g., office workers, sales people, restaurant workers) or outdoors (e.g., maintenance workers, landscapers/groundskeepers). Some outdoor workers, such as a landscaper/groundskeeper at a park, could be engaged in activities such as digging in surface soils, mowing/raking lawns, planting shrubs and flowers, and other related activities. We evaluated an outdoor worker

because these workers would have a greater potential than indoor workers for exposure to soil contaminants.

The key aspects of the adult outdoor worker exposure scenario are:

- The person works outdoors performing landscaping/groundskeeping/maintenance activities at a park four days/week, during the warmer months of the year. On these days, the worker's activities result in contact with contaminated soil (e.g., landscaping activities). On the remaining workday, the worker performs duties that do not involve contact with contaminated soil (e.g., work indoors at park facilities). During the cooler months of the year the person works indoors, or at another occupation (i.e., a seasonal employee).
- The worker ingests outdoor soil, during groundskeeping/landscaping work, four days/week during the warmer months of the year.
- Dermal contact occurs on the same days as outdoor soil ingestion.
- Inhalation exposure occurs during the outdoor working days (during the warmer months of the year) for volatile and particulate-bound contaminants, released from soil to outdoor air.

5.2.1.5 Industrial Land Use

Industrial land uses may include manufacturing, production, fabrication or assembly processes and ancillary services. While young children could visit industrial facilities, such visits would probably be rare or at least quite infrequent. Also, opportunities for a young child to contact soil contaminants during any such visits probably would be minimal because the child would likely be under strict adult supervision and the areas of the industrial facility visited by the child would likely be offices or other indoor locations. Therefore, we did not evaluate a child scenario for this land use category. While industrial facilities may have security measures in place to restrict/prevent unauthorized access, the possibility exists that older children (e.g., adolescents) could trespass at these facilities (e.g., to play, ride bicycles etc.). Therefore, we included an evaluation of an adolescent trespasser scenario in the development of SCOs for this land use. Adults could be present at industrial facilities as customers/patrons, visitors, or workers. As described for the commercial land use category, workers at industrial facilities could spend time indoors (e.g., office workers, production line workers) or outdoors (e.g., maintenance workers,

groundskeepers). While working outdoors, people at industrial facilities could be engaged in the activities that could lead to exposure to soil contaminants (e.g., outdoor workers at railroad switching yards, lumberyards, or mining operations). The key aspects of the adolescent trespasser and adult worker exposure scenarios are described below.

Adolescent Trespasser

- An adolescent trespasses at an industrial facility one day/week, during the warmer months of the year. The adolescent spends all of his or her time outdoors and has the opportunity to contact soil contaminants.
- While on the facility grounds, the adolescent incidentally ingests outdoor soil.
- Dermal contact with outdoor soil occurs on the same days as outdoor soil ingestion (one day/week during the warmer months of the year).
- While on the facility grounds, inhalation exposure occurs for volatile and particulate-bound contaminants, released from outdoor soil to air.

Adult Worker

- The person is a groundskeeper/maintenance worker at an industrial facility. On two days/week during the warmer months of the year, the worker performs groundskeeping/maintenance duties outdoors at locations where residual soil contamination remains. On all other working days of the year, the person works in facility locations where exposure to residual soil contaminants does not occur.
- The worker ingests outdoor soil two days/week during the warmer months of the year.
- Dermal contact with soil occurs on the same days as outdoor soil ingestion.
- The worker has inhalation exposure to volatile and particulate-bound soil contaminants while working outdoors.

5.2.1.5 Summary of Receptors and Pathways Across Land Uses

The exposure pathways that were assessed in developing the SCOs for the exposure scenarios in each of the four land-use categories are summarized in Table 5.2.1.5-1.

Table 5.2.1.5-1. Summary of Exposure Pathways for Developing SCOs.

	Unrestricted Land Use		Residential Land Use		Restricted Residential Land Use		Commercial Land Use		Industrial Land Use	
	Adult Resident	Child Resident	Adult Resident	Child Resident	Adult Resident	Child Resident	Adult Worker	Child Visitor	Adult Worker	Adolescent Trespasser
Soil Ingestion	√	√	√	√	√	√	√	√	√	√
Inhalation (Particle and Vapor)	√	√	√	√	√	√	√	√	√	√
Dermal Contact	√	√	√	√	√	√	√	√	√	√
Home-grown Vegetable Ingestion	√	√	√	√						
Home-produced animal product consumption	√	√								

5.2.2 Exposure Assessment Parameters and Values

In order to estimate the exposure associated with contaminants in a medium such as soil, one typically needs to assign values to various exposure parameters. Such parameters include medium intake rates, exposure frequency, exposure duration, and body weight. The following sections identify the exposure assessment parameters and parameter values that were used to develop the human health-based SCOs.

5.2.2.1 Soil Ingestion Pathway

People can be exposed to contaminants in soil through the incidental ingestion of soil (i.e., unintentional soil ingestion that occurs through activities such as hand-to-mouth behavior, mouthing of toys or other objects that have been in contact with soil, etc.). Some people, especially young children, also may sometimes deliberately ingest larger amounts of soil. People may ingest soil outdoors during the warm months of the year during activities such as gardening, yard work, and play. People may also ingest outdoor soil that has been transported into buildings (e.g., tracked in on shoes or by pets, carried in on clothing) and incorporated in indoor dust. Evaluating this pathway requires information on soil and dust ingestion rates, the time over which exposure occurs (exposure frequency and duration) and body weight. The data used to assign values to the parameters necessary for developing soil ingestion SCOs are described below.

1. Soil/Dust Ingestion Rates

Children

Although incidental soil ingestion by children has been widely acknowledged, relatively few investigators have conducted studies to yield quantitative estimates of soil ingestion rates. The US EPA reviewed the available studies in the Exposure Factors Handbook (US EPA, 1997) and identified “key studies” for use in deriving estimates of chronic soil ingestion rates. These studies used data on levels of trace elements (e.g., aluminum, silicon, titanium) in feces and soil to estimate soil/dust ingestion rates. The key studies identified by US EPA are Binder et al.

(1986), Clausing et al. (1987), Calabrese et al. (1989), Davis et al. (1990), VanWijnen et al. (1990), and Stanek and Calabrese (1995a and 1995b). US EPA (1997) considered the strengths and weaknesses of the available data and recommended 100 milligrams per day (mg/day) as the best estimate of a mean soil ingestion rate for young children (less than six years of age).

NYS DOH staff evaluated the same studies evaluated by the US EPA. The usefulness of the Binder et al., Clausing et al., and VanWijnen et al. studies for estimating a soil/dust ingestion rate is limited because the investigators did not account for tracers in food and medicines. The Stanek and Calabrese studies rely primarily on reanalysis of the original Calabrese et al. data. Therefore, we concluded that the Calabrese et al. (1989) and Davis et al. (1990) studies are the most useful for estimating a soil/dust ingestion rate. The Calabrese et al. study evaluated soil and dust ingestion for 64 children, ages one to four years, over eight days during a two-week period. Calabrese et al. determined that three of the eight tracer elements assessed (aluminum, silicon and yttrium) provided the most stable and reliable results. Mean soil ingestion estimates based on these tracers are 153 mg/day (aluminum tracer; 95^{th} percentile = 223 mg/day; SD = 852 mg/day); 154 mg/day (silicon tracer; 95^{th} percentile = 276 mg/day; SD = 693 mg/day) and 85 mg/day (yttrium tracer; 95^{th} percentile = 106 mg/day; SD = 890 mg/day). Mean soil/dust ingestion estimates based on these tracers are 154 mg/day (aluminum tracer; 95^{th} percentile = 478 mg/day; SD = 629 mg/day); 483 mg/day (silicon tracer; 95^{th} percentile = 653 mg/day; SD = 3105 mg/day) and 65 mg/day (yttrium tracer; 95^{th} percentile = 159 mg/day; SD = 717 mg/day). These estimates include data for one child with very high soil ingestion rates (10 to 14 grams/day during the second week of the study). Excluding data for this child yields soil/dust ingestion rates of 132 (aluminum tracer), 288 (silicon tracer) and 55 (yttrium tracer) mg/day. The Davis et al. study evaluated soil and dust ingestion for 104 children, ages two to seven years, over seven days. Mean soil ingestion rates estimated in this study are 39 mg/day (aluminum tracer; range 279 to 905 mg/day), 82 mg/day (silicon tracer; range –404 to 535 mg/day) and 246 mg/day (titanium tracer; range –5821 to 6182 mg/day). Mean soil/dust ingestion rates estimated in this study are 65 mg/day (aluminum tracer), 160 mg/day (silicon tracer) and 268 mg/day (titanium tracer) (ranges were not provided for combined soil/dust ingestion rates). The average of the mean soil/dust ingestion rates estimated by Calabrese et al. (excluding the child with very high soil ingestion rates) and Davis et al. is 160 mg/day.

Stanek and Calabrese (1992) reported that for children, approximately 50 percent of the total combined soil and dust exposure is due to ingestion of dust while indoors and 50 percent is due to ingestion of soil while outdoors. Chaney and Mielke (1986), citing sixteen separate source apportionment studies dating from the period 1975 to 1986, concluded that 50 percent of household dust originates as outdoor soil, on average. Allott et al. (1992) reported that the larger the particle size, the greater the proportion of indoor dust which originates from outdoor soil. They also found that the proportion of indoor dust that originates as outdoor soil is related to the location in the house. A value of 50 percent for the proportion of indoor dust that originates from outdoor soil is in the middle of the range reported in their work. This is consistent with results published by Trowbridge and Burmaster (1997) for the "transfer coefficient" (their term describing the fraction of outdoor soil in indoor dust). Using a series of tracers determined to have no sources inside homes other than outdoor soil, they reported the interquartile range (25^{th} to 75^{th} percentiles, or the middle "half" of the data) of their data ranged from 35 to 51 percent. The mean value was 44 percent and the median 43 percent, indicating that the bulk of the distribution is not strongly skewed. The 95 percent confidence interval around the mean value was 37.4 to 51.6 percent. A value of 50 percent is consistent with the middle of the range of values from this study.

Using these reported relationships, it is possible to account for the contribution of outdoor soil to total soil and dust exposures for children. Applying the findings of Stanek and Calabrese (1992) to an estimated soil/dust ingestion rate of 160 mg/day yields an ingestion rate of 80 mg/day for outdoor soil, and the same for total indoor dust. Based on the conclusions of Chaney and Mielke (1986), Allott et al. (1992) and Trowbridge and Burmaster (1997), approximately 50 percent of the 80 mg/day of indoor dust (or 40 mg/day) is assumed to originate as outdoor soil, and the remaining 40 mg/day is dust which originates from non-soil sources within the home.
Using the above information, we selected 120 mg/day (80 mg/day + 40 mg/day) as the soil ingestion rate for developing unrestricted, residential and restricted residential soil ingestion SCOs for children. For commercial settings, we recognized that children may spend less time outdoors than in unrestricted/residential/restricted residential settings and assumed that opportunities for soil ingestion may be less frequent. For example, if hand-to-mouth activity

leads to at least some incidental soil ingestion, less time spent outdoors in contact with contaminated soil could correspond to fewer hand-to-mouth events and a reduced daily soil ingestion rate. Calabrese et al. (1989) reported that the children in their study averaged 1.5 to 2 hours/day outdoors. Davis et al. (1990) reported that the children in their study averaged four hours/day outdoors. Based on this information, we assumed that an outdoor soil ingestion rate of 80 mg/day is associated with three hours/day of outdoor activity. The commercial exposure scenario we are evaluating for children assumes that a child spends two hours/day outdoors at a commercial site. We therefore assumed that daily outdoor soil ingestion by children in commercial settings will be two-thirds of the daily outdoor soil ingestion by children in unrestricted/residential/restricted residential settings (53 mg/day).

Data on soil ingestion rates for children who deliberately ingest soil are limited. To evaluate the potential for health effects from acute soil ingestion by children, we used a soil ingestion value of 10 grams per event. This value is within the range of observations reported by Calabrese et al. (1997) for children deliberately ingesting soil during a single event. It also is the value recommended by the US EPA (2002a) as a reasonable value for use in acute exposure assessments. The estimate is based on data from one child from one study who was observed deliberately ingesting soil over a two week period (US EPA, 2002a).

Adults

Limited information is available from which to derive soil ingestion rates for adults. The US EPA summarizes two published studies (Hawley (1985) and Calabrese et al. (1990)) in the Exposure Factors Handbook (US EPA, 1997). Hawley (1985) estimated that adults ingest outdoor soil at a rate of 480 mg/day (during yard work), house dust from living spaces at a rate of 0.56 mg/day, and house dust from working in attics at a rate of 110 mg/day. Calabrese et al. (1990) employed a tracer methodology (as was done in the child soil ingestion study) to estimate adult soil ingestion rates. Data for the most reliable tracers yielded estimated adult soil ingestion rates of 110 mg/day (aluminum tracer), 30 mg/day (silicon tracer), and 63 mg/day (yttrium tracer). In developing a recommended soil ingestion rate for adults, the US EPA (1997) did not specifically rely upon either of these studies. The US EPA indicates that many US EPA risk

assessments have assumed an adult soil ingestion rate of 50 mg/day for industrial settings and 100 mg/day for residential and agricultural scenarios, noting that these value are within the range of estimates from the available studies. The US EPA recommends 50 mg/day as a "central estimate of adult soil ingestion" and states that this estimate is highly uncertain. In its guidance for developing soil-screening levels, US EPA (1996; 2002b) uses an adult ingestion rate of 100 mg/day for residential settings, 100 mg/day for an outdoor worker with "substantial soil exposure" and 50 mg/day for an indoor worker with "minimal soil exposure."

In developing the SCOs for unrestricted, residential and restricted residential land uses, we used an adult soil ingestion rate of 100 mg/day (consistent with the value the US EPA has used in risk assessments for residential scenarios and with the value used by the US EPA in developing soil-screening levels). In developing SCOs for commercial and industrial land uses, we assumed that workers in both settings have similar opportunities for soil ingestion and we used a soil ingestion rate of 50 mg/day (consistent with the value the US EPA has used in risk assessments for industrial scenarios and with the US EPA's overall recommendation of an adult soil ingestion rate).

Adolescents

Data/studies upon which to base a soil/dust ingestion rate for older children (e.g., adolescents) are not available and the US EPA has not developed any recommendations. In developing the SCOs for an "adolescent trespasser" we used the same value used for an adult in an unrestricted/residential/restricted residential setting (100 mg/day).

2. Exposure Frequency

The term exposure frequency refers to how often people contact (e.g., ingest) a contaminated medium (e.g., soil) in a given period of time (e.g., events per day or per week). In calculating the soil ingestion SCOs, we recognized that people are not likely to ingest soil at a site each and every day. To determine soil ingestion exposure frequency values for each of the land use categories, we reviewed the information on activity factors in the US EPA's Exposure Factors

Handbook (1997), but determined that this information was not sufficient for deriving exposure frequency values. Therefore, the exposure frequency values described below were based on assumed activity patterns.

Unrestricted, Residential and Restricted Residential Land Use

As described above, people may incidentally ingest soil outdoors as well as outdoor soil that has been transported indoors. In calculating SCOs for children and adults for the unrestricted, residential and restricted residential land-use categories, we assumed that ingestion of outdoor soil only occurs during the warmer months of the year. To define this time period, we examined maps (developed by the Cornell University Cooperative Extension and available at http://www.cce.cornell.edu) showing the dates of the last spring frost and the first fall frost. These maps indicate that the latest date for first fall frost (after November 10) and earliest date for last spring frost (before April 10) occur in Kings, Nassau, Queens, Richmond and Suffolk Counties. Based on this information we assumed that outdoor soil ingestion only will occur during a 31-week period (217 days) from early April through early November.

We also assumed that activity patterns of children and adults will further limit the number of days on which soil ingestion may occur. For unrestricted, residential and restricted residential land uses, we assumed that children are not outdoors every day (due to inclement weather, travel away from home, etc.), and that they ingest outdoor soil five days per week. We assumed that indoor dust ingestion by children occurs each day of the year. For adults we assumed that soil ingestion occurs two days per week, through activities such as gardening and lawn care. We also assumed that adults may ingest some indoor dust derived from outdoor soil and that this exposure is included in the assumed ingestion of 100 mg of soil per day, two days per week.

Applying the above time-weighting assumptions to the child soil ingestion rate (80 mg/day soil; 40 mg/day dust) and adult soil ingestion rate (100 mg/day) yields average daily soil ingestion rates of 74 mg/day for children and 17 mg/day for adults.

Commercial Land Use

As in the calculation of unrestricted, residential and restricted residential land use SCOs, we assumed that soil ingestion only occurs during a 31-week period (217 days). We also assumed that a child visitor will be present outdoors at a commercial site (e.g., a park) two days per week and will ingest outdoor soil on those days. We assumed that an adult worker at a commercial park will have exposure to contaminated soil on four of five working days per week. Applying these time-weighting assumptions to the child soil ingestion rate (53 mg/day soil) and adult commercial worker soil ingestion rate (50 mg/day) yields average daily soil ingestion rates of 9 mg/day for children and 17 mg/day for adults.

Industrial Land Use

As in the calculation of unrestricted, residential, restricted residential, and commercial land use SCOs, we assumed that soil ingestion only occurs during a 31-week period (217 days). We assumed that an adolescent trespasser will be present at an industrial site one day per week, and that an adult worker at an industrial site will have exposure to contaminated soil on two of five working days per week. Applying these assumptions to the adolescent soil ingestion rate (100 mg/day) and adult industrial worker soil ingestion rate (50 mg/day) yields average daily soil ingestion rates of 8.5 mg/day for adolescents and 8.5 mg/day for adults.

3. Exposure Duration

In calculating cancer risks, exposure duration often is assumed to be shorter than the averaging time (e.g., a 25-year exposure duration averaged over a 70-year (lifetime) averaging time). An averaging time of 70 years is used in calculating cancer risks because the toxicological values used to calculate cancer risk are based on lifetime exposure. To calculate unrestricted, residential and restricted residential soil ingestion SCOs for cancer endpoints, we assumed a 70-year exposure duration and a 70-year averaging time. To calculate commercial and industrial soil ingestion SCOs for cancer endpoints we assumed a 25-year exposure duration and a 70-year averaging time. The 25-year exposure duration is based on information on occupational mobility in the US EPA's Exposure Factors Handbook (US EPA, 1997). The US EPA summarized "key

studies" that measured "occupational tenure" which was defined as the cumulative number of years a person worked in an occupation, regardless of number of employers, interruptions in employment, or time spent in other occupations. The US EPA recommends a median value of 6.6 years for working men and women 16 years and older. We did not consider this value because it is derived, in part, from data on younger workers who might have frequently changed occupations. For persons 70 years and older, the US EPA recommends a median value of 21.9 years for all workers (30.5 years for men only, and 18.8 years for women only). We chose the average of the values for men and women (25 years), which is approximately the same as the recommended value of 21.9 years. We used this value to derive adult worker (commercial and industrial land-use categories) soil ingestion SCOs for cancer endpoints.

4. Body Weight

In its Exposure Factors Handbook, the US EPA (1997) discusses published studies on body weight for the general US population. Based on an evaluation of "key studies," the US EPA recommends body weight values for children and adults of various ages. The US EPA's recommended mean body weight values for children (6 months to 19 years of age) and adults (18 to <75 years of age) are derived from 1987 National Center for Health Statistics data. The US EPA does not provide a single recommended value for children in general. To calculate non-cancer SCOs for children we used 13.3 kilograms (kg), which is the recommended body weight value for a 2-year-old child (mean value for boys and girls). To calculate non-cancer SCOs for adolescents, we used 58.1 kg, which is the recommended body weight value for a 15-year-old child (mean value for boys and girls). For adults (18 to <75 years), the US EPA recommends a value of 71.8 kg (mean value for men and women), and notes that this differs from the value of 70 kg commonly used in risk assessment. The US EPA also states that risk assessors who choose to use a value other than 70 kg should consider if dose-response relationships were derived using an assumed body weight of 70 kg. The use of 71.8 kg rather than 70 kg to calculate adult SCOs would have a minimal effect on the SCO values and, consistent with typical risk assessments, we used a body weight of 70 kg to calculate non-cancer SCOs for adults. To calculate SCOs for cancer endpoints, we used the body weight data shown in Table 5.3.1.1-1.

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5.2.2.2 Inhalation Pathway

People may be exposed to contaminants in soils by inhalation. Inhalation exposure may occur if 1) particulate-bound contaminants in surface soils are entrained in air (e.g., by the wind as fugitive dust) or 2) contaminants volatilize from soils. Individual exposures will depend on factors including, but not limited to, the chemical-specific air concentration at the time of exposure, the duration and frequency of exposure, an individual's breathing rate (which depends, in part, on an individual's activity level), and the amount of chemical inhaled and absorbed across the lung.

Evaluating the inhalation pathway requires information on the air concentrations of particulate-bound and volatile contaminants associated with contaminant concentrations in soil, exposure frequency and exposure duration. The approach we used to develop this information for deriving SCOs is described below.

1. Particulate-Bound Contaminants

We reviewed the methods used by other states to develop soil cleanup standards based on inhalation exposures to particulate-bound contaminants. Numerous states have used the approach developed by the US EPA in its Soil Screening Guidance documents (US EPA, 1996; 2002a). We were unable to identify any validation studies for this approach. However, the US EPA's soil screening guidance documents were subject to internal US EPA review and external peer-review for which the US EPA solicited comments from States, non-governmental and environmental organizations, industry representatives, and the general public. Also, the US EPA held outreach meetings and asked a Science Advisory Board to review the technical aspects of the documents (personal communication with Janine Dinan, US EPA, May 2005).

We used the approach presented by the US EPA in its Soil Screening Guidance documents for evaluating chemical-specific soil and air concentrations for particulate-bound contaminants. This approach yields a "particulate emission factor" (PEF) which relates the concentration of particulate-bound contaminants in soil to air concentration estimates for respirable soil particles

(particulate matter with a mass-median aerodynamic diameter of 10 microns or less, PM_{10}) entrained by winds in the outdoor air. The derivation of the PEF is based on an approach developed for assessments of exposure to particulates from a contaminated site. This approach used empirically-based relationships, based on field measurements and climatic conditions, to characterize the erosion potential of soils and estimate exposures to respirable particulates (US EPA, 1985).

Inhalation of particulate-bound contaminants was evaluated for contaminants, likely to be associated with surface soils (e.g., the top 2-3 centimeters, about an inch). We limited our evaluation of particulate-bound inhalation exposures to inorganic contaminants (such as metals) and semi-volatile compounds because surface soils are likely to be depleted of volatile organic compounds for sites with aged spills and/or chemicals with high mobility. This is consistent with the approach in the Soil Screening Guidance documents (US EPA 1996; 2002a).

According to US EPA (2002a), inhalation of fugitive dusts (and particulate-bound soil contaminants) are most likely to occur for site conditions such as dry soils, finely divided or dusty soils (high silt or clay content), high average annual wind speeds, and site parcels with less than 50 percent vegetative cover. We did not evaluate exposures due to activities that are likely to generate high dust levels including heavy truck traffic on unpaved roads and construction-related activities. PEF is calculated as shown in the following equation.

$$PEF = Q/C \times \frac{3{,}600\ s/hr}{R \times (1 - V) \times \left(U_m / U_t \right)^3 \times F(x)}$$

Where:

PEF = particulate emission factor (m^3/kg)
Q/C = dispersion term (the inverse of the mean air concentration at the center of square 0.5-acre area source, g/m^2-s per kg/m^3)
R = respirable fraction emission rate (g/m^2-hr)

V = fraction of vegetative cover (unitless)

U_m = mean annual wind speed (m/s)

U_t = equivalent threshold friction velocity value of the wind speed at 7-meters (m/s)

F(x) = wind speed distribution function dependent on U_m/U_t (unitless)

Using the PEF equation and these parameter values (discussed below), the PEF is 1.21 E+9 m^3/kg.

Dispersion Term - Q/C

This term accounts for the dispersion (i.e., mixing) of contaminants in air after they are emitted from soil. The US EPA developed this term using a dispersion model to estimate on-site long-term (annual) air concentrations for square area sources of various sizes (US EPA, 1996; 2002a). A dispersion model is a computerized set of mathematical equations that merges contaminant emissions data with meteorological data, such as wind speed and direction, to estimate contaminant concentrations in air. The dispersion term (in units of g/m^2-s per kg/m^3) is the mathematical inverse of the mean concentration at the center of a square source on a unit area emission rate basis (i.e., the predicted air concentration is based on an emission rate of 1 g/m^2-s).

The dispersion model that the US EPA used to develop the Q/C term is the Industrial Source Complex model. The US EPA used this model to estimate ground-level ambient air concentrations of soil contaminants released from ground-level area sources. (The term area source refers to contaminant sources that are dispersed over an area, in contrast to a point source such as a smokestack). The US EPA ran the area source model for a number of square source areas ranging from 0.5-acre to 600-acres (US EPA, 1996). In running the model, the US EPA used a full year of meteorological data from 29 US locations, chosen to be representative of the national range of meteorological conditions (US EPA, 1996). In its Supplemental Soil Screening Guidance, the US EPA (2002a) updated the dispersion model based on five years of meteorological data and provided algorithms to estimate dispersion terms for each of the 29 US locations and a variety of site sizes.

The US EPA approach estimated ground-level concentrations. However, people would typically be exposed to site-related contaminants at heights above ground-level. Therefore, we evaluated possible differences in ground-level versus breathing height level concentrations. To do this, we used US EPA's Screen3 (Lakes Environmental, 1995) model to estimate the difference between ground-level concentrations and air concentrations in the breathing zone of an adult (5-6 feet). These estimates suggest that the breathing zone air concentrations may be approximately two-times lower than the predicted ground-level concentrations. We did not account for this difference in developing the inhalation SCOs.

In the Soil Screening Guidance, the US EPA divides the country into "climatic zones" within which the meteorological conditions of the 29 assessed locations apply. New York State falls into two climatic zones, #7 and #8. The US EPA conducted dispersion modeling using meteorological data for four cities within these zones (Cleveland, OH; Harrisburg, PA; Hartford, CT; and Philadelphia, PA). We derived Q/C for each of these four cities using US EPA methods (2002a) assuming a site size of 0.5 acre. That is, we assumed that the area of contamination at a Brownfield site is 0.5 acre. This site size is consistent with the site size for the US EPA's default dispersion term for the Soil Screening Guidance (US EPA, 1996). In its Soil Screening Guidance, US EPA states that the point of maximum ground-level concentration was located at the center of the source and that the maximum concentration represented by the 600 acre source is 2.9 times higher than that of the 0.5 acre source (US EPA, 1996). We averaged the four Q/C values (85.63, 87.17, 73.95, and 87.37 g/m^2-s per kg/m^3 for Cleveland, Harrisburg, Hartford, and Philadelphia, respectively) to represent possible meteorological conditions in New York State. Using this approach we estimated that the dispersion term (or Q/C term) for a 0.5 acre site is 83.53 g/m^2-s per kg/m^3.

Respirable Fraction Emission Rate

US EPA defines this parameter (R) as the emission rate of respirable particulate matter (PM_{10}). In its Soil Screening Guidance documents (US EPA, 1996; 2002a), the US EPA identifies a default value for this parameter (i.e., 0.036 grams per square meter per hour, g/m^2-hr). We used the US EPA's default value for the emission rate of the respirable fraction of fugitive dust.

Vegetative Cover

The amount of vegetative cover present at a site (V) will affect the amount of bare soil available to become entrained in the wind as fugitive dust. In its Soil Screening Guidance documents (US EPA, 1996; 2002a), the US EPA identifies a default value of 0.5 for this parameter (i.e., half of the site has vegetative cover). We decided to use the US EPA's default value. Although the US EPA uses the term "vegetative cover," the value for this parameter would also account for other areas of a site that are not exposed to winds (e.g., paved areas).

Meteorological Conditions

Meteorological conditions (e.g., intensity of winds) affect emissions and dispersion of particulate matter. The approach developed by the US EPA (1985) relates the effects of wind speed on fugitive dust generation using certain meteorological parameters. These parameters are the mean annual wind speed (U_m), the threshold friction velocity wind speed at 7-meters above ground surface (U_t), and an empirically-based function dependent on these terms ($F(x)$).

For mean annual wind speed, we used US EPA default value of 4.69 m/s (US EPA, 1996; 2002a). This value is consistent with long-term average meteorological conditions in New York State (5 m/s) (Sedefian, 1982).

For the threshold velocity wind speed (at a height of 7-meters), we used the US EPA default value of 11.32 m/s (US EPA, 1996; 2002a). This parameter, which describes the wind speed necessary to generate fugitive dust, is a function of the size of surface soil aggregates and accounts for non-erodible elements at a site (e.g., grass and stones) that would consume the frictional forces of the wind (US EPA, 1996).

US EPA (1985) derived a function dependent on the mean annual wind speed and the threshold friction velocity wind speed. We used the US EPA default value for this term (i.e., 0.194). The method used to derive this term is described in US EPA, 1985.

2. Volatile Contaminants

People can be exposed to contaminants that volatilize from soils and mix with ambient air. Volatilization of contaminants in soils may be an important exposure pathway for chemicals that have a tendency to volatilize (such as VOCs[1] and elemental mercury). This section describes the approach used to evaluate inhalation exposure to contaminants that volatilize from soils to outdoor air. Vapor intrusion of volatile contaminants to indoor air is discussed in Section 5.2.4.1 (Vapor Intrusion Pathway).

Contaminant fate and transport in unsaturated subsurface soils (i.e., the vadose zone) is complex and dynamic. At equilibrium and steady-state conditions, subsurface contaminants are distributed between the soil organic matter and the water- and air- filled pore spaces in the soil matrix. Chemicals are more readily transported to the surface by diffusion through the air-filled spaces in the soil matrix than the water-filled spaces. Predictions of VOC emissions from the soil surface depends primarily on the initial soil concentrations, followed by the air-filled porosity (i.e., the air in the pore spaces of the soil matrix) (US EPA, 1996). Thus, in general, the higher the air-filled porosity, the greater the estimated rate of volatilization (US EPA, 1996).

Because soil characteristics differ, it is difficult to generically predict chemical behavior in the vadose zone without site-specific information. Relevant site-specific information includes soil moisture content, soil organic content, soil bulk densities, volume of contaminated soils at a site, and depth to groundwater. Chemical-specific information (e.g., chemical-specific properties and soil/soil gas concentrations) also can affect volatilization rates. The chemical-specific sorption partition coefficient[2] (K_d) and Henry's Law constant[3] (K_H) can help describe the distribution of a

[1] For the purpose of estimating inhalation exposures to volatile organic chemicals, we limited our evaluation to the priority chemicals listed as volatile in Section 4.0.

[2] The sorption partition coefficient (K_d) represents the distribution of a contaminant between the liquid and solid phases of the soils. Different soil types have different sorption capacities that are controlled, in part, by the amount of organic matter present in the soil (or fraction of organic carbon, f_{oc}). Sorption coefficients are usually normalized to be independent of organic carbon content and are known as the organic-carbon partition coefficient (K_{oc}). A soil-specific K_d is estimated as the product of the fraction of organic carbon (f_{oc}) and the organic-carbon partition coefficient (K_{oc}).

chemical between the soil organic matter and the water- and air- filled pore spaces in the soil matrix, provided that the soil saturation concentration is not reached.

The soil saturation concentration (C_{sat}) is the point 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. At soil concentrations above C_{sat}, soil contaminants may be present in free-phase and in some instances, non-aqueous phase liquids (NAPLs) may have a tendency to form in the soil matrix. In these cases, the K_H no longer is a valid description of the air-water partitioning (US EPA, 1996). However, volatilization rates probably do not increase substantially when soil concentrations exceed C_{sat} because when NAPL is present, the vapor density in the soil matrix is saturated and emissions plateau.

Mathematical relationships and chemical-specific properties can be used to estimate chemical behavior in subsurface soils and volatilization (or flux, mass emitted per unit area and time) to the soil surface. The available vapor-phase flux models can vary in complexity, accuracy and validity. We researched the methods used by other states to develop soil cleanup standards based on inhalation exposures to volatile chemicals in soils. Several states relied upon the methods presented in the US EPA's Soil Screening Guidance documents. Therefore, we focused our review on the methods presented by US EPA in its Soil Screening Guidance. The US EPA held outreach meetings with State, industry and environmental groups and solicited external peer-review comments on the soil screening guidance documents. The US EPA also convened a Science Advisory Board to review technical aspects of the documents (personal communication with Janine Dinan, US EPA 2005). In that guidance, mathematical constructs by Jury et al. (1984; 1990) serve as the basis for estimating chemical-specific volatilization factors[4] and inhalation exposures to soil contaminants.

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[3] The Henry's Law constant represents the distribution of a contaminant between the air and water phases. There are two forms of Henry's Law constants – one with dimensions (K_H, atm-m^3/mol) and one without dimensions (H). The correct format of this constant must be considered. Dimensionless forms of Henry's Law constants are estimated using the Ideal Gas Law Constant (R = 8.21×10^{-5} m^3-atm/K-mol) and temperature (298 degrees Kelvin); H = $(K_H)/(R)(T)$.

[4] A volatilization factor (VF) relates the soil concentration to the chemical-specific concentration in the ambient air due to volatilization.

The US EPA's generic screening method for estimating inhalation-based soil screening levels assumes that an "infinite source" of a volatile chemical in subsurface soils is available to volatilize. In its Soil Screening Guidance documents, the US EPA used this method to develop soil screening levels (SSLs) for inhalation exposures. The method employs several site-specific parameters (e.g., fraction of organic carbon, dry bulk density and soil moisture content), and the average time over which volatilization occurs (the US EPA refers to this term as an "exposure interval"). The approach does not require information on the depth of soil contamination (as it assumes soil contamination is uniform from the surface to an infinite depth), and the area of contamination is approximated in the dispersion term, Q/C (described previously under "Particulate-Bound Contaminants"). Chemical-specific parameters include K_d, H, and air and water diffusion rates. The US EPA acknowledges that the infinite source method may violate the mass balance assumption for certain contaminants (e.g., very volatile) and for certain site conditions (e.g., small contaminated areas) (US EPA, 1996). Although some chemicals may be more slow to volatilize because of strong interactions with soil organic matter or its solubility, assuming a volatile contaminant is present in the soil for an infinite amount of time may be unreasonable.

The US EPA also provides an approach to estimate contaminant volatilization rates for a "finite source" of contamination, where site contamination is well characterized (in terms of both the extent (e.g., depth of contamination) and magnitude of contamination), as are soil characteristics at the site. Many of the model assumptions for the finite source model are similar to the infinite source model however, the finite source model cannot account for subsurface contamination covered by a layer of clean soil (US EPA, 1996). Neither the infinite or finite source approaches account for the effects of site-specific meteorological and hydrological conditions on volatilization rates, nor, do they consider contaminant loss via microbial action or leaching to groundwater via rainfall percolation through soils. Additionally, the US EPA's approaches do not account for a high initial rate of volatilization (such as may occur after a recent spill) and the infinite and finite source approaches tend to incorrectly predict emissions prior to equilibrium conditions (US EPA, 1996). Both approaches assume that free-phase chemical is absent (i.e., no NAPL), and both approaches yield estimates of VF for individual chemicals; the validity of these models for interactions between multiple chemicals at a site is not well characterized. Although

the US EPA's finite source approach is more realistic than the infinite source approach for estimating volatilization from soils (particularly in situations where the infinite source model may violate the mass balance assumption), the approach relies heavily upon site-specific information such as the depth and magnitude of contamination.

In its 1996 guidance, the US EPA presents a limited validation of the Jury-based methods for infinite and finite sources. In this assessment, the US EPA used data from both bench-scale (controlled conditions) and pilot-scale (field conditions) experiments to evaluate predictions of volatilization rates for the several chemicals (dieldrin, lindane, benzene, toluene, ethylbenzene, and triallate) over relatively short time periods (approximately 7-40 days). Based on this limited assessment, the US EPA concluded that both models (infinite and finite source) showed good agreement with measured experimental emissions data, with mean modeled-to-measured ratios ranges of 0.42 to 0.81 for the bench-scale tests and 2.5 to 7.8 for the pilot-scale tests (US EPA, 1996). Our review of this limited assessment suggests that the models often over-predicts volatilization, particularly for the petroleum contaminants. Furthermore, because the available relevant research for the assessment was limited to a few contaminants studied over short time periods, we were not confident that the models are applicable to the range of volatile contaminants of interest (e.g., vinyl chloride) and the variety of conditions encountered in the field at contaminated sites. Additionally, we considered the assumption of an infinite source not to be reasonable for the development of inhalation SCOs. And, because the finite source approach required extensive site-specific information, we determine that it was not appropriate for development of SCOs statewide.

Given the uncertainties associated with the finite and infinite source approaches to estimate volatilization, we used the US EPA's "mass-limit" approach to develop inhalation SCOs for volatile chemicals. This approach does not rely extensively on site-specific characteristics and does not assume an infinite amount of contamination at the site. Although similar to a finite source model in some respects, the "mass limit" approach does not estimate the chemical-specific volatilization and does not require specific information about site conditions.

The "mass-limit" approach assumes that contaminant release from soil occurs at a constant rate over a specified duration of exposure. The US EPA uses the "mass-limit" approach to constrain the infinite source model (US EPA, 1996). We chose to use the mass-limit approach to estimate VF for all volatile contaminants to avoid the uncertainties and mass violations associated with the infinite source model. However, the mass-limit approach also has some weaknesses such as lack of chemical specificity and the potential to overestimate inhalation exposures for some chemicals (e.g., for those that are not very volatile, easily leach to groundwater, interact with the soil matrix). Also, the approach is not suitable when soil concentrations exceed Csat. The mass-limit approach is also sensitive to assumptions for the values of dry soil bulk density, depth of contamination and time over which volatilization occurs. The "mass-limit" VF is calculated as shown in the following equation (US EPA, 1996; 2002a).

$$VF = Q/C \times \left[\frac{\left(T \times 3.15E+7 \ s/yr \right)}{\left(\rho_b \times d_s \times 10^6 \ g/Mg \right)} \right]$$

Where:

VF = mass-limit volatilization factor (m^3/kg)
Q/C = dispersion term (the inverse of the mean air concentration at the center of square 0.5-acre area source, g/m^2-s per kg/m^3)
T = average duration of volatilization (years)
ρ_b = dry soil bulk density (Mg/m^3)
d_s = depth of contamination (meters)

Using the "mass-limit" VF equation and these parameter values, the VF is 2.67 E+4 m^3/kg.

Dispersion Term - Q/C

The dispersion term used to estimate the "mass-limit" VF is the same as that used to estimate PEF, 83.53 g/m^2-s per kg/m^3 (see above).

Average Duration of Volatilization

The "mass-limit" approach requires information on the amount of time over which volatilization occurs. The US EPA describes this term as an exposure interval (US EPA, 1996; 2002a). We assumed that the average duration of volatilization (or exposure interval) is 70 years, which is consistent with the exposure duration for residential land uses. Choosing seventy years may overestimate volatilization rates for chemicals that are less likely to volatilize and it may underestimate volatilization rates for chemicals that are more likely to volatilize. Choosing a lower value for this parameter would result in higher estimates of exposure for shorter duration of time. However, given that many other factors may influence volatilization rates as well, and some of these may result in overestimates of volatilization (e.g., not accounting for contaminant depletion over time and interactions with soil organic matter), shortening the exposure interval in the absence of data was not done.

Dry Soil Bulk Density

The dry soil bulk density is defined as the ratio the mass of dried soils to total soil volume. This variable is dependent on soil structure, type and moisture content (US EPA, 1998). We chose a soil bulk density of 1.5 kilogram of soil per liter (Kg/L; equivalent to Mg/m^3) consistent with the default value recommended by the US EPA (1996, 1998, 2002a). Soil bulk densities generally occur within a limited range, 1.3 – 1.7 Kg/L (US EPA, 1996).

Depth of Contamination

We assumed homogenous contamination to a depth of 4.6 meters (15 feet) below surface because the SCOs, developed under the proposed regulation for commercial and industrial land uses, are applicable to this depth.

3. Exposure Frequency

a. Unrestricted Land Use

Children and adults may be exposed to soil contaminants by inhalation at residential locations during the warmer months of the year. During the cooler months of the year (late fall, winter, and early spring), surface soils can be moist and may be frozen or the ground may be covered with snow. These conditions reduce the likelihood of fugitive dust generation and impede volatilization from soils. Therefore, we did not evaluate inhalation exposure to particulate-bound and volatile contaminants during the cooler months of the year.

Exposure to volatile and particulate-bound contaminants can occur outdoors and indoors. Many factors can affect the transport of outdoor contaminants to indoor spaces (e.g., building characteristics and the chemical and physical properties of the contaminant). The process of outdoor contaminants entering indoor air can be generally referred to as infiltration. For volatile contaminants, we assumed that contaminant infiltration from outdoor air to indoor air readily occurs. That is, we assumed no difference between contaminant concentrations indoors and outdoors for exposure to volatile contaminants. Infiltration of particulate-bound contaminants is variable and a function of particle sizes, in addition to building characteristics and ventilation rates (US EPA, 2004). We assumed that exposure to particulate-bound contaminants occurs outdoors (i.e., we did not account for particle infiltration to indoor spaces). Additionally, although some particle-bound contaminants may infiltrate homes (e.g., blow into home through open windows), a portion of these particles is incorporated into household dust. In Section 5.2.2.1 (Soil Ingestion Pathway), the child's soil ingestion rate accounts for a portion of indoor dust derived from outdoor soil and we assumed that this accounted for all indoor exposure to particulate-bound contaminants.

Adult and child exposures to volatile chemicals were assumed to occur seven days per week, 24 hours per day, during the warmer months of the year (total of 217 days/year). Adult exposures to particulate-bound contaminants was assumed to occur five days per week for three hours per day and two days per week for six hours a day, during the warmer months of the year (total of 35

days/year). We assumed that children are exposed to particulate-bound contaminants five days per week for three hours per day, during the warmer months of the year (total of 19 days/year). These exposure frequencies are consistent with those assumed for the Soil Ingestion Pathway (see Section 5.2.2.1)

b. Residential Land Use

Exposure frequencies used to develop inhalation SCOs for residential use are the same as those described for the unrestricted land use.

c. Restricted Residential Land Use

Exposure frequencies used to develop inhalation SCOs for restricted residential use are the same as those described for unrestricted and residential land use.

d. Commercial Land Use

We assumed that adult workers have inhalation exposure to both particulate-bound contaminants and volatile contaminants during outdoor work activities, and we assumed that these activities occur on four days of a five-day workweek during the warmer months of the year. We accounted for an increased respiration rate during work activities (i.e., the individual inhales half of their daily intake of air during work) (US EPA, 1994; 2002b) by assuming a 12-hour, rather than an 8-hour workday. Based on these assumptions, the exposure frequency for the adult worker is 62 days per year. We assumed a child visitor will be present outdoors at a commercial site two days a week, two hours per day during the warmer months of the year (total of five days per year), and during these times is exposed to particulate-bound and volatile contaminants by inhalation.

e. Industrial Land Use

We assumed that an adult worker at an industrial site works outdoors two days per week during the warmer months of the year (total of 31 days per year) and during these times is exposed to particulate-bound and volatile contaminants by inhalation. We accounted for an increased respiration rate while working by assuming a 12-hour, rather than an 8-hour workday (US EPA, 1994; 2002b). We also assumed that an adolescent trespasser will inhale particulate-bound and volatile contaminants while at an industrial site one day per week, for four hours per day during the warmer months of the year (total of five days per year).

4. Exposure Duration

In developing the inhalation SCOs, we assumed the same exposure durations that were used to develop the soil ingestion SCOs (Section 5.2.2.1).

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5.2.2.3 Dermal Pathway

People can be exposed to contaminants in soil through dermal contact with soil. Once in contact with the skin, some contaminants can be absorbed into the body and have the potential to cause systemic health effects. Some contaminants can be absorbed into the skin and have the potential to cause irritant contact dermatitis. In developing the SCOs, we assumed that people may dermally contact soil outdoors during the warmer months of the year during activities such as gardening, yardwork and play. Evaluating the dermal absorption pathway requires information on the amount of skin surface area in contact with soil, the amount of soil that adheres to the skin (called a soil adherence factor), the amount of chemical that is absorbed into the body from the soil on the skin (called an absorption fraction), exposure frequency, exposure duration and body weight. Evaluating irritant contact dermatitis requires soil adherence factors and an estimate of the amount of chemical that is absorbed into the skin itself. The data used to assign values to the parameters necessary for developing dermal SCOs are described below.

1. Exposed Surface Area

The magnitude of dermal exposure to chemicals in soil depends, in part, on the amount of surface area that may come into contact with soil. In its Exposure Factors Handbook, the US EPA (1997) summarizes a number of studies that used various approaches, including direct measurement techniques, to determine total body surface area and body part surface areas for people of different ages. In its recently published Supplemental Guidance for Dermal Risk Assessment (part of the Risk Assessment Guidance for Superfund (RAGS)), the US EPA (2004) used the information in the Exposure Factors Handbook to develop estimates of exposed surface area. We relied upon information in that document to define values for child, adult, and adolescent exposed surface areas.

<u>Children</u>

The US EPA (2004) assumes that children who play outdoors in residential settings wear a short-sleeved shirt and shorts, but no shoes. Therefore, the US EPA assumes that the head, hands, forearms, lower legs, and feet are potentially exposed to soil. We assumed that a child would not

routinely experience dermal exposure to soil for the entire surface area of the head (e.g., because of the presence of hair or possibly wearing a hat or cap) and that a smaller area, equivalent to that of the face, is exposed. Using US EPA (2004) data for the fraction of total surface for each of these body parts for a 2- to 3-year-old child (face – 0.0473; forearms – 0.0531; hands – 0.053; lower legs – 0.093; feet – 0.0707) and an average of the 50th percentile values of total surface areas for 2 to 3 year-old males and females (5900 square centimeters (cm^2)) yields an exposed surface area estimate of 1870 cm^2. We used this value to calculate dermal absorption SCOs for children for the unrestricted, residential, restricted residential, and commercial land-use categories.

Adults

The US EPA (2004) assumes that adults in residential settings wear a short-sleeved shirt, shorts, and shoes, and that the head, hands, forearms, and lower legs are potentially exposed to soil. As was the case for children, we assumed that adults would not routinely experience dermal exposure to soil for the entire surface area of the head. The sum of 50th percentile body part surface areas for males and females greater than 18 years of age (face – 402 cm^2; forearms – 1173 cm^2; hands – 904 cm^2; lower legs – 2370 cm^2) is 4850 cm^2. We used this value to calculate dermal absorption SCOs for adults for the unrestricted, residential and restricted residential land-use categories.

The US EPA (2004) assumes that adult workers in commercial or industrial settings wear short-sleeved shirts, long pants, and shoes. Assuming that the potentially exposed surface area includes the face (402cm^2), forearms (1173 cm^2) and hands (904 cm^2) yields a total estimated surface area of 2480 cm^2. We used this value to calculate dermal absorption SCOs for adult workers for the commercial and industrial land-use categories.

Adolescents

The US EPA (2004) did not describe a dermal exposure scenario for adolescents. We assumed that adolescents who might occasionally trespass at industrial sites wear a short-sleeved shirt,

shorts, and shoes, and that the face, hands, forearms, and lower legs are potentially exposed to soil. Using US EPA (2004) data for the fraction of total surface area for each of these body parts for a 15-year-old adolescent (face – 0.0265; forearms – 0.0590; hands – 0.0568; lower legs – 0.134) and an average of the 50th percentile values of total surface areas for 15-year-old males and females (16,400 cm^2) yields an exposed surface area estimate of 4530 cm^2. We used this value to calculate dermal absorption SCOs for adolescents for the industrial land-use category.

2. Soil Adherence Factors

The term "soil adherence factor" describes the amount of soil that adheres to skin (i.e., mass per unit area, such as milligrams of soil per square centimeter of skin (mg/cm^2)). The US EPA (1997) summarizes a number of studies in which the investigators measured the amount of soil adhering to the skin of people engaged in various soil contact activities. Collectively, these studies contain information on activity-specific and body part-specific soil adherence factors for males and females of various ages. The US EPA (1997 and 2004) concluded that these studies demonstrate that: (1) soil properties (such as moisture content and particle size) influence adherence;, (2) soil adherence varies for different parts of the body; and (3) soil adherence varies with activity. In the RAGS Dermal Risk Assessment Guidance, the US EPA (2004) uses the data presented in the Exposure Factors Handbook (US EPA, 1997) to develop recommended soil adherence factors for adults and children in different exposure settings. In deriving these recommendations, the US EPA calculated adult and child surface area-weighted soil adherence factors for a number of different activity patterns. The surface area-weighted soil adherence factors account for the differential soil adherence of the various exposed body parts (e.g., hands) and the surface area of those body parts. We relied upon this analysis to define the adult and child soil adherence factors used to develop dermal SCOs.

<u>Children</u>

For children in residential settings, US EPA (2004) calculated surface area-weighted soil adherence factors (geometric mean and 95th percentile) for the following exposure scenarios/activities: playing indoors, day-care children playing indoors and outdoors, playing in

dry soil, playing in wet soil, and playing in mud. In all cases, the surface area-weighted activity factor was calculated using soil adherence data for the face, hands, forearms, lower legs, and feet. In selecting a value to represent the soil adherence factor for children in residential settings, the US EPA considered two options: (1) select a central tendency (typical) soil contact activity and use the high end (95^{th} percentile) weighted soil adherence factor for that activity; or (2) select a high-end soil contact activity and use the central tendency (geometric mean) weighted soil adherence factor for that activity. (The US EPA considered these same options in selecting soil adherence factors for adults.) As indicated by the US EPA, the geometric mean adherence factors are more stable estimates of the true adherence factors than the 95^{th} percentile values because outlier values can more significantly affect the 95^{th} percentile values. The US EPA determined that a child playing in wet soil represents a reasonable high-end activity; the geometric mean weighted soil adherence factor for this activity is 0.2 mg/cm^2. We used a soil adherence factor of 0.2 mg/cm^2 to develop unrestricted, residential, restricted residential, and commercial dermal SCOs for children.

Adults

For adults in residential settings, the US EPA (2004) calculated surface area-weighted soil adherence factors (geometric mean and 95^{th} percentile) for the following exposure scenarios/activities: groundskeeping, landscaping, and gardening. In all cases, the surface area-weighted soil adherence factor was calculated using adherence data for the face, hands, forearms, and lower legs. The US EPA concluded that gardening represents a reasonable high-end activity pattern for residential adults; the geometric mean soil adherence factor for this activity is 0.07 mg/cm^2 and this is the value that the US EPA recommends for adults in residential settings. For comparison, a 95^{th} percentile soil adherence factor of 0.06 mg/cm^2 is associated with groundskeeping work (a central tendency activity). We used a soil adherence factor of 0.07 mg/cm^2 to develop unrestricted, residential and restricted residential dermal SCOs for adults.

For adults in commercial/industrial settings, the US EPA (2004) calculated surface area-weighted soil adherence factors (geometric mean and 95^{th} percentile) for the following exposure scenarios/activities: groundskeepers, landscapers, gardeners, pipe installers (dry and wet soil),

irrigation installers, construction workers, heavy equipment operators, and utility workers. In all cases, the surface area-weighted soil adherence factor was calculated using adherence data for the face, hands, and forearms. The US EPA concluded that utility work represents a reasonable high-end activity pattern for adults working in commercial/industrial settings; the geometric mean soil adherence factor for this activity is 0.2 mg/cm^2 and this is the value that the US EPA recommends for adult commercial/industrial workers. This is the same geometric mean soil adherence factor for another high-end activity pattern – heavy equipment operators. For comparison, 95^{th} percentile soil adherence factors range from 0.1 to 0.3 mg/cm^2 for commercial/industrial activities such as groundskeeping, landscaping, pipe laying (dry soil), installing irrigation systems, and gardening (activities that the US EPA considered to be more representative of central tendency soil contact for workers in commercial/industrial settings). We used a soil adherence factor of 0.2 mg/cm^2 to develop commercial and industrial dermal SCOs for adult workers.

Adolescents

The US EPA has not developed a recommended soil adherence factor for older children (e.g., adolescents) that would be directly applicable to the adolescent trespasser scenario. However, the US EPA (2004) calculated weighted soil adherence factors of 0.04 mg/cm^2 (geometric mean) and 0.3 mg/cm^2 (95^{th} percentile) for teenagers playing soccer (males only, ages 13 to 15). In the exposure scenarios for dermal SCOs, both residential adults and adolescent trespassers are assumed to have dermal contact with soil on the face, hands, forearms, and lower legs. The residential adult soil adherence factor of 0.07 mg/cm^2 is a weighted value that represents these body parts. The value also represents reasonable high-end soil contact activity. We therefore used a soil adherence factor of 0.07 mg/cm^2 to develop dermal SCOs for adolescent trespassers in industrial settings.

3. Absorption Fraction

The term “absorption fraction” refers to the amount of a chemical that is absorbed into the body from the soil on the skin. The Exposure Factors Handbook (US EPA, 1997) describes this term,

but provides no information on chemical-specific absorption fractions; readers of the handbook are referred to the US EPA's (1992) "Dermal Exposure Assessment: Principles and Applications." In that document, the US EPA discusses some methods that have been used to estimate exposure to chemicals in a soil matrix and uncertainties that exist in estimating the extent to which a chemical is transferred from soil into the skin. For example, absorption of a chemical from soil may depend on characteristics of the soil (e.g., particle size and organic carbon content), processes occurring in soil (e.g., resorption to and diffusion through the soil, volatilization), and the amount of soil in contact with the skin.

In its review of information on chemical absorption from soil, the US EPA (1992) indicates that experimentally derived absorption fractions should be given priority in developing estimates of dermal exposure for chemicals in soil. The US EPA also indicates that predictive procedures can be used in the absence of experimentally derived factors, but such procedures are not well developed. The 1992 dermal exposure document recommends ranges of absorption fractions for several chemicals (i.e., 0.001 to 0.03 for 2,3,7,8-TCDD; 0.006 to 0.06 for 3,3',4,4'-tetrachlorobiphenyl; 0.001 to 0.01 for cadmium) and indicates that those ranges are appropriate for exposure times equal to or less than 24 hours. In its 2004 RAGS document, the US EPA indicates that it considered the recommendations in its 1992 dermal exposure document and, based upon a review of the literature, developed recommended absorption fraction values for a larger set of chemicals. The US EPA indicates that the values it recommends are experimental mean values and are applicable to the exposure assumptions (e.g., exposed surface areas, soil adherence factors) recommended in the RAGS document. The absorption fraction values recommended by the US EPA (2004) are shown in Table 5.2.2.3-1. We used these US EPA-recommended absorption fractions to estimate dermal exposure in developing dermal absorption SCOs for all land use categories. The absorption fractions we used are summarized Table 5.2.2.3-2.

As described in Section 5.1.4 (Toxicity Values for Non-allergic Skin Irritation) and Appendix C-1, we evaluated irritant contact dermatitis for phenol, a surrogate SVOC, nickel, and chromium VI. This analysis requires an estimate of the amount of a chemical that is absorbed into the skin (i.e., epidermis and dermis) from the soil on the skin. For phenol and a surrogate SVOC, we

used the generic absorption fraction recommended by the US EPA (2004) for the systemic absorption of SVOCs (0.1). We used the recommended generic value for all SVOCs rather than the recommended value for individual SVOCs, including pesticides, (see Table 5.2.2.3-1) because the goal of the analysis (a generic evaluation applicable to all SVOCs) does not warrant the use of contaminant-specific absorption fractions with surrogate SVOC irritancy data.

The US EPA did not recommend systemic absorption fractions for nickel or chromium VI. For these chemicals, we used epidermis absorption fractions derived from limited studies of nickel salts and sodium chromate. For nickel, we used 0.01, which was derived from studies (Hostynek et al., 2001; Tanojo et al., 2001) that estimated the percentage of applied nickel salts that penetrated various layers of the human epidermis both *in vivo* and *in vitro*. For chromium, we used 0.04, which was derived from studies that estimated the percentage of applied aqueous solutions of sodium chromate that disappeared from the skin of guinea pigs (Wahlberg and Skog, 1963). These studies did not determine the percentage of the applied dose that entered general circulation within the body. They did not provide data that could be used to calculate a fraction of the applied nickel or chromium that entered into the body. Thus, we did not use these data to derive dermal absorption SCOs.

4. Exposure Frequency, Exposure Duration and Body Weight

In developing the dermal absorption SCOs, we assumed the same exposure frequencies, exposure durations, and body weights that were used to develop the soil ingestion SCOs. These parameters are described in Section 5.2.2.1 (Soil Ingestion Pathway).

References

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Table 5.2.2.3-1. US EPA Recommended Dermal Absorption Fractions.

Chemical	Dermal Absorption Fraction
Arsenic	0.03
Cadmium	0.001
Chlordane	0.04
2,4-Dichlorophenoxyacetic acid	0.05
DDT	0.03
Lindane	0.04
Benzo[a]pyrene and other PAHs	0.13
Pentachlorophenol	0.25
Semivolatile organic compounds	0.1

Source: US EPA (2004)

Table 5.2.2.3-2. Dermal Absorption Fractions Used to Develop Dermal Absorption SCOs.

Volatile Organic Compounds	Absorption Fraction	Volatile Organic Compounds	Absorption Fraction
Acetone	--	1,2-Dichloroethene (cis)	--
Benzene	--	1,2-Dichloroethene (trans)	--
2-Butanone (methyl ethyl ketone)	--	1,4-Dioxane	--
n-Butylbenzene	--	Ethylbenzene	--
sec-Butylbenzene	--	Methylene chloride	--
tert-Butylbenzene	--	Methyl tert-butyl ether	--
Carbon tetrachloride	--	n-Propylbenzene	--
Chlorobenzene	--	Tetrachloroethene	--
Chloroform	--	Toluene	--
1,2-Dichlorobenzene	--	1,1,1-Trichloroethane	--
1,3-Dichlorobenzene	--	Trichloroethene	--
1,4-Dichlorobenzene	--	1,2,4-Trimethylbenzene	--
1,1-Dichloroethane	--	1,3,5-Trimethylbenzene	--
1,2-Dichloroethane	--	Vinyl chloride	--
1,1-Dichloroethene	--	Xylenes	--

Semi Volatile Organic Compounds	Absorption Fraction	Semi Volatile Organic Compounds	Absorption Fraction
Acenaphthene	0.13[2]	Fluorene	0.13[2]
Acenaphthylene	0.13[2]	Hexachlorobenzene	0.1[1]
Anthracene	0.13[2]	Indeno[1,2,3-cd]pyrene	0.13[2]
Benz[a]anthracene	0.13[2]	2-Methylphenol	0.1[1]
Benzo[b]fluoranthene	0.13[2]	3-Methylphenol	0.1[1]
Benzo[k]fluoranthene	0.13[2]	4-Methylphenol	0.1[1]
Benzo[g,h,i]perylene	0.13[2]	Naphthalene	0.1[1]
Benzo[a]pyrene	0.13[2]	Pentachlorophenol	0.25
Chrysene	0.13[2]	Phenanthrene	0.13[2]
Dibenzofuran	0.1[1]	Phenol	0.1[1]
Dibenz[a,h]anthracene	0.13[2]	Pyrene	0.13[2]
Fluoranthene	0.13[2]		

Table 5.2.2.3-2. Dermal Absorption Fractions Used to Develop Dermal Absorption SCOs. (continued)

Pesticides	Absorption Fraction	Pesticides	Absorption Fraction
Aldrin	--	delta-Hexachlorocyclohexane	--
alpha-Hexachlorocyclohexane	--	Dieldrin	--
beta-Hexachlorocyclohexane	--	Endosulfan (I, II and Sulfate)	--
alpha Chlordane	0.04	Endrin	--
4,4'-DDD	--	Heptachlor	--
4,4'-DDE	--	gamma-Hexachlorocyclohexane (lindane)	0.04
4,4'-DDT	0.03	2-(2,4,5-Trichlorophenoxy) propionic acid	--

Inorganics	Absorption Fraction	Inorganics	Absorption Fraction
Arsenic	0.03	Lead	--
Barium	--	Manganese	--
Beryllium	--	Mercury (elemental)	--
Cadmium	0.001	Mercury (inorganic salts)	--
Chromium III	--	Nickel	--
Chromium VI	--	Selenium	--
Copper	--	Silver	--
Cyanide	--	Zinc	--

(1) Assigned the US EPA (2004) value of 0.1 for semivolatile organic compounds.
(2) Assigned the US EPA (2004) value of 0.13 for benzo[a]pyrene and other PAHs.

5.2.2.4 Dietary Exposure Pathways

People can have ingestion exposures to soil contaminants through consumption of foods. For example, vegetables that are harvested from plants grown in contaminated soil can contain contaminants that originated in the soil. Foods that are produced from farm animals like cows, pigs and chickens also can contain contaminants that originated from soil. Similarly, fish can contain contaminants that originated in the soil. Consumption of these foods can result in the dietary ingestion exposure pathways, as discussed below.

Children and adults can be exposed to soil contaminants through consumption of garden vegetables that were grown in the soil. Soil contaminants can be incorporated into vegetable plants in a number of different ways. For example, contaminants can be taken up into plants through the roots (along with water and nutrients), and either remain stored in the roots or be distributed to other plant parts. Some contaminants can volatilize from soil and then sorb to leaves and other plant parts. Soil particles can also adhere to roots and above ground parts of vegetable plants. Quantitatively evaluating the magnitude of soil contaminant exposure through the vegetable consumption pathway requires information about rates of consumption of vegetables, the relationship between contaminant concentrations in soil and those in vegetables grown in the soil, as well as body weight, and exposure frequency and duration for hypothetically exposed individuals. A large amount of information was considered in an effort to assign values to these parameters for developing vegetable pathway SCOs. This section of the Technical Support Document contains a discussion of the parameters – homegrown vegetable consumption, body weight, exposure duration, exposure frequency, and vegetable contaminant concentrations.

People can also be exposed to soil contaminants through consumption of meat and dairy products that are produced from animals raised on a site (i.e., a farm) with contaminated soil. As farm animals incidentally ingest soil, and consume locally growing grasses and other forage plants that may have incorporated the soil contaminants, the contaminants can enter their bodies - and thus become associated with meats (e.g., beef, pork, chicken). Once a contaminant enters the body of an animal (e.g., a cow or a chicken), it can be also be distributed within the body in such

a way as to become part of other animal product foods (e.g., cow's milk or chicken eggs). Quantitatively evaluating the magnitude of soil contaminant exposure through consumption of meat and dairy products produced on a site with soil contamination requires knowledge of the relationship between soil contaminant concentrations and contaminant concentrations in pasture grass or forage plants, the various types of meats and dairy products that are consumed, as well as the rates at which these foods are consumed.

Finally, people can be exposed to soil contaminants through consumption of fish. Soil contaminants can enter the bodies of fish directly from the water, or through the food chain, from microorganisms and algae, to zooplankton, invertebrates, and smaller fish. Quantitatively evaluating the magnitude of soil contaminant exposure to receptors through consumption of fish requires knowledge of the relationship between soil contaminant concentrations and water concentrations, the amount of the contaminant that is present in the various types of fish that are consumed, and the rates at which these fish are consumed.

1. Meat, Dairy, Fish and Vegetable Consumption Rates

Calculation of SCOs that quantitatively account for the consumption of meats, dairy products, fish and vegetables requires estimates of relevant food consumption rates for the appropriate child and adult receptors. The US EPA Exposure Factors Handbook (US EPA, 1989, 1997) is a source of data from which such estimates may be obtained.

The 1989 Exposure Factors Handbook (EFH) presents consumption rates for vegetables, broken down into three categories (protected, leafy, and exposed) based on a nationwide survey. These data, while useful, require adjustment for the development of residential land use SCOs to reflect only the homegrown portion of vegetable consumption rates. The 1997 Exposure Factors Handbook (EFH) provides data on homegrown vegetable consumption rates, including that specifically for the Northeastern United States. For the unrestricted land use SCOs, these EFH vegetable consumption rates may need to be adjusted upwards to account for the generally higher rates of vegetable consumption for adults and children living on a farm compared to those for people living on non-agricultural residential properties (e.g., see Moya and Phillips, 2001).

Human consumption rates for beef, pork, chicken, fish, cow's milk, and chicken eggs can also be found in the EFH. These same rates are recommended for use in risk assessment practice by EPA (US EPA, 1998). However, some of these rates may need to be adjusted to account for higher consumption for adults and children living on a farm (Moya and Phillips, 2001).

In general, consumption rates for meat, dairy products, fish and vegetables are provided on a per-kilogram-body-weight basis, and it is assumed that they are relevant to both children and adults. There is some uncertainty implicit in this assumption.

2. Body Weight, Exposure Duration, and Exposure Frequency

Both adult and child residents can be exposed to soil contaminants via dietary ingestion exposures including consumption of garden vegetables. Adults and children who live on farms may be exposed to onsite soil contaminants through consumption of vegetables and fish, as well as meats and dairy products produced from animals, that have been exposed to the soil contaminants. Contaminant exposures through these dietary ingestion pathways are considered to be additive with concurrent exposure through incidental soil ingestion, dermal contact, and inhalation (for all contaminants associated with systemic toxicity by inhalation). For this reason, it is appropriate to estimate multiple pathway exposure for the same hypothetical individuals ("receptors") for each pathway. For the sake of consistency and simplicity, body weights for children and adult receptors in the estimation of exposure for the dietary ingestion pathways, can be considered identical to those chosen for the incidental soil ingestion pathway. However, as mentioned previously, data on rates of consumption of meat, dairy products, fish and vegetables are reported on a per-kg-body-weight basis. Therefore, the receptor's body weight is actually of no consequence in estimating exposure with these consumption rates. Exposure durations for these pathways can also be set equal to those for the residential category soil ingestion pathway, as exposures for each of these pathway coincides with periods of residence for the hypothetical receptors. However, exposure frequency, while limited to the warmer months of the year for soil ingestion, may not be so limited for consumption of locally produced meats, dairy products, fish and vegetables. Current practices of animal husbandry, as well as freezing, canning, and other

long-term storage options suggest that locally produced foodstuffs may be available for consumption throughout the year. Thus, daily consumption rates can be applied to all 365 days of the year.

3. Contaminant Concentrations in Vegetables

The transfer of soil contaminants to plants has been well documented. Field and greenhouse studies have demonstrated that, for the most part, contaminant concentrations in plants increase with increasing soil contaminant concentration. However, plant contaminant concentrations can be difficult to predict from soil contaminant concentration alone. Aside from soil concentration, the level of a contaminant in a plant may vary with properties of the contaminant (e.g., lipophilicity, solubility, volatility), soil characteristics (e.g., pH, fraction organic matter, mineral content), environmental factors (temperature, wind, rain), plant-related characteristics (species, plant part), and other factors. Scientists have attempted to construct conceptual and mathematical models to account for many of these factors to predict plant tissue concentration (Trapp and McFarlane, 1995). However, the complexity of these models, and their requirement for highly detailed and specific input data (which are often unavailable), tend to limit their usefulness and applicability in environmental health practice.

A more commonly used approach to estimating vegetable concentrations is based on calculation of a central tendency ratio of plant concentration to soil concentration as measured and reported in one or more studies. Assuming a linear relationship between soil and plant concentrations, these "uptake factors" can be used to predict concentrations for other plants in other soils. This approach is particularly well established for metals and semi-metals. Many papers have been published with data that can be used to calculate uptake factors, and other documents have summarized such findings. One of the more commonly cited summaries of uptake factors was published by Oak Ridge National Laboratory (Baes et al., 1984). This summary reports uptake factors for vegetative and non-vegetative (reproductive) plant parts for each of 88 elements.

A variation of calculating a central tendency ratio of plant concentration to soil concentration is based on a linear regression of these ratios versus soil concentration. This approach yields a

linear equation – rather than a single factor - that can be used to predict uptake. This approach is put forth in another comprehensive review of elemental uptake compiled by a consultant for the US Department of Energy (Bechtel Jacobs Company, 1988).

For organic contaminants, some studies have suggested that there is a relationship between observed uptake and the lipophilicity of the chemical. For example, Travis and Arms (1988) aggregated previously reported soil and above-ground plant concentration data for 29 chemicals and found a log-linear relationship between the uptake factor and the octanol-water partition coefficient (K_{ow}), with less lipophilic chemicals having the most uptake. In contrast, Briggs et al. (1982) conducted experiments with barley seedlings growing in water, and found that the most lipophilic compounds exhibited high uptake in barley roots. However, the authors of this paper also observed that uptake did not vary much with $\log K_{ow}$ for the most polar compounds. The Briggs model can be readily adapted to predict plant concentrations from soil concentrations instead of water concentrations, by assuming equilibrium partitioning between soil and water, and applying a chemical-specific organic carbon-water partition coefficient (K_{oc}) and an assumed soil fraction organic carbon (foc). Because both K_{ow} and K_{oc} are related to lipophilicity, a compound that has a lower tendency to partition from water to roots (lower K_{ow}), also has a lower tendency to partition to organic carbon (lower K_{oc}). Less partitioning to organic carbon (and organic matter) results in greater availability of a compound in the dissolved phase (porewater). In the adaptation of the Briggs equation to soil for the most polar compounds, this greater availability more than compensates for the lesser tendency to sorb to roots. Thus the adaptation of the Briggs et al., approach to soil results in the estimated highest root uptake for compounds with the highest and lowest K_{ow}s, with a minimum uptake at a $\log K_{ow}$ of around 2. The adaptation of the Briggs et al. approach to soil is explored in more detail in Ryan et al. (1988). Travis and Arms and Briggs et al. reported the equations for these relatively simple models, thus providing a mechanism for estimation of chemical-specific uptake factors in the absence of measured values.

Quantitative approaches to estimating the magnitude of exposure to soil contaminants through consumption of garden vegetables have been employed in several US federal guidance documents, and are sometimes used in human health risk assessments. US EPA documents

evaluating the risk associated with agricultural application of sewage sludge estimated plant uptake with a single uptake factor (0.001) for all organic chemicals and inorganic chemicals for which there were no empirical data. These documents also estimated uptake for several metals with chemical-specific uptake factors (US EPA, 1995). The *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* ("Combustor Guidance"), published by US EPA's Office of Research and Development (1999), includes methods to estimate exposure to air pollutants that have been deposited on and mixed into soil, taken up by edible plants, and ingested by human receptors. This guidance document generally employs Baes, et al. uptake factors for metals, and for organics, the Travis and Arms model to predict above ground plant part concentrations and the Briggs, et al. model for plant roots and tubers. The US EPA draft Dioxin Reassessment (US EPA, 2004) also puts forth a method of estimating plant uptake that accounted for root uptake, particle deposition, and vapor phase partitioning to above ground plant parts. The US EPA's recently revised draft *Guidance for Developing Ecological Soil Screening Levels* ("Eco-SSL Guidance", US EPA 2003, 2005) estimates exposure of ecological receptors to soil contaminants via consumption of above ground plant forage. For organic compounds evaluated in the Eco-SSL Guidance, the US EPA and their consultants reconstituted the Travis and Arms data set and added more recent data. The resulting combined data set was used to identify empirically based chemical-specific uptake factors for a number of contaminants and reassess the slope of the log-linear relationship of uptake factors with K_{ow}. Several European nations have accounted for exposure through consumption of vegetables in the development of soil standards or guideline values (Ferguson, 1999). For example, the United Kingdom, in published soil guideline values that account for home gardening, estimates vegetable uptake primarily with the Ryan et al. approach for organic contaminants and Bechtel Jacobs regression equations for metals (Environment Agency, 2002). However, few US states have included homegrown vegetable consumption in the derivation of soil standards or guideline values. The recently revised Massachusetts Contingency Plan (MCP) utilizes empirically derived vegetable uptake factors for several metals, chlordane, and PCBs (MA DEP, 2004). The CA EPA has included vegetable uptake in their soil screening number for lead (CA EPA, 2005). Texas, in their guidance for calculating soil protective concentrations, provides the reader with above-ground and below-ground uptake factors for several contaminants (TX NRCC, 1999). Thus far, the US EPA's Office of Solid Waste and Emergency

Response has included the vegetable uptake pathway for only six metals in their Soil Screening Guidance (US EPA, 1996).

4. Uncertainty in Contaminant Concentration Estimates for Vegetables

Although there are estimation methods available and there is precedent for quantification of the vegetable consumption exposure pathway, there is also considerable uncertainty in these methods. Much of the uncertainty associated with the vegetable consumption pathway stems from the need to estimate a concentration in one medium (vegetables) from that in another (soil).

First, neither one medium nor the other can be considered homogenous in any sense. Soil can vary greatly from one site or one region to another. Properties such as predominant mineral type, clay content, phosphate level, organic matter content, pH, cation exchange capacity, and particle size distribution are among those that differentiate one soil from another. These soil-specific properties can affect the availability of a chemical for transfer to other media (Miner et al., 1997; Millis et al., 2004). Vegetables are not homogeneous either. Vegetable "properties" including those of the parent plant such as species, variety, morphology, age, and vigor can affect rate of uptake of (or "receptiveness" to) an available contaminant, as well as its rate of translocation within (and loss from) the plant (Cataldo and Wildung, 1978). Concentrations in the vegetable itself can be a function of relative rates of uptake and loss, distribution in the plant, as well as physiological function and relative location of the plant part, length of growing season, the stage of harvest, and post-harvest treatment (e.g., washing, peeling, drying, cooking).

Second, a number of chemical, physical, and biological processes mediate the transfer (uptake) of a chemical from one heterogeneous medium to the other. Each of these processes can be influenced by many factors in addition to properties of the media, including environmental conditions (e.g., temperature, wind, and rain) (Dreicer et al., 1984).

Third, the relative amount of plant uptake of soil contaminants can vary with the contaminant, the absolute contaminant concentration, the contaminant species or form, and the aging or weathering status of the contaminant in the soil matrix (Lunney et al., 2004).

Each of the many factors that influence contaminant availability, translocation, plant uptake, and vegetable concentration has its own inherent level of influence on the vegetable concentration, and its own inherent variability. The extent to which the variability in any one factor translates into variability in the vegetable concentration depends, in part, on the relative influence of that factor, and this in turn is a function of the presence or magnitude of the other factors.

Under ideal circumstances, an appropriate central tendency value and measure of variability (e.g., variance) for each factor would be known. In addition, the quantitative relationships between the factors themselves and the vegetable concentrations would be understood. With this knowledge, a central tendency vegetable concentration and the expected variance in concentrations could be predicted with little uncertainty. Unfortunately, this level of understanding does not currently exist. The quantitative relationships between the factors and vegetable concentration are poorly understood. Only estimates of the central tendency and variability for each factor are available at best. Each estimate has inherent uncertainty, leading to substantial uncertainty in the final vegetable concentration estimate.

Many compilations of soil and plant concentration data contain little or no information on potentially influential factors. Instead, assumptions must be made that the factor values inherent in the reported concentration data are reasonably representative of appropriate central tendencies. For example, the values of many of these potentially influential variables are not reported along with plant-soil concentration ratios in the February 2005 revision of the US EPA's Eco-SSL Guidance. Table 5.2.2.4-1 presents data from the Eco-SSL Guidance that illustrate chemical-specific variability in uptake factors for three chemicals. The data for these chemicals based on a limited number of observations indicate that uptake factors range over one to two orders of magnitude. There is little to no accompanying information to suggest that the inherent values of influential factors are representative of appropriate central tendency values. That is, there is no information on soil type, organic matter content, environmental conditions, etc. The Eco-SSL Guidance does indicate that the data represent uptake into plant foliage. It is not apparent how well these data represent uptake in the kind of foliage that humans might consume. Furthermore,

it is not known whether foliage data are sufficiently representative of uptake for the range of vegetables that people consume (including root vegetables and fruits).

The problem of uncertainty in estimates based on empirical data is compounded for chemicals for which empirical data are completely unavailable – as is the case for many organic contaminants. In this case, observed log-linear relationships between available plant-soil ratios and K_{ow} have been used to predict plant-soil ratios for chemicals lacking empirical data, but with known K_{ow} values. Estimates based on a regression model can be heavily influenced by the empirical data for the chemicals with the lowest and highest K_{ow} values. This is illustrated in Figure 5.2.2.4-1, taken from the Eco-SSL Guidance, where the uptake factors ("BAF") for a number of different organic chemicals are regressed against $\log K_{ow}$. It is readily apparent that the data for the chemical(s) with the lowest $\log K_{ow}$ on the left of the graph can have a considerable influence on both the strength of the correlation (r^2) and the slope of the regression line. Clearly, uncertainty in the empirical data upon which the regression model is based can result in uncertainty in the regression-based estimates. Furthermore, an inherent assumption of the model is that K_{ow} is the primary chemical-specific factor associated with the plant-soil ratio. Such an assumption may not be valid for all chemicals. For example, recent studies have suggested that some chemicals may undergo significant biotransformation within the plant (Schnabel et al., 1997). The concentrations of such chemicals in plants may be overestimated by a K_{ow}-based uptake model.

5. Contaminant Concentrations in Meats, Dairy Products and Fish

It has been demonstrated that animal products, such as meats, eggs, and milk, can contain contaminants as a result of the animals' exposure to contaminated soil (Bruce et al, 2003, Harnly et al, 2000, Fries, 1985, Stachel et al. 2005). For example, eggs produced by chickens raised in yards containing soil contaminated with polychlorinated dioxins and furans had clearly elevated levels of these compounds when compared with eggs from chickens that had no contact with contaminated soil (Schuler et al., 1997). In addition to consuming grasses, grains or other vegetation that may contain soil contaminants, animals can take in soil contaminants by incidentally ingesting soil. Building upon available data on incidental soil ingestion rates (e.g.,

Fries and Paustenbach, 1990), feeding studies have also demonstrated significant soil contaminant bioavailability, with implications for efficient incorporation of contaminants into the tissues of grazing farm animals (Stephens et al, 1995).

In general, compounds that are found in the highest relative concentrations in animal products are those that are persistent and bioaccumulative. Most of the data in the literature pertaining to soil-to-animal transfer primarily suggest the availability of bioaccumulative chemicals for transfer into animal products - e.g., PCBs available for transfer into cow's milk, as described by Gill et al., (1992), or mercury available for transfer into fish, as described by Balogh et al., (2003). The potential for organic chemicals to bioaccumulate can be crudely predicted using values for chemical parameters found in the literature such as octanol-water partition coefficients. However, the accuracy of these methods is limited, as they do not take into account a number of factors, including the persistence of the chemical in the environment or in biota. Empirically derived estimates of potential for bioaccumulation can be found in the literature for some chemicals. However, these empirically derived estimates are often based on aquatic bioconcentration, are not directly applicable to terrestrial bioaccumulation. They are also not available for all contaminants. In spite of these impediments to identifying chemicals that bioaccumulate, there are a number of contaminants of concern that have been unequivocally identified as bioaccumulative and persistent by environmental or health organizations for various regulatory or programmatic purposes. For example, the United Nations Environmental Program identifies a number of these chemicals on its list of Persistent Organic Pollutants (POP) list (UNEP, 2006). Another list that identifies known bioaccumulative chemicals is the US EPA's group of 13 *Priority Persistent Bioaccumulative Toxics* (PBTs) (US EPA, 2006) currently being addressed under the Persistent Bioaccumulative and Toxic Chemical Program. Most of the data in the literature illustrate transfer from soil into animal products for compounds like those included on these lists. There are, however, data in the literature that illustrate that other less bioaccumulative chemicals (e.g., various metals) are also transferred into animal product foods (Manske, 2002).

Data in the literature provide a means of directly estimating food concentrations from soil concentrations by calculating soil-to-food concentration ratios or transfer factors for each

contaminant. While the data are generally limited to a few chemicals and a limited range of site conditions, this approach can be useful in developing a crude estimate of food concentrations.

There are also mathematical models available that can be used to estimate food concentrations from soil concentrations. One commonly used and relatively simple model allows the calculation of animal intake-to-animal product concentration biotransfer factors for beef and milk based on a positive log-linear relationship with the chemical's octanol-water partitioning coefficient (Travis and Arms, 1988). These transfer factors relate beef or milk concentration to the intake of a chemical through incidental ingestion of soil and consumption of forage, grain and silage. Estimated rates of ingestion of soil and consumption of forage, grain and silage are available in the literature. Contaminant concentrations in forage, grain and silage could be estimated using empirical or model-based methods described in the previous discussion of concentrations in vegetables. This overall approach (including the Travis and Arms model) was included in EPA's draft risk assessment guidance for hazardous waste combustion facilities (US EPA, 1998). Other models have been compared to the Travis and Arms model and been found to be no better at predicting biotransfer factors. Furthermore, increasing the number of parameters included as predictors in models does not appear to improve the accuracy of predictions (EPA, 2005). Models that use multiple input parameters are also more data- and computationally intensive. In the final Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (EPA, 2005), EPA presented a polynomial regression model that characterizes the relationship between biotransfer factors and chemical lipophilicity. This model was based on a dataset that included only those chemicals which are not readily oxidizable or hydrolyzable. The model is based on data that show an apparent increase in transfer factors with increasing lipophilicity that reaches a maximum for compounds with a logKow of around 5 to 6, followed by a decrease as lipophilicity increases beyond that point.

Fish contaminant concentrations that result from contaminated soil can also be modeled. US EPA (2005) presents a model that uses site-specific and chemical-specific information to estimate water column dissolved contaminant concentrations. The model then allows fish concentrations to be estimated with chemical-specific bioconcentration factors that are reported in the literature.

6. Uncertainty in Contaminant Concentration Estimates for Meats, Dairy Products and Fish

While there are some empirical data available to estimate the levels of contaminants in food that result from levels in local soils, these data are generally limited to a few highly bioaccumulative compounds. Even for these compounds, the exact contribution of the soil intake to animal body burden tends to be difficult to differentiate from contributions from other sources like atmospheric deposition to pasture grass or consumption of contaminated feed brought in from offsite. Furthermore, results reported in various studies suggest a range of possible food-to-soil ratios that spans several orders of magnitude (Schuler, et al., 1997, Harnly et al., 2000, McKone and Ryan, 1989).

As illustrated in the discussion of uncertainty in estimated vegetable concentrations, models that predict chemical concentrations in one heterogeneous medium from those in another based on limited data, have a high degree of inherent uncertainty. This is also true for estimates of contaminant concentrations in foods from those in soil. In fact, the variability in transfer from soil to animal product is likely to be much higher than that for soil to plants. For example, soil-to-animal transfer models require predictions of soil-derived plant contaminant concentrations in order to estimate contaminant intake by animals that results from consumption of plants. Any uncertainty or error in the estimation of plant concentrations is then compounded with the uncertainty associated with estimated ingestion rates for soil, forage, silage and grain, and the estimated biotransfer factors. If variability and uncertainty result in estimated and measured plant concentrations that differ by orders of magnitude, it is likely that variability and uncertainty can result in even greater disparities between actual and estimated animal product concentrations. It has been argued that even as additional information on important parameters affecting transfer is obtained, uncertainty in estimation will not be reduced below two to three orders of magnitude (Price et al, 1996)

7. Considerations in Accounting for the Dietary Ingestion Pathways

In evaluating the available data concerning the influence of soil contaminant concentration on food contaminant concentration, the following can be acknowledged:

- Soil contaminants can be transferred to foods from contaminated soil;
- Empirical data and quantitative models are available in the literature that allow prediction of concentrations in some kinds of food from soil concentration for a number of contaminants; and
- Based on the current state of the science, uncertainty associated with predictions of these concentrations in food is considerable.

Considerations for the Vegetable Consumption Pathway

For most contaminants, concentrations in vegetables, as estimated by empirically-derived or model-based uptake factors, are low - considerably lower than concentrations in the soil. However, estimated rates of vegetable consumption are much higher on a mass-per-day basis than those for incidental ingestion of soil. For this reason, there is a possibility of significant exposure to soil contaminants via homegrown vegetable consumption.

If the significant uncertainties inherent in estimates of vegetable concentrations derived from empirical uptake factors or K_{ow}-based models could be ignored, exposure via vegetable consumption could be quantified by combining estimated vegetable concentrations, consumption rates, exposure durations, exposure frequencies, and body weight. Calculations carried out in this manner would reveal that - for soil present at sites where exposure may occur by incidental ingestion, dermal exposure, ambient air inhalation - the relative contribution of homegrown produce consumption to total exposure can vary widely. This variation reflects not only differences among chemicals in estimated vegetable concentration, but also the relative contributions of the other pathways. For non-carcinogenic chemicals, exposures are evaluated for children who have higher incidental soil ingestion rates, and the soil ingestion pathway can be fairly significant. Calculations suggest that for some of these chemicals, estimated exposure via the homegrown vegetable consumption pathway is similar to exposure via soil ingestion. For others, however, even though estimated vegetable concentrations are low, exposure via vegetable

consumption estimated with the empirical or model-derived uptake factors can be considerable. These calculations, along with other data and assessments reported in the literature, suggest that decisions concerning exposure to soil contaminants in residential settings should reflect the possibility that exposure via garden vegetable consumption may be significant.

However, as discussed earlier, the estimation of exposure to soil contaminants via consumption of homegrown produce incorporates considerable uncertainty. Much of this uncertainty originates in the estimation of contaminant concentrations in vegetables. Uncertainty in prediction may be reduced as more data become available in the literature.

Similar conclusions regarding unacceptable levels of uncertainty associated with incorporation of this pathway into standards or fixed guidelines have been made by other authoritative bodies. The US EPA's 1996 Soil Screening Guidance (US EPA, 1996) did not incorporate the vegetable consumption pathway for organic contaminants because "a lack of empirical data." The CA EPA did not include this pathway in their recent soil screening numbers (with the exception of lead), in part because of the "paucity of data" on the ratios of concentrations in vegetables and soils, and the "enormous uncertainty in models" that are commonly used for vegetable contaminant concentration estimation (CA EPA, 2005).

Due to the high degree of uncertainty in prediction of concentrations in garden vegetables grown in contaminated soil, quantitative estimates of exposure via the vegetable consumption pathway are not included in the calculation of SCOs. However, SCOs based on incidental ingestion, ambient air inhalation, and dermal exposures alone would fail to acknowledge a potentially significant exposure pathway. For this reason, the unrestricted and residential land use SCOs are adjusted to acknowledge a significant - though not quantified - portion of total exposure to soil contaminants from the vegetable consumption pathway. Because of the uncertainties in estimating vegetable concentration, this adjustment does not account for any factors or variables associated with the vegetable pathway. Rather, it is an across-the-board proportional reduction in the unrestricted and residential land use SCOs. Because of the common oral exposure route shared between vegetable consumption and incidental soil ingestion, it was decided that the

adjustment – the proportional reduction – be made to the unrestricted and residential land use SCOs already calculated for the incidental soil ingestion pathway.

Considerations for the Meat, Dairy and Fish Consumption Pathways

While it is difficult to precisely ascertain the exact amounts of soil contaminants that can become incorporated into and retained by the tissues of cattle, pigs, chickens or fish, it is clear that the process of incorporation can and does occur. This process has been established for terrestrial farm animals through a combination of studies which have demonstrated incidental soil ingestion, bioavailability of chemical soil constituents, transfer of chemicals to grain, silage and forage crops, and associations between chemical concentrations in soil and the tissue (and milk or eggs) of grazing animals. Similarly, incorporation of soil contaminants into fish has been indicated by studies showing the contribution of soil-derived chemical constituents to water concentrations, and associations between water concentrations and fish concentrations (Roulet et al., 1999).

The chemicals for which this process of incorporation has been demonstrated are relatively few, being largely limited to the most persistent and bioaccumulative contaminants. Nonetheless, even for those chemical constituents that are not generally considered bioaccumulative (e.g., metals), the process of incorporation and retention of chemical soil constituents in animal tissue has been shown to occur (Wilkinson et al., 2003). The limited available data suggest that this process of incorporation can result in very different concentrations in food products even under apparently similar conditions and soil contaminant concentrations. While a number of factors that might influence the magnitude of this process have been identified (e.g., feeding regimes and plant concentrations for farm animals; land slope characteristics, general water chemistry, and local ecosystem characteristics for fish), assignment of values for these factors and development of equations that characterize their influence is challenging. Models have been developed and are used for risk assessment purposes, but there are apparently no regulatory programs that incorporate these methods in the development of standards or guidelines. EPA's Supplementary Soil Screening Guidance (US EPA, 2001) suggests that exposure scenarios for sites with potential future agricultural uses should address a wider range of potential receptors

including children and adults exposed to contamination through consumption of agricultural products. However, no attempt was made to quantitatively account for this exposure in EPA's generic Soil Screening Levels.

Because estimates of concentrations of chemicals in animal products that originated from soil are highly uncertain, likely even more so than those of contaminants in vegetables, the calculation of SCOs does not quantitatively account for this exposure pathway. However, because of the potential significance of these exposures, especially for bioaccumulative contaminants, it is important that unrestricted land use SCOs are adjusted to account for them. Because these exposures can be concurrent with the similarly unquantified vegetable ingestion exposure, a single across-the-board adjustment is made to the unrestricted land use SCOs to account for all potential dietary ingestion exposures in aggregate.

8. Accounting for the Dietary Ingestion Pathways

In determining the absolute value of adjustments to the SCOs based on unquantified dietary ingestion exposure pathways, not only were the available data on contaminant concentrations in food considered, but also precedents set by analogous adjustments in a regulatory context. In regulating drinking water quality, the US EPA has set standards that recognize the potential for additional exposures beyond those quantified in the inherent exposure assessment of the regulated medium. Specifically, the standards for some contaminants allow the quantified drinking water exposure to contribute only a portion of the non-cancer oral reference dose. The remaining portion of the reference dose is reserved for an unquantified additional exposure source. For some drinking water contaminants, the great majority of an individual's total exposure may originate from these unquantified sources. To avoid regulating a quantified *de minimus* exposure based on the allowance for a dominant contribution from an additional unquantified source, the regulatory body has traditionally set an 80% ceiling for this allowance (for more detailed discussion, see Section 5.2.3).

This traditional regulatory approach can serve as a template for the development of analogous exposure adjustments for additional unestimated exposures to soil contaminants. The SCOs'

inherent estimate of soil ingestion exposure can be upwardly adjusted to an aggregate exposure, that attributes a percentage of the total to unestimated supplemental (although site-related) sources. Using this approach, the unrestricted and residential land use soil ingestion SCOs incorporate an adjusted estimate of exposure that attributes 20% to the quantified soil ingestion pathway and allows an additional 80% for the unestimated vegetable consumption pathway. Application of the traditional ceiling threshold of 80% is a reasonable approach to account for exposure to soil contaminants through consumption of vegetables from residential gardens. However, the even greater potential exposures that may occur as a result of agricultural land use - higher rates of vegetable consumption and other additional dietary ingestion pathways of meat, dairy product and fish consumption - suggest that the 80% adjustment may not be sufficient for the unrestricted SCOs. In order to account for possible additional exposures that may occur on land for which there are no restrictions placed on use, an additional adjustment is appropriate. Because data in the literature suggest that these pathways are much more significant for some chemicals (i.e., highly bioaccumulative chemicals) than for others, a single greater adjustment can not reasonably account for the wide range of possible exposures across all Priority List contaminants. Most contaminants on the Priority List are not expected to accumulate appreciably in animal tissue, and unrestricted land use SCOs for these contaminants are calculated with a moderately higher allowance for unquantified dietary ingestion pathways. Therefore, the quantified soil ingestion pathway for the unrestricted land use category is further increased by a factor of 2, allowing for 90% of total ingestion exposures to be allocated to exposures due to the consumption of meat, dairy products, fish and vegetables. However, a greater adjustment to the quantified soil ingestion pathway is appropriate for Priority List contaminants that have been identified as highly bioaccumulative chemicals. For Priority List contaminants that are identified on either the EPA Persistent Bioaccumulative and Toxic Chemical Program list, or the United Nations Environmental Program *Persistent Organic Pollutant* list, unrestricted health-based SCOs reflect an increase in the soil ingestion pathway by an additional factor of 10, allocating a total of 98% of ingestion exposure for the unquantified dietary ingestion exposures which could take place on land for which there are no restrictions placed on use. The Priority List contaminants to which this factor was applied are listed below.

- Aldrin
- Benzo(a)pyrene

- Chlordane
- Dieldrin
- DDT, DDD, DDE
- Endrin
- Hexachlorobenzene
- Heptachlor
- Mercury (elemental and inorganic salts)
- PCBs

Incorporation of these adjusted ingestion SCOs into the combined pathway unrestricted and residential SCOs is discussed in Section 5.3.5 (Combined Pathway Chronic SCOs).

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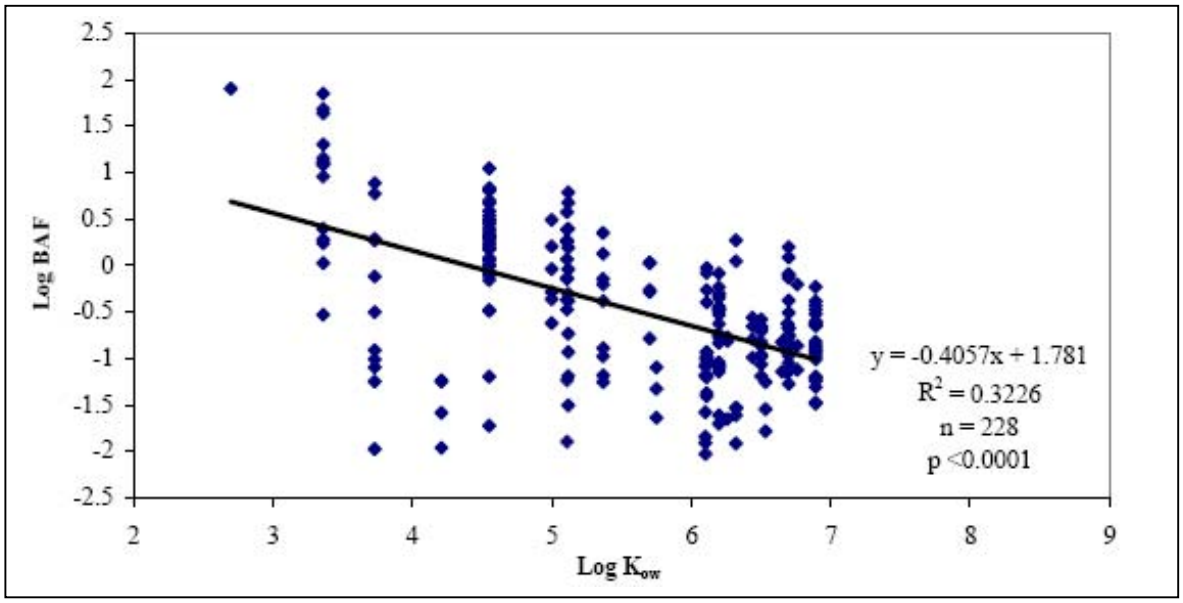
Table 5.2.2.4-1. Uptake Factors Presented in the US EPA Guidance for Developing Ecological Soil Screening Levels (US EPA, 2003, 2005).

	Dieldrin	DDT	DDE
N	18	6	3
Minimum	0.005	0.016	0.075
Median	0.41	0.037	0.136
Maximum	11.0	0.079	0.62

Figure 5.2.2.4-1. Log-linear Regression Plot of Empirical Uptake Factors (BAF) for Various Organic Chemicals Versus LogKow (US EPA, 2003, 2005).



5.2.3 Compensating for the Potential of Non-Site Exposures to Contaminants (Non-Cancer Human Health Effects)

The legislation (§ 27-1415.6(b)) states that "…the department shall consider:…exposure to the same contaminant or group of contaminants from other routes;..." in the development of tables of contaminant-specific SCOs based on the potential health effects of exposures.

Aggregate exposure is the combined exposures of an individual to a single contaminant or a group of contaminants from various routes, pathways, or sources of exposure (US EPA, 2003a). Aggregate exposure can be divided into exposures (doses) associated with a specific contaminated site and those not associated with that site. Methods to evaluate the contaminant dose and potential health risk from major exposure routes (ingestion, inhalation, and dermal) and pathways (soil, air, vegetables) associated with contaminated soil at a particular site are discussed in Sections 5.1 Toxicity Assessment and 5.2 Exposure Assessment. A method to compensate for additional exposures, and perhaps additional health risks, from sources not associated with a contaminated site is discussed in this section. These exposures could include, but are not limited to, those due to the presence of contaminants in the water, air, food, and in the consumer products used by the general US population.

Non-site contributions to aggregate exposure are considered only in the derivation of SCOs based on non-cancer effects because of a fundamental difference in risk assessment methods typically used to evaluate the potential for non-cancer and cancer effects (e.g., US EPA, 2000a,b).

Most non-cancer effects are thought to have a threshold dose, i.e., a dose below which no deleterious effect is expected to occur. At doses above zero, but below the threshold, the risk of a non-cancer health effect is assumed to be zero. At doses above the threshold, the risk of a non-cancer effect typically increases with dose. Thus, aggregate exposure has been considered in the derivation of environmental guidelines (e.g., ambient surface water or groundwater, drinking water, air) based on the non-cancer effects of contaminants (CA EPA, 2003; FL DEP, 2004; MN DOH, 2004; NYS DEC, 1999; US EPA, 1991, 1998, 2000a,b). In practice, agencies typically

allocate only a portion of the reference dose or reference concentration to the environmental guideline. The goal of such allocations is to reduce the likelihood that aggregate exposure (i.e., exposure at the medium-specific environmental guideline and exposures from other potential sources) exceeds the reference dose or reference concentration.

For many carcinogens, however, a single interaction between it and a cellular molecule can theoretically cause changes in the cell that can eventually lead to cancer. This mechanism does not have a threshold dose because any dose level, no matter how small, may pose a small but finite probability of initiating a carcinogenic effect. Risk is assumed to be zero only at zero dose. Theoretically, it is not possible to keep doses below a threshold dose. Guidelines for carcinogenic contaminants in an environmental medium (e.g., water or soil) are typically based on a level of excess risk from exposure to the contaminant in that environmental medium (FL DEP, 2004; MN PCA, 1999; NJ DEP, 2004; NYS DEC, 1999; US EPA, 1991, 2000a,b). Thus, non-site contributions to aggregate exposure are not considered in the derivation of SCOs based on cancer effects.

5.2.3.1 Current Methods to Compensate for Aggregate Exposure In the Derivation of Medium-Specific Guidelines Based on Non-Cancer Effects

The US EPA has consistently considered multiple sources of exposures in their derivation and promulgation of drinking water standards and ambient water quality standards (US EPA, 1991, 2000a,b). The US EPA has used two methods to account for non-water sources of exposures. In the subtraction method, which is less commonly used, exposures from non-water sources (e.g., diet) are subtracted from the reference dose and the remaining dose is allocated to water. In the percentage method, which is more commonly used, the percentage of total exposure typically expected from water is applied to the reference dose to determine the amount of the reference dose that is allocated to water. This percentage is the relative source contribution (RSC) factor.

The US EPA ambient and drinking-water programs have similar approaches to estimating a RSC factor (US EPA, 1990, 1991, 2000a,b). When quantitative data are adequate to determine the relative contribution of each exposure source (e.g., ambient water, drinking water, diet, air) to aggregate exposure, both programs specify that the data should be used to determine a chemical-

specific RSC factor. Both programs also specify that the contaminant-specific factor should be within the range of 20% to 80%. In other words, exposures at the drinking-water or ambient water standard should not account for less than 20% of the reference dose nor more than 80% of the reference dose.

When the water exposure is less than 20% of aggregate exposure, the US EPA recommends a 20% RSC factor. According to the US EPA (2000a), "the 20 percent floor has been traditionally rationalized to prevent a situation where small fractional exposures are being controlled. That is, below that point, it is more appropriate to reduce other sources of exposure, rather than promulgating standards for *de minimus* reductions in overall exposure." When the estimated water exposure is between 20% and 80% of the aggregate exposure, the US EPA recommends a chemical-specific RSC factor. When the estimated exposure from drinking water or ambient water exceeds 80% of the aggregate exposure, the US EPA recommends an 80% RSC factor. This provides adequate protection for those individuals whose aggregate exposure to a contaminant may be higher than that indicated by the available data (US EPA, 1990, 1991, 2000a). More often than not, there have been inadequate quantitative data adequate to determine the relative contribution of each exposure source to aggregate exposure, and the US EPA has defaulted to a 20% RSC factor (Howd et al., 2004; US EPA, 2002, 2003b,c).

Other regulatory programs also consider aggregate exposure in the derivation and promulgation of environmental guidelines. An RSC factor is used by the US EPA Region 6 for purposes of establishing health-based screening levels for air contaminants from hazardous waste combustion facilities (US EPA, 1998). The NYS DEC is required to use an RSC factor in the derivation of ambient water quality guidance values and standards that are based on non-cancer effects of water contaminants (NYS DEC, 1999). Many agencies use a RSC factor in the derivation of drinking water guidelines or standards (e.g., CA EPA, 2003; Health Canada, 1995; MN DOH, 2004; WHO, 1996).

5.2.3.2 Method to Compensate for Non-Site Contributions to Aggregate Exposure in the Derivation of SCOs Based on Non-Cancer Effects

Monitoring programs of US populations consistently show the presence of environmental chemicals in human tissues collected from the general population (e.g., CDC, 2003; ATSDR, 1997, 1999, 2002) and in samples of environmental media, including drinking water (Squillace et al., 2002; US EPA, 1991), ambient water (Kolpin et al., 2002), indoor air (Adgate et al., 2004; NYS DOH, 2005; Sexton et al., 2004), outdoor air (US EPA, 1988; NYS DOH, 2005), and house dust (Butte and Heinzow, 2002; Rudel et al., 2003). Exposure may also occur from consumption of food (Dougherty et al., 2000), nutritional supplements (Bayer, 2005; Wyeth, 2005), or from the use of household products (HPDB, 2005). These data indicate that US population is exposed to many chemicals from a variety of environmental sources.

Similar types of data for the contaminants on the priority list indicate that the US population is exposed to them from a variety of environmental sources. Many of them were or are widely used in industrial, commercial, or consumer products and are present at measurable levels in samples of water, air, and food (Table 5.2.3-1). In addition, many of the priority contaminants are present at measurable levels in human tissues, including adipose tissue, blood/serum, breath, urine, or breastmilk (Table 5.2.3-1). These data have raised concerns about the potential health effects of these exposures (measured or potential), and environmental criteria, guidelines, or standards (ambient water, drinking water, air, and soil) have been proposed or promulgated to control or reduce exposures to many of the priority contaminants. Collectively, these data suggest human exposure to priority list contaminants may come from many sources.

For almost all contaminants, however, the quantitative data on environmental and dietary levels are likely to be inadequate to determine accurately the relative contribution of each exposure source to the aggregate exposure for populations of concern (adults and children). Whether data are adequate depends, to a limited extent, on professional judgement, but the US EPA (2000a, 2003c) recommended minimum requirements for the development of contaminant-specific RSC factors are not likely to be met for most contaminants. Thus, contaminant-specific RSC factors within the range of 20% to 80% are not used in the calculations of SCOs. Instead, a default RSC factor of 20% is used in the calculation of SCOs based on non-cancer effects.

A 20% RSC factor is consistent with US EPA (2000a,b) default procedures for use with exposure databases of differing quality. The US EPA recommends a 20% RSC factor when data are inadequate to characterize aggregate exposure. It is likely that the database for many of the lesser-studied priority contaminants could be placed in this category. A 20% RSC factor is also recommended when data indicate that there might be significant potential or known exposures from sources other than the source of concern or when exposure data are adequate to determine that sources other than the source of concern are a major source (>80%) of aggregate exposure. It is likely that the databases for those priority contaminants that are commonly found in food, such as organic compounds that that bioaccumulate or metals naturally found in soil, could be placed in one or both of these categories.

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Table 5.2.3-1. Presence of Priority Contaminants in Samples of Human Tissues/Fluids, Environmental Media or in Consumer Products.
(see end of table for key to notations)

Analyte	CAS	Adipose	Blood/ Serum	Breath	Urine	Breast Milk	Household Products	Indoor Air	Outdoor Air	Food/ Water	Vitamin Pills	Gasoline Volatiles
1,1,1-Trichloroethane	71-55-6		A1995	A1995	A1995	A1995	A1995,2	A1995, 3,4a,4b	A1995, 3,4a,4b	A1995		
1,1-Dichloroethane	75-34-3			A1990					4b	A1990		
1,1-Dichloroethene	75-35-4						A1994	A1994	A1994	A1994		
1,2,4-Trimethylbenzene	95-63-6						2	3,4a,4b	3,4a,4b			7
1,2-Dichlorobenzene	95-50-1						2		4b			
1,2-Dichloroethane	107-06-2			A1999	A1999	A1999	A1999	A1999	A1999,4b	A1999		A1999
1,3,5-Trimethylbenzene	108-67-8						2	3,4b	3,4b			7
1,3-Dichlorobenzene	541-73-1			A2005				4b	A2005,4b	A2005		
1,4-Dichlorobenzene	106-46-7	A2005	A2005	A2005			A2005,2	A2005	A2005	A2005		
1,4-Dioxane	123-91-1						A2005,2	A2005	A2005	A2005		7
2-Butanone	78-93-3		A1992	A1992		A1992	A1992,2	A1992	A1992	A1992		A1992
2-Methylphenol	95-48-7						2					
4,4-DDD	72-54-8	A2002							A2002	A2002		
4,4-DDE	72-55-9	A2002	A2002,1			A2002			A2002	A2002		
4,4-DDT	50-29-3	A2002	A2002,1			A2002			A2002	A2002,5		
Acenaphthene	83-32-9						2					
Acenaphthylene	208-96-8						2					
Acetone	67-64-1		A1994	A1994	A1994	A1994	A1994,2	A1994,3,4b	A1994, 3,4a,4b	A1994		A1994
Aldrin	309-00-2									A2002		
BHC		A2003	A2003			A2003			A2003	A2003		
alpha-Chlordane	5103-71-9	A1994	A1994			A1994		A1994	A1994	A1994		
Anthracene	120-12-7						2					
Arsenic			A2000		A2000	A2000	A2000	A2000	A2000	A2000,5		
Barium		A1992	A1992		A1992,1		A1992,2		A1992	A1992		
Benzene	71-43-2	A1997	A1997	A1997	A1997		A1992,2	A1997, 3,4a,4b	A1997, 3,4a,4b	A1997		A1997,7

Analyte	CAS	Adipose	Blood/ Serum	Breath	Urine	Breast Milk	Household Products	Indoor Air	Outdoor Air	Food/ Water	Vitamin Pills	Gasoline Volatiles
Benzo[a]anthracene	56-55-3				1		2					
Benzo[a]pyrene	50-32-8						2					
Benzo[b]fluoranthene	205-99-2						2					
Benzo[g,h,i]perylene	191-24-2						2					
Benzo[k]fluoranthene	207-08-9						2					
Beryllium			A2000		A2000				A2000	A2000		
Cadmium			A1999,1		A1999,1	A1999	A1999,2		A1999	A1999,5		
Carbon Tetrachloride	86-74-8		A2003	A2003	A2003		2	A2003,3,4b	A2003, 3,4b	A2003		
Chlorobenzene	108-90-7	A1990		A1990	A1990		A1990,2	A1990	A1990,4b	A1990		
Chloroform	67-66-3		A1997	A1997		A1997	A1997,2	A1997,3,4b	A1997,4b	A1997		
Chromium (Total)			A2000		A2000	A2000	A2000	A2000	A2000	A2000	A2000,6	
Chrysene	218-01-9				1		2					
cis-1,2-Dichloroethene	156-59-2						A1996	A1996	A1996,4b	A1996		
Copper						A2004	A2004,2		A2004	A2004	A2004,6	A2004
Cyanide			A2005		A2005				A2005	A2005		
Dibenz[a,h]anthracene	53-70-3						2					
Dibenzofuran	132-64-9											
Dieldrin	60-57-1	A2002	A2002		A2002	A2002		A2002	A2002	A2002,5		
Endosulfans			A2000		A2000	A2000			A2000	A2000,5		
Endrin	72-20-8	A1996				A1996				A1996		
Ethylbenzene	100-41-4	A1999	A1999		A1999	A1999	A1999,2	A1999, 3,4a,4b	A1999,3, 4a,4b	A1999		A1999,7
Fluoranthene	206-44-0				1		2					
Fluorene	86-73-7				1		2					
Heptachlor	76-44-8	A1993	A1993,1			A1993		A1993	A1993	A1993,5		
Hexachlorobenzene	118-74-1	A2002	A2002,1		A2002	A2002		A2002	A2002	A2002,5		
Indeno[1,2,3-cd]pyrene	193-39-5						2					
Lead			A1999,1		A1999,1	A1999	A1999,2	A1999	A1999	A1999		
Xylene			A1995	A1995	A1995		A1995,2	A1995, 3,4a,4b	A1995, 3,4a,4b	A1995		A1995,7
Manganese			A2000		A2000	A2000	A2000,2	A2000	A2000	A2000,5	A2000,6	

Analyte	CAS	Adipose	Blood/ Serum	Breath	Urine	Breast Milk	Household Products	Indoor Air	Outdoor Air	Food/ Water	Vitamin Pills	Gasoline Volatiles
Mercury	7439-97-6		A1999		A1999,1	A1999	A1999	A1999	A1999	A1999.5		
Methyl tert-butyl Ether	1634-04-4							A1996,3,4a	A1996,3	A1996		A1996,7
Methylene Chloride	75-09-2		A2000			A2000	A2000,2	A2000,4a	A2000, 3,4a,4b	A2000		
Naphthalene	91-20-3	A2004			1	A2004	A2004,2	A2004	A2004,4b	A2004		
n-Butylbenzene	104-51-8											7
Nickel					A2004	A2004	2	A2004	A2004	A2004	6	
Pentachlorophenol	87-86-5	A2001	A2001,1		A2001			A2001	A2001	A2001		
Phenanthrene	85-01-8				1		2					
Phenol	108-95-2						A1998,2		A1998	A1998		
Pyrene	129-00-0				1		2					
sec-Butylbenzene	135-98-8											7
Selenium			A2003		A2003	A2003	A2003,2		A2003	A2003,5	A2003,6	
Silver			A1990		A1990		A1990,2		A1990	A1990		
tert-Butylbenzene	98-06-6											7
Tetrachloroethene	127-18-4		A1997	A1997	A1997	A1997	A1997,2	A1997, 3,4a,4b	A1997, 4a,4b	A1997		
Toluene	108-88-3	A2000	A2000	A2000		A2000	A2000,2	A2000,3,4a	A2000, 3,4a,4b	A2000		A2000,7
trans-1,2-Dichloroethene	156-60-5						A1996	A1996	A1996	A1996		
Trichloroethene	79-01-6		A1997			A1997	A1997,2	A1997, 4a,4b	A1997,4b	A1997		
Vinyl Chloride	75-01-4			A2005			A2005,2	A2005	A2005,4b	A2005		
Zinc		A2003	A2003		A2003	A2003	A2003,2		A2003	A2003	A2003,6	

Key to Table 5.2.3-1 Notations:

A(YEAR) - Agency for Toxic Substance Substances and Disease Registry (ATSDR). Toxicological Profile Series. Atlanta, GA: U.S. Department of Health and Human Services. Entry is year of publication for toxicological profile on chemical or substance. Accessed on March 15, 2005 at <http//www.atsdr.cdc.gov>

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2. HPDB (Household Products Database). 2005. National Institutes of Health, National Library of Medicine. Chemical in found in product. Accessed on March 15, 2005 at <http://householdproducts.nlm.nih.gov/index.htm>.

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4a. US EPA (US Environmental Protection Agency). Building Assessment and Survey Evaluation (BASE '94-'98). Unpublished. Washington, DC: US Environmental Protection Agency.

4b. US EPA (US Environmental Protection Agency). 1988. National Ambient Volatile Organic Compounds (VOCs) Data Base Update. EPA/600/3-88-010a. NTIS No. PB88-195631. Research Triangle Park, NC: Office of Research and Development.

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5.3 Calculation of Chronic Human Health-based Soil Cleanup Objectives

Chronic health-based SCOs for unrestricted, residential, restricted residential, commercial, and industrial land uses were calculated using the toxicity values described in Section 5.1 (Toxicity Assessment) and the exposure parameters and parameter values described in Section 5.2 (Exposure Assessment). SCOs were calculated for two health endpoints – non-cancer and cancer. Pathway-specific SCOs for non-cancer endpoints define the chemical-specific soil concentrations at which the dose that a person receives through the pathway is equivalent to the reference dose or the dose-equivalent of the reference concentration (modified as previously described in Section 5.2.3). The pathway-specific SCOs for cancer endpoints define the chemical-specific soil concentrations at which the dose that a person receives from the pathway is equivalent to an excess cancer risk of one-in-one-million. Final chronic health-based SCOs were calculated by combining the exposures associated with all relevant pathways.

While the methods for calculating the chronic health-based SCOs (as well as the acute and irritant contact dermatitis SCOs) were chosen in consideration of the available science, their usefulness and applicability have limitations. In addition to uncertainty in the inherent estimates of exposure and toxicity, there are general factors and issues that are not considered in the equations, and there may be underlying assumptions that are not valid under all commonly encountered conditions for all chemicals. Many of these issues remain poorly understood.

Some of the general factors that are not considered in the equations may have impacts on protection of public health, or they may have impacts on other beneficial or favorable attributes of soil that people value. Examples of some potential chemical impacts to soil that are less directly related to health, and are not quantitatively accounted for in the SCO equations, include impacts to soil appearance, texture, odor, and aerability. It is possible that there are also other factors that are more directly related to protection of health that remain unaccounted for.

The equations and parameter values used to calculate chronic health-based SCOs are also only as valid as the assumptions upon which they are based. Inherent assumptions relate to chemical species, contaminant availability, and sorptive limitations of the soil matrix. Some of these

assumptions effectively impose boundary conditions upon the applicability of the equations. For example, exposure calculations for the SCOs are based on the assumption that the contaminant is part of the soil matrix. This may not be true at soil concentrations that exceed the soil saturation level for the contaminant. The soil saturation level of a contaminant ("C_{sat}") corresponds to the contaminant 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 all been reached (US EPA, 1996). At higher concentrations in soil, the contaminant may not be incorporated into the soil matrix. Rather, it is likely to be present in a free phase (e.g., non-aqueous phase liquid) (US EPA, 1996). At concentrations that exceed C_{sat}, assumed values for some parameters (e.g., soil dermal adherence factors and dermal absorption fractions) used to calculate SCOs may not be appropriate.

In addition to the assumptions that soil contaminants are part of the soil matrix, there may be other assumptions that may not be valid under all commonly encountered circumstances. Thus, while the SCO equations incorporate reasonable estimates of exposure and toxicity, they rely upon assumptions that are not universally applicable, and they might not account for all possible conditions or factors that could be important for determination of protective soil cleanup objectives. In some instances, not accounting for these factors or relying on inappropriate assumptions may result in calculation of SCO values that are unreasonably high. For the reasons mentioned, maximum acceptable soil contaminant concentrations - or "caps" - were developed for each land-use category.

The methods used to calculate the chronic health-based SCOs are presented below. Pathway-specific human chronic health-based SCOs are listed in Table 5.3.6-1 and the final human chronic health-based SCOs are listed in Table 5.3.6-2. The development of caps and the underlying rationale for their development is discussed in more detail in Section 9.3.

5.3.1 Chronic Soil Ingestion SCOs

5.3.1.1 Unrestricted Land Use

1. Soil Ingestion SCOs for Non-Cancer Endpoints – Child

$$SCO_{soil} = \frac{RfD_{adj} \times D}{\frac{CF \times 74\ mg_{soil}/day}{13.3 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6\ mg_{soil}$)

74 mg_{soil}/day = child soil ingestion rate (time-weighted average)

13.3 kg_{bw} = child body weight

D = factor to account for dietary exposure pathways (see Section 5.2.2.4):

- 0.02 for homegrown vegetable consumption pathway ***and*** home produced animal product consumption pathway (persistent, bioaccumulative, toxic (PBT) chemicals)

or

- 0.1 for homegrown vegetable consumption pathway ***and*** home produced animal product consumption pathway (non-PBT chemicals)

2. Soil Ingestion SCOs for Non-Cancer Endpoints – Adult

$$SCO_{soil} = \frac{RfD_{adj} \times D}{\frac{CF \times 17\ mg_{soil}/day}{70 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

17 mg_{soil}/day = adult soil ingestion rate (time-weighted average)

70 kg_{bw} = adult body weight

D = factor to account for dietary exposure pathways (see Section 5.2.2.4):

- 0.02 for homegrown vegetable consumption pathway ***and*** home produced animal product consumption pathway (persistent, bioaccumulative, toxic (PBT) chemicals)

or

- 0.1 for homegrown vegetable consumption pathway ***and*** home produced animal product consumption pathway (non-PBT chemicals)

3. Soil Ingestion SCOs for Cancer Endpoints

In deriving the unrestricted, residential and restricted residential SCOs for cancer endpoints, we assumed that exposure occurs over an entire lifetime. The information presented in Section 5.2.2.1 (Soil Ingestion Pathway) shows that children have higher estimated soil ingestion rates and lower body weights than adults, and therefore potentially higher exposures than adults. We accounted for this potentially increased exposure during childhood in deriving the soil ingestion SCOs for cancer endpoints, using an approach that is generally consistent with the approach used by the US EPA in its Soil Screening Guidance (US EPA, 1996). To calculate these SCOs, we used exposure parameters for four age classes: 1 year old, 2 to 5 years, 6 to 15 years, and 16 to 69 years. The body weight values for each age class are mean values for males and females derived from the US EPA (1997). We also accounted for the potentially increased sensitivity of children to early-life exposures to carcinogens that act through a mutagenic mode-of-action (i.e., carcinogenic PAHs) using the approach described in Section 5.1.1.6. Table 5.3.1.1-1 shows the parameter values that we used to calculate these SCOs. The SCOs were calculated as follows:

$$SCO_{soil} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF} \times D}{\sum_{i=1}^{5} \frac{ADAF_i \times IR_i \times ED_i}{BW_i}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

i = age class number; see Table 5.3.1.1-1

IR_i = soil ingestion rate (mg/day) for age class *i* (time-weighted average)

ED_i = exposure duration (years) for age class *i*

BW_i = average body weight (kg) for age class *i*

$ADAF_i$ = age-dependent CPF adjustment factor for age class *i*

CF = conversion factor ($1\ kg_{soil}/10^6\ mg_{soil}$)

70 years = averaging time

D = factor to account for dietary exposure pathways (see Section 5.2.2.4):

- 0.02 for homegrown vegetable consumption pathway ***and*** home produced animal product consumption pathway (persistent, bioaccumulative, toxic (PBT) chemicals)

or

- 0.1 for homegrown vegetable consumption pathway ***and*** home produced animal product consumption pathway (non-PBT chemicals)

5.3.1.2 Residential Land Use

1. Soil Ingestion SCOs for Non-Cancer Endpoints – Child

$$SCO_{soil} = \frac{RfD_{adj} \times D}{\frac{CF \times 74\ {}^{mg_{soil}}/_{day}}{13.3 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor ($1\ kg_{soil}/10^6\ mg_{soil}$)

74 mg_{soil}/day = child soil ingestion rate (time-weighted average)

13.3 kg_{bw} = child body weight

D = factor to account for dietary exposure pathway (see Section 5.2.2.4):

- 0.2 for homegrown vegetable consumption pathway

2. Soil Ingestion SCOs for Non-Cancer Endpoints – Adult

$$SCO_{soil} = \frac{RfD_{adj} \times D}{\frac{CF \times 17\ ^{mg_{soil}}/_{day}}{70 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

17 mg_{soil}/day = adult soil ingestion rate (time-weighted average)

70 kg_{bw} = adult body weight

D = factor to account for dietary exposure pathway (see Section 5.2.2.4):

- 0.2 for homegrown vegetable consumption pathway

3. Soil Ingestion SCOs for Cancer Endpoints

The approach for deriving residential SCOs for cancer endpoints is the same as the approach used to derive unrestricted SCOs for cancer endpoints (described above), except the factor for the dietary consumption pathways only accounts for the homegrown vegetable pathway. The SCOs were calculated as follows:

$$SCO_{soil} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF} \times D}{\sum_{i=1}^{5} \frac{ADAF_i \times IR_i \times ED_i}{BW_i}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

i = age class number; see Table 5.3.1.1-1

IR_i = soil ingestion rate (mg/day) for age class *i* (time-weighted average)

ED_i = exposure duration (years) for age class *i*

BW_i = average body weight (kg) for age class *i*

$ADAF_i$ = age-dependent CPF adjustment factor for age class *i*

CF = conversion factor ($1\ kg_{soil}/10^6\ mg_{soil}$)

70 years = averaging time

D = factor to account for dietary exposure pathway (see Section 5.2.2.4):

- 0.2 for homegrown vegetable consumption pathway

5.3.1.3 Restricted Residential Land Use

1. Soil Ingestion SCOs for Non-Cancer Endpoints – Child

$$SCO_{soil} = \frac{RfD_{adj}}{\frac{CF \times 74\ {}^{mg_{soil}}\!/_{day}}{13.3 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor ($1\ kg_{soil}/10^6\ mg_{soil}$)

74 mg_{soil}/day = child soil ingestion rate (time-weighted average)

13.3 kg_{bw} = child body weight

2. Soil Ingestion SCOs for Non-Cancer Endpoints – Adult

$$SCO_{soil} = \frac{RfD_{adj}}{\frac{CF \times 17\ ^{mg_{soil}}/_{day}}{70kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

17 mg_{soil}/day = adult soil ingestion rate (time-weighted average)

70 kg_{bw} = adult body weight

3. Soil Ingestion SCOs for Cancer Endpoints

The approach for deriving restricted residential SCOs for cancer endpoints is the same as the approach used to derive unrestricted SCOs for cancer endpoints (described above), except the factor to account for the dietary consumption pathways is not used. The SCOs were calculated as follows:

$$SCO_{soil} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF}}{\sum_{i=1}^{5} \frac{ADAF_i \times IR_i \times ED_i}{BW_i}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

i = age class number; see Table 5.3.1.1-1

IR_i = soil ingestion rate (mg/day) for age class *i* (time-weighted average)

ED_i = exposure duration (years) for age class *i*

BW_i = average body weight (kg) for age class *i*

$ADAF_i$ = age-dependent CPF adjustment factor for age class *i*

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

70 years = averaging time

5.3.1.4 Commercial Land Use

1. Soil Ingestion SCOs for Non-Cancer Endpoints – Child

$$SCO_{soil} = \frac{RfD_{adj}}{\frac{CF \times 9\ ^{mg_{soil}}/_{day}}{13.3 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

9 mg_{soil}/day = child soil ingestion rate (time-weighted average)

13.3 kg_{bw} = child body weight

2. Soil Ingestion SCOs for Non-Cancer Endpoints – Adult

$$SCO_{soil} = \frac{RfD_{adj}}{\frac{CF \times 17\ ^{mg_{soil}}/_{day}}{70 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

17 mg_{soil}/day = adult soil ingestion rate (time-weighted average)

70 kg_{bw} = adult body weight

3. Soil Ingestion SCOs for Cancer Endpoints

$$SCO_{soil} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF}}{\frac{17\ ^{mg_{soil}}/_{day} \times 25 years}{70 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

17 mg_{soil}/ day = adult soil ingestion rate (time-weighted average)

25 years = exposure duration

70 years = averaging time

70 kg_{bw} = adult body weight

5.3.1.5 Industrial Land Use

1. Soil Ingestion SCOs for Non-Cancer Endpoints – Adolescent

$$SCO_{soil} = \frac{RfD_{adj}}{\frac{CF \times 8.5\ ^{mg_{soil}}/_{day}}{58.1 kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

8.5 mg_{soil}/day = adolescent soil ingestion rate (time-weighted average)

58.1 kg_{bw} = adolescent body weight

2. Soil Ingestion SCOs for Non-Cancer Endpoints – Adult

$$SCO_{soil} = \frac{RfD_{adj}}{\frac{CF \times 8.5\,^{mg_{soil}}/_{day}}{70kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

8.5 mg_{soil}/day = adult soil ingestion rate (time-weighted average)

70 kg_{bw} = adult body weight

3. Soil Ingestion SCOs for Cancer Endpoints

$$SCO_{soil} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF}}{\frac{8.5\,^{mg_{soil}}/_{day} \times 25years}{70kg_{bw}}}$$

Where:

SCO_{soil} = soil cleanup objective (mg/kg) for the soil ingestion pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

8.5 mg_{soil}/ day = adult soil ingestion rate (time-weighted average)

25 years = exposure duration

70 years = averaging time

70 kg_{bw} = adult body weight

References

US EPA (US Environmental Protection Agency). 1996. Soil Screening Guidance: Technical Background Document. Washington, DC: Office of Solid Waste and Emergency Response, EPA/540/R95/128.

US EPA (US Environmental Protection Agency). 1997. Exposure Factors Handbook. National Center for Environmental Assessment.

Table 5.3.1.1-1. Exposure Factors Used to Calculate Unrestricted, Residential and Restricted Residential Soil Ingestion SCOs for Cancer Endpoints.

Age Class *i*	Ages in Class	ED (years)	BW (kg)	IR (mg/day)	ADAF[1]
1	<1	1	9.1	0	10
2	1	1	12.3	74	10
3	2-5	4	16.2	74	3
4	6-15	10	39.8	17	3
5	16-69	54	69.3	17	1

[1] For carcinogens that act with a mutagenic mode of action. For all other carcinogens, ADAF is equal to one for ALL age classes.

5.3.2 Chronic Inhalation SCOs

5.3.2.1 Unrestricted, Residential and Restricted Residential Land Use

1. Particulate-Bound Contaminant Inhalation SCOs for Non-Cancer Endpoints - Child

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{19 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{1.21E+9 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

19 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

2. Particulate-Bound Contaminant Inhalation SCOs for Non-Cancer Endpoints – Adult

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{35 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{1.21E+9 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

35 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

3. Particulate-Bound Contaminant Inhalation SCOs for Cancer Endpoints

$$SCO_{inhalation} = \frac{10^{-6} \times 25550\ \text{days}}{\text{ADAF} \times \text{URF} \times 10^{3}\ \mu\text{g/mg} \times 35\ \text{days/year} \times 70\ \text{years} \times \left[\dfrac{1}{1.21\text{E}+9\ \dfrac{\text{m}^3}{\text{kg}}}\right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

10^{-6} = one-in-one-million risk level

25550 days = averaging time (equal to 70 years)

URF = inhalation unit risk factor $(\mu g/m^3)^{-1}$

10^3 µg/mg = conversion factor

35 days/year = exposure frequency

70 years = exposure duration

ADAF = age-dependent CPF adjustment factor averaged over a lifetime (ADAF = 1.66 for carcinogens that act with a mutagenic mode of action; ADAF = 1 for all other carcinogens)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

4. Volatile Contaminant Inhalation SCOs for Non-Cancer Endpoints - Child

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{217 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{2.67 \text{ E} + 4 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

217 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

5. Volatile Contaminant Inhalation SCOs for Non-Cancer Endpoints - Adult

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{217 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{2.67 \text{ E} + 4 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

217 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

6. Volatile Contaminant Inhalation SCOs for Cancer Endpoints

$$SCO_{inhalation} = \frac{10^{-6} \times 25550 \text{ days}}{ADAF \times URF \times 10^3 \text{ µg/mg} \times 217 \text{ days/year} \times 70 \text{ years} \times \left[\frac{1}{2.67 \text{ E} + 4 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

10^{-6} = one-in-one-million risk level

25550 days = averaging time (equal to 70 years)

URF = inhalation unit risk factor $(\mu g/m^3)^{-1}$

10^3 µg/mg = conversion factor

217 days/year = exposure frequency

70 years = exposure duration

ADAF = age-dependent CPF adjustment factor averaged over a lifetime (ADAF = 1.66 for carcinogens that act with a mutagenic mode of action; ADAF = 1 for all other carcinogens)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

5.3.2.2. Commercial Land Use

1. Particulate-Bound Contaminant Inhalation SCOs for Non-Cancer Endpoints - Child

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{5 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{1.21E + 9 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

5 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

2. Particulate-Bound Contaminant Inhalation SCOs for Non-Cancer Endpoints – Adult

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{62 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{1.21E+9 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

62 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

3. Particulate-Bound Contaminant Inhalation SCOs for Cancer Endpoints

$$SCO_{inhalation} = \frac{10^{-6} \times 25550 \text{days}}{URF \times 10^{3}\ \mu g/mg \times 62 \text{days/year} \times 25\ \text{years} \times \left[\frac{1}{1.21E+9 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

10^{-6} = one-in-one-million risk level

25550 days = averaging time (equal to 70 years)

URF = inhalation unit risk factor, $(\mu g/m^3)^{-1}$

10^3 µg/mg = conversion factor

62 days/year = exposure frequency

25 years = exposure duration

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

4. Volatile Contaminant Inhalation SCOs for Non-Cancer Endpoints - Child

$$SCO_{inhalation} = \frac{1 \times 365 \text{ days}}{5 \text{ days/year} \times 1 \text{ year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{2.67\ E+4 \frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

5 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

5. Volatile Contaminant Inhalation SCOs for Non-Cancer Endpoints - Adult

$$SCO_{inhalation} = \frac{1 \times 365\,\text{days}}{62\,\text{days/year} \times 1\,\text{year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{2.67\,E+4\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

62 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

6. Volatile Contaminant Inhalation SCOs for Cancer Endpoints

$$SCO_{inhalation} = \frac{10^{-6} \times 25550\text{days}}{URF \times 10^3\ \mu g/mg \times 62\,\text{days/year} \times 25\,\text{years} \times \left[\frac{1}{2.67\,E+4\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

10^{-6} = one-in-one-million risk level

25550 days = averaging time (equal to 70 years)

URF = inhalation unit risk factor, $(\mu g/m^3)^{-1}$

10^3 µg/mg = conversion factor

62 days/year = exposure frequency

25 years = exposure duration

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

5.3.2.3 Industrial Land Use

1. Particulate-Bound Contaminant Inhalation SCOs for Non-Cancer Endpoints - Adolescent

$$SCO_{inhalation} = \frac{1 \times 365\,\text{days}}{5\,\text{days/year} \times 1\,\text{year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{1.21E+9\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

5 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

2. Particulate-Bound Contaminant Inhalation SCOs for Non-Cancer Endpoints – Adult

$$SCO_{inhalation} = \frac{1 \times 365\,\text{days}}{31\,\text{days/year} \times 1\,\text{year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{1.21E+9\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

31 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

3. Particulate-Bound Contaminant Inhalation SCOs for Cancer Endpoints

$$SCO_{inhalation} = \frac{10^{-6} \times 25550\text{days}}{URF \times 10^3\ \mu g/mg \times 31\text{days/year} \times 25\ \text{years} \times \left[\frac{1}{1.21E+9\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the particulate inhalation pathway

10^{-6} = one-in-one-million risk level

25550 days = averaging time (equal to 70 years)

URF = inhalation unit risk factor, $(\mu g/m^3)^{-1}$

10^3 μg/mg = conversion factor

31 days/year = exposure frequency

25 years = exposure duration

1.21E+9 m^3/kg = particulate emission factor (see Section 5.2.2.2-1)

4. Volatile Contaminant Inhalation SCOs for Non-Cancer Endpoints - Adolescent

$$SCO_{inhalation} = \frac{1 \times 365\,\text{days}}{5\,\text{days/year} \times 1\,\text{year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{2.67\,E+4\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

5 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

5. Volatile Contaminant Inhalation SCOs for Non-Cancer Endpoints - Adult

$$SCO_{inhalation} = \frac{1 \times 365\,\text{days}}{31\,\text{days/year} \times 1\,\text{year} \times \left[\frac{1}{RfC_{adj}} \times \frac{1}{2.67\,E+4\,\frac{m^3}{kg}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

1 = target hazard quotient (unitless)

365 days = averaging time

31 days/year = exposure frequency

1 year = exposure duration

RfC_{adj} = inhalation reference concentration, adjusted to account for relative source contribution (mg/m^3)

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

6. Volatile Contaminant Inhalation SCOs for Cancer Endpoints

$$SCO_{inhalation} = \frac{10^{-6} \times 25550 \text{days}}{\text{URF} \times 10^3\ \mu\text{g/mg} \times 31\,\text{days/year} \times 25\ \text{years} \times \left[\frac{1}{2.67\ \text{E} + 4\ \frac{\text{m}^3}{\text{kg}}} \right]}$$

Where:

$SCO_{inhalation}$ = soil cleanup objective (mg/kg) for the vapor-phase inhalation pathway

10^{-6} = one-in-one-million risk level

25550 days = averaging time (equal to 70 years)

URF = inhalation unit risk factor, $(\mu g/m^3)^{-1}$

10^3 µg/mg = conversion factor

62 days/year = exposure frequency

25 years = exposure duration

2.67 E+4 m^3/kg = soil-to-air volatilization factor (see Section 5.2.2.2-2)

5.3.3 Chronic Dermal Absorption SCOs

5.3.3.1 Unrestricted, Residential and Restricted Residential Land Use

1. Dermal Absorption SCOs for Non-Cancer Endpoints – Child

$$SCO_{dermal} = \frac{RfD_{adj}}{\frac{1870 cm^2 \times 0.2\,{}^{mg}\!/_{cm^2} \times AF \times CF \times \frac{155}{365} days}{13.3 kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

1870 cm^2 = exposed surface area

0.2 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

155 days/365 days = exposure frequency

13.3 kg_{bw} = child body weight

2. Dermal Absorption SCOs for Non-Cancer Endpoints – Adult

$$SCO_{dermal} = \frac{RfD_{adj}}{\frac{4850 cm^2 \times 0.07\,{}^{mg}\!/_{cm^2} \times AF \times CF \times \frac{62}{365} days}{70 kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

4850 cm^2 = exposed surface area

0.07 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

62 days/365 days = exposure frequency

70 kg_{bw} = adult body weight

3. Dermal Absorption SCOs for Cancer Endpoints

In deriving the unrestricted, residential and restricted residential SCOs for cancer endpoints, we assumed that exposure occurs over an entire lifetime. The information presented in Section 5.2.2.3 (Dermal Pathway) shows that children have higher estimated soil adherence factors, greater exposure frequencies, and lower body weights than adults, and therefore potentially higher exposures than adults. We accounted for this potentially increased exposure during childhood in deriving the dermal absorption SCOs for cancer endpoints, using an approach that is generally consistent with the approach used by the US EPA in its Supplemental Guidance for Dermal Risk Assessment (US EPA, 2004). To calculate these SCOs, we used exposure parameters for five age classes: 1 year old, 2 to 5 years, 6 to 15 years, 16 to 17 years and 18 to 69 years. The body weight values for each age class are mean values for males and females derived from the US EPA (1997). We used the child, adolescent and adult surface areas and soil adherence factors described in Section 5.2.2.3 (Dermal Pathway) and the exposure frequencies described in Section 5.2.2.1 (Soil Ingestion Pathway). We also accounted for the potentially increased sensitivity of children to early-life exposures to carcinogens that act through a mutagenic mode-of-action (i.e., carcinogenic PAHs) using the approach described in Section 5.1.1.6. Table 5.3.3.1-1 shows the parameter values that we used to calculate these SCOs. The SCOs were calculated as follows:

$$SCO_{dermal} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF}}{AF \times \sum_{i=1}^{6} \frac{ADAF_i \times SAF_i \times SA_i \times \frac{EF_i}{365} \times ED_i}{BW_i}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

i = age class number; see Table 5.3.3.1-1

SA_i = skin surface area available for contact (cm^2) for age class *i*

SAF_i = soil adherence factor (mg/cm^2) for age class *i*

ED_i = exposure duration (years) for age class *i*

EF_i = exposure frequency (days/365 days) for age class *i*

BW_i = body weight (kg) for age class *i*

$ADAF_i$ = age-dependent CPF adjustment factor for group *i*

AF = absorption fraction (unitless)

CF = conversion factor ($1\ kg_{soil}/10^6\ mg_{soil}$)

70 years = averaging time

5.3.3.2 Commercial Land Use

1. Dermal Absorption SCOs for Non-Cancer Endpoints – Child

$$SCO_{dermal} = \frac{RfD_{adj}}{\frac{1870 cm^2 \times 0.2\ {}^{mg}\!/_{cm^2} \times AF \times CF \times \frac{62}{365} days}{13.3 kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

1870 cm^2 = exposed surface area

0.2 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor ($1\ kg_{soil}/10^6\ mg_{soil}$)

62 days/365 days = exposure frequency

13.3 kg_{bw} = child body weight

2. Dermal Absorption SCOs for Non-Cancer Endpoints – Adult

$$SCO_{dermal} = \frac{RfD_{adj}}{\frac{2480cm^2 \times 0.2\,{}^{mg}\!/_{cm^2} \times AF \times CF \times \frac{124}{365} days}{70kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

2480 cm^2 = exposed surface area

0.2 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6\,mg_{soil}$)

124 days/365 days = exposure frequency

70 kg_{bw} = adult body weight

3. Dermal Absorption SCOs for Cancer Endpoints

$$SCO_{dermal} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF}}{\frac{2480cm^2 \times 0.2\,{}^{mg}\!/_{cm^2} \times AF \times \frac{124}{365} days \times 25 years}{70kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

2480 cm^2 = exposed surface area

0.2 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

124 days/365 days = exposure frequency

25 years = exposure duration

70 years = averaging time

70 kg_{bw} = adult body weight

5.3.3.3 Industrial Land Use

1. Dermal Absorption SCOs for Non-Cancer Endpoints – Adolescent

$$SCO_{dermal} = \frac{RfD_{adj}}{\frac{4530 cm^2 \times 0.07\, {}^{mg}\!/_{cm^2} \times AF \times CF \times \frac{31}{365} days}{58.1 kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

4256 cm^2 = exposed surface area

0.07 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6 mg_{soil}$)

31 days/365 days = exposure frequency

58.1 kg_{bw} = adolescent body weight

2. Dermal Absorption SCOs for Non-Cancer Endpoints – Adult

$$SCO_{dermal} = \frac{RfD_{adj}}{\frac{2480 cm^2 \times 0.2\, {}^{mg}\!/_{cm^2} \times AF \times CF \times \frac{62}{365} days}{70 kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

RfD_{adj} = reference dose, adjusted to account for relative source contribution (mg/kg/day)

2480 cm^2 = exposed surface area

0.2 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

62 days/365 days = exposure frequency

70 kg_{bw} = adult body weight

3. Dermal Absorption SCOs for Cancer Endpoints

$$SCO_{dermal} = \frac{\frac{10^{-6}}{CPF} \times \frac{70}{CF}}{\frac{2480 cm^2 \times 0.2\,{}^{mg}\!/_{cm^2} \times AF \times \frac{62}{365} days \times 25 years}{70 kg_{bw}}}$$

Where:

SCO_{dermal} = soil cleanup objective (mg/kg) for the dermal absorption pathway

10^{-6} = one-in-one-million risk level

CPF = cancer potency factor $(mg/kg/day)^{-1}$

2480 cm^2 = exposed surface area

0.2 mg/cm^2 = soil adherence factor

AF = absorption fraction (unitless)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

62 days/365 days = exposure frequency

25 years = exposure duration

70 years = averaging time

70 kg_{bw} = adult body weight

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US EPA (US Environmental Protection Agency). 2004. Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Washington, DC: Office of Emergency and Remedial Response.

Table 5.3.3.1-1. Exposure Factors Used to Calculate Unrestricted, Residential and Restricted Residential Dermal Absorption SCOs for Cancer Endpoints.

Age Class *i*	Ages in Class	ED (years)	BW (kg)	SA (cm^2)	SAF (mg/cm^2)	EF (days/365 days)	ADAF[1]
1	<1	1	9.1	1870	0.2	0	10
2	1	1	12.3	1870	0.2	155	10
3	2-5	4	16.2	1870	0.2	155	3
4	6-15	10	39.8	4526	0.07	155	3
5	16-17	2	61.3	4526	0.07	155	1
6	18-69	52	69.6	4850	0.07	62	1

[1] For carcinogens that act with a mutagenic mode of action. For all other carcinogens, ADAF is equal to one for ALL age classes.

5.3.4 Chronic Lead SCOs

The derivation of SCOs (non-cancer effects) for lead is substantially different from the approach used to derive SCOs for other soil contaminants. Most public health agencies, including the US EPA (2005b) and the ATSDR (1999) do not have non-cancer toxicity values (i.e., reference dose, and reference concentration) for lead. Rather, these agencies base their environmental guidelines on a blood lead level (see Non-Cancer Effects in Section 5.1.1.7 Toxicity Values for Inorganic Lead). A blood lead level of 10 mcg/dL (10 micrograms of lead per deciliter of blood) is typically used in the guideline derivations. This value is the US Centers for Disease Control and Prevention's level of concern for blood lead in young children (ATSDR, 1999; CDC, 1991). Most guidelines, however, are derived so that the blood levels of almost all children exposed at the guideline would be below 10 mcg/dL. This is the approach taken in the derivation of the SCOs for lead for all land-use categories (see Section 5.3.4.1 to 5.3.4.3).

The human data on lead are inadequate for use in developing cancer toxicity values (i.e., cancer potency factor or inhalation unit risk) for lead (see Cancer Effects in Section 5.1.1.7, Toxicity Values for Inorganic Lead). Some lead compounds are carcinogenic in animals. However, the US EPA (2005b) has not derived cancer toxicity values (i.e., cancer potency factor or inhalation unit risk) for lead (see discussion in Cancer Effects in Section 5.1.1.7, Toxicity Values for Inorganic Lead). Thus, lead SCOs based on cancer effects are not derived.

5.3.4.1 Unrestricted, Residential and Restricted Residential Land Use

1. Children

Many public health agencies (e.g., CA EPA, 1997, 2005; Health Canada, 1992; RIVM, 2001; US EPA, 2000, 2001; WHO, 1996) base their environmental guidelines/standards on maintaining blood lead levels in children below a specific level. However, each agency has taken a different approach to the development of their environmental guideline for lead. We reviewed the approaches and selected the US EPA (2001) approach as the basis for setting lead SCOs (non-cancer effects) for the unrestricted, residential and restricted residential land use categories.

This selection was based on several factors. (1) The US EPA (2001) approach is the only one specifically designed for evaluating the non-cancer health risks of lead in soil and deriving a soil guideline for lead. (2) It allows for an evaluation of multiple routes of exposures, which is an important consideration for a ubiquitous contaminant such as lead. (3) It is a "state-of-the-art" approach that uses recent data on the toxicity and pharmacokinetics of lead in humans. (4) It has undergone peer-review as part of the development of lead cleanup standards for residential soils. (5) It is widely used by federal (US EPA) and state agencies (e.g., FL DEP, 2004; NJ DEP, 2004; MN PCA, 1999) in soil cleanup programs.

The SCOs for the unrestricted, residential and restricted residential land use categories are based on US EPA's (2001) residential soil lead standard of 400 parts per million (ppm) in the bare soil of play areas. In its analysis supporting this rulemaking, the US EPA analyzed the relationship between soil lead levels and blood lead levels in young children using its Integrated Exposure Uptake Biokinetic (IEUBK) Model (US EPA, 2002b). This model is designed to predict the probable blood-lead concentrations for children between six months and seven years of age who have been exposed to lead through several environmental media (air, water, soil, dust, and diet). The model can be used to estimate the risk (i.e., probability) that a child's or a population of children's PbB concentration will exceed 10 mcg/dL when soil lead levels are at a specified level. At 400 ppm, this chance is no greater than 5%. The US EPA used the results of this analysis and consideration of other factors to support 400 ppm as a hazard standard for bare residential soil (see US EPA, 2001 for additional details).

2. Adults

Most assessments of the risk posed by lead in soil in residential settings focus on children (e.g., using the IEUBK model), since children have greater exposures than adults to lead in soil and are more sensitive than adults to the effects of lead. The US EPA (2003) has published an approach (the Adult Lead Methodology or ALM) to evaluate non-residential adult exposures to lead in soil (e.g., workers in commercial or industrial settings). This methodology also can be use to evaluate residential adult lead exposures. The approach focuses on estimating fetal blood lead levels for

women exposed to lead-contaminated soils. The ALM uses a simplified pharmacokinetic model to predict quasi-steady state blood lead concentrations among adult females who have relatively steady patterns of site-related lead exposures. The approach is generally similar to that used by The US EPA to evaluate childhood lead exposures (the IEUBK model). The sensitive population is the fetus, the critical effect is neurotoxicity, and the critical blood lead level for the fetus is 10 mcg/dL. However, the exposed individual is a pregnant woman rather than a young child. The ALM generates estimates of soil lead concentrations at which there would be less than a 5% probability that fetal blood lead levels would exceed 10 mcg/dL (the same cutoff point used by US EPA (2001) in establishing the lead soil standard of 400 ppm).

We used the ALM to calculate the adult SCO for lead for the unrestricted, residential and restricted residential land-use categories. The ALM equation, descriptions of the equation parameters, and values for the parameters used to calculate this SCO are shown below.

$$SCO = \frac{\left(\frac{PbB_{95,\,fetal,goal}}{R \times GSD_i^{1.645}} - PbB_0 \right) \times AT}{BKSF \times IR \times AF \times EF}$$

Where:

SCO = Soil cleanup objective (mcg/g, equivalent to mg/kg).

$PbB_{95,fetal,goal}$ = Goal for the 95th percentile blood lead concentration (mcg/dL) among fetuses born to women having site-related exposures to lead in soil. That is, the likelihood that the fetal blood lead concentration will exceed the goal is less than 5%. ($PbB_{95,fetal,goal}$ = 10 mcg/dL)

R = Constant of proportionality between fetal blood lead concentration at birth and maternal blood lead concentration (dimensionless). (R = 0.9)

$GSD_i^{1.645}$ = Estimated value of the individual geometric standard deviation (dimensionless). The GSD among adults (i.e., women of childbearing age) that have exposures to similar on-site lead concentrations, but that have non-uniform response (intake, biokinetics) site lead and non-uniform off-site lead exposures. The exponent (1.645) is the value of the standard normal deviate used to calculate the 95th percentile from a lognormal distribution of blood lead concentration. (GSD_i = 2.00)

PbB_0 = Typical blood lead concentration (mcg/dL) in adults (i.e., women of childbearing age in the absence of exposures to the site. (PbB_0 = 1.98)

AT = Averaging time (days/year). (AT = 365 days/year)

BKSF = Biokinetic slope factor relating increase in typical adult blood lead concentration to average daily lead uptake (mcg/dL blood lead increase per mcg/day lead uptake). (BKSF = 0.4)

IR = Soil ingestion rate (g/day). (IR = 0.1 g/day)

AF = Absolute gastrointestinal absorption fraction for ingested lead in soil (dimensionless). (AF = 0.12)

EF = Exposure frequency (days/year). (EF = 62 days/year)

In using the ALM approach, we used US EPA (2003) recommended (default) values for $PbB_{95,fetal,goal}$, R, AT, BKSF, and AF (see US EPA, 2003 for information on the basis for each of these values). For the parameters PbB_0 and GSD_i, the US EPA recommends using data from Phases 1 and 2 of the Third National Health and Nutrition Evaluation Survey (NHANES III). The US EPA (2002a) provides summary statistics for these data and from that report we selected values representative of all races/ethnicities for the Northeast region of the US (geometric mean blood lead concentration for women 17 to 45 years of age = 1.98 mcg/dL; geometric standard

deviation = 2.00). For the parameter IR, we used the same value (100 mg/day) used to calculate adult unrestricted and restricted residential soil ingestion SCOs for other chemicals. For the parameter EF we used the same value (62 days/year) that was to calculate the pathway-specific unrestricted and restricted residential land use SCOs for other chemicals. Using the approach described above, we calculated an SCO of 1900 ppm for adults in the unrestricted, residential and restricted residential categories.

5.3.4.2 Commercial Land Use

1. Children

As described in Section 5.2.1 (Exposure Scenarios and Exposure Pathways), the exposure scenarios for unrestricted and restricted land uses assume that a child plays outdoors and has contact with soil five days per week during the warmer months of the year. The exposure scenario for commercial land use assumes that a child is present at a commercial facility two days per week during the warmer months of the year. To derive the commercial SCO for children, we used this difference in exposure frequency (a factor of 2.5) to adjust the unrestricted/restricted residential SCOs (400 ppm). Thus, the lead SCO (non-cancer) for the commercial land use category is 1000 ppm.

2. Adults

To calculate the adult SCO for the commercial land-use category, we used the ALM in the same manner as described above (Section 5.3.4.1, Unrestricted and Restricted Residential Land Use), except that we used an ingestion rate (IR) of 50 mg/day and an exposure frequency (EF) of 124 days/year (see Section 5.2.2.1, Soil Ingestion Pathway). The resulting SCO is 1900 ppm.

5.3.4.3 Industrial Land Use

1. Adolescents

The US EPA (2005a) indicates that the adolescent population may be considered sensitive, since exposures during these years may result in a body burden of lead that is available to transfer to

the fetus later in life. The US EPA (2005a) also indicates that given the limitations of currently available modeling tools, it is reasonable to apply the ALM to adolescent receptors (e.g., trespasser scenarios), provided that appropriate values can be selected for important model parameters. The important model parameters that the US EPA identifies include exposure frequency, exposure duration, baseline blood lead, absorption fraction and biokinetic slope factor.

For the ALM, US EPA (2003) recommends a minimum exposure frequency of one day per week. This value is consistent with exposure frequency we used to calculate adolescent trespasser SCOs for other chemicals (e.g., see Section 5.2.2.1, Soil Ingestion Pathway). The US EPA also recommends a minimum exposure duration of three months (90 days). For other chemical SCOs, we assumed that exposures may occur during the warmer months of the year (i.e., 217 days per year) (see Section 5.2.2.1, Soil Ingestion Pathway). Data on blood lead levels in adolescents are available from CDC (2003). This report identifies a geometric mean blood lead level of 1.1 mcg/dL for 12 to 19 year olds (95^{th}% CI = 1.03 – 1.18 mcg/dL; sample size = 2135), from 1999-2000 NHANES data. However, as described by US EPA (2005a), there are uncertainties in selecting an appropriate baseline blood lead value (PbB_0) for adolescents (e.g., low reported blood lead values may be associated with growth spurts during which there may be increased bone deposition of lead, resulting in lower blood lead concentrations, even though the total body burden of lead may be increasing). The CDC (2003) report did not identify a geometric standard deviation (GSD). The GSD value that we used in applying the ALM to adults is 2.0 (see Section 5.3.4.1). In its IEUBK model for children, the US EPA uses a GSD of 1.6. The absorption fraction (AF) for adults in the ALM is 0.12. In its IEUBK model for children, the US EPA assumes an AF of 0.3 for lead in soil. The US EPA (2005a) indicates that lead absorption may be higher in adolescents than in adults. The US EPA (2005a) also indicates that while biokinetic slope factors (BKSF) for young children and adults appear to be similar, there is uncertainty in identifying estimates for adolescents.

Since uncertainty exists in identifying appropriate values for certain ALM parameters, there would be corresponding uncertainty and variability in adolescent SCOs. For example, if we applied the ALM using an exposure frequency (EF) and soil ingestion rate (IR) as described in

Section 5.2.2.1 (Soil Ingestion Pathway) (31 days/year and 100 mg/day, respectively), PbB_0 = 1.1 mcg/dL, GSD = 1.6, BKSF = 0.4, AF = 0.3, with values of $PbB_{95,fetal,goal}$, R and AT set equal to the values used for adults, an SCO of 3950 ppm is calculated. Changing AF to the adult value of 0.12 yields an SCO of 9880 ppm. If a GSD of 2.0 and an AF of 0.3 are used, the resulting SCO is 2410 ppm. Using a GSD of 2.0 and an AF of 0.12 yields an SCO of 6020.

Given the uncertainty and variability in possible values for adolescent SCO, a non-cancer lead SCO was not developed for the adolescent trespasser scenario.

2. Adults

To calculate the adult SCO for the industrial land-use category, we used the ALM in the same manner as described above (Section 5.3.4.1, Unrestricted and Restricted Residential Land Use), except that we used an ingestion rate (IR) of 50 mg/day and an exposure frequency (EF) of 62 days/year (see Section 5.2.2.1, Soil Ingestion Pathway). The resulting SCO is 3900 ppm.

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5.3.5 Combined Pathway Chronic SCOs

SCOs that are based on combined exposure from the soil ingestion, dermal, and inhalation pathways were calculated for each land use, receptor, and health endpoint. When the equations used to calculate each individual pathway SCO are mathematically combined, they reduce to:

$$SCO_{combined} = \frac{1}{\frac{1}{SCO_{soil}} + \frac{1}{SCO_{dermal}} + \frac{1}{SCO_{inhalation}}}$$

Where:

$SCO_{combined}$ = Soil cleanup objective (mg/kg) based on combined pathways

SCO_{soil} = Soil cleanup objective (mg/kg) based on soil ingestion

SCO_{dermal} = Soil cleanup objective (mg/kg) based on dermal contact

$SCO_{inhalation}$ = Soil cleanup objective (mg/kg) based on inhalation

It is important to note that only SCOs calculated for *the same receptor and health endpoint* can be combined in this way. Because the lowest SCO for a given pathway may not be based on the same receptor or health endpoint as the lowest SCO for another pathway, the lowest combined pathway SCO is not necessarily a combination of the lowest SCO from each pathway. In addition, inhalation-based SCOs for contaminants with non-systemic inhalation toxicity were not combined with SCOs based on ingestion and dermal exposure. The reasoning behind this approach is presented in Section 5.1.2 (Combining Toxicity Values for Systemic Effects). The lowest of the combined pathway SCOs for each land use is presented in Section 5.3.6 (Chronic Human Health-based SCOs).

5.3.6 Chronic Human Health-based SCOs

With the toxicity information and exposure assessment approaches described in the previous sections, chronic health-based SCOs were calculated for each receptor, exposure pathway, and health endpoint (cancer and non-cancer, as appropriate). These values are shown in Table 5.3.6-1 (a-e). We also calculated combined pathway chronic health-based SCOs (cancer and non-cancer, as appropriate) for each receptor and health endpoint for the five land use categories. These SCOs account for combined exposure via soil ingestion (adjusted to allow for dietary consumption pathways for unrestricted and residential land uses), soil particle and soil vapor contaminant inhalation, and dermal exposure. The SCOs are based on estimated chronic exposures of adults, and children or adolescents, as is appropriate for the each exposure scenario and land use. Table 5.3.6-2 presents the chronic health-based SCOs for cancer and non-cancer health endpoints.

Table 5.3.6-1(a). Exposure Pathway-Specific Soil Cleanup Objectives – Unrestricted

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
ACENAPHTHENE	83-32-9		4900	220		110000	7700		-- [2]	-- [2]		4700	210
ACENAPHTHYLENE	208-96-8		4900	220		110000	7700		-- [2]	-- [2]		4700	210
ACETONE	67-64-1		74000	3200					270000	270000		58000	3200
ALDRIN	309-00-2	0.0019	0.49	0.022				2600	250000	460000	0.0019	0.49	0.022
ANTHRACENE	120-12-7		25000	1100		560000	39000		-- [2]	-- [2]		24000	1000
ARSENIC		0.11	25	1.1	11	2400	170	8400	76000	140000	0.11	24	1.1
BARIUM			1600	72					-- [2]	-- [2]		1600	72
BENZENE	71-43-2	3	330	14				5.8	270	270	2	150	14
BENZ(a)ANTHRACENE	56-55-3	0.054	2500	110	1.5	56000	3900	69000	-- [2]	-- [2]	0.052	2400	100
BENZO(a)PYRENE	50-32-8	0.0011	490	22	0.15	56000	3900	6900	-- [2]	-- [2]	0.0011	490	21
BENZO(b)FLUORANTHENE	205-99-2	0.054	2500	110	1.5	56000	3900	69000	-- [2]	-- [2]	0.052	2400	100
BENZO(k)FLUORANTHENE	207-08-9	0.54	2500	110	15	56000	3900	-- [2]	-- [2]	-- [2]	0.52	2400	100
BENZO(g,h,i)PERYLENE	191-24-2		2500	110		56000	3900		-- [2]	-- [2]		2400	100
BERYLLIUM	7440-41-7		160	7.2				5300	18000	33000	5300	160	7.2
n-BUTYLBENZENE	104-51-8		8200	360					3600	3600		2500	330
sec-BUTYLBENZENE	135-98-8		8200	360					3600	3600		2500	330
tert-BUTYLBENZENE	98-06-6		8200	360					3600	3600		2500	330
CADMIUM		0.43	58	2.5	1300	170000	12000	3000	50000	93000	0.43	58	2.5
CARBON TETRACHLORIDE	56-23-5	1.3	58	2.5				3	18	18	0.89	14	2.2
CHLORDANE (alpha)	5103-71-9	0.094	8.2	0.36	36	3000	210	130000	-- [2]	-- [2]	0.094	8.2	0.36
CHLOROBENZENE	108-90-7		1600	72					540	540		410	63
CHLOROFORM	67-66-3	5.3	820	36				660	450	450	5.3	290	33
CHROMIUM (III)			410 [3]	18 [3]					-- [2]	-- [2]		410	18
CHROMIUM (VI)			250	11				250	250000	460000	250	250	11
CHRYSENE	218-01-9	0.54	2500	110	15	56000	3900	690000	-- [2]	-- [2]	0.52	2400	100
COPPER			12000	500					-- [2]	-- [2]		12000	500
CYANIDE			1600	72					-- [2]	-- [2]		1600	72
4,4’-DDD	72-54-8	0.26	8.2	0.36				350000	-- [2]	-- [2]	0.26	8.2	0.36
4,4’-DDE	72-55-9	0.18	200	8.6				240000	-- [2]	-- [2]	0.18	200	8.6
4,4’-DDT	50-29-3	0.17	8.2	0.36	90	4000	280	230000	-- [2]	-- [2]	0.17	8.2	0.36
DIBENZ(a,h)ANTHRACENE	53-70-3	0.0054	2500	110	0.15	56000	3900	6900	-- [2]	-- [2]	0.0052	2400	100

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
DIBENZOFURAN	132-64-9		160	7.2		4900	330		-- [2]	-- [2]		160	7
1,2-DICHLOROBENZENE	95-50-1		1700	76					1800	1800		880	72
1,3-DICHLOROBENZENE	541-73-1		250	11					90	90		66	9.6
1,4-DICHLOROBENZENE	106-46-7	15	2500	110				14	7200	7200	7.3	1800	110
1,1-DICHLOROETHANE	75-34-3	29						28			14		
1,2-DICHLOROETHANE	107-06-2	3.5	3700	160				3.5	3600	3600	1.7	1800	150
1,1-DICHLOROETHENE	75-35-4		4100	180					1800	1800		1300	160
cis-1,2-DICHLOROETHENE	156-59-2		820	36					310	310		230	32
trans-1,2-DICHLOROETHENE	156-60-5		1600	72					540	540		410	63
DIELDRIN	60-57-1	0.0039	0.82	0.036				5300	450000	840000	0.0039	0.82	0.036
1,4-DIOXANE	123-91-1	15	8200	360				14	32000	32000	7.3	6600	360
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--		55 [4]	2.4 [4]					-- [2]	-- [2]		55 [4]	2.4 [4]
ENDRIN	72-20-8		4.9	0.22					-- [2]	-- [2]		4.9	0.22
ETHYLBENZENE	100-41-4	47	8200	360				45	18000	18000	23	5600	350
FLUORANTHENE	206-44-0		3300	140		75000	5200		-- [2]	-- [2]		3200	140
FLUORENE	86-73-7		3300	140		75000	5200		-- [2]	-- [2]		3200	140
HEPTACHLOR	76-44-8	0.042	25	1.1				55000	-- [2]	-- [2]	0.042	25	1.1
HEXACHLOROBENZENE	118-74-1	0.033	13	0.58	5.1	1900	130	44000	-- [2]	-- [2]	0.033	13	0.57
beta-HEXACHLOROCYCLOHEXANE	319-85-7	0.17	0.82	0.036				47000	88000	160000	0.17	0.82	0.036
delta-HEXACHLOROCYCLOHEXANE	319-86-8		2100	90					-- [2]	-- [2]		2100	90
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	0.23	3.3	0.14	18	240	17	63000	350000	650000	0.23	3.3	0.14
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	0.048	41	1.8				13000	-- [2]	-- [2]	0.048	41	1.8
INDENO(1,2,3-cd)PYRENE	193-39-5	0.054	2500	110	1.5	56000	3900	69000	-- [2]	-- [2]	0.052	2400	100
LEAD [5]													
MANGANESE			4100	180					380000	700000		4100	180
MERCURY (ELEMENTAL)	7439-97-6								0.81	0.81		0.81	0.81
MERCURY (INORGANIC SALTS)			2.6	0.12								2.6	0.12
METHYL TERT-BUTYL ETHER	1634-04-4	48	2700	120				170	72000	72000	38	2600	120
METHYLENE CHLORIDE	75-09-2	26	4900	220				1200	3600	3600	26	2100	200
METHYL ETHYL KETONE	78-93-3		49000	2200					45000	45000		24000	2100
2-METHYLPHENOL (o-CRESOL)	95-48-7		4100	180		120000	8400		-- [2]	-- [2]		4000	180
3-METHYLPHENOL (m-CRESOL)	108-39-4		4100	180		120000	8400		-- [2]	-- [2]		4000	180

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
4-METHYLPHENOL (p-CRESOL)	106-44-5		410	18		12000	840		-- [2]	-- [2]		400	18
NAPHTHALENE	91-20-3		1600	72		49000	3300		-- [2]	-- [2]		1600	70
NICKEL			1600	72				26000	230000	420000	26000	1600	72
PENTACHLOROPHENOL	87-86-5	1.4	82	3.6	17	970	67	370000	-- [2]	-- [2]	1.3	76	3.4
PHENANTHRENE	85-01-8		2500	110		56000	3900		-- [2]	-- [2]		2400	100
PHENOL	108-95-2		25000	1100		730000	50000		-- [2]	-- [2]		24000	1100
POLYCHLORINATED BIPHENYLS [6]	1336-36-3												
n-PROPYLBENZENE	103-65-1		8200	360					3600	3600		2500	330
PYRENE	129-00-0		2500	110		56000	3900		-- [2]	-- [2]		2400	100
SELENIUM			410	18					-- [2]	-- [2]		410	18
SILVER			410	18					-- [2]	-- [2]		410	18
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1		660	29					-- [2]	-- [2]		660	29
TETRACHLOROETHENE	127-18-4	3.2	820	36				45	900	900	2.9	430	35
TOLUENE	108-88-3		16000	720					2700	2700		2300	570
1,1,1-TRICHLOROETHANE	71-55-6		23000	1000					20000	20000		11000	960
TRICHLOROETHENE	79-01-6	29	120	5.3				22	360	360	13	90	5.2
1,2,4-TRIMETHYLBENZENE	95-63-6		4100	180					54	54		53	41
1,3,5-TRIMETHYLBENZENE	108-67-8		4100	180					54	54		53	41
VINYL CHLORIDE	75-01-4	0.11	250	11				5.1	900	900	0.11	190	11
XYLENES	1330-20-7		16000 [7]	720 [7]					900 [7]	900 [7]		850 [7]	400 [7]
ZINC			25000	1100					-- [2]	-- [2]		25000	1100

Footnotes:

[1] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

[2] Calculated value equals or exceeds one million milligrams of substance per kilogram of soil.

[3] Calculated values based on the soluble salts oral reference dose of 0.005 mg/kg/day (Table 5.1.1-2). If the insoluble salts oral reference dose of 1.5 mg/kg/day (Table 5.1.1-2) is used, the calculated values will be higher.

[4] As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.

[5] See Section 5.3.4 for a discussion of SCOs for lead.

[6] Based on the US EPA regulation "Disposal of Polychlorinated Biphenyls (PCBs)" (40 CFR 716) (US EPA, 1998); see Section 6.0.

[7] As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

Table 5.3.6-1(b). Exposure Pathway-Specific Soil Cleanup Objectives – Residential

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
ACENAPHTHENE	83-32-9		9900	430		110000	7700		-- [2]	-- [2]		9100	410
ACENAPHTHYLENE	208-96-8		9900	430		110000	7700		-- [2]	-- [2]		9100	410
ACETONE	67-64-1		150000	6500					270000	270000		96000	6300
ALDRIN	309-00-2	0.019	4.9	0.22				2600	250000	460000	0.019	4.9	0.22
ANTHRACENE	120-12-7		49000	2200		560000	39000		-- [2]	-- [2]		45000	2000
ARSENIC		0.22	49	2.2	11	2400	170	8400	76000	140000	0.21	48	2.1
BARIUM			3300	140					-- [2]	-- [2]		3300	140
BENZENE	71-43-2	6	660	29				5.8	270	270	2.9	190	26
BENZ(a)ANTHRACENE	56-55-3	0.11	4900	220	1.5	56000	3900	69000	-- [2]	-- [2]	0.1	4500	200
BENZO(a)PYRENE	50-32-8	0.011	4900	220	0.15	56000	3900	6900	-- [2]	-- [2]	0.01	4500	200
BENZO(b)FLUORANTHENE	205-99-2	0.11	4900	220	1.5	56000	3900	69000	-- [2]	-- [2]	0.1	4500	200
BENZO(k)FLUORANTHENE	207-08-9	1.1	4900	220	15	56000	3900	-- [2]	-- [2]	-- [2]	1	4500	200
BENZO(g,h,i)PERYLENE	191-24-2		4900	220		56000	3900		-- [2]	-- [2]		4500	200
BERYLLIUM	7440-41-7		330	14				5300	18000	33000	5300	330	14
n-BUTYLBENZENE	104-51-8		16000	720					3600	3600		2900	600
sec-BUTYLBENZENE	135-98-8		16000	720					3600	3600		2900	600
tert-BUTYLBENZENE	98-06-6		16000	720					3600	3600		2900	600
CADMIUM		0.86	120	5	1300	170000	12000	3000	50000	93000	0.86	120	5
CARBON TETRACHLORIDE	56-23-5	2.5	120	5				3	18	18	1.4	16	3.9
CHLORDANE (alpha)	5103-71-9	0.94	82	3.6	36	3000	210	130000	-- [2]	-- [2]	0.91	80	3.5
CHLOROBENZENE	108-90-7		3300	140					540	540		460	110
CHLOROFORM	67-66-3	11	1600	72				660	450	450	10	350	62
CHROMIUM (III)			820 [3]	36 [3]					-- [2]	-- [2]		820	36
CHROMIUM (VI)			490	22				250	250000	460000	250	490	22
CHRYSENE	218-01-9	1.1	4900	220	15	56000	3900	690000	-- [2]	-- [2]	1	4500	200
COPPER			23000	1000					-- [2]	-- [2]		23000	1000
CYANIDE			3300	140					-- [2]	-- [2]		3300	140
4,4’-DDD	72-54-8	2.6	82	3.6				350000	-- [2]	-- [2]	2.6	82	3.6
4,4’-DDE	72-55-9	1.8	2000	86				240000	-- [2]	-- [2]	1.8	2000	86
4,4’-DDT	50-29-3	1.7	82	3.6	90	4000	280	230000	-- [2]	-- [2]	1.7	81	3.6
DIBENZ(a,h)ANTHRACENE	53-70-3	0.011	4900	220	0.15	56000	3900	6900	-- [2]	-- [2]	0.01	4500	200

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
DIBENZOFURAN	132-64-9		330	14		4900	330		-- [2]	-- [2]		310	14
1,2-DICHLOROBENZENE	95-50-1		3500	150					1800	1800		1200	140
1,3-DICHLOROBENZENE	541-73-1		490	22					90	90		76	17
1,4-DICHLOROBENZENE	106-46-7	30	4900	220				14	7200	7200	9.8	2900	210
1,1-DICHLOROETHANE	75-34-3	58						28			19		
1,2-DICHLOROETHANE	107-06-2	7	7400	320				3.5	3600	3600	2.3	2400	300
1,1-DICHLOROETHENE	75-35-4		8200	360					1800	1800		1500	300
cis-1,2-DICHLOROETHENE	156-59-2		1600	72					310	310		260	59
trans-1,2-DICHLOROETHENE	156-60-5		3300	140					540	540		460	110
DIELDRIN	60-57-1	0.039	8.2	0.36				5300	450000	840000	0.039	8.2	0.36
1,4-DIOXANE	123-91-1	30	16000	720				14	32000	32000	9.8	11000	700
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--		110 [4]	4.8 [4]					-- [2]	-- [2]		110 [4]	4.8 [4]
ENDRIN	72-20-8		49	2.2					-- [2]	-- [2]		49	2.2
ETHYLBENZENE	100-41-4	94	16000	720				45	18000	18000	30	8600	690
FLUORANTHENE	206-44-0		6600	290		75000	5200		-- [2]	-- [2]		6100	270
FLUORENE	86-73-7		6600	290		75000	5200		-- [2]	-- [2]		6100	270
HEPTACHLOR	76-44-8	0.42	250	11				55000	-- [2]	-- [2]	0.42	250	11
HEXACHLOROBENZENE	118-74-1	0.33	130	5.8	5.1	1900	130	44000	-- [2]	-- [2]	0.31	120	5.5
beta-HEXACHLOROCYCLOHEXANE	319-85-7	0.34	1.6	0.072				47000	88000	160000	0.34	1.6	0.072
delta-HEXACHLOROCYCLOHEXANE	319-86-8		4100	180					-- [2]	-- [2]		4100	180
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	0.46	6.6	0.29	18	240	17	63000	350000	650000	0.45	6.4	0.28
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	0.097	82	3.6				13000	-- [2]	-- [2]	0.097	82	3.6
INDENO(1,2,3-cd)PYRENE	193-39-5	0.11	4900	220	1.5	56000	3900	69000	-- [2]	-- [2]	0.1	4500	200
LEAD [5]													
MANGANESE			8200	360					380000	700000		8100	360
MERCURY (ELEMENTAL)	7439-97-6								0.81	0.81		0.81	0.81
MERCURY (INORGANIC SALTS)			26	1.2								26	1.2
METHYL TERT-BUTYL ETHER	1634-04-4	97	5400	240				170	72000	72000	62	5100	240
METHYLENE CHLORIDE	75-09-2	53	9900	430				1200	3600	3600	51	2600	390
METHYL ETHYL KETONE	78-93-3		99000	4300					45000	45000		31000	3900
2-METHYLPHENOL (o-CRESOL)	95-48-7		8200	360		120000	8400		-- [2]	-- [2]		7700	340
3-METHYLPHENOL (m-CRESOL)	108-39-4		8200	360		120000	8400		-- [2]	-- [2]		7700	340

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
4-METHYLPHENOL (p-CRESOL)	106-44-5		820	36		12000	840		-- [2]	-- [2]		770	34
NAPHTHALENE	91-20-3		3300	140		49000	3300		-- [2]	-- [2]		3100	140
NICKEL			3300	140				26000	230000	420000	26000	3300	140
PENTACHLOROPHENOL	87-86-5	2.7	160	7.2	17	970	67	370000	-- [2]	-- [2]	2.4	140	6.5
PHENANTHRENE	85-01-8		4900	220		56000	3900		-- [2]	-- [2]		4500	200
PHENOL	108-95-2		49000	2200		730000	50000		-- [2]	-- [2]		46000	2100
POLYCHLORINATED BIPHENYLS [6]	1336-36-3												
n-PROPYLBENZENE	103-65-1		16000	720					3600	3600		2900	600
PYRENE	129-00-0		4900	220		56000	3900		-- [2]	-- [2]		4500	200
SELENIUM			820	36					-- [2]	-- [2]		820	36
SILVER			820	36					-- [2]	-- [2]		820	36
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1		1300	58					-- [2]	-- [2]		1300	58
TETRACHLOROETHENE	127-18-4	6.3	1600	72				45	900	900	5.5	580	67
TOLUENE	108-88-3		33000	1400					2700	2700		2500	940
1,1,1-TRICHLOROETHANE	71-55-6		46000	2000					20000	20000		14000	1800
TRICHLOROETHENE	79-01-6	57	240	11				22	360	360	16	140	10
1,2,4-TRIMETHYLBENZENE	95-63-6		8200	360					54	54		54	47
1,3,5-TRIMETHYLBENZENE	108-67-8		8200	360					54	54		54	47
VINYL CHLORIDE	75-01-4	0.22	490	22				5.1	900	900	0.21	320	21
XYLENES	1330-20-7		33000 [7]	1400 [7]					900 [7]	900 [7]		870 [7]	550 [7]
ZINC			49000	2200					-- [2]	-- [2]		49000	2200

Footnotes:

[1] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

[2] Calculated value equals or exceeds one million milligrams of substance per kilogram of soil.

[3] Calculated values based on the soluble salts oral reference dose of 0.005 mg/kg/day (Table 5.1.1-2). If the insoluble salts oral reference dose of 1.5 mg/kg/day (Table 5.1.1-2) is used, the calculated values will be higher.

[4] As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.

[5] See Section 5.3.4 for a discussion of SCOs for lead.

[6] Based on the US EPA regulation "Disposal of Polychlorinated Biphenyls (PCBs)" (40 CFR 716) (US EPA, 1998); see Section 6.0.

[7] As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

Table 5.3.6-1(c). Exposure Pathway-Specific Soil Cleanup Objectives – Restricted Residential

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
ACENAPHTHENE	83-32-9		49000	2200		110000	7700		-- [2]	-- [2]		34000	1700
ACENAPHTHYLENE	208-96-8		49000	2200		110000	7700		-- [2]	-- [2]		34000	1700
ACETONE	67-64-1		740000	32000					270000	270000		200000	29000
ALDRIN	309-00-2	0.097	25	1.1				2600	250000	460000	0.097	25	1.1
ANTHRACENE	120-12-7		250000	11000		560000	39000		-- [2]	-- [2]		170000	8400
ARSENIC		1.1	250	11	11	2400	170	8400	76000	140000	1	220	10
BARIUM			16000	720					-- [2]	-- [2]		16000	720
BENZENE	71-43-2	30	3300	140				5.8	270	270	4.8	250	94
BENZ(a)ANTHRACENE	56-55-3	0.54	25000	1100	1.5	56000	3900	69000	-- [2]	-- [2]	0.39	17000	840
BENZO(a)PYRENE	50-32-8	0.054	25000	1100	0.15	56000	3900	6900	-- [2]	-- [2]	0.039	17000	840
BENZO(b)FLUORANTHENE	205-99-2	0.54	25000	1100	1.5	56000	3900	69000	-- [2]	-- [2]	0.39	17000	840
BENZO(k)FLUORANTHENE	207-08-9	5.4	25000	1100	15	56000	3900	-- [2]	-- [2]	-- [2]	3.9	17000	840
BENZO(g,h,i)PERYLENE	191-24-2		25000	1100		56000	3900		-- [2]	-- [2]		17000	840
BERYLLIUM	7440-41-7		1600	72				5300	18000	33000	5300	1600	72
n-BUTYLBENZENE	104-51-8		82000	3600					3600	3600		3400	1800
sec-BUTYLBENZENE	135-98-8		82000	3600					3600	3600		3400	1800
tert-BUTYLBENZENE	98-06-6		82000	3600					3600	3600		3400	1800
CADMIUM		4.3	580	25	1300	170000	12000	3000	50000	93000	4.3	570	25
CARBON TETRACHLORIDE	56-23-5	13	580	25				3	18	18	2.4	17	10
CHLORDANE (alpha)	5103-71-9	4.7	410	18	36	3000	210	130000	-- [2]	-- [2]	4.2	360	17
CHLOROBENZENE	108-90-7		16000	720					540	540		520	310
CHLOROFORM	67-66-3	53	8200	360				660	450	450	49	430	200
CHROMIUM (III)			4100 [3]	180 [3]					-- [2]	-- [2]		4100	180
CHROMIUM (VI)			2500	110				250	250000	460000	250	2500	110
CHRYSENE	218-01-9	5.4	25000	1100	15	56000	3900	690000	-- [2]	-- [2]	3.9	17000	840
COPPER			120000	5000					-- [2]	-- [2]		120000	5000
CYANIDE			16000	720					-- [2]	-- [2]		16000	720
4,4’-DDD	72-54-8	13	410	18				350000	-- [2]	-- [2]	13	410	18
4,4’-DDE	72-55-9	8.9	9900	430				240000	-- [2]	-- [2]	8.9	9900	430
4,4’-DDT	50-29-3	8.7	410	18	90	4000	280	230000	-- [2]	-- [2]	7.9	370	17
DIBENZ(a,h)ANTHRACENE	53-70-3	0.054	25000	1100	0.15	56000	3900	6900	-- [2]	-- [2]	0.039	17000	840

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
DIBENZOFURAN	132-64-9		1600	72		4900	330		-- [2]	-- [2]		1200	59
1,2-DICHLOROBENZENE	95-50-1		17000	760					1800	1800		1600	530
1,3-DICHLOROBENZENE	541-73-1		2500	110					90	90		87	49
1,4-DICHLOROBENZENE	106-46-7	150	25000	1100				14	7200	7200	13	5600	940
1,1-DICHLOROETHANE	75-34-3	290						28			26		
1,2-DICHLOROETHANE	107-06-2	35	37000	1600				3.5	3600	3600	3.1	3300	1100
1,1-DICHLOROETHENE	75-35-4		41000	1800					1800	1800		1700	900
cis-1,2-DICHLOROETHENE	156-59-2		8200	360					310	310		300	170
trans-1,2-DICHLOROETHENE	156-60-5		16000	720					540	540		520	310
DIELDRIN	60-57-1	0.2	41	1.8				5300	450000	840000	0.2	41	1.8
1,4-DIOXANE	123-91-1	150	82000	3600				14	32000	32000	13	23000	3200
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--		550 [4]	24 [4]					-- [2]	-- [2]		550 [4]	24 [4]
ENDRIN	72-20-8		250	11					-- [2]	-- [2]		250	11
ETHYLBENZENE	100-41-4	470	82000	3600				45	18000	18000	41	15000	3000
FLUORANTHENE	206-44-0		33000	1400		75000	5200		-- [2]	-- [2]		23000	1100
FLUORENE	86-73-7		33000	1400		75000	5200		-- [2]	-- [2]		23000	1100
HEPTACHLOR	76-44-8	2.1	1200	54				55000	-- [2]	-- [2]	2.1	1200	54
HEXACHLOROBENZENE	118-74-1	1.6	660	29	5.1	1900	130	44000	-- [2]	-- [2]	1.2	490	24
beta-HEXACHLOROCYCLOHEXANE	319-85-7	1.7	8.2	0.36				47000	88000	160000	1.7	8.2	0.36
delta-HEXACHLOROCYCLOHEXANE	319-86-8		21000	900					-- [2]	-- [2]		21000	900
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	2.3	33	1.4	18	240	17	63000	350000	650000	2	29	1.3
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	0.48	410	18				13000	-- [2]	-- [2]	0.48	410	18
INDENO(1,2,3-cd)PYRENE	193-39-5	0.54	25000	1100	1.5	56000	3900	69000	-- [2]	-- [2]	0.39	17000	840
LEAD [5]													
MANGANESE			41000	1800					380000	700000		37000	1800
MERCURY (ELEMENTAL)	7439-97-6								0.81	0.81		0.81	0.81
MERCURY (INORGANIC SALTS)			130	5.8								130	5.8
METHYL TERT-BUTYL ETHER	1634-04-4	480	27000	1200				170	72000	72000	130	20000	1200
METHYLENE CHLORIDE	75-09-2	260	49000	2200				1200	3600	3600	220	3300	1300
METHYL ETHYL KETONE	78-93-3		490000	22000					45000	45000		41000	15000
2-METHYLPHENOL (o-CRESOL)	95-48-7		41000	1800		120000	8400		-- [2]	-- [2]		31000	1500
3-METHYLPHENOL (m-CRESOL)	108-39-4		41000	1800		120000	8400		-- [2]	-- [2]		31000	1500

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
4-METHYLPHENOL (p-CRESOL)	106-44-5		4100	180		12000	840		-- [2]	-- [2]		3100	150
NAPHTHALENE	91-20-3		16000	720		49000	3300		-- [2]	-- [2]		12000	590
NICKEL			16000	720				26000	230000	420000	26000	16000	720
PENTACHLOROPHENOL	87-86-5	14	820	36	17	970	67	370000	-- [2]	-- [2]	7.6	450	23
PHENANTHRENE	85-01-8		25000	1100		56000	3900		-- [2]	-- [2]		17000	840
PHENOL	108-95-2		250000	11000		730000	50000		-- [2]	-- [2]		180000	8900
POLYCHLORINATED BIPHENYLS [6]	1336-36-3												
n-PROPYLBENZENE	103-65-1		82000	3600					3600	3600		3400	1800
PYRENE	129-00-0		25000	1100		56000	3900		-- [2]	-- [2]		17000	840
SELENIUM			4100	180					-- [2]	-- [2]		4100	180
SILVER			4100	180					-- [2]	-- [2]		4100	180
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1		6600	290					-- [2]	-- [2]		6600	290
TETRACHLOROETHENE	127-18-4	32	8200	360				45	900	900	19	810	260
TOLUENE	108-88-3		160000	7200					2700	2700		2700	2000
1,1,1-TRICHLOROETHANE	71-55-6		230000	10000					20000	20000		18000	6700
TRICHLOROETHENE	79-01-6	290	1200	53				22	360	360	21	280	46
1,2,4-TRIMETHYLBENZENE	95-63-6		41000	1800					54	54		54	52
1,3,5-TRIMETHYLBENZENE	108-67-8		41000	1800					54	54		54	52
VINYL CHLORIDE	75-01-4	1.1	2500	110				5.1	900	900	0.9	660	96
XYLENES	1330-20-7		160000 [7]	7200 [7]					900 [7]	900 [7]		890 [7]	800 [7]
ZINC			250000	11000					-- [2]	-- [2]		250000	11000

Footnotes:

[1] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

[2] Calculated value equals or exceeds one million milligrams of substance per kilogram of soil.

[3] Calculated values based on the soluble salts oral reference dose of 0.005 mg/kg/day (Table 5.1.1-2). If the insoluble salts oral reference dose of 1.5 mg/kg/day (Table 5.1.1-2) is used, the calculated values will be higher.

[4] As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.

[5] See Section 5.3.4 for a discussion of SCOs for lead.

[6] Based on the US EPA regulation "Disposal of Polychlorinated Biphenyls (PCBs)" (40 CFR 716) (US EPA, 1998); see Section 6.0.

[7] As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

Table 5.3.6-1(d). Exposure Pathway-Specific Soil Cleanup Objectives – Commercial

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
ACENAPHTHENE	83-32-9		49000	18000		38000	19000		-- [2]	-- [2]		22000	9200
ACENAPHTHYLENE	208-96-8		49000	18000		38000	19000		-- [2]	-- [2]		22000	9200
ACETONE	67-64-1		740000	270000					940000	-- [2]		420000	260000
ALDRIN	309-00-2	0.68	25	8.9				4100	140000	-- [2]	0.68	25	8.9
ANTHRACENE	120-12-7		250000	89000		190000	97000		-- [2]	-- [2]		110000	46000
ARSENIC		7.7	250	89	26	830	420	13000	43000	530000	5.9	190	73
BARIUM			16000	5900					710000	-- [2]		16000	5900
BENZENE	71-43-2	210	3300	1200				56	940	12000	44	730	1100
BENZ(a)ANTHRACENE	56-55-3	13	25000	8900	9.9	19000	9700	180000	-- [2]	-- [2]	5.6	11000	4600
BENZO(a)PYRENE	50-32-8	1.3	25000	8900	0.99	19000	9700	18000	-- [2]	-- [2]	0.56	11000	4600
BENZO(b)FLUORANTHENE	205-99-2	13	25000	8900	9.9	19000	9700	180000	-- [2]	-- [2]	5.6	11000	4600
BENZO(k)FLUORANTHENE	207-08-9	130	25000	8900	99	19000	9700	-- [2]	-- [2]	-- [2]	56	11000	4600
BENZO(g,h,i)PERYLENE	191-24-2		25000	8900		19000	9700		-- [2]	-- [2]		11000	4600
BERYLLIUM	7440-41-7		1600	590				8300	10000	120000	8300	1600	590
n-BUTYLBENZENE	104-51-8		82000	30000					13000	160000		11000	25000
sec-BUTYLBENZENE	135-98-8		82000	30000					13000	160000		11000	25000
tert-BUTYLBENZENE	98-06-6		82000	30000					13000	160000		11000	25000
CADMIUM		30	580	210	3100	58000	29000	4700	28000	350000	30	560	210
CARBON TETRACHLORIDE	56-23-5	89	580	210				29	63	780	22	57	160
CHLORDANE (alpha)	5103-71-9	33	410	150	83	1000	520	200000	-- [2]	-- [2]	24	290	120
CHLOROBENZENE	108-90-7		16000	5900					1900	23000		1700	4700
CHLOROFORM	67-66-3	370	8200	3000				6500	1600	19000	350	1300	2600
CHROMIUM (III)			4100 [3]	1500 [3]					-- [2]	-- [2]		4100	1500
CHROMIUM (VI)			2500	890				400	140000	-- [2]	400	2500	890
CHRYSENE	218-01-9	130	25000	8900	99	19000	9700	-- [2]	-- [2]	-- [2]	56	11000	4600
COPPER			120000	41000					-- [2]	-- [2]		120000	41000
CYANIDE			16000	5900					-- [2]	-- [2]		16000	5900
4,4’-DDD	72-54-8	92	410	150				550000	-- [2]	-- [2]	92	410	150
4,4’-DDE	72-55-9	62	9900	3500				380000	-- [2]	-- [2]	62	9900	3500
4,4’-DDT	50-29-3	61	410	150	210	1400	700	370000	-- [2]	-- [2]	47	320	120
DIBENZ(a,h)ANTHRACENE	53-70-3	1.3	25000	8900	0.99	19000	9700	18000	-- [2]	-- [2]	0.56	11000	4600

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
DIBENZOFURAN	132-64-9		1600	590		1700	840		-- [2]	-- [2]		830	350
1,2-DICHLOROBENZENE	95-50-1		17000	6200					6300	78000		4600	5700
1,3-DICHLOROBENZENE	541-73-1		2500	890					310	3900		280	720
1,4-DICHLOROBENZENE	106-46-7	1000	25000	8900				140	25000	310000	130	12000	8600
1,1-DICHLOROETHANE	75-34-3	2000						280			240		
1,2-DICHLOROETHANE	107-06-2	250	37000	13000				34	13000	160000	30	9400	12000
1,1-DICHLOROETHENE	75-35-4		41000	15000					6300	78000		5500	12000
cis-1,2-DICHLOROETHENE	156-59-2		8200	3000					1100	14000		970	2400
trans-1,2-DICHLOROETHENE	156-60-5		16000	5900					1900	23000		1700	4700
DIELDRIN	60-57-1	1.4	41	15				8300	260000	-- [2]	1.4	41	15
1,4-DIOXANE	123-91-1	1000	82000	30000				140	110000	-- [2]	130	48000	29000
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--		550 [4]	200 [4]					-- [2]	-- [2]		550 [4]	200 [4]
ENDRIN	72-20-8		250	89					-- [2]	-- [2]		250	89
ETHYLBENZENE	100-41-4	3300	82000	30000				440	63000	780000	390	36000	28000
FLUORANTHENE	206-44-0		33000	12000		26000	13000		-- [2]	-- [2]		14000	6200
FLUORENE	86-73-7		33000	12000		26000	13000		-- [2]	-- [2]		14000	6200
HEPTACHLOR	76-44-8	15	1200	440				87000	-- [2]	-- [2]	15	1200	440
HEXACHLOROBENZENE	118-74-1	12	660	240	12	660	330	69000	-- [2]	-- [2]	5.8	330	140
beta-HEXACHLOROCYCLOHEXANE	319-85-7	12	8.2	3				74000	50000	620000	12	8.2	3
delta-HEXACHLOROCYCLOHEXANE	319-86-8		21000	7400					-- [2]	-- [2]		21000	7400
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	16	33	12	41	83	42	100000	200000	-- [2]	12	24	9.2
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	3.4	410	150				21000	-- [2]	-- [2]	3.4	410	150
INDENO(1,2,3-cd)PYRENE	193-39-5	13	25000	8900	9.9	19000	9700	180000	-- [2]	-- [2]	5.6	11000	4600
LEAD [5]													
MANGANESE			41000	15000					210000	-- [2]		35000	15000
MERCURY (ELEMENTAL)	7439-97-6								2.8	35		2.8	35
MERCURY (INORGANIC SALTS)			130	47								130	47
METHYL TERT-BUTYL ETHER	1634-04-4	3400	27000	9800				1700	250000	-- [2]	1100	25000	9700
METHYLENE CHLORIDE	75-09-2	1900	49000	18000				12000	13000	160000	1600	10000	16000
METHYL ETHYL KETONE	78-93-3		490000	180000					160000	-- [2]		120000	160000
2-METHYLPHENOL (o-CRESOL)	95-48-7		41000	15000		42000	21000		-- [2]	-- [2]		21000	8700
3-METHYLPHENOL (m-CRESOL)	108-39-4		41000	15000		42000	21000		-- [2]	-- [2]		21000	8700

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
4-METHYLPHENOL (p-CRESOL)	106-44-5		4100	1500		4200	2100		-- [2]	-- [2]		2100	870
NAPHTHALENE	91-20-3		16000	5900		17000	8400		-- [2]	-- [2]		8300	3500
NICKEL			16000	5900				42000	130000	-- [2]	42000	16000	5900
PENTACHLOROPHENOL	87-86-5	96	820	300	39	330	170	590000	-- [2]	-- [2]	28	240	110
PHENANTHRENE	85-01-8		25000	8900		19000	9700		-- [2]	-- [2]		11000	4600
PHENOL	108-95-2		250000	89000		250000	130000		-- [2]	-- [2]		120000	52000
POLYCHLORINATED BIPHENYLS [6]	1336-36-3												
n-PROPYLBENZENE	103-65-1		82000	30000					13000	160000		11000	25000
PYRENE	129-00-0		25000	8900		19000	9700		-- [2]	-- [2]		11000	4600
SELENIUM			4100	1500					-- [2]	-- [2]		4100	1500
SILVER			4100	1500					-- [2]	-- [2]		4100	1500
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1		6600	2400					-- [2]	-- [2]		6600	2400
TETRACHLOROETHENE	127-18-4	220	8200	3000				440	3100	39000	150	2300	2700
TOLUENE	108-88-3		160000	59000					9400	120000		8900	39000
1,1,1-TRICHLOROETHANE	71-55-6		230000	83000					69000	860000		53000	75000
TRICHLOROETHENE	79-01-6	2000	1200	430				220	1300	16000	200	610	420
1,2,4-TRIMETHYLBENZENE	95-63-6		41000	15000					190	2300		190	2000
1,3,5-TRIMETHYLBENZENE	108-67-8		41000	15000					190	2300		190	2000
VINYL CHLORIDE	75-01-4	15	2500	890				100	3100	39000	13	1400	870
XYLENES	1330-20-7		160000 [7]	59000 [7]					3100 [7]	39000 [7]		3100 [7]	23000 [7]
ZINC			250000	89000					-- [2]	-- [2]		250000	89000

Footnotes:

[1] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

[2] Calculated value equals or exceeds one million milligrams of substance per kilogram of soil.

[3] Calculated values based on the soluble salts oral reference dose of 0.005 mg/kg/day (Table 5.1.1-2). If the insoluble salts oral reference dose of 1.5 mg/kg/day (Table 5.1.1-2) is used, the calculated values will be higher.

[4] As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.

[5] See Section 5.3.4 for a discussion of SCOs for lead.

[6] Based on the US EPA regulation "Disposal of Polychlorinated Biphenyls (PCBs)" (40 CFR 716) (US EPA, 1998); see Section 6.0.

[7] As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

Table 5.3.6-1(e). Exposure Pathway-Specific Soil Cleanup Objectives – Industrial

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
ACENAPHTHENE	83-32-9		99000	82000		77000	200000		-- [2]	-- [2]		43000	58000
ACENAPHTHYLENE	208-96-8		99000	82000		77000	200000		-- [2]	-- [2]		43000	58000
ACETONE	67-64-1		-- [2]	-- [2]					-- [2]	-- [2]		830000	-- [2]
ALDRIN	309-00-2	1.4	49	41				8100	280000	-- [2]	1.4	49	41
ANTHRACENE	120-12-7		490000	410000		380000	-- [2]		-- [2]	-- [2]		220000	290000
ARSENIC		15	490	410	52	1700	4300	27000	85000	530000	12	380	370
BARIUM			33000	27000					-- [2]	-- [2]		32000	27000
BENZENE	71-43-2	420	6600	5500				110	1900	12000	89	1500	3700
BENZ(a)ANTHRACENE	56-55-3	26	49000	41000	20	38000	100000	360000	-- [2]	-- [2]	11	22000	29000
BENZO(a)PYRENE	50-32-8	2.6	49000	41000	2	38000	100000	36000	-- [2]	-- [2]	1.1	22000	29000
BENZO(b)FLUORANTHENE	205-99-2	26	49000	41000	20	38000	100000	360000	-- [2]	-- [2]	11	22000	29000
BENZO(k)FLUORANTHENE	207-08-9	260	49000	41000	200	38000	100000	-- [2]	-- [2]	-- [2]	110	22000	29000
BENZO(g,h,i)PERYLENE	191-24-2		49000	41000		38000	100000		-- [2]	-- [2]		22000	29000
BERYLLIUM	7440-41-7		3300	2700				17000	20000	120000	17000	3300	2700
n-BUTYLBENZENE	104-51-8		160000	140000					25000	160000		22000	73000
sec-BUTYLBENZENE	135-98-8		160000	140000					25000	160000		22000	73000
tert-BUTYLBENZENE	98-06-6		160000	140000					25000	160000		22000	73000
CADMIUM		61	1200	960	6100	120000	300000	9500	57000	350000	60	1100	950
CARBON TETRACHLORIDE	56-23-5	180	1200	960				59	130	780	44	110	430
CHLORDANE (alpha)	5103-71-9	66	820	680	170	2100	5400	400000	-- [2]	-- [2]	47	590	610
CHLOROBENZENE	108-90-7		33000	27000					3800	23000		3400	13000
CHLOROFORM	67-66-3	740	16000	14000				13000	3100	19000	700	2600	8000
CHROMIUM (III)			8200 [3]	6800 [3]					-- [2]	-- [2]		8200	6800
CHROMIUM (VI)			4900	4100				800	280000	-- [2]	800	4900	4100
CHRYSENE	218-01-9	260	49000	41000	200	38000	100000	-- [2]	-- [2]	-- [2]	110	22000	29000
COPPER			230000	190000					-- [2]	-- [2]		230000	190000
CYANIDE			33000	27000					-- [2]	-- [2]		33000	27000
4,4’-DDD	72-54-8	180	820	680				-- [2]	-- [2]	-- [2]	180	820	680
4,4’-DDE	72-55-9	120	20000	16000				750000	-- [2]	-- [2]	120	20000	16000
4,4’-DDT	50-29-3	120	820	680	410	2800	7200	740000	-- [2]	-- [2]	94	640	620
DIBENZ(a,h)ANTHRACENE	53-70-3	2.6	49000	41000	2	38000	100000	36000	-- [2]	-- [2]	1.1	22000	29000

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
DIBENZOFURAN	132-64-9		3300	2700		3300	8600		-- [2]	-- [2]		1700	2100
1,2-DICHLOROBENZENE	95-50-1		35000	29000					13000	78000		9200	21000
1,3-DICHLOROBENZENE	541-73-1		4900	4100					630	3900		560	2000
1,4-DICHLOROBENZENE	106-46-7	2100	49000	41000				280	50000	310000	250	25000	36000
1,1-DICHLOROETHANE	75-34-3	4000						550			480		
1,2-DICHLOROETHANE	107-06-2	490	74000	62000				68	25000	160000	60	19000	44000
1,1-DICHLOROETHENE	75-35-4		82000	68000					13000	78000		11000	36000
cis-1,2-DICHLOROETHENE	156-59-2		16000	14000					2200	14000		1900	6800
trans-1,2-DICHLOROETHENE	156-60-5		33000	27000					3800	23000		3400	13000
DIELDRIN	60-57-1	2.8	82	68				17000	510000	-- [2]	2.8	82	68
1,4-DIOXANE	123-91-1	2100	160000	140000				280	230000	-- [2]	250	95000	120000
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--		1100 [4]	920 [4]					-- [2]	-- [2]		1100 [4]	920 [4]
ENDRIN	72-20-8		490	410					-- [2]	-- [2]		490	410
ETHYLBENZENE	100-41-4	6600	160000	140000				880	130000	780000	780	71000	120000
FLUORANTHENE	206-44-0		66000	55000		51000	130000		-- [2]	-- [2]		29000	39000
FLUORENE	86-73-7		66000	55000		51000	130000		-- [2]	-- [2]		29000	39000
HEPTACHLOR	76-44-8	29	2500	2100				170000	-- [2]	-- [2]	29	2500	2100
HEXACHLOROBENZENE	118-74-1	23	1300	1100	23	1300	3500	140000	-- [2]	-- [2]	12	660	830
beta-HEXACHLOROCYCLOHEXANE	319-85-7	24	16	14				150000	100000	620000	24	16	14
delta-HEXACHLOROCYCLOHEXANE	319-86-8		41000	34000					-- [2]	-- [2]		41000	34000
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	33	66	55	82	170	430	200000	400000	-- [2]	23	47	49
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	6.8	820	680				41000	-- [2]	-- [2]	6.8	820	680
INDENO(1,2,3-cd)PYRENE	193-39-5	26	49000	41000	20	38000	100000	360000	-- [2]	-- [2]	11	22000	29000
LEAD [5]													
MANGANESE			82000	68000					430000	-- [2]		69000	67000
MERCURY (ELEMENTAL)	7439-97-6								5.7	35		5.7	35
MERCURY (INORGANIC SALTS)			260	220								260	220
METHYL TERT-BUTYL ETHER	1634-04-4	6800	54000	45000				3400	500000	-- [2]	2300	49000	45000
METHYLENE CHLORIDE	75-09-2	3700	99000	82000				24000	25000	160000	3200	20000	54000
METHYL ETHYL KETONE	78-93-3		990000	820000					310000	-- [2]		240000	580000
2-METHYLPHENOL (o-CRESOL)	95-48-7		82000	68000		83000	220000		-- [2]	-- [2]		41000	52000
3-METHYLPHENOL (m-CRESOL)	108-39-4		82000	68000		83000	220000		-- [2]	-- [2]		41000	52000

Chemical	CAS Number [1]	Ingestion (mg/kg)			Dermal (mg/kg)			Inhalation (mg/kg)			Combined (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
4-METHYLPHENOL (p-CRESOL)	106-44-5		8200	6800		8300	22000		-- [2]	-- [2]		4100	5200
NAPHTHALENE	91-20-3		33000	27000		33000	86000		-- [2]	-- [2]		17000	21000
NICKEL			33000	27000				83000	260000	-- [2]	83000	33000	27000
PENTACHLOROPHENOL	87-86-5	190	1600	1400	78	660	1700	-- [2]	-- [2]	-- [2]	55	470	760
PHENANTHRENE	85-01-8		49000	41000		38000	100000		-- [2]	-- [2]		22000	29000
PHENOL	108-95-2		490000	410000		500000	-- [2]		-- [2]	-- [2]		250000	310000
POLYCHLORINATED BIPHENYLS [6]	1336-36-3												
n-PROPYLBENZENE	103-65-1		160000	140000					25000	160000		22000	73000
PYRENE	129-00-0		49000	41000		38000	100000		-- [2]	-- [2]		22000	29000
SELENIUM			8200	6800					-- [2]	-- [2]		8200	6800
SILVER			8200	6800					-- [2]	-- [2]		8200	6800
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1		13000	11000					-- [2]	-- [2]		13000	11000
TETRACHLOROETHENE	127-18-4	440	16000	14000				880	6300	39000	300	4600	10000
TOLUENE	108-88-3		330000	270000					19000	120000		18000	82000
1,1,1-TRICHLOROETHANE	71-55-6		460000	380000					140000	860000		110000	260000
TRICHLOROETHENE	79-01-6	4000	2400	2000				440	2500	16000	400	1200	1800
1,2,4-TRIMETHYLBENZENE	95-63-6		82000	68000					380	2300		380	2300
1,3,5-TRIMETHYLBENZENE	108-67-8		82000	68000					380	2300		380	2300
VINYL CHLORIDE	75-01-4	31	4900	4100				200	6300	39000	27	2800	3700
XYLENES	1330-20-7		330000 [7]	270000 [7]					6300 [7]	39000 [7]		6200 [7]	34000 [7]
ZINC			490000	410000					-- [2]	-- [2]		490000	410000

Footnotes:

[1] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

[2] Calculated value equals or exceeds one million milligrams of substance per kilogram of soil.

[3] Calculated values based on the soluble salts oral reference dose of 0.005 mg/kg/day (Table 5.1.1-2). If the insoluble salts oral reference dose of 1.5 mg/kg/day (Table 5.1.1-2) is used, the calculated values will be higher.

[4] As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.

[5] See Section 5.3.4 for a discussion of SCOs for lead.

[6] Based on the US EPA regulation "Disposal of Polychlorinated Biphenyls (PCBs)" (40 CFR 716) (US EPA, 1998); see Section 6.0.

[7] As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

Table 5.3.6-2. Chronic Human Health-based Soil Cleanup Objectives.

Chemical	CAS Number [1]	Unrestricted (mg/kg)			Residential (mg/kg)			Restricted Residential (mg/kg)			Commercial (mg/kg)			Industrial (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
ACENAPHTHENE	83-32-9		4700	210		9100	410		34000	1700		22000	9200		43000	58000
ACENAPHTHYLENE	208-96-8		4700	210		9100	410		34000	1700		22000	9200		43000	58000
ACETONE	67-64-1		58000	3200		96000	6300		200000	29000		420000	260000		830000	-- [2]
ALDRIN	309-00-2	0.0019	0.49	0.022	0.019	4.9	0.22	0.097	25	1.1	0.68	25	8.9	1.4	49	41
ANTHRACENE	120-12-7		24000	1000		45000	2000		170000	8400		110000	46000		220000	290000
ARSENIC		0.11	24	1.1	0.21	48	2.1	1	220	10	5.9	190	73	12	380	370
BARIUM			1600	72		3300	140		16000	720		16000	5900		32000	27000
BENZENE	71-43-2	2	150	14	2.9	190	26	4.8	250	94	44	730	1100	89	1500	3700
BENZ(a)ANTHRACENE	56-55-3	0.052	2400	100	0.1	4500	200	0.39	17000	840	5.6	11000	4600	11	22000	29000
BENZO(a)PYRENE	50-32-8	0.0011	490	21	0.01	4500	200	0.039	17000	840	0.56	11000	4600	1.1	22000	29000
BENZO(b)FLUORANTHENE	205-99-2	0.052	2400	100	0.1	4500	200	0.39	17000	840	5.6	11000	4600	11	22000	29000
BENZO(k)FLUORANTHENE	207-08-9	0.52	2400	100	1	4500	200	3.9	17000	840	56	11000	4600	110	22000	29000
BENZO(g,h,i)PERYLENE	191-24-2		2400	100		4500	200		17000	840		11000	4600		22000	29000
BERYLLIUM	7440-41-7	5300	160	7.2	5300	330	14	5300	1600	72	8300	1600	590	17000	3300	2700
n-BUTYLBENZENE	104-51-8		2500	330		2900	600		3400	1800		11000	25000		22000	73000
sec-BUTYLBENZENE	135-98-8		2500	330		2900	600		3400	1800		11000	25000		22000	73000
tert-BUTYLBENZENE	98-06-6		2500	330		2900	600		3400	1800		11000	25000		22000	73000
CADMIUM		0.43	58	2.5	0.86	120	5	4.3	570	25	30	560	210	60	1100	950
CARBON TETRACHLORIDE	56-23-5	0.89	14	2.2	1.4	16	3.9	2.4	17	10	22	57	160	44	110	430
CHLORDANE (alpha)	5103-71-9	0.094	8.2	0.36	0.91	80	3.5	4.2	360	17	24	290	120	47	590	610
CHLOROBENZENE	108-90-7		410	63		460	110		520	310		1700	4700		3400	13000
CHLOROFORM	67-66-3	5.3	290	33	10	350	62	49	430	200	350	1300	2600	700	2600	8000
CHROMIUM (III)			410	18		820	36		4100	180		4100	1500		8200	6800
CHROMIUM (VI)		250	250	11	250	490	22	250	2500	110	400	2500	890	800	4900	4100
CHRYSENE	218-01-9	0.52	2400	100	1	4500	200	3.9	17000	840	56	11000	4600	110	22000	29000
COPPER			12000	500		23000	1000		120000	5000		120000	41000		230000	190000
CYANIDE			1600	72		3300	140		16000	720		16000	5900		33000	27000
4,4’-DDD	72-54-8	0.26	8.2	0.36	2.6	82	3.6	13	410	18	92	410	150	180	820	680

Chemical	CAS Number [1]	Unrestricted (mg/kg)			Residential (mg/kg)			Restricted Residential (mg/kg)			Commercial (mg/kg)			Industrial (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
4,4'-DDE	72-55-9	0.18	200	8.6	1.8	2000	86	8.9	9900	430	62	9900	3500	120	20000	16000
4,4'-DDT	50-29-3	0.17	8.2	0.36	1.7	81	3.6	7.9	370	17	47	320	120	94	640	620
DIBENZ(a,h)ANTHRACENE	53-70-3	0.0052	2400	100	0.01	4500	200	0.039	17000	840	0.56	11000	4600	1.1	22000	29000
DIBENZOFURAN	132-64-9		160	7		310	14		1200	59		830	350		1700	2100
1,2-DICHLOROBENZENE	95-50-1		880	72		1200	140		1600	530		4600	5700		9200	21000
1,3-DICHLOROBENZENE	541-73-1		66	9.6		76	17		87	49		280	720		560	2000
1,4-DICHLOROBENZENE	106-46-7	7.3	1800	110	9.8	2900	210	13	5600	940	130	12000	8600	250	25000	36000
1,1-DICHLOROETHANE	75-34-3	14			19			26			240			480		
1,2-DICHLOROETHANE	107-06-2	1.7	1800	150	2.3	2400	300	3.1	3300	1100	30	9400	12000	60	19000	44000
1,1-DICHLOROETHENE	75-35-4		1300	160		1500	300		1700	900		5500	12000		11000	36000
cis-1,2-DICHLOROETHENE	156-59-2		230	32		260	59		300	170		970	2400		1900	6800
trans-1,2-DICHLOROETHENE	156-60-5		410	63		460	110		520	310		1700	4700		3400	13000
DIELDRIN	60-57-1	0.0039	0.82	0.036	0.039	8.2	0.36	0.2	41	1.8	1.4	41	15	2.8	82	68
1,4-DIOXANE	123-91-1	7.3	6600	360	9.8	11000	700	13	23000	3200	130	48000	29000	250	95000	120000
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--		55 [3]	2.4 [3]		110 [3]	4.8 [3]		550 [3]	24 [3]		550 [3]	200 [3]		1100 [3]	920 [3]
ENDRIN	72-20-8		4.9	0.22		49	2.2		250	11		250	89		490	410
ETHYLBENZENE	100-41-4	23	5600	350	30	8600	690	41	15000	3000	390	36000	28000	780	71000	120000
FLUORANTHENE	206-44-0		3200	140		6100	270		23000	1100		14000	6200		29000	39000
FLUORENE	86-73-7		3200	140		6100	270		23000	1100		14000	6200		29000	39000
HEPTACHLOR	76-44-8	0.042	25	1.1	0.42	250	11	2.1	1200	54	15	1200	440	29	2500	2100
HEXACHLOROBENZENE	118-74-1	0.033	13	0.57	0.31	120	5.5	1.2	490	24	5.8	330	140	12	660	830
beta-HEXACHLOROCYCLOHEXANE	319-85-7	0.17	0.82	0.036	0.34	1.6	0.072	1.7	8.2	0.36	12	8.2	3	24	16	14
delta-HEXACHLOROCYCLOHEXANE	319-86-8		2100	90		4100	180		21000	900		21000	7400		41000	34000
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	0.23	3.3	0.14	0.45	6.4	0.28	2	29	1.3	12	24	9.2	23	47	49
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	0.048	41	1.8	0.097	82	3.6	0.48	410	18	3.4	410	150	6.8	820	680
INDENO(1,2,3-cd)PYRENE	193-39-5	0.052	2400	100	0.1	4500	200	0.39	17000	840	5.6	11000	4600	11	22000	29000
LEAD [4]																
MANGANESE			4100	180		8100	360		37000	1800		35000	15000		69000	67000
MERCURY (ELEMENTAL)	7439-97-6		0.81	0.81		0.81	0.81		0.81	0.81		2.8	35		5.7	35

Chemical	CAS Number [1]	Unrestricted (mg/kg)			Residential (mg/kg)			Restricted Residential (mg/kg)			Commercial (mg/kg)			Industrial (mg/kg)		
		Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic	Carcin-ogenic	Adult Non-carcin-ogenic	Child Non-carcin-ogenic
MERCURY (INORGANIC SALTS)			2.6	0.12		26	1.2		130	5.8		130	47		260	220
METHYL TERT-BUTYL ETHER	1634-04-4	38	2600	120	62	5100	240	130	20000	1200	1100	25000	9700	2300	49000	45000
METHYLENE CHLORIDE	75-09-2	26	2100	200	51	2600	390	220	3300	1300	1600	10000	16000	3200	20000	54000
METHYL ETHYL KETONE	78-93-3		24000	2100		31000	3900		41000	15000		120000	160000		240000	580000
2-METHYLPHENOL (o-CRESOL)	95-48-7		4000	180		7700	340		31000	1500		21000	8700		41000	52000
3-METHYLPHENOL (m-CRESOL)	108-39-4		4000	180		7700	340		31000	1500		21000	8700		41000	52000
4-METHYLPHENOL (p-CRESOL)	106-44-5		400	18		770	34		3100	150		2100	870		4100	5200
NAPHTHALENE	91-20-3		1600	70		3100	140		12000	590		8300	3500		17000	21000
NICKEL		26000	1600	72	26000	3300	140	26000	16000	720	42000	16000	5900	83000	33000	27000
PENTACHLOROPHENOL	87-86-5	1.3	76	3.4	2.4	140	6.5	7.6	450	23	28	240	110	55	470	760
PHENANTHRENE	85-01-8		2400	100		4500	200		17000	840		11000	4600		22000	29000
PHENOL	108-95-2		24000	1100		46000	2100		180000	8900		120000	52000		250000	310000
POLYCHLORINATED BIPHENYLS [5]	1336-36-3															
n-PROPYLBENZENE	103-65-1		2500	330		2900	600		3400	1800		11000	25000		22000	73000
PYRENE	129-00-0		2400	100		4500	200		17000	840		11000	4600		22000	29000
SELENIUM			410	18		820	36		4100	180		4100	1500		8200	6800
SILVER			410	18		820	36		4100	180		4100	1500		8200	6800
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1		660	29		1300	58		6600	290		6600	2400		13000	11000
TETRACHLOROETHENE	127-18-4	2.9	430	35	5.5	580	67	19	810	260	150	2300	2700	300	4600	10000
TOLUENE	108-88-3		2300	570		2500	940		2700	2000		8900	39000		18000	82000
1,1,1-TRICHLOROETHANE	71-55-6		11000	960		14000	1800		18000	6700		53000	75000		110000	260000
TRICHLOROETHENE	79-01-6	13	90	5.2	16	140	10	21	280	46	200	610	420	400	1200	1800
1,2,4-TRIMETHYLBENZENE	95-63-6		53	41		54	47		54	52		190	2000		380	2300
1,3,5-TRIMETHYLBENZENE	108-67-8		53	41		54	47		54	52		190	2000		380	2300
VINYL CHLORIDE	75-01-4	0.11	190	11	0.21	320	21	0.9	660	96	13	1400	870	27	2800	3700
XYLENES	1330-20-7		850 [6]	400 [6]		870 [6]	550 [6]		890 [6]	800 [6]		3100 [6]	23000 [6]		6200 [6]	34000 [6]
ZINC			25000	1100		49000	2200		250000	11000		250000	89000		490000	410000

Footnotes:
[1] CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.
[2] Calculated value equals or exceeds one million milligrams of substance per kilogram of soil.
[3] As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.
[4] See Section 5.3.4 for a discussion of SCOs for lead.
[5] Based on the US EPA regulation "Disposal of Polychlorinated Biphenyls (PCBs)" (40 CFR 716) (US EPA, 1998); see Section 6.0.
[6] As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

5.4 Calculation of Acute Soil Ingestion SCOs

Acute soil ingestion soil cleanup objectives (SCO_{acute}) are applicable to the unrestricted, restricted residential, and commercial land use categories since young children may be present in these settings. Because almost all of the total intake under this exposure scenario would be from soil ingestion during a single event, the equation for calculating an SCO_{acute} does not consider dermal or inhalation exposures or a relative source contribution factor. The SCO_{acute} values were calculated as shown below.

$$SCO_{acute} = \frac{RfD_{acute}}{\frac{CF \times 10 g_{soil}}{13.3 kg_{bw}}}$$

Where:

SCO_{acute} = soil cleanup objective (mg/kg)

RfD_{acute} = acute oral reference dose (See Appendix B)

CF = conversion factor (1 $kg_{soil}/10^3$ g_{soil})

10 g_{soil} = amount of soil ingested

13.3 kg_{bw} = child body weight

5.4.1. Acute Soil Ingestion SCOs

Acute soil ingestion SCOs, calculated according to the methods described in Section 5.4, are shown in Table 5.4.1-1.

Table 5.4.1-1. Acute Soil Ingestion SCOs.

Contaminant	SCO_{acute} (mg/kg)
Barium	400
Cadmium	9.3
Copper	270
Cyanide (free)	27.0
Nickel	310
Pentachlorophenol	6.7
Phenol	800

5.5 Calculation of Irritant Contact Dermatitis SCOs

SCOs for irritant contact dermatitis (SCO_{ICD}) were calculated for all land use categories (unrestricted, restricted residential, commercial, and industrial). The SCO_{ICD} values were calculated as shown below.

$$SCO_{ICD} = \frac{SkinRfD_{ICD}}{CF \times SAF \times AF}$$

Where:

SCO_{ICD} = soil cleanup objective (mg/kg)

Skin RfD_{ICD} = skin reference dose based on irritant contact dermatitis ($mg_{contaminant}/cm^2_{skin}$)

CF = conversion factor (1 $kg_{soil}/10^6$ mg_{soil})

SAF = soil adherence factor (mg_{soil}/cm^2_{skin})

AF = absorption fraction (unitless)

The values for the parameters used calculating irritant contact dermatitis SCOs are shown in Table 5.5-1.

Table 5.5-1. Parameter Values Used in Calculating Irritant Contact Dermatitis SCOs.

Land Use Category	Population	Skin RfD_{ICD} (1)	Exposure Parameter	
			SAF (2)	AF (3)
Phenol				
unrestricted and residential restricted	child	0.012	0.2	0.1
	adult		0.07	0.1
commercial	child and adult		0.2	0.1
industrial	adolescent		0.07	0.1
	adult		0.2	0.1
Nickel				
unrestricted and residential restricted	child	0.0023	0.2	0.01
	adult		0.07	0.01
commercial	child and adult		0.2	0.01
industrial	adolescent		0.07	0.01
	adult		0.2	0.01
Chromium VI				
unrestricted and residential restricted	child	0.0016	0.2	0.04
	adult		0.07	0.04
commercial	child and adult		0.2	0.04
industrial	adolescent		0.07	0.04
	adult		0.2	0.04
Surrogate SVOC (including SVOCs and pesticides)				
unrestricted and residential restricted	child	0.0013	0.2	0.1
	adult		0.07	0.1
commercial	child and adult		0.2	0.1
industrial	adolescent		0.07	0.1
	adult		0.2	0.1

(1) skin reference dose based on irritant contact dermatitis ($mg_{contaminant}/cm^2_{skin}$)
(2) soil adherence factor (mg_{soil}/cm^2_{skin})
(3) absorption fraction (unitless)

5.5.1 Irritant Contact Dermatitis SCOs

The irritant contact dermatitis SCO for anthracene (surrogate SVOC) for an adult in industrial settings is the only value that is lower than a chronic SCO. The value of this SCO is 65,000 ppm.

5.6 Final Human Health-based SCOs

Final health-based SCOs were determined for each of the five land use categories. For each Priority List chemical, the final health-based SCO was determined as the lowest of all the SCOs calculated for the chemical considering, as applicable, chronic exposure (Section 5.3), acute soil ingestion (Section 5.4), and irritant contact dermatitis (Section 5.5). Table 5.6-1 presents the final health-based SCOs.

Table 5.6-1. Final Human Health-based Soil Cleanup Objectives.

Chemical	CAS Number [1]	Unrestricted (mg/kg)	Residential (mg/kg)	Restricted Residential (mg/kg)	Commercial (mg/kg)	Industrial (mg/kg)
ACENAPHTHENE	83-32-9	210	410	1700	9200	43000
ACENAPHTHYLENE	208-96-8	210	410	1700	9200	43000
ACETONE	67-64-1	3200	6300	29000	260000	830000
ALDRIN	309-00-2	0.0019	0.019	0.097	0.68	1.4
ANTHRACENE	120-12-7	1000	2000	8400	46000	65000 [2]
ARSENIC		0.11	0.21	1	5.9	12
BARIUM		72	140	400 [3]	400 [3]	27000
BENZENE	71-43-2	2	2.9	4.8	44	89
BENZ(a)ANTHRACENE	56-55-3	0.052	0.1	0.39	5.6	11
BENZO(a)PYRENE	50-32-8	0.0011	0.01	0.039	0.56	1.1
BENZO(b)FLUORANTHENE	205-99-2	0.052	0.1	0.39	5.6	11
BENZO(k)FLUORANTHENE	207-08-9	0.52	1	3.9	56	110
BENZO(g,h,i)PERYLENE		100	200	840	4600	22000
BERYLLIUM	7440-41-7	7.2	14	72	590	2700
n-BUTYLBENZENE	104-51-8	330	600	1800	11000	22000
sec-BUTYLBENZENE	135-98-8	330	600	1800	11000	22000
tert-BUTYLBENZENE	98-06-6	330	600	1800	11000	22000
CADMIUM		0.43	0.86	4.3	9.3 [3]	60
CARBON TETRACHLORIDE	56-23-5	0.89	1.4	2.4	22	44
CHLORDANE (alpha)	5103-71-9	0.094	0.91	4.2	24	47
CHLOROBENZENE	108-90-7	63	110	310	1700	3400
CHLOROFORM	67-66-3	5.3	10	49	350	700
CHROMIUM (III)		18	36	180	1500	6800
CHROMIUM (VI)		11	22	110	400	800
CHRYSENE	218-01-9	0.52	1	3.9	56	110
COPPER		270 [3]	270 [3]	270 [3]	270 [3]	190000
CYANIDE		27 [3]	27 [3]	27 [3]	27 [3]	27000
4,4’-DDD	72-54-8	0.26	2.6	13	92	180

Chemical	CAS Number [1]	Unrestricted (mg/kg)	Residential (mg/kg)	Restricted Residential (mg/kg)	Commercial (mg/kg)	Industrial (mg/kg)
4,4'-DDE	72-55-9	0.18	1.8	8.9	62	120
4,4'-DDT	50-29-3	0.17	1.7	7.9	47	94
DIBENZ(a,h)ANTHRACENE	53-70-3	0.0052	0.01	0.039	0.56	1.1
DIBENZOFURAN	132-64-9	7	14	59	350	1700
1,2-DICHLOROBENZENE	95-50-1	72	140	530	4600	9200
1,3-DICHLOROBENZENE	541-73-1	9.6	17	49	280	560
1,4-DICHLOROBENZENE	106-46-7	7.3	9.8	13	130	250
1,1-DICHLOROETHANE	75-34-3	14	19	26	240	480
1,2-DICHLOROETHANE	107-06-2	1.7	2.3	3.1	30	60
1,1-DICHLOROETHENE	75-35-4	160	300	900	5500	11000
cis-1,2-DICHLOROETHENE	156-59-2	32	59	170	970	1900
trans-1,2-DICHLOROETHENE	156-60-5	63	110	310	1700	3400
DIELDRIN	60-57-1	0.0039	0.039	0.2	1.4	2.8
1,4-DIOXANE	123-91-1	7.3	9.8	13	130	250
ENDOSULFAN I, ENDOSULFAN II and ENDOSULFAN SULFATE	--	2.4 [4]	4.8 [4]	24 [4]	200 [4]	920 [4]
ENDRIN	72-20-8	0.22	2.2	11	89	410
ETHYLBENZENE	100-41-4	23	30	41	390	780
FLUORANTHENE	206-44-0	140	270	1100	6200	29000
FLUORENE	86-73-7	140	270	1100	6200	29000
HEPTACHLOR	76-44-8	0.042	0.42	2.1	15	29
HEXACHLOROBENZENE	118-74-1	0.033	0.31	1.2	5.8	12
beta-HEXACHLOROCYCLOHEXANE	319-85-7	0.036	0.072	0.36	3	14
delta-HEXACHLOROCYCLOHEXANE	319-86-8	90	180	900	7400	34000
gamma-HEXACHLOROCYCLOHEXANE	58-89-9	0.14	0.28	1.3	9.2	23
alpha-HEXACHLOROCYCLOHEXANE	319-84-6	0.048	0.097	0.48	3.4	6.8
INDENO(1,2,3-cd)PYRENE	193-39-5	0.052	0.1	0.39	5.6	11
LEAD [5]		200 [6]	400	400	1000	3900
MANGANESE		180	360	1800	15000	67000
MERCURY (ELEMENTAL)	7439-97-6	0.81	0.81	0.81	2.8	5.7

Chemical	CAS Number [1]	Unrestricted (mg/kg)	Residential (mg/kg)	Restricted Residential (mg/kg)	Commercial (mg/kg)	Industrial (mg/kg)
MERCURY (INORGANIC SALTS)		0.12	1.2	5.8	47	220
METHYL TERT-BUTYL ETHER	1634-04-4	38	62	130	1100	2300
METHYLENE CHLORIDE	75-09-2	26	51	220	1600	3200
METHYL ETHYL KETONE	78-93-3	2100	3900	15000	120000	240000
2-METHYLPHENOL (o-CRESOL)	95-48-7	180	340	1500	8700	41000
3-METHYLPHENOL (m-CRESOL)	108-39-4	180	340	1500	8700	41000
4-METHYLPHENOL (p-CRESOL)	106-44-5	18	34	150	870	4100
NAPHTHALENE	91-20-3	70	140	590	3500	17000
NICKEL		72	140	310 [3]	310 [3]	27000
PENTACHLOROPHENOL	87-86-5	1.3	2.4	6.7 [3]	6.7 [3]	55
PHENANTHRENE	85-01-8	100	200	840	4600	22000
PHENOL	108-95-2	800 [3]	800 [3]	800 [3]	800 [3]	250000
POLYCHLORINATED BIPHENYLS [7]	1336-36-3	0.1 [6]	1	1	1	25
n-PROPYLBENZENE	103-65-1	330	600	1800	11000	22000
PYRENE	129-00-0	100	200	840	4600	22000
SELENIUM		18	36	180	1500	6800
SILVER		18	36	180	1500	6800
2-(2,4,5-TRICHLOROPHENOXY) PROPRIONIC ACID	93-72-1	29	58	290	2400	11000
TETRACHLOROETHENE	127-18-4	2.9	5.5	19	150	300
TOLUENE	108-88-3	570	940	2000	8900	18000
1,1,1-TRICHLOROETHANE	71-55-6	960	1800	6700	53000	110000
TRICHLOROETHENE	79-01-6	5.2	10	21	200	400
1,2,4-TRIMETHYLBENZENE	95-63-6	41	47	52	190	380
1,3,5-TRIMETHYLBENZENE	108-67-8	41	47	52	190	380
VINYL CHLORIDE	75-01-4	0.11	0.21	0.9	13	27
XYLENES	1330-20-7	400 [8]	550 [8]	800 [8]	3100 [8]	6200 [8]
ZINC		1100	2200	11000	89000	410000

Footnotes:

(1) CAS Registry Numbers are not included for metals, except for elemental mercury. The toxicity values for metals are intended for use with various inorganic forms found in the environment.

(2) SCO based on dermal irritancy

(3) SCO based on acute toxicity

(4) As described in the Technical Support Document, the SCO is applicable to the sum of the soil concentrations of endosulfan I, endosulfan II, and endosulfan sulfate.

(5) See Section 5.3.4 for a discussion of SCOs for lead.

(6) The residential value was adjusted to account for the animal product consumption pathway as described in Section 5.2.2.4.

(7) Based on the US EPA regulation “Disposal of Polychlorinated Biphenyls (PCBs)”(40 CFR 716) (US EPA, 1998); see Section 6.0.

(8) As described in the Technical Support Document, the SCO is applicable to total xylenes measured in soil, or to the sum of individual isomers of xylene measured in soil.

6.0 SCOs for Polychlorinated Biphenyls (PCBs)

Soil may be contaminated with PCBs from many different types of disposal[1] activities. Before the manufacture of PCBs was discontinued in 1977, various formulations of this class of compounds were a component of many different products that were used for a variety of purposes. The different formulations, known as Aroclors, are composed of many individual chemicals that had differing physical properties and toxicities. While the largest volumes of PCBs disposed in the environment are associated with the power industry (dielectric fluid in transformers and capacitors), PCBs also were used in hydraulic fluid, paints, inks, and carbonless forms (NCR paper).

In deriving the SCOs required by the Brownfield Cleanup Program legislation, toxicological data are necessary for the compounds under consideration. The availability of these toxicological data, coupled with New York State's experience with compounds typically encountered at remedial sites, was a significant factor in establishing the list of compounds for which SCOs were to be developed. Much of the available toxicological information for PCBs is based on the specific formulations (Aroclors) of the material as it was produced. Since the PCB contamination encountered in site remediation has, for the most part, existed in the environment for an extended period of time, transformation by natural forces (e.g., volatilization, degradation) of the PCBs in the contaminated media has occurred. Degradation can alter the chemical nature of the Aroclors (e.g., remove chlorine atoms). The number of chlorine atoms and their arrangement in the PCB molecule are believed to be a major factor in determining the toxicity of the compound.

Since much of the toxicological data that exist for PCBs are based on original chemical formulations that may differ substantially from the PCBs actually found in the environment, we considered an alternate approach to using Aroclor-specific toxicity data. In 1998, under the authority of the Toxic Substances Control Act (TSCA), US EPA promulgated a regulation (Disposal of Polychlorinated Biphenyls (PCBs), 40 CFR 761), as the primary nation-wide regulation to address PCB remediation, treatment, and disposal (US EPA, 1998). The regulation

[1] Disposal means the abandonment, discharge, deposit, injection, dumping, spilling, leaking, or placing of any substance so that such substance or any related constituent thereof may enter the environment.

specifies cleanup levels for "PCB remediation waste" (defined at 40 CFR 761.3) for "high occupancy areas" (i.e., areas where people may be present for 335 hours or more per year; see 40 CFR 761.3) and "low occupancy areas" (i.e., areas where people may be present for fewer than 335 hours per year; see 40 CFR 761.3). The cleanup levels (40 CFR 761.61(a)(4)(i)) are:

- high occupancy areas: <= 1 part per million (ppm), without further conditions
- high occupancy areas: >1 ppm to <= 10 ppm, covered with a cap
- low occupancy areas: <= 25 ppm
- low occupancy areas: > 25 ppm to <= 50 ppm, with site secured by a fence and marked with a sign
- low occupancy areas: >25 ppm to <= 100 ppm, covered with a cap.

These levels have been used in many PCB remediation projects across the country as the cleanup levels for PCBs in soils. The TSCA regulations do not directly address protection of ecological resources, although 40CFR 761.61(a)(4)(v) of the regulation states that where the exposure of animal life is expected to be a concern at a particular site, the cleanup of the area should be in accordance with the higher occupancy cleanup levels noted above.

On the basis of these cleanup levels, the following are established as SCOs for the Brownfield Cleanup Program:

- unrestricted land use: 0.1[2] ppm
- residential land use: 1 ppm
- restricted residential land use: 1 ppm
- commercial land use: 1 ppm
- industrial land use: 25 ppm
- ecological resources: 1 ppm

[2] The residential value of 1 ppm was adjusted to account for the animal product consumption pathway as described in Section 5.2.2.4.

These SCOs for total PCBs are therefore being included in the Brownfield Cleanup Program tables for the following reasons:

- After a specific Aroclor has been in the environment, the congener composition will no longer be the same as in the initial Aroclor
- The Aroclor-specific toxicity data may not be an accurate measure of the toxicity of the PCB contamination
- The US EPA TSCA regulation identifies cleanup levels which can be used as SCOs for total PCBs
- The ecological soil cleanup objective was assigned after consideration of other technical and practical concerns

References

US EPA (US Environmental Protection Agency). 1998. Disposal of Polychlorinated Biphenyls (PCBs). Federal Register 63 (124): 35384 - 35474. June 29, 1998.

7.0 Groundwater

7.1 Introduction

Protection of groundwater addresses the potential for residual soil contamination to leach and act as a long-term source of groundwater contamination. This section describes the basis for selecting the approach used to predict the SCO necessary for the protection of groundwater.

7.2 Background

In order for residual soil contamination to contribute to groundwater contamination, the contamination first has to leach from the soil, travel through the vadose zone (soil above the groundwater table), then mix with groundwater. There are many predictive tests and theoretical models that have been developed to predict leachate quality given a known value of soil contamination. These models vary widely in their degree of sophistication, with the more sophisticated requiring a larger amount of site-specific information relative to soil characteristics (e.g., porosity, grain size distribution, bulk density, moisture content) for the model to function as expected. In our experience, much of the information relative to soil characteristics and architecture required by these sophisticated models is not normally collected during site investigations. Further, due to the heterogeneity of most sites, these characteristics vary widely across the site, making it almost impossible to set a value that is representative for the entire site. The remedy for this problem is to select default values for the soil characteristics required by the model but that defeats the purpose of the refined model. Use of a sophisticated model with default inputs is no more valuable than the use of a simpler model that does not rely on as many inputs.

7.3 History

The Division of Environmental Remediation has had guidance for the determination of soil cleanup levels since 1992. This guidance (referred to as TAGM 4046) considers the protection of groundwater pathway, in addition to direct ingestion of soil, in developing soil cleanup levels.

TAGM 4046 incorporates the soil-water partitioning theory (described in detail below) along with a dilution attenuation factor (also described below). The division's experience with this approach has been that when the soil cleanup levels calculated using this approach are achieved, the contaminated groundwater responds favorably. The soil-water partitioning theory, along with a dilution attenuation factor, has also been used by a number of other governmental agencies, including US EPA in their Soil Screening Guidance.

7.4 Determination of Leaching

The soil-water partitioning theory is used in determining soil concentrations or cleanup objectives that would be protective of groundwater quality for its best use, which is as a source of drinking water. This theory is conservative and assumes that contaminated soil and groundwater are in direct contact.

For organic chemicals, this theory is based upon the ability of organic matter in soil to adsorb organic chemicals and prevent them from leaching out of the soil. The approach predicts the maximum amount of contamination that can be remain in the soil and the leachate from the contaminated soil not violate groundwater and/or drinking water standards. Using a water quality value not to be exceeded in leachate (typically class GA groundwater standards) and the partition coefficient method, the equilibrium concentration in soil (C_s) is expressed (in the same units as the water standards) as follows:

Allowable Soil Concentration $C_s = f_{oc} \times K_{oc} \times C_w$

Where:

f_{oc} = fraction of organic carbon of the natural soil medium.

K_{oc} = partition coefficient between water and soil media.

C_w = groundwater / drinking water standard

For calculating the SCOs, values for K_{oc} were selected from authoritative bodies based on the hierarchy shown in Section 7.7 below. If the authoritative body shown first listed a value, it was used. If the first body authoritative body did not but the second did, the value from the second

authoritative body was used. The process was used for each chemical until K_{oc}s were found for all of the chemicals on the SCO priority list.

In TAGM 4046, an assumption of 1% organic carbon content was used. While it is recognized that in some areas of the state the organic carbon content of the soil is less than 1% (and the resulting value for C_s would be lower), it is also recognized that the organic soil water partitioning theory itself is very conservative and probably overestimates the concentration of contaminants in the leachate generated from contaminated soil. Further, this theory assumes a continuous flow of leachate and an infinite source of contamination, which is seldom the case.

For inorganic chemicals, while the soil-water partitioning theory still applies, there is no simple relationship between soil organic carbon content and sorption of organic chemicals. For this reason, a parameter known as the soil-water distribution coefficient (K_d) was used in the development of SCOs for metals and other inorganic compounds. K_d is affected by numerous geochemical parameters and processes, including pH; sorption to clays, organic matter, iron oxide, iron oxides, and other soil constituents; oxidation/reduction conditions; major iron chemistry; and the valent state of the metal. Similar to K_{oc}, values for K_d were selected from authoritative bodies based on the hierarchy shown in Section 7.7. If the authoritative body shown first listed a value, it was used. If the first body authoritative body did not but the second did, the value from the second authoritative body was used. The process was used for each chemical until K_ds were found for all of the chemicals on the SCO priority list. As with organic chemicals, the approach predicts the maximum amount of contamination that may remain in soil so that leachate from the contaminated soil will not violate groundwater and/or drinking water standards. Using a water quality value which may not be exceeded in leachate and the partition coefficient method, the equilibrium concentration (C_s) will be expressed in the same units as the water standards. The following expression is used:

Allowable Soil Concentration $C_s = K_d \times C_w$

Where:

K_d = soil water distribution coefficient

C_w = groundwater / drinking water standard

7.5 Determination of Impact on Groundwater

The second part of the problem is to determine how much of that contamination will actually contribute to a violation of groundwater standards upon reaching and dispersing into groundwater. When contamination leaves a particle of soil in the form of leachate, there are many mechanisms at work that prevent all of the contamination that leaves the contaminated soil from impacting groundwater. For instance, some of the contamination which initially leaches from the soil will be absorbed by other soil particles before it reaches groundwater, while some will be reduced through natural attenuation or other mechanism. These mechanisms occur during transport and may work simultaneously. They include: 1) volatilization; 2) sorption and desorption; 3) leaching and diffusion; 4) transformation and degradation; and 5) change in concentration of contaminants after reaching and/or mixing with the groundwater surface.

To account for these mechanisms, a correction factor or dilution attenuation factor (DAF) of 100 was used to establish soil cleanup objectives. This value of 100 is consistent with the logic used by the US EPA in its DAF approach for EP Toxicity and TCLP (Federal Register/Vol. 55, No. 61, March 29, 1990/Pages 11826-27). SCOs are calculated by multiplying the allowable soil concentration (C_s, as determined in Section 7.4) by the DAF or correction factor. This value is also discussed in the US EPA's Soil Screening Effort (EPA/540/R-95/128) which occurred after the NYS DEC developed TAGM 4046. The DAF of 100 was established in 1992 when the NYS DEC's soil cleanup guidance was first established and has been used continuously since that time. Based on our experience applying this guidance at hundreds of sites, when a site is cleaned up to the SCOs in accordance with TAGM 4046, the groundwater responds favorably. While the existing guidance does include a note of caution for situations where the contamination is close (within 3-5 ft) to the groundwater table, our experience is that even in those situations, remediation to the SCOs (using a DAF of 100) results in the groundwater responding favorably.

Further, the brownfields law, as well as the other programs that this Division oversees, requires consideration of a groundwater remedy as well as the prevention of migration of contaminated groundwater from the site. Any residual contamination that may leach from the soil will be controlled by a groundwater remedy or migration control.

Lastly, in addition to predicting groundwater impacts using partitioning theory and modeling, the Division also relies on empirical evidence obtained through direct monitoring of groundwater quality adjacent to or beneath an area of contaminated soil. The idea is that if groundwater has not been impacted by the soil prior to remediation, it is unlikely that it will be impacted after remediation.

7.6 Summary

Preventing the contamination of groundwater from leachate is an important consideration that must be addressed when determining the appropriate soil cleanup level. The NYS DEC addressed this pathway in its soil cleanup guidance, which has existed since 1992. This guidance assumes an organic carbon content of soil (used for organic chemicals only) and a DAF of 100. Our experience with this approach has been that it is effective in protecting groundwater.

While there are more sophisticated approaches to determining an SCO that is protective of groundwater, they require significantly more data in terms of soil characteristics, most of which is not routinely required when investigating sites or analyzed at the laboratories that accept environmental samples. The laboratories that do analyze for soil characteristics, often do not accept contaminated soils. Given the difficulties and added expense of obtaining the data required to run the more sophisticated models and the fact that the approach used by the Division for more than 10 years produces acceptable results, we are proposing continuing to use the existing approach as described in TAGM 4046.

7.7 Hierarchy of Authoritative Bodies

1. US EPA (United States Environmental Protection Agency). 1998. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Region 6: Office of Solid Waste and Emergency Response. EPA530D-D-98-001A. July, 1998. (updated by Errata)

2. ATSDR (Agency for Toxic Substances and Disease Registry). Toxicological Profiles for various chemicals.

3. US National Library of Medicine. 2004. HSDB (Hazardous Substances Data Base). Bethesda MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

4. Syracuse Research Corporation. 2004. Environmental Fate Data Base (EFDB). Syracuse, NY. http://www.syrres.com/esc/efdb_info.htm

5. US EPA. 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. OSWER 9355.4-24. December 2002. (Prepared for US EPA Office of Emergency and Remedial Response)

The order of the references listed above was used as a hierarchy for finding the chemical-specific parameters of: $\log K_{ow}$, K_{oc}, and solubility. For any parameters not found in the first reference, the second reference was consulted and so forth until a value for the parameter was found, or the hierarchy of references was exhausted.

The US EPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (Reference #1) provided a single K_{oc} value for a majority of the chemicals. For any chemical in which a single reference provided multiple K_{oc} values, one K_{oc} was derived from the geometric mean (applies to g-BHC [Lindane], n-Butylbenzene, Methyl tert butyl ether [MTBE], n-Propylbenzene, 4,6-Dinitro-o-cresol, Endrin aldehyde, Toxaphene, Acenaphthylene, Silvex, and 2-Methylnapththalene). The K_{oc} for Xylene (mixed) is a geometric mean of experimental K_{oc}s from various soil types, pH, and organic carbon content. A list of K_{oc}s based on pH was given for the following: Pentachlorophenol, Phenol, 2,4-Dichlorophenol, 2,4-Dinitrophenol, 2,4-Dimethylphenol, 2,4,5-Trichlorophenol, and 2,4,6-Trichlorophenol; the K_{oc} used in the calculation is based on a pH of 7.0. K_{oc} values for p-Chloroaniline and n-Nitrosodiphenylamine were reported as a single value but applies to a pH range of 4.9 – 8.0.

The equation to estimate K_d using K_{oc}, taken from EPA's Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (Reference #1), is $K_d = f_{oc} * K_{oc}$ where f_{oc} is estimated between 0.002 and 0.024 but the mid-range value of 0.01 is generally used.

If K_{ow} was not found in the reference hierarchy, K_{ow} was calculated with one of the following equations:

if Solubility (S) was known, then $K_{ow} = 10(4.186 - \log S) / 0.922$ (Lyman, 1990)

else if K_{oc} was known, then Kow = 10(log Koc - 1.377) / 0.544 (Dragun, 1988)

If K_{oc} was not found in the reference hierarchy, K_{oc} was calculated with one of the following equations:

If K_{ow} was available, $K_{oc} = 100.544 * \log K_{ow} + 1.377$

else if S was available, $K_{oc} = 103.847 - 0.59 * \log S$

If S was not found in the reference hierarchy, S was calculated with one of the following equations:

If K_{ow} was available, $S = 104.186-0.933*\log K_{ow}$

else if, K_{oc} was available $S = 10(3.847-\log K_{oc})/0.59$

Table 7-1. Groundwater SCOs.

Chemical	CAS RN [(1)]	Groundwater SCO (mg/kg)
acenaphthene	83-32-9	98
acenaphthylene	208-96-8	107
acetone	67-64-1	0.05
aldrin	309-00-2	0.19
anthracene	120-12-7	1175
arsenic		15
barium		820
benz[a]anthracene	56-55-3	0.52
benzene	71-43-2	0.06
benzo[a]pyrene	50-32-8	22
benzo[b]fluoranthene	205-99-2	1.7
benzo[g,h,i]perylene	191-24-2	79,000
benzo[k]fluoranthene	207-08-9	1.7
beryllium		47
n-butylbenzene	104-51-8	12
sec-butylbenzene	135-98-8	11
tert-butylbenzene	98-06-6	5.9
cadmium		7.5
carbon tetrachloride	56-23-5	0.76
chlordane (alpha)	12789-03-6	2.9
chlorobenzene	56-23-5	1.1
chloroform	67-66-3	0.37
chromium III		NS[(2)]
chromium VI		19
chrysene	218-01-9	0.59
copper		1720
cyanide		40
DDD	72-54-8	14
DDE	72-55-9	17
DDT	50-29-3	136
dibenz[a,h]anthracene	53-70-3	89,500
dibenzofuran	132-64-9	210
1,2-dichlorobenzene	95-50-1	1.1
1,3-dichlorobenzene	541-73-1	2.4
1,4-dichlorobenzene	106-46-7	1.8
1,1-dichloroethane	75-34-3	0.27
1,2-dichloroethane	107-06-2	0.01
1,1-dichloroethene	75-35-4	0.33
cis-1,2-dichloroethene	156-59-2	0.25

trans-1,2-dichloroethene	156-60-5	0.19
dieldrin	60-57-1	0.1
1,4-dioxane	123-91-1	0.04
endosulfan I	959-98-8	102
endosulfan II	33213-65-9	102
endosulfan sulfate	1031-07-8	1,600
endrin	72-20-8	0.06
ethyl benzene	100-41-4	1
fluoranthene	206-44-0	2,455
fluorene	86-73-7	386
heptachlor	76-44-8	0.38
hexachlorobenzene	118-74-1	3.2
alpha-hexachlorocyclohexane	319-84-6	0.02
beta-hexachlorocyclohexane	319-85-7	0.09
delta-hexachlorocyclohexane	319-86-8	0.25
gamma-hexachlorocyclohexane	58-89-9	0.1
indeno[1,2,3-cd]pyrene	193-39-5	8.2
lead		450
manganese		390
mercury (elemental)	7439-97-6	0.73
mercury (inorganic salts)		NS[(2)]
methylene chloride	1634-04-4	0.05
methyl ethyl ketone	78-93-3	0.12
2-methylphenol	95-48-7	0.05
3-methylphenol	108-39-4	0.04
4-methylphenol	106-44-5	0.05
methyl *tert*-butyl ether	1634-04-4	0.93
naphthalene	91-20-3	12
nickel		130
pentachlorophenol	87-86-5	0.51
phenanthrene	85-01-8	1045
phenol	108-95-2	0.02
polychlorinated biphenyls	1336-36-3	3.2
n-propylbenzene	103-65-1	3.9
pyrene	129-00-0	3,400
selenium		1
silver		8.3
tetrachloroethene	127-18-4	1.3
toluene	108-88-3	0.7
1,1,1-trichloroethane	71-55-6	0.68
trichloroethene	79-01-6	0.47
2-(2,4,5-trichlorophenoxy)propionic acid	93-72-1	3.8

1,2,4-trimethylbenzene	95-63-6	3.6
1,3,5-trimethylbenzene	108-67-8	8.4
vinyl chloride	75-01-4	0.02
xylenes	1330-20-7	1.6
zinc		2,480

(1) CAS RN: Chemical Abstracts Service Registry Number.
(2) Not specified.

8.0 Protection of Ecological Resources

Ecological resources are here defined as all flora and fauna and the habitats that support them, excluding such species as pets or livestock, and agricultural and horticultural crops. While it is generally considered that brownfield sites occur primarily in developed or blighted areas with limited ecological resources, this is not always the case; some brownfield sites may be found in rural and undeveloped areas with more extensive ecological resources. Brownfield sites can contain or be situated adjacent to habitats such as forests, fields, wetlands, streams, and rivers. Given the potential for flora, fauna, and habitats to be impacted by contamination from brownfield sites, ecological SCOs were developed to meet the requirement in the Brownfield Cleanup Program to protect ecological resources. These SCOs are referred to as Ecological Soil Cleanup Objectives or ESCOs. The ESCOs derived by the methods described herein are presented in Table 8.6-1.

8.1 Risk Levels and Exposure Scenarios

The Brownfield Cleanup Program legislation specifies that the level of risk associated with the remedial action objectives "shall not exceed an excess cancer risk of one-in-one million for carcinogenic endpoints and a hazard index of one for non-cancer endpoints"[§27-1415.6.(b)]. Carcinogenic risk is not generally considered in ecological risk assessment and there is no guidance on developing cancer risk levels in fish and wildlife. The concept of a hazard index or hazard quotient, however, is often used in ecological impact analysis and risk assessment. The NYS DEC has determined that the ESCOs should be based on a low level of overall risk to ecological resources rather than a no-risk level. For this reason, ESCOs were based on lowest observed adverse effects levels taken from the ecotoxicological literature. In order to meet the requirement of a hazard index of one, the overall concentration of a contaminant in soils at a site must be equal to or less than the ESCO value.

The development of any ESCO is dependent upon the availability of data on the toxicity of various contaminants to a diverse range of organisms that dwell in or on the soil. Different contaminants have widely differing modes of action. Some contaminants are rapidly, acutely

toxic to plants or animals that are directly exposed to them. Others are not immediately toxic to exposed organisms, but can accumulate to toxic levels over time. Still others have low toxicity, but because of their high affinity for organic substances, are magnified as they are taken up by plants and/or soil invertebrates such as earthworms or benthic organisms, which are in turn consumed by fish, birds, and mammals. Such compounds may never directly kill an animal, but they may produce chronic effects such as impaired growth and modified behavior, or cause reproductive effects, such as birth defects, reduced number and/or survival of offspring, or eggshell thinning. Acceptable ESCOs must address all exposure scenarios that could potentially result in adverse effects.

8.2 Derivation Methodology for ESCOs

The NYS DEC adopted the US EPA Eco-SSLs model for calculating hazard quotients, plant bioaccumulation models, and earthworm bioaccumulation models (US EPA, 2003a). Eco-SSL methodologies were modified somewhat, because Eco-SSLs were specifically designed to be used as screening values and not cleanup levels. Eco-SSL methodologies (not Eco-SSL values themselves) were adopted for use in deriving NYS DEC ESCO values because they represent the best, most current, accepted scientific methods for assessing the uptake and bioaccumulation of soil-borne contaminants by plants and soil invertebrates and for estimating food chain risks to birds and terrestrial wildlife. These methodologies were modified to address cleanup needs rather than screening by changing some of the variables and parameters that tend to be more conservative; for example, using lowest observed effects concentrations (LOECs) for the calculation of hazard quotients (HQs) rather than no observed effects concentrations (NOECs).

ESCO values were derived by first estimating risk thresholds for chemicals in soil for three groups of organisms via two different pathways of exposure:

- Toxicity to plants via direct exposure;
- Toxicity to soil invertebrates via direct exposure;
- Toxicity to birds and mammals via food chain (bioaccumulation) exposure.

If a risk threshold for only one group of organisms could be derived, that risk threshold was selected as the ESCO. If risk thresholds were available for more than one group of organisms, the lowest risk threshold was selected as the ESCO.

8.2.1 Toxicity to Plants via Direct Exposure

The toxic effects of 45 chemicals (27 inorganic, 18 organic compounds) to plants were assessed by Efroymson et al., (1997a), and described as toxicity benchmarks. The toxicity benchmarks determined by Efroymson et al., (1997a) were adopted as risk thresholds for plants. Efroymson et al., (1997a) also reported toxic effects of various compounds to plants rooted in aqueous solutions instead of soil. Toxicity thresholds based on exposure to aqueous solutions were not used to derive ESCO values, because exposure conditions were not consistent with the exposure to chemicals that plants growing in contaminated brownfield soils would experience.

Risk thresholds for plant uptake of additional contaminants can be estimated using the same methodology used by Efroymson et al., (1997a) to derive toxicity benchmarks. That methodology can be summarized[1] as follows:

1. Collect (or conduct) studies of the toxicity of the chemical of interest to plants. Efroymson et al., (1997a) defined a significant effect as a greater than 20% reduction in plant growth or yield. Thus, a LOEC would be defined as the lowest chemical concentration tested that caused a greater than 20% reduction in growth or yield.

2. An LC_{50} is defined as the concentration that is lethal to 50% of the exposed organisms. If a study reported a plant LC_{50}, the LC_{50} was divided by 5 to estimate a LOEC based on a 20% effect concentration from a concentration that caused a 50% decrease in survival.

[1] The summary is intended to only describe the procedure employed by Efroymson et al., (1997), and to provide a general overview of the methodology for developing plant risk thresholds for additional chemical contaminants in soil.

3. If ten or more suitable studies were identified, the LOECs were organized in rank order, and the concentration equivalent to the 10^{th} percentile of the range of LOECs was selected as the toxicity benchmark for that chemical. If less than 10 studies were available, the lowest LOEC was selected as the risk threshold.

If no plant toxicity data are available for a particular chemical, a toxicity assessment may be needed to develop risk thresholds for that chemical. Any required soil toxicity testing for such an assessment should be based on at least three toxicity tests using different plant species native to New York State. Plant species used should be species that would be expected to grow in the type of soil, hydrology, and climatic conditions similar to that of the site being evaluated, or plant species used in standard phytotoxicity test methodologies as approved by the NYS DEC.

8.2.2 Toxicity to Soil Invertebrates via Direct Exposure

The toxic effects of 35 chemicals (9 inorganic, 26 organic) to earthworms were assessed by Efroymson et al., (1997b), and described as toxicity benchmarks. The intent of Efroymson et al., (1997b) was to derive toxicity benchmarks for the protection of soil and litter invertebrates and heterotrophic processes. However, earthworms were the only organisms for which enough toxicity studies were consistently available to derive benchmarks. Efroymson et al., (1997b) did derive toxicity benchmarks for an additional 32 chemicals for soil microorganisms and microbial processes. Those benchmarks were on the whole, less sensitive than either the earthworm benchmarks or the plant benchmarks derived by Efroymson et al., (1997a), so they were not used. The earthworm toxicity benchmarks derived by Efroymson et al., (1997b) were adopted as risk thresholds for soil invertebrates.

Additional risk thresholds for earthworms exposed to other contaminants via direct uptake from the soil can be determined using the same methodology used by Efroymson et al., (1997b) to derive toxicity benchmarks. That methodology can be summarized[2] as follows:

[2] The summary is intended to only describe the procedure employed by Efroymson et al., (1997a), and to provide a general overview of the methodology for developing earthworm risk thresholds for additional chemical contaminants in soil.

1. Collect (or conduct) studies of the toxicity of the contaminant of interest to earthworms. Earthworm toxicity studies typically evaluate effects such as survival, growth, reproduction, or changes in behavior. Efroymson et al., (1997b) defined a LOEC as the lowest chemical concentration tested that did cause a greater than 20% effect.

2. An LC_{50} is defined as the concentration that is lethal to 50% of the exposed organisms. If a study reported an earthworm LC_{50}, the LC_{50} was divided by 5 to estimate a LOEC based on a 20% effect concentration from a concentration that caused a 50% decrease in survival.

3. If ten or more suitable studies were identified, the LOECs were organized in rank order, and the concentration equivalent to the 10^{th} percentile of the range of LOECs was selected as the toxicity benchmark for that chemical. If less than 10 studies were available, the lowest LOEC was selected as the risk threshold.

If no earthworm toxicity information is available for a particular chemical of concern, a toxicity assessment may be needed to develop new risk thresholds. Any required soil toxicity testing for such an assessment should be based on at least three replicate toxicity tests, or tests with different species of earthworms that are native to New York using standard earthworm test protocols as approved by the NYS DEC.

8.2.3 Toxicity to Birds and Mammals via Food Chain Exposure (Bioaccumulation)

Chemicals in soil can be taken up and accumulate in plants and soil invertebrates. Those contaminants can then be passed to birds and animals[3] that consume plants and soil invertebrates, where they can cause toxic effects. To address this route of exposure, a simplified food chain bioaccumulation model was constructed consisting of three parameters:

[3] There are very few studies of the bioaccumulation of soil-borne contaminants by amphibians and reptiles, so the food chain bioaccumulation model described herein only addresses impacts to birds and mammals.

- a simplified food chain,
- the concentration of chemicals in wildlife forage, and
- toxic reference values for those chemicals.

Simplified Food Chain

The members of the simplified food chain are listed in Table 8.2-1 which shows the species and the corresponding biological characteristics used to model risks of contaminants in soil to birds and wildlife. These species are surrogates for organisms with similar feeding and foraging behaviors (e.g., herbivores, carnivores, etc.) and the resulting ESCO values are intended to protect all members of a particular feeding guild. A detailed description of the derivation of these parameters is included in Section 8.3.

Body weights and daily rations were estimated from literature values (US EPA, 1993; Sample and Suter, 1994; Baker, 1993). Soil ingestion rates are from Sample and Suter, (1994). The food ingestion rate (FIR) in kg dw/kg bw was estimated by converting the daily ration in wet weight to dry weight using conversion factors for plants and earthworms from US EPA (1993), and dividing by the body weight.

Table 8.2-1. Simplified Food Chain for Calculation of Bioaccumulation Based ESCOs

Species	Diet composition (%)		Body weight kg ww	Daily ration kg ww	Food Ingestion Rate (FIR) (kg dw/kg bw)	soil ingestion rate kg dw/day
	plants	earthworms				
meadow vole	100		0.044	0.01523	0.0547	0.00012
white-footed mouse	50	50	0.022	0.00455	0.0354	0.000068
short-tailed shrew		100	0.0186	0.01403	0.183	0.00117
white-tailed deer	100		56.5	1.74	0.0052	0.0348
American woodcock		100	0.184	0.146	0.212	0.0156

Chemical Concentrations in Wildlife Forage

To estimate wildlife risk thresholds from food chain bioaccumulation, the concentration of chemicals in wildlife forage must first be determined. In the simplified food chain model used here, the diet of the representative birds and animals was limited to plants and earthworms. The US EPA (2003b) published models for estimating the uptake of contaminants from soil by plants. For arsenic, barium, chromium, manganese, DDT, and pentachlorophenol chemical-specific median BAFs published in US EPA (2003b) were used to estimate plant concentrations from soil concentrations. For cadmium, copper, lead, nickel, selenium, zinc, dieldrin, and benzo[a]pyrene, natural log regression models published in US EPA (2003b) were used to estimate plant concentrations from soil concentrations. For other nonpolar organic chemicals, a general model published in US EPA (2003b) was used to calculate a soil-to-plant bioaccumulation factor (BAF) from each chemical's *n*-octanol water partitioning coefficient (K_{OW}):

$$\log(BAF) = -0.4965 * (\log K_{OW}) + 2.53 \qquad (1)$$

US EPA (2003b) also published models for estimating the uptake of contaminants from soil by earthworms. For barium, chromium, copper, nickel, dieldrin, and DDT, chemical-specific median BAFs published in US EPA (2003b) were used to estimate earthworm concentrations from soil concentrations. For arsenic, cadmium, lead, manganese, selenium, and zinc, natural log regression models published in US EPA (2003b) were used to estimate plant concentrations from soil concentrations. For other nonpolar organic chemicals, a general model published in US EPA (2003b) was used to calculate a soil-to-earthworm bioaccumulation factor (BAF) from each chemical's *n*-octanol water partitioning coefficient (K_{OW}):

$$BAF = \frac{10^{(\log K_{OW} - 0.6)}}{f_{oc} * K_{oc}} \qquad (2)$$

where f_{OC} = % of organic carbon in the soil, set at 1% (0.01);
K_{OC} = soil organic carbon partitioning coefficient.

US EPA (2003b) published several different models for deriving the K_{OC} from a chemical's K_{OW}, depending upon the type of chemical:

PAHs:

$$\text{Log } K_{OC} = 0.8903 * (\log K_{OW}) + 0.2794 \quad (3)$$

Halogenated aromatic hydrocarbons:

$$\text{Log } K_{OC} = 0.9739 * (\log K_{OW}) - 0.2238 \quad (4)$$

Non-halogenated aromatic hydrocarbons:

$$\text{Log } K_{OC} = 0.5289 * (\log K_{OW}) + 0.9182 \quad (5)$$

Chlorophenols:

$$\text{Log } K_{OC} = 01.0757 * (\log K_{OW}) - 0.8006 \quad (6)$$

General model for other nonpolar organic compounds:

$$\text{Log } K_{OC} = 0.983 * (\log K_{OW}) +0.00028 \quad (7)$$

A detailed listing of the different BAFs and regression values from the US EPA (2003b) used to estimate plant and earthworm uptake of soil-borne contaminants is included in Section 8.3.

Toxic Reference Values

The last piece of information needed to estimate the risks of soil-borne contaminants to the animals in the simplified food chain model described above is the toxic reference values (TRVs) for various soil contaminants. Sample et al., (1996) conducted an extensive review of the literature, and published a summary of toxic effects of different chemicals to birds and wildlife. They identified both NOECs and LOECs. The LOECs published by Sample et al., (1996) were used as TRVs. Additional risk thresholds for the exposure of birds and animals to other soil-borne chemicals can be derived using toxicity data for LOECs from other literature sources as well.

Calculating Food Chain ESCO Values

US EPA (2003a) published a general model for estimating the risk to wildlife from contaminants in soil. That equation is:

$$HQ_j = \frac{\left\{\left[Soil_j * P_s * FIR\right] + \left[\sum_{i=1}^{j} B_i * P_i * FIR\right]\right\}}{TRV_j} \qquad (10)$$

Where:

HQ_j = Hazard Quotient for chemical j;
$Soil_j$ = concentration of chemical in soil, mg/kg dry weight;
N = number of different items in diet (n=maximum of 2, earthworms and/or plants);
P_s = soil ingestion rate as a percentage of diet;
FIR = Food ingestion rate in kg food (dry weight) / kg body weight (wet weight);
B_i = contaminant concentration in biota type (i.e., food type, plants or earthworms);
P_i = Percentage of food type B_i in diet;
TRV_j = LOEC for chemical j in mg/kg body weight.

For each chemical of interest, the concentration in plants was calculated using a chemical specific BAF or regression equation from US EPA (2003b), or equation 1, above. The concentration in earthworms was then calculated using a chemical specific BAF or regression equation from US EPA (2003b), or equations 2 - 8, above. Once the chemical concentrations in plants and earthworms were estimated, equation 10 was solved for each of the five representative species (meadow vole, short-tailed shrew, white-footed mouse, white-tailed deer, American woodcock) by iteratively substituting different values for $Soil_j$ until the hazard quotient (HQ_j) equaled 1.

ESCO values for additional chemicals can be derived using the methodology described above. The required elements of information for a nonpolar organic chemical of interest are its K_{OW} and an appropriate TRV derived experimentally or from the literature. For inorganic chemicals, appropriate plant and earthworm uptake factors (BAFs or uptake regression equation variables) derived either experimentally or from the literature are needed instead of a K_{OW}. An example of the calculation for a food chain based ESCO value is included in Section 8.4.

8.3 Derivation of Parameters Used for ESCO Wildlife Modeling

This section explains the derivation of the parameters and variables used to conduct wildlife toxicity modeling to develop ESCOs.

8.3.1 Body Weights

A. Meadow vole: The body weight of 44 g (0.044 kg) was taken from Sample and Suter (1994). Baker (1983) reported the range of body weights for adult meadow voles to be between 30 - 60 g. The mean of 30 g and 60 g is 45 g, which is consistent with Sample and Suter (1994). The US EPA (1993) reported 10 values for meadow vole body weights for both sexes over different seasons. The mean of the 10 values was 32.8 g, however, 8 of the ten values were from Canada, where because of a colder climate and shorter summer, values might be biased slightly lower.

B. White-footed mouse: The body weight of 22 g (0.022 kg) was taken from Sample and Suter (1994). The US EPA (1993) reported a range of body weights for the white-footed mouse of 14 - 31 g; the mean of which is 22.5 g, consistent with Sample and Suter (1994). Baker (1983) reported the same as the US EPA (1993).

C. Short-tailed shrew: Sample and Suter (1994) reported a body weight for the short-tailed shrew as 15 g (0.015 kg). Baker (1983) reported a range of body weights for short-tailed shrew of 18 - 30 g; the mean of which is 24 g. The US EPA (1993) reported five values for short-tailed shrew body weights for both sexes collected in the summer and fall from New Hampshire and Pennsylvania. The mean of these five values was 16.8 g. The mean of those three estimates (15g, 24g, and 16.8g) was 18.6 g (0.0186 kg). This value was used as the estimate of body weight for the short-tailed shrew.

D. White-tailed deer: The body weight of 56.5 kg (56500 g) was taken from Sample and Suter (1994). The US EPA (1993) did not report data for white-tailed deer. Baker (1983) reported two ranges of body weights, 68.6 - 140.9 kg and 40.9 - 95.5 kg. The mean of those two ranges was

86.4 kg. Despite the difference, the body weight from Sample and Suter was selected for use, as the body weight ranges from Baker (1983) seem to be under-representative of young deer in the population.

E. American woodcock: Sample and Suter (1994) reported a mean body weight for American woodcock as 198 g (0.198 kg). The US EPA (1993) reported 13 adult body weights for both sexes over the spring, summer and fall. The mean of those 13 values was 169.969 or 170g. The mean of those two estimates (170 g and 198 g) was 184 g (0.184 kg), which was the value used herein as the American woodcock body weight.

8.3.2 Food Consumption Rates

A. Meadow vole: Baker (1983) reported that a vole consumed approximately 60% of its body weight daily. The US EPA (1993) reported the food consumption rate for meadow voles was 0.3 - 0.35 g/g bw ww. Sample and Suter (1994) reported the daily food consumption rate for meadow voles to be 5 g ww. Using a body weight of 44 grams, the literature values can be used to produce 3 estimates of meadow vole daily ration: 26.4g; 14.3 g; and 5 g ww. The mean of the three estimates is 15.23 g ww. Using the wet weight to dry weight conversion factor for plants from the US EPA (1993), the mean daily ration for meadow voles was estimated to be 2.285 g dw.

B. White-footed mouse: Sample and Suter (1994) reported a daily ration of 3.4 g ww. The US EPA (1993) reported seven food ingestion rates for the deer mouse (which has the same average size and diet preferences as a white-footed mouse). The mean of those seven values was 0.26 g/g bw ww. Using a body weight of 22 grams, a daily ration of 5.72 g ww can be estimated. The mean of the two estimates (3.4g & 5.72 g) is 4.55 g ww. Assuming that 50% of the white-footed mouse's diet is earthworms and 50% is plants, a dw daily ration of 0.71 g can be estimated, using the wet weight to dry weight conversion factors from the US EPA (1993).

C. Short-tailed shrew: Sample and Suter (1994) reported a daily ration of 9 g ww. The US EPA (1993) reported ww food ingestion rates of 7.95 g/day; 0.49 g/g bw; and 0.62 g/g bw. Baker (1983) reported that short-tailed shrews will consume from ½ - 3 times their body weight daily. The average of 0.5 and 3 is 1.75 g/g bw ww. Using a body weight of 18.6 grams, ww daily ration estimates of 9 g; 7.95 g; 32.6 g; 9.1; and 11.5 g were made. The mean of these five estimates is 14.03 g ww. Using the wet weight to dry weight conversion factor for earthworms from the US EPA (1993), the mean daily ration for the short-tailed shrew is 3.41 g dw.

D. White-tailed deer: Sample and Suter (1994) reported a daily ration of 1.74 kg ww. Baker (1983) reported that a deer must consume 4 pounds of forage per day to maintain its body weight. Baker (1983) did not apply this ration size to any particular size of deer. Four pounds (1.8 kg) is consistent with Sample and Suter's (1994) estimate of 1.74 kg, so that estimate was adopted as the ww daily ration for white-tailed deer. Using the wet weight to dry weight conversion factor for plants from the US EPA (1993), the mean daily ration for the white-tailed deer is 261 g dw.

E. American woodcock: Sample and Suter (1994) reported a daily ration of 150 grams ww. The US EPA (1993) reported a food ingestion rate of 0.77 g/g bw ww. Using a body weight of 184 grams, a ww daily ration estimate of 141.68 g ww can be made. The mean of the two estimates (150 g and 141.68 g) results in a estimated ww daily ration of 146 g for the American woodcock. Using the wet weight to dry weight conversion factor for plants from the US EPA (1993), the mean daily ration for the American woodcock is 23.4 g dw.

8.3.3 Uptake Factors

Factors used to estimate bioaccumulation of soil-borne contaminants by plants and earthworms are presented in Table 8.3-1.

Table 8.3-1. Uptake Factors for Calculation of Bioaccumulation Based ESCOs.

Chemical	Plant uptake			Earthworm uptake		
	BAF	Slope	Intercept	BAF	Slope	Intercept
Arsenic	0.03752				0.706	-1.421
Barium	0.156			0.091		
Cadmium		0.546	-0.475		0.795	2.114
Chromium	0.041			0.306		
Copper		0.394	0.668	0.515		
Lead		0.561	-1.328		0.807	-0.218
Manganese	0.079				0.682	-0.809
Nickel		0.748	-2.223	1.059		
Selenium		1.104	-0.677		0.733	-0.075
Zinc		0.554	1.575		0.328	4.449
dieldrin		0.841	-3.271	267.08		
DDT	0.028			116.61		
Pentachlorophenol	9.615071			74.68		
benzo[a]pyrene		0.635	-2.053	31.47		

This table provides a list of the variables needed to model the uptake of some soil borne contaminants by plants and earthworms. If a BAF for a chemical is provided, then the concentration of the chemical in plant or earthworm tissue ($C_{P/E}$) can be estimated from the concentration in soil (C_{Soil}) by:

$$C_{P/E} = C_{Soil} * BAF$$

If a slope and intercept are provided, then the concentration of the chemical in plant or earthworm tissue ($C_{P/E}$) can be estimated from the concentration in soil (C_{Soil}) by:

$$\ln(C_{P/E}) = \text{slope} * \ln(C_{Soil}) + \text{intercept}$$

For all other chemicals, the concentration in plant or earthworm tissue was estimated from the concentration in soil using the general uptake model for plants (equation 1) or the general uptake model for earthworms (equations 2 - 8).

8.4 Example Calculation for Bioaccumulation Based ESCOs

The following example illustrates the calculation of a wildlife threshold value for benzo[a]pyrene.

1. Collect required information on the chemical of interest:

Chemical: Benzo[a]pyrene (CAS - 50-32-8)
$\log K_{OW}$: 6.11
TRV: The mammalian LOEC from Sample et al., (1996) is 10 mg/kg bw. No avian LOEC was available.

2. Determine uptake factors:

Plant: US EPA (2003b) provides a plant uptake regression equation of

$$\ln(C_{BAP\text{-}P)} = 0.635\ln(C_{BAP\text{ - }Soil}) - 2.035$$

Earthworm: US EPA (2003b) provides an earthworm uptake BAF of 31.47:

$$C_{BAP\text{-}E} = 31.47 * C_{BAP\text{-}Soil}$$

3. Determine food ingestion rates and dietary preferences as listed in Table 8.2-1.

4. Select initial soil concentration to be tested: 3 mg/kg dw.

5. Calculate plant uptake:

$\ln(C_{BAP\text{-}P}) = 0.635(\ln(3) - 2.035 = 0.6976 - 2.035 = -1.355$; $e^{(-1.355)} = 0.258$ mg/kg dw[4]

6. Calculate earthworm uptake:

$$C_{BAP\text{-}E} = 31.47 * 3 = 94.41 \text{ mg/kg dw}^4$$

7. Use the bioaccumulation data derived above and the food ingestion / dietary preference data from Table 8.2-1 to calculate the hazard quotient using equation 10. In the example below, the hazard quotient corresponding to a soil concentration of 3 mg/kg dw for the white-footed mouse is calculated.

$$HQ = \frac{\overset{\text{soil}}{(3 * 0.09 * 0.0354)} + \overset{\text{plants}}{(0.258 * 0.455 * 0.0354)} + \overset{\text{earthworms}}{(94.41 * 0.455 * 0.0354)}}{10} =$$

$$HQ = \frac{0.009558 + 0.004156 + 1.5207}{10} = 0.1534$$

In this example, the hazard quotient is less than 1.0, so more benzo[a]pyrene could persist in the soil without harming white-footed mice. However, the white-footed mouse is not the most sensitive animal exposed to benzo[a]pyrene through its food chain. An examination of the plant and earthworm bioaccumulation rates shows that earthworms are much higher bioaccumulators of benzo[a]pyrene than plants. The short-tailed shrew has a much greater dietary preference for earthworms than the white-footed mouse. When the model is run for the short-tailed shrew, the hazard quotient equals 1 at a benzo[a]pyrene soil concentration of 2.5 mg/kg.

[4] USEPA (2003b) indicates that for plants and earthworms, regression models are derived using dw tissue concentrations. It is assumed that dw is also applicable to BAFs as well, as USEPA (2003b) does not show a conversion step.

8.5 Ecological Soil Cleanup Objectives (ESCOs)

Table 8.5-1. Ecological Soil Cleanup Objectives

Contaminant[1]	CAS	ESCO mg/kg	Basis[2]
acenaphthene	83-32-9	20	plant
acetone	67-64-1	2.2	vole
aldrin	309-00-2	0.14	shrew
arsenic	7440-38-2	10	plant
barium	7440-39-3	433	woodcock
benzene	71-43-2	70	shrew
benzo[a]pyrene	50-32-8	2.6	shrew
beryllium	7440-41-7	10	plant
BHC-beta	319-85-7	0.6	shrew
BHC-mixed isomers	608-73-1	0.04	shrew
cadmium	7440-43-9	4	plant
chlordane (alpha)	57-74-9	1.3	shrew
chlorobenzene	108-90-7	40	earthworm
chloroform	67-66-3	12	shrew
chromium, hexavalent	18540-29-9	0.4	earthworm
chromium, trivalent	18540-29-9	41	woodcock
copper	7440-50-8	50	earthworm
ΣDDT	50-29-3	0.002	woodcock
1,4-dichlorobenzene	106-46-7	20	earthworm
1,2-dichloroethane	107-06-2	10	woodcock
dieldrin	60-57-1	0.006	shrew
1,4-dioxane	123-91-1	0.042	deer

Contaminant[1]	CAS	ESCO mg/kg	Basis[2]
endrin	72-20-8	0.014	shrew
fluorene	86-73-7	30	plant
heptachlor	76-44-8	0.14	shrew
lead	7439-92-1	50	plant
lindane (gamma BHC)	58-89-9	6	woodcock
manganese	7439-96-5	500	plant
mercury		0.1	earthworm
methylene chloride	75-09-2	12	vole
methyl ethyl ketone (2 butanone)	78-93-3	360	vole
nickel	7440-02-0	30	plant
pentachlorophenol	87-86-5	0.27	shrew
phenol	108-95-2	30	earthworm
selenium	7782-49-2	1	woodcock
silver	7440-22-4	2	plant
tetrachloroethene	127-18-4	2	shrew
toluene	108-88-3	36	shrew
trichloroethylene	79-01-6	2	shrew
xylene (mixed)	1330-20-7	0.26	shrew
zinc	7440-66-6	50	plant

[1] Unless otherwise indicated, this list of compounds was developed from the list of compounds included in Efroymson et al., (1997a), Efroymson et al., (1997b), and Sample et al., (1996). These are the compounds which comprise the ORNL database of toxicological benchmarks for which toxicological data were readily available.

[2] Where the basis for the ESCO is plant or earthworm, the value is based on direct toxicity and may not be modified. All others are based on food chain bioaccumulation and may be modified using site-specific total organic carbon and the formula shown in Equation 2 of Section 8.2.3.

8.6 Limitations

The ESCOs described herein were derived specifically for use as cleanup levels in the Brownfields Cleanup Program. They represent concentrations of contaminants in soils that, if not exceeded, should not cause measurable harm to exposed organisms in the context of a remediated site.

The ESCOs should not be used as SSLs, and should not be used as input parameters for screening level ecological risk assessments; nor should they be used generically to define soils as uncontaminated. They are less conservative than, and may not be as protective as, ecological soil screening criteria because they are derived from low effect levels rather than no effect levels. Residual risks will still be present at these concentrations not only because the ESCOs are derived from threshold effect levels, but also because the currently available data may not necessarily be protective of the most sensitive species.

The ESCOs were developed based upon current, publicly available toxicity and bioaccumulation data from two sources: Efroymson et al., (1997a and 1997b) for direct toxicity data and Sample et al. (1996) for bioaccumulation data. Data were not always available for both toxicity and bioaccumulation; nor were data available for more than a single species in most cases. In addition, neither toxicity nor bioaccumulation data were available from these sources for some of the contaminants on the SCO priority list. These contaminants are designated with an “NS” (not specified) in the SCO tables in the draft proposed 6 NYCRR Part 375-6.8.

The ESCO values were derived without regard for background concentrations, soil type, or analytical detection limits. Other provisions of the Brownfield Cleanup Program law and proposed regulations address this limitation.

Because of the wide range of organisms that must be protected, the impossibility of characterizing toxicity thresholds for all exposure scenarios, and the necessity of using general models for deriving ESCOs, there is uncertainty associated with the calculated risk thresholds.

To minimize the influence of uncertainty, reasonable assumptions that would balance each other were selected, such as overprotective dietary assumptions balanced by the use of LOECs as TRVs. Another measure used to minimize the influence of uncertainty was the use of mean or median values for model variables whenever possible. The use of median (or near median) values reduces the likelihood that the risk thresholds would be overprotective, but increases the chance that some level of toxicity might occur when soil concentrations are very close to ESCO values.

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8.7 Acronyms

BAF	Bioaccumulation Factor
BW	Body Weight
CAS	Chemical Abstract Society
DDT	Dichlorodiphenyltrichloroethane
DW	Dry Weight
Eco-SSL	Ecological Soil Screening Level
ESCO	Ecological Soil Cleanup Objective
FIR	Food Ingestion Rate
HQ	Hazard Quotient
KG	Kilograms
K_{oc}	Organic Carbon Partitioning Factor
K_{ow}	*n*-Octanol Water Partitioning Factor
LC_{50}	50% Lethal Concentration
LOEC/LOEL	Lowest Observed Effects Concentration or Level
NOEC/NOEL	No Observed Effects Concentration or Level
NYCRR	New York Code of Rules and Regulations
NYS DEC	New York State Department of Environmental Conservation
PAHs	Polyaromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
TRV	Toxic Reference Value
US EPA	United States Environmental Protection Agency
WW	Wet Weight

8.8 Definitions and Abbreviations

Bioaccumulation Factor (BAF): The concentration of a chemical in an organism divided by the concentration of the same contaminant in the surrounding media (in this document, the media is soil).

Dry weight (DW or dw): The weight of a substance (soil, plant tissue, earthworm tissue, etc.) after the water content has been removed.

ESCO: Ecological Soil Cleanup Objective - the highest concentration of a contaminant in soil that can be present without causing significant harm to ecological resources.

Hazard Quotient (HQ): The concentration of a chemical in soil divided by a risk threshold. If a hazard quotient is greater than one, the concentration of a chemical in soil exceeds its corresponding risk threshold and a toxic effect should be anticipated. If a hazard quotient is less than one, the concentration of a chemical in soil does not exceed the corresponding risk threshold and toxic effects are not anticipated.

LOEC/LOEL: Lowest Observed Effects Concentration; Lowest Observed Effects Level: The lowest concentration of a chemical actually measured in a toxicity test at which statistically significant effects resulting from exposure to the chemical being tested were documented to occur.

NOEC/LOEL: No Observed Effects Concentration; No Observed Effects Level: The highest concentration of a chemical actually measured in a toxicity test at which no statistically significant effects resulting from exposure to the chemical being tested were documented to occur.

Risk Threshold: The concentration of a contaminant in soil that represents the point where toxic effects begin to occur to the most sensitive organisms in the ecosystem (or in a given group of organisms such as plants, soil invertebrates, etc.), based on the data available.

Route of exposure: The pathway through which a chemical contaminant in soil comes in contact with, and is taken up by, an organism.

Toxicity Assessment: A field study, laboratory study and/or literature review conducted to determine the concentration at which a contaminant becomes toxic to exposed organisms. A contaminant is considered toxic if it causes death, morbidity or sub-lethal effects on growth, reproduction, behavior or physiology, whether through direct or indirect toxicity or through bioaccumulation.

Toxic Reference Value (TRV): The dose of a contaminant that caused a toxic effect in a test animal.

Wet weight (WW or ww): The weight of a substance (soil, plant tissue, earthworm tissue, etc.) without removing the water content.

9.0 Modification of SCOs

SCOs were modified, when appropriate, based on background concentrations of Priority List analytes in rural soils, analytical limits of detection and maximum allowable analyte concentrations ("caps").

In order to provide a basis for comparing risk-based and groundwater protection SCOs to rural soil background concentrations, the NYS DEC and the NYS DOH conducted a statewide rural surface soil survey. The statewide rural surface soil survey determined concentration ranges for 179 commonly assessed analytes in discrete surface soil samples collected at randomly selected rural properties in New York State. The survey is described in greater detail in Section 9.1.2. A summary report on the survey is attached as Appendix D. SCOs for public health protection were modified, when appropriate, as set forth in Section 9.1. SCOs for protection of ecological resources were modified, when appropriate, as set forth in Section 9.2.

Maximum allowable analyte concentrations ("caps") are discussed in Section 9.3.

9.1 Background Soil Concentrations (Public Health)

Section 27-1415.6(b) of the legislation states that Soil Clean-up Objectives "... shall not exceed an excess cancer risk of one-in-one million for carcinogenic endpoints and a hazard index of one for non-cancer endpoints; provided, however, that if the background soil concentration for a contaminant in rural soils in New York state exceeds such risk level the contaminant specific action objective for such contaminant may be established equal to such background concentration." This section describes the methods used to determine background concentrations for SCO Priority List analytes in rural soils in New York State.

9.1.1 Definition of "Background Soil Concentration"

There is no widely accepted definition of "background soil concentration." In establishing a Rural Soil Background Concentration (RSBC), we selected a concentration that approximated

the 98th percentile concentration for that analyte in rural New York State soils. The 98th percentile was used because it is the nearest whole percentile to the 97.5th percentile, which is the upper bound of the "reference range" (2.5th to 97.5th percentile)[1] often employed to define values that are considered typical. For example, the 97.5th percentile was used by the Ontario Typical Range Model to establish upper bounds for organic and inorganic analytes in various environmental media including soil (OMEE, 1993).

9.1.2 The Statewide Rural Surface Soil Survey

The Statewide Rural Surface Soil Survey ("Rural Survey") determined concentration ranges for 170 commonly assessed analytes in discrete surface soil samples collected at randomly selected rural properties in New York State. A report on the survey is attached as Appendix D.

Rural Survey samples were of three types: "source-distant," "near source," and "remote." Source-distant samples were collected from points of human contact with soil that were a distance of approximately five meters (about 15 feet) or more from any identifiable source of contamination including roadways, pavement, and structures.[2] Near source samples were collected along an imaginary line extending from the source-distant sampling location to the nearest road or driveway, and at a distance of two to three meters (about 6.5 to 10 feet) from the road or driveway.[3] Remote samples, collected for purposes of ecological assessment, were obtained from points that were a distance of about 15 meters (about 50 feet) or more from areas of human activity such as lawns, cultivated land, or trails if possible, and otherwise from portions of designated rural properties that were the least influenced by human activities.

Sampling depths were consistent with NYS DEC guidance for investigating contaminated sites (NYS DEC, 2002). Source-distant and near source soil samples were collected at a depth interval of zero to five centimeters (zero to two inches) below ground surface (b.g.s.). Remote samples were collected at a depth interval of zero to 15 centimeters (zero to six inches) b.g.s.

[1] The range between the 2.5th and 97.5th percentile will include 95 percent of observed values.
[2] The original survey requirement of 20 paces (approximately 15 to 20 meters) distance was relaxed soon after soil sampling began, because field staff could not identify suitable sampling locations.
[3] A distance of two meters was obtained in most cases.

Also consistent with NYS DEC guidance for investigating contaminated sites, organic materials such as leaves, pine needles, grass, and roots were removed prior to sampling, but no attempt was made to completely exclude the organic soil horizon.

The Rural Survey provided analytical data for source-distant and remote soil samples collected at 120 randomly selected rural properties in New York State. Data were also available for a remote soil sample collected at one additional property (total of 121 properties). In addition, data were available for near source soil samples collected at a randomly selected subset of 28 properties, for a total of 269 samples (all types). Data for two source-distant and two remote soil samples collected at known orchards were excluded, consistent with the Rural Survey protocol, reducing the total number of available samples to 265.

The Rural Survey data were used in two ways. First, 98th percentile analyte concentrations from the Rural Survey data were used for screening purposes to identify "focus analytes" -- Priority List analytes most likely to achieve concentrations in rural soils greater than the lowest health-based SCOs. Next, RSBCs were designated for focus analytes using the Rural Survey data and, if available, other relevant survey data.

9.1.3 Screening Process to Select Focus Analytes

Analyte concentration data from the Rural Survey were used for screening because the Rural Survey obtained a high degree of geographic coverage (i.e., all regions of New York State were sampled). The survey also employed a uniform study design with rigorous quality assurance, reducing uncertainty associated with estimates of analyte concentration ranges in rural soils. Furthermore, the survey employed a restrictive definition of "background" that precluded sampling near readily discernable contamination (e.g., trash, discoloration, odors), and avoided other areas where contaminants may be enriched in soil (e.g., drainage swales, waste disposal sites). The degree of conformance with the survey protocol was confirmed through reviews of field notes, photo-documentation, and aerial photographs. As previously mentioned, soil samples were collected at a depth consistent with draft NYS DEC guidance for investigating contaminated sites (NYS DEC, 2002).

Screening values from the Rural Survey data set were developed using analyte concentrations for source-distant and near source rural soil samples. Data for remote samples were not used for screening to ensure that screening values would be based on analyte concentrations in soils at points of human contact (see Appendix D).

Analyte levels for source-distant and near source samples were combined to form a single data set consisting of 146 observations per analyte.[4] Then, the 98th percentile concentration was calculated for each analyte by employing the empirical distribution function with averaging. The 98th percentile concentration for an analyte is a concentration that exceeds 98 percent of the reported values.

The empirical distribution function was used to calculate 98th percentiles because that approach accommodated aspects of the data such as multi-modal distributions for metals (due, in part, to inclusion of data from different soil orders) and left-censored data (due to detection limits). This method of calculating percentiles also avoided data filling, data transformations, and sequential outlier removal. Distribution-free approaches like the one selected tend to be more robust to extreme observations than parametric alternatives, a desirable characteristic when, as in this case, the goal is to employ all quality assured data.

Table 9.1-1 provides screening values for most analytes on the SCO Priority List. Screening values were not developed for 3-methylphenol (*m*-cresol), 4-methylphenol (*p*-cresol) and *n*-propylbenzene because concentrations of these were not determined in Rural Survey samples.

Levels of individual mercury and chromium species were not determined by the Rural Survey, so screening values for all mercury and chromium species were based on total mercury and total chromium levels, respectively, in Rural Survey samples.

9.1.4 Results of Screening Process

The results of the screening process are summarized in Table 9.1-2. Screening values for 22 Priority List analytes exceeded candidate SCOs (health-based or groundwater protection SCOs) for those analytes, so rural soil concentrations for those analytes were further evaluated. The 22 "focus" analytes were:

Metals and Metal Compounds

Arsenic

Barium

Cadmium

Chromium(III)[5]

Chromium(VI)[5]

Manganese

Mercury(Inorganic)[6]

Selenium

Polycyclic Aromatic Hydrocarbons

Benzo[a]anthracene

Benzo[a]pyrene

Benzo[b]fluoranthene

Benzo[k]fluoranthene

Chrysene

Dibenz[a,h]anthracene

Indeno[1,2,3-cd]pyrene

[4] Only 49 observations for the organochlorine pesticide delta-BHC and 145 observations for 1,4-dioxane were available, due to incomplete reporting by the contract laboratory. Fewer than 146 observations were available for several other analytes after data quality review.

[5] The screening value for total chromium was greater than the lowest SCOs for chromium (III) and chromium (VI).

[6] The screening value for total mercury was greater than the lowest SCO for inorganic mercury.

Other Organic Compounds

Acetone

Aldrin

1,2-Dichlorethane

Dieldrin

1,4-Dioxane

2-Methylphenol (*o*-cresol)

Phenol

Screening values for other analytes were below health-based and groundwater protection SCOs, so rural soil concentrations for those analytes were not evaluated further.

9.1.5 Process for Establishing RSBCs

An RSBC for chromium (VI) could not be established because no data were identified indicating levels of chromium (VI) in rural New York State soils (see Section 9.1.5.1). To determine RSBCs for the 21 remaining focus analytes, we examined Rural Survey data and other available survey data. Other survey data were used if the survey approach resulted in the collection of soil samples from points on rural properties that were likely points of human contact and distant from any identifiable sources of contamination. If no alternative Rural Survey data sets were available, other data sets were sought to help guide selection of appropriate data from the Rural Survey. When no alternative data sources were identified, the Rural Survey data were employed exclusively to establish RSBCs.

9.1.5.1 RSBCs for Metals

1. Data Sources

The following surveys were used to establish RSBCs for metals. Summary information for each survey is provided in Table 9.1-3.

Rural Survey (2005). This survey was described previously. Data for each of the three sample types (source-distant, near source, and remote) were used. Differences in concentrations of focus metals among the three sample types were evaluated using SAS version 9.1 (SAS Institute, Cary, N.C.) to perform Kruskal-Wallis tests (Table 9.1-4). Differences were not statistically significant for focus metals other than mercury ($p > 0.05$). Therefore, the data for each analyte other than mercury were pooled to create a single Rural Survey data set for each analyte. Concentrations of mercury differed among the three Rural Survey sample types and the differences were statistically significant (KW $p = 0.01$, see Table 9.1-4). Data for remote samples were not used to evaluate an RSBC for inorganic mercury to ensure that the RSBC would be based on analyte concentrations in soils at points of human contact.

Data from two source-distant and two remote Rural Survey soil samples collected at known orchards were not included in the analyses. Excluding these orchard samples is consistent with the Rural Survey protocol.

NYS DEC Region 3 (2003). This survey determined elemental concentrations in soil samples from 20 publicly owned properties in the Lower Hudson Valley region of New York State. The properties were managed by the NYS DEC, the State Office of Parks, Recreation and Historic Preservation, or the Westchester County Department of Parks, Recreation and Conservation. Properties were not designated for soil sampling using a probabilistic approach, but rather at the discretion of the investigators. Special care was taken to sample only undeveloped sites exhibiting mature natural vegetation, with no apparent signs of pollution. At each site, three replicate samples were collected within a 10-foot radius using a corer. Samples were collected at a depth of zero to 15 centimeters (zero to six inches) b.g.s. The organic soil horizon or "litter layer," the soil layer that is rich in organic materials, was excluded if present.

Al-Wardy (2002). This survey summarized data on elemental concentrations in 922 "mineral" and 28 "organic" soil samples submitted by farmers to the Cornell Nutrient Analysis Laboratories during 1998 and 1999. The author did not indicate sampling depths, the number of lots sampled, and distances from sampling points to pollution sources, so data from these "farmers' samples" were not considered further.

Al-Wardy also reported concentration values for soil samples collected from agricultural and forest areas in two geographic regions: central and western New York. Samples were collected at 51 locations. At each of these locations, a "plow zone" sample was collected at a depth of 23 to 25 centimeters (nine to ten inches) b.g.s.; deeper "subsoil" samples were collected at 61 centimeters (24 inches) b.g.s at 50 of the locations. Properties were not designated for soil sampling using a probabilistic approach. Instead, the investigator designated locations that appeared to adequately characterize four common soil types.

Elemental concentrations reported by Al-Wardy for 51 plow zone samples from agricultural and forest areas were considered during determination of RSBCs. However, as actual data points were not available, the 98th percentile was assumed to be the maximum concentration reported (100th percentile).

Elemental concentrations for subsoil samples from agricultural and forest areas were not considered during the determination of RSBCs because the sampling depth of 61 centimeters (two feet) b.g.s. was well-below the surface soils that people frequently contact.

Clarke *et al*. (1985). This NYS DOH survey provided elemental concentration data for 40 composite samples collected from established lawns in Albany, Broome, Essex, Franklin, Onondaga, St. Lawrence, and Ulster Counties. These included 11 samples from five rural (described as "outlying farmland") properties. Properties were not designated for soil sampling using a probabilistic approach. Instead, NYS DOH regional staff identified property owners willing to allow sampling.

One to three samples were collected at each designated property. Each sample was made up of two soil subsamples collected about one foot apart using a coring device. The subsamples consisted of soil from the zero to 10-centimeter (zero to four-inch) b.g.s. soil horizon. The organic soil horizon was not excluded. Grass remained on the subsample after sampling but was trimmed down to a length of about 0.6 centimeters (0.25 inches). Property owners confirmed

that sampled lawn areas were free of known contamination and fulfilled the following survey requirements:

- no significant quantities of topsoil removed within the past 20 years;
- no excavation work performed within the past 20 years;
- no more than one inch of topsoil added within the past 20 years;
- no excessive application of pesticides and chemical fertilizers;
- no burn barrels or cooking grills in the area for the past 20 years;
- no oil contamination as by rupture of a utility transformer, or due to vehicle repair work.

In addition, field staff selected sampling locations that were not near a shed or garage where chemicals may have been stored, and at least five meters (about 15 feet) from a road.

The 11 rural soil samples included three samples from one property each in Broome, Onondaga and St. Lawrence counties, and one sample from one property each in Ulster and Essex counties.

Shacklette and Boerngen (1984). United States Geologic Survey (USGS) staff analyzed 527 soil samples collected from locations scattered across the United States, including 25 soil samples from sites in New York State. Most soil samples were collected at a depth of approximately 20 centimeters (eight inches) b.g.s., although shallower samples were obtained when rock was encountered during sampling. The samples were typically collected from undisturbed locations but included some cultivated fields. Properties were not designated for soil sampling using a probabilistic approach. Instead, USGS staff collected samples of convenience while attempting to achieve a desired distance between sampling sites. The investigators noted that samples were often collected along newly constructed thruways (i.e., within 100 meters of roadways).

2. RSBC Values

Arsenic. Five surveys indicated 98th percentile arsenic concentrations ranging from 14.1 ppm to 19.1 ppm. Due to limited reporting or small sample sizes, three 98th percentile values (19, 19.1 and 16.0 ppm) were survey maxima. The survey data are summarized in Table 9.1-3.

The lowest value (14.1 ppm) was derived from the Rural Survey data, which excluded samples taken at orchards. However, arsenical pesticides such as lead arsenate were historically used at some orchards in rural New York State, and some former orchards have been developed as residential subdivisions. Had we included data from these orchard samples in the Rural Survey data set, the 98th percentile arsenic concentration would have increased to 17.1 ppm. Al-Wardy did not report 98th percentile elemental concentrations, but reported a maximum arsenic concentration (100th percentile) of 19.1 ppm for 51 surface soil samples collected from rural agricultural and forest lands. Clarke et al. reported a similar maximum (19 ppm) for 11 soil samples from rural lawns.

Arsenic concentrations will be higher in soils derived from arsenical rock. Soil surveys that included areas of known mineral deposits may therefore produce arsenic concentration distributions that are higher relative to statewide arsenic levels. Arsenical mineral deposits are known in some of the former iron mining areas in New York State, including the Edenville deposit (Orange County) and the Putnam County Mining Corp. property (Putnam County).[5] The NYS DEC survey provided data on arsenic concentrations in surface soils of the lower Hudson Valley, including Putnam County, an area known for mineral deposits containing arsenic, and reported a 98th percentile arsenic value of 17.7 ppm. The NYS DEC data are useful in that they, at least in part, may include naturally occurring elevated levels of arsenic in rural settings. However the NYS DEC survey only included one of the five Rural Survey regions. Also, fewer lots were sampled (20 vs. 119) and a smaller number of samples were collected (60 vs. 265) compared with the Rural Survey.

[5] Mineral deposit information obtained from mindat.org (http://www.mindat.org), accessed July 19, 2005.

The Shacklette and Boerngen (1984) survey included fewer samples than the NYS DEC Region 3 survey (25 vs. 60), but it covered all five of the regions represented by the Rural Survey. The sampling depth (about 20 centimeters b.g.s) was somewhat greater than either the NYS DEC survey (zero to 15 centimeters b.g.s) or the Rural Survey (zero to 5 centimeters b.g.s. for source-distant and near source samples, zero to 15 centimeters b.g.s. for remote samples). The 98th percentile value from the Shacklette and Boergnen survey (16.0 ppm) fell between the 98th percentile values from the Rural Survey (14.1 ppm excluding orchards and 17.1 ppm including orchards).

The Al-Wardy (2002) data reflect samples collected from agricultural and forest areas, and how well the sample locations represent areas of typical human contact is uncertain. Also, these samples were collected at a depth (23 to 25 centimeters b.g.s.), greater than any of the other survey samples. The maximum value (98th percentile value not reported) from this survey (19.1 ppm) was higher than 98th percentile values from the other surveys. The Clark et al. (1985) survey yielded a similar 98th percentile value (19.0) ppm. However, the Clarke et al. survey collected only 11 samples from five locations, so confidence in the survey results is low.

Giving greater weight to the Rural Survey (2005), the NYS DEC (2003) survey, and the Shacklette and Boerngen (1984) survey, the above information suggests that an RSBC of 14.1 to 17.7 is reasonable for arsenic. An RSBC value of 16 ppm is established for arsenic.

Barium. Three rural surface soil surveys included data for barium. The data are summarized in Table 9.1-3. The 98th percentile concentration values from these surveys range from 187 ppm to 500 ppm. Due to small sample size, one 98th percentile value (500 ppm) was a survey maximum. The survey data are summarized in Table 9.1-3.

The lowest value was derived from data on elemental concentrations in 60 Lower Hudson Valley soil samples collected at 20 public parks by NYS DEC Region 3 staff. The highest value was the maximum concentration from data on 25 soil samples collected by USGS staff (Shacklette and Boerngen, 1984). The Rural Survey 98th percentile concentration (278 ppm) fell between these values.

Shacklette and Boerngen (1984) reported a substantially higher median barium concentration than that reported for shallower soils collected by the Rural Survey (300 ppm vs. 64 ppm). Like the Rural Survey, the Shacklette and Boerngen findings were based on statewide sampling. However, the manner in which Shacklette and Boerngen reported barium concentrations, rounded to the nearest 100 ppm versus 1 ppm or less for other surveys, makes it somewhat difficult to directly compare these results to the results of other surveys. The Rural Survey was considerably larger than the other surveys in terms of sample numbers and parcels sampled, so the Rural Survey receives greater weight when establishing an RSBC. However, some potential remains for the Rural Survey data to underestimate concentrations of barium in some mineral soils. Based on these considerations, an RSBC of 350 ppm is established for barium.

Cadmium. Four rural surface soil surveys provided 98th percentile cadmium concentrations ranging from 1.0 ppm to 2.7 ppm. Due to limited reporting and small sample size, two 98^{th} percentile values (0.9 and 1.0 ppm) were survey maxima. The data are summarized in Table 9.1-3.

The lowest value was the maximum concentration reported for 11 rural lawn soil samples collected from five locations by Clarke et al. (1985). The highest value was derived from the Rural Survey results. The 98th percentile value from the NYS DEC Region 3 survey was 1.2 ppm. A fourth survey that evaluated only Western/Central New York agricultural and forest soils (Al-Wardy, 2002) did not report 98th percentile elemental concentrations, but reported a maximum cadmium concentration (100th percentile) of 0.9 ppm for 51 surface soil samples collected from rural agricultural and forest lands.

Only the Rural Survey obtained statewide coverage and included sampling of roadside soils (which are points of possible human contact). In addition, the Rural Survey included considerably more soil samples, regions and properties than the other surveys. Therefore, the Rural Survey is given greater weight in establishing the RSBC for cadmium. Based on these considerations, an RSBC of 2.5 ppm is established for cadmium.

Chromium (III). No survey data were identified reflecting concentrations of the two Priority List chromium species, chromium (III) and chromium (VI), in rural New York State surface soils. In establishing an RSBC for chromium (III) the Department considered survey data reflecting levels of total chromium in soils. The assumption that most chromium in rural soils is in the trivalent form is supported by studies indicating that trivalent chromium is environmentally stable and the most commonly occurring natural form (ATSDR, 2000). The Department did not establish an RSBC for chromium (VI) because hexavalent chromium is not a commonly occurring natural form (ATSDR, 2000).

Five soil surveys provided 98th percentile chromium concentrations ranging from 22.8 to 100 ppm. Three of the 98th percentile values (18.3, 72 and 100 ppm) for chromium were survey maxima, and the highest of these (72 and 100 ppm) were derived from small data sets (n=11 and 25, respectively). The survey data are summarized in Table 9.1-3.

The lowest value (18.3 ppm) was derived from 51 surface soil samples collected from rural agricultural and forest lands in two regions of New York State by Al-Wardy (2002). The highest value (100 ppm) was derived from the Shacklette and Boerngen (1984) survey, which included 25 soil samples collected from all five regions of New York State. The NYS DEC Region 3 survey, which included 60 samples collected from 20 properties in the lower Hudson Valley, reported a 98th percentile value of 39.1 ppm. Clarke et al. (1985) reported a 100th percentile (maximum value) of 72 ppm from data on 11 rural lawn soil samples collected from five locations. The 98th percentile value reported in the Rural Survey was 22.8 ppm.

The Rural Survey was considerably larger than the other surveys in terms of sample size and parcels sampled, so it receives the greatest weight when establishing an RSBC. The 98th percentile value from the Shacklette and Boerngen survey, which also demonstrated statewide coverage, is considerably higher than the Rural Survey value, but is based on only 25 soil samples. The Clarke et al. survey also reported a considerably higher value than the Rural Survey, but this survey is given little weight due to its small sample size. The Al-Wardy survey reported a value similar to that of the Rural Survey, but the NYS DEC data lend support to the

selection of a somewhat higher RSBC. Given these considerations, an RSBC of 30 ppm is established for chromium.

Manganese. Three rural surface soil surveys obtained statewide coverage and provided 98th percentile manganese concentrations of 1,930 and 2,000 ppm. The lower value was derived from the Rural Survey results. The higher value was derived from the Shacklette and Boerngen survey (1984). A third survey that evaluated only Western/Central New York agricultural and forest soils (Al-Wardy, 2002) did not report 98th percentile elemental concentrations, but reported a maximum manganese concentration (100th percentile) of 2,285 ppm for 51 surface soil samples collected from rural agricultural and forest lands.

As both the Rural Survey and Shacklette and Boerngen survey obtained statewide coverage, they are given greater weight than the Al-Wardy data. The 98th percentile/maximum values from Shacklette and Boerngen and Al-Wardy are higher than the 98th percentile value from the Rural Survey. Based on these considerations, an RSBC of 2,000 ppm is established for manganese.

Mercury (Inorganic). Health-based and groundwater protection SCOs for elemental mercury were above the background screening value for mercury, so we did not evaluate an RSBC for elemental mercury. Candidate SCOs for inorganic mercury were below the background screening value, so we evaluated an RSBC for inorganic mercury.

No survey data were identified reflecting concentrations of inorganic mercury in rural New York State surface soils, so in establishing an RSBC for inorganic mercury, the Department considered survey data reflecting levels of total mercury in soils.

Concentrations of mercury differed among the three Rural Survey sample types (near source, source-distant and remote) and the differences were statistically significant (KW p = 0.01, see Table 9.1-4). Data for remote samples were not used to evaluate an RSBC for inorganic mercury to ensure that the RSBC would be based on analyte concentrations in soils at points of human contact.

Three of the rural surface soil surveys that we identified included data for total mercury. The data are summarized in Table 9.1-3. The 98th percentile concentration values from these surveys were 0.28, 0.60 and 0.69 ppm. The lowest value (0.28 ppm) was derived from 146 Rural Survey samples. The highest value (0.69 ppm) was derived from data on metal concentrations in 60 Lower Hudson Valley soil samples collected at 20 public parks by NYS DEC Region 3 staff. Shacklette and Boerngen reported a 98th percentile concentration of 0.60, derived from data on 25 soil samples collected across New York State.

The Shacklette and Boerngen (1984) survey included fewer samples, and the NYS DEC Region 3 survey considered only one of the five regions represented by the Rural Survey. The Rural Survey was considerably larger than the other surveys in terms of sample size and parcels sampled, so it receives the greatest weight when establishing an RSBC. Based on these considerations, an RSBC of 0.3 ppm is established for inorganic mercury.

***Selenium*.** Four rural surface soil surveys provided 98th percentile selenium concentrations ranging from <0.1 ppm to 4.4 ppm. Table 9.1-3 contains a summary of the data. The lowest value was derived from data on 11 rural lawn soil samples collected from five locations by Clarke et al. (1985). The highest value was derived from the Rural Survey results. A 98th percentile value of 0.64 ppm was derived from the 25 soil samples of the Shacklette and Boerngen survey (1984). The 98th percentile value from the NYS DEC Region 3 survey was 2.6 ppm.

As the Rural Survey included considerably more soil samples, regions and properties than any other survey, it receives the greatest weight when deriving the RSBC for selenium. However, some potential remains for the Rural Survey data to overestimate concentrations of selenium in some mineral soils. This potential overestimation is indicated by the low concentration obtained from the Shacklette and Boerngen survey, which also demonstrated statewide coverage and thus is given greater weight than the Clarke or DEC Region 3 surveys. Nevertheless, the DEC Region 3 survey data supported the selection of a higher RSBC than indicated by the Clarke et al. or Shacklette and Boerngen data sets. Given these considerations, and giving the greatest weight to the Rural Survey value, an RSBC of 4 ppm is established for selenium.

9.1.5.2 RSBCs for PAHs

1. Data Sources

The review process identified only the Rural Survey as a source of information regarding concentrations of PAHs in rural New York State soils. Focus PAHs were detected in very few remote soil samples, and were rarely detected in source-distant soil samples, so the statistical procedure previously employed to evaluate differences in metals concentrations was not used. Instead, all concentration data for a focus PAH were combined, and the 95th percentile value was determined. Then, the Cochran-Armitage Exact Trend Test (SAS version 9.1, SAS Institute, Cary, N.C.) was employed to compare frequencies of elevated (> 95th percentile) concentrations in the three sample types. For each focus PAH, frequencies of elevated concentrations followed the same trend of near source>source-distant>remote, and the trend was statistically significant (p = 0.01 to 0.03).[7] PAH concentration data from remote soil samples were not considered in establishing RSBCs for PAHs because PAH concentrations in remote soils differed significantly from levels in source-distant and near source soils, and remote soils contribute relatively little to peoples' overall exposure to soil.

PAHs are components of road dust, vehicle exhaust, tire wear particles, pavement, and various other petroleum and combustion products (Pengchai et al., 2005). Some of these may be sources of PAHs in soil. These types of sources are present in rural areas of New York State, and the contribution of these sources to soil PAH levels is probably greater near highways, roads, and driveways than at more distant locations such as backyards and fields (Rural Survey, 2005; Crepineau, 2003). As people in rural areas may have contact with soil near highways, roads, and driveways, the near source samples from the Rural Survey were included in the determination of RSBCs for PAHs.

[7] Concentration data for the focus PAH dibenz[a,h]anthracene were not evaluated due to the low number of detections.

One approach for using the near source data is to use only those data (28 observations). However, this "near source" approach ignores the source distant data (118 observations) which also reflect points of human contact with rural soils. Another approach (the "combined approach") is to pool the near source and source distant data into one combined data set (28 + 118 = 146 observations). Such an approach considers data representative of points of human contact. A third approach is to average the analytical results for the near source and source distant samples collected at 28 properties and to combine those average results with the analytical results for the 90 properties at which only source distant samples were collected (118 observations). This "blended approach" may be reasonable because the average results for the 28 properties at which both types of samples were collected may reflect people's exposure both near and away from roads and driveways.

The source-distant data were not considered alone because they do not include the near source data, which represents points of potential exposure.

Table 9.1-5 summarizes the Rural Survey data with regard to the focus PAH analytes using the three approaches described above. The different approaches generated different 98^{th} percentile concentrations for PAHs, reflecting the different degrees to which the near source data influenced the overall data distributions.

Each approach gives a certain "weight" to the near source data. To further examine the appropriate weight to give to the near source data in deriving the RSBCs, additional data sets were evaluated. Because no other rural PAH survey data were available and because urban soils, like rural soils, are influenced by some of the same nonpoint sources of PAHs, including automobiles, we examined several sets of data on urban PAH levels in soils. These include the "Seneca-Babcock," "Mineral Springs," and "Hickory Woods" data sets.

The Seneca-Babcock and Mineral Springs data are contained in a NYS DOH report that summarizes the results of soil sampling performed in these two Buffalo neighborhoods in 1994 (NYS DOH, 1998). These data are summarized in Table 9.1-6. The Hickory Woods data were obtained by the US EPA and are discussed in an ATSDR report that summarizes the results of

soil sampling performed in this Buffalo subdivision (ATSDR, 2001). The Hickory Woods data set includes PAH concentrations for soil samples collected from yards of older homes built before 1931, and newer homes built after 1989. The analytical data from the Hickory Woods investigations were obtained from the US EPA by the NYS DOH and independently evaluated for this assessment (unpublished data). These data are summarized in Table 9.1-7.

2. RSBC Values

Benz[a]anthracene. As shown in Table 9.1-5, an RSBC based on one of the three approaches described above could range from 730 ppb to 2,900 ppb. The higher end of this range represents the "near source approach" which does not consider points of potential human exposure at source-distant sampling locations. Also, the near source 98th percentile is higher than the 90th percentile values from both the pre-1931 and post-1989 Hickory Woods data (1,490 ppb and 1,035 ppb, respectively), and the averages from both the Seneca-Babcock and Mineral Springs data (2,400 ppb and 1,200 ppb, respectively). Thus, the 98th percentile values for the "blended approach" and/or the "combined approach" are given more weight. The 90th percentile values from the pre-1931 and post-1989 Hickory Woods data, and the Seneca-Babcock and Mineral Springs averages all exceed the 98th percentile from the "blended approach" (730 ppb). The 98th percentile value from the "combined approach" (1,200 ppb) is close to the 90th percentile value from the pre-1931 Hickory Woods data and the 90th percentile from the post-1989 Hickory Woods data. The "combined approach" value is one-half of the Seneca-Babcock average value and the same as the Mineral Springs average value. This information suggests that an RSBC value between the 98th percentile values generated by the "blended approach" and "combined approach" is reasonable. Based on this analysis, an RSBC of 1,000 ppb is established for benz[a]anthracene.

Benzo[a]pyrene. As shown in Table 9.1-5, an RSBC based on one of the three approaches described above could range from 554 ppb to 2,400 ppb. The higher end of this range represents the "near source approach" which does not consider points of potential human exposure at source-distant sampling locations. Also, this near source value is higher than the 90th percentile values from both the pre-1931 and post-1989 Hickory Woods data (1,500 ppb and 995 ppb,

respectively), and the averages from both the Seneca-Babcock and Mineral Springs data (2,300 ppb and 1,100 ppb, respectively). Thus the 98th percentile values from the "blended approach" and/or the "combined approach" are given more weight. The 90th percentile values from the pre-1931 and post-1989 Hickory Woods data, and the Seneca-Babcock and Mineral Springs averages all exceed the 98th percentile from the "blended approach" (554 ppb). Thus a somewhat higher RSBC may be selected. The 98th percentile value from the "combined approach" (1,100 ppb) is close to the 90th percentile value from the post-1989 Hickory Woods data, and is the same as the Mineral Springs average value. This information suggests that an RSBC value between the 98th percentile values generated by the "blended approach" and "combined approach" is reasonable. Based on this analysis, an RSBC of 1,000 ppb is established for benzo[a]pyrene.

Benzo[b]fluoranthene. As shown in Table 9.1-5, an RSBC based on one of the three approaches could range from 640 ppb to 3,300 ppb. The higher end of this range represents the "near source approach" which does not consider points of potential human exposure at source-distant sampling locations. Also, the near source 98th percentile is higher than the 90th percentile values from both the pre-1931 and post-1989 Hickory Woods data (2,300 ppb and 1,550 ppb, respectively), and the averages from both the Seneca-Babcock and Mineral Springs data (2,400 ppb and 1,300 ppb, respectively). Thus, the RSBC derived from the "blended approach" and/or the "combined approach" is given more weight. The 90th percentile values from the pre-1931 and post-1989 Hickory Woods data, and the Seneca-Babcock and Mineral Springs averages all exceed the 98th percentile from the "blended approach" (640 ppb). Thus a somewhat higher RSBC may be selected. The 98th percentile from the "combined approach" (1,200 ppb) is close to the 90th percentile from the post-1989 Hickory Woods data and the Mineral Springs average. This suggests that an RSBC value between the 98th percentile values generated by the "blended approach" and "combined approach" is reasonable. Based on this analysis, an RSBC of 1,000 ppb is established for benzo[b]fluoranthene.

Benzo[k]fluoranthene. As shown in Table 9.1-5, an RSBC based on one of the three approaches could range from 420 ppb to 1,500 ppb. The higher end of this range represents the "near source approach" which does not consider points of potential human exposure at source-distant sampling locations. Also, the near source 98th percentile is higher than the 90th percentile values

from both the pre-1931 and post-1989 Hickory Woods data (1,400 ppb and 796 ppb, respectively) and the average from the Mineral Springs data (980 ppb). Thus, an RSBC derived from the "blended approach" and/or the "combined approach" is given more weight. The 90th percentile values from the pre-1931 and post-1989 Hickory Woods data, and the Seneca-Babcock (2,300 ppb) and Mineral Springs averages all greatly exceed the 98th percentile from the "blended approach" (420 ppb). Thus a higher RSBC may be appropriate. The 98th percentile from the "combined approach" (740 ppb) is close to the 90th percentile from the post-1989 Hickory Woods data and the Mineral Springs average. This suggests that an RSBC value similar to the 98th percentile value generated by the "combined approach" is reasonable. Based on this analysis, an RSBC of 800 ppb is established for benzo[k]fluoranthene.

Chrysene. As shown in Table 9.1-5, an RSBC based on one of the three approaches could range from 657 ppb to 1,300 ppb. The higher end of this range represents the "near source approach" which does not consider points of potential human exposure at source-distant sampling locations. The 90th percentile values from both the pre-1931 and post-1989 Hickory Woods data (2,000 ppb and 1,160 ppb, respectively) as well as the Seneca-Babcock and Mineral Springs averages (2,900 ppb and 1,400 ppb, respectively) all greatly exceed the 98th percentile from the "blended approach" (657 ppb), which suggests that the "combined approach" and/or the "near source approach" should be given more weight. The near source 98th percentile is higher than the 90th percentile from the post-1989 Hickory Woods data and is similar to the average from the Mineral Springs data, but is much lower than the 90th percentile from the pre-1931 Hickory Woods data and the average from the Seneca Babcock data. Based on this comparison, a value somewhat lower than the near source 98th percentile value seems reasonable. Collectively, this information suggests that an RSBC value falling between the combined and near source 98th percentiles may be appropriate. Therefore, an RSBC of 1,000 ppb is established for chrysene.

Dibenz[a,h]anthracene. As shown in Table 9.1-5, an RSBC based on one of the three approaches could range from 47 ppb to less than 110 ppb. The 110 ppb is the highest Method Detection Limit (MDL) reported for a sample; the analyte was not detected in the sample. Unlike the other PAHs discussed in this subsection, dibenz[a,h]anthracene was rarely observed in rural soils. Only two source-distant samples contained detectable concentrations of the

analyte, at concentrations of 47 and 230 ppb. MDLs for the analyte ranged from 10 to 110 ppb. Thus the 98th percentile value for rural soils appears to be within the range of detection limits for the laboratory analytical method employed by the Rural Survey -- a method commonly used during investigations of contaminated sites. Based on these observations, an RSBC equal to the upper limit of MDLs reported for Rural Survey samples rounded to one significant digit (100 ppb) is established for dibenz[a,h]anthracene.

Indeno[1,2,3-cd]pyrene. As shown in Table 9.1-5, an RSBC based on one of the three approaches could range from 315 ppb to 660 ppb. The lower end of this range is the 98th percentile value from the "blended approach." The 90th percentile values for the pre-1931 (1,130 ppb) and post-1989 (700 ppb) Hickory Woods data, as well as the Seneca-Babcock and Mineral Springs averages (1,900 ppb and 970 ppb, respectively) are all higher than the "blended approach" value. The "combined approach" and "near source approach" 98th percentile values are similar (620 ppb and 660 ppb, respectively). Both of these values are similar to the 90th percentile value from the post-1989 Hickory Woods data (700 ppb) and the Mineral Springs average (970 ppb). Collectively, this information suggests that the "combined approach" may be given more weight in selecting an RSBC. Therefore, an RSBC of 500 ppb is established for indeno[1,2,3-cd]pyrene.

9.1.5.3 RSBCs for Other Organic Chemicals

1. Data Sources

The Department identified only the Rural Survey as a source of information regarding concentrations of acetone, aldrin, 1,2-dichloroethane, dieldrin, 1,4-dioxane, 2-methylphenol (*o*-cresol) and phenol and in rural New York State surface soils. These compounds were rarely detected in rural soil samples, so differences among rural soil sample types (*e.g.*, source-distant, near source) were not evaluated. Rather, the full Rural Survey data set was employed to establish RSBCs.

2. RSBC Values

Each of these compounds were detected in 2.4 percent or less of Rural Survey samples, so their 98th percentile concentrations are near or below ranges for limits of detection reported by the Rural Survey. Therefore, RSBCs for these anlaytes are the highest corresponding Method Detection Limits (MDLs) reported by the Rural Survey, rounded to one significant digit (see Table 9.1-8).

9.1.6 Summary of RSBCs

The final RSBCs for the analytes with screening values exceeding candidate SCOs are summarized in Table 9.1-9. SCOs after consideration of health risk, groundwater protection and RSBCs are summarized in Table 9.1-10.

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Table 9.1-1. Screening Values for SCO Priority List Analytes.

Organic Analyte	*n*	Screening Value	Units
Acenaphthene	146	100	ppb
Acenaphthylene	146	120	ppb
Acetone	146	280*	ppb
Aldrin	146	<4.8	ppb
Anthracene	146	180	ppb
Benzene	146	<0.92	ppb
Benz[a]anthracene	146	1,200	ppb
Benzo[a]pyrene	146	1,100	ppb
Benzo[b]fluoranthene	146	1,200	ppb
Benzo[k]fluoranthene	146	740	ppb
Benzo[g,h,i]perylene	146	550	ppb
n-Butylbenzene	146	3.3	ppb
sec-Butylbenzene	146	<1.1	ppb
tert-Butylbenzene	146	<1.3	ppb
Carbon Tetrachloride	146	<1.4	ppb
alpha-Chlordane	146	<7.0	ppb
Chlorobenzene	146	<1.6	ppb
Chloroform	146	<1.1	ppb
Chrysene	146	850	ppb
4,4-DDD	146	<4.9	ppb
4,4-DDE	146	<5.8	ppb
4,4-DDT	146	<8.4	ppb
Dibenz[a,h]anthracene	146	47	ppb
Dibenzofuran	146	93	ppb
1,2-Dichlorobenzene	146	<1.9	ppb
1,3-Dichlorobenzene	146	<0.96	ppb
1,4-Dichlorobenzene	146	<1.6	ppb
1,1-Dichloroethane	146	<1.6	ppb
1,2-Dichloroethane	146	<14.0	ppb
1,1-Dichloroethene	146	<0.98	ppb
cis-1,2-Dichloroethene	146	<1.6	ppb
trans-1,2-Dichloroethene	146	<1.7	ppb
Dieldrin	146	<4.6	ppb
1,4-Dioxane	145	<91	ppb
Endosulfan I	146	<7.0	ppb
Endosulfan II	146	<6.0	ppb
Endosulfan Sulfate	146	<6.7	ppb
Endrin	146	<8.3	ppb
Ethyl Benzene	146	<1.1	ppb

Table 9.1-1. Screening Values for SCO Priority List Analytes (continued).

Organic Analyte (contd.)	*n*	Screening Value	Units
Fluoranthene	146	2,000	ppb
Fluorene	146	130	ppb
Heptachlor	146	<6.0	ppb
Hexachlorobenzene	146	<29.0	ppb
beta-BHC	146	<5.2	ppb
delta-BHC	49	<4.2	ppb
gamma-BHC	146	<5.5	ppb
alpha-BHC	146	<5.1	ppb
Indeno[1,2,3-cd]pyrene	146	620	ppb
Methyl tert-butyl Ether	146	<1.0	ppb
Methylene Chloride	146	6.5*	ppb
2-Butanone	146	<10.0	ppb
2-Methylphenol	146	<99	ppb
4-Chloro-3-methylphenol	146	<47	ppb
Naphthalene	146	24.0	ppb
Pentachlorophenol	146	<49	ppb
Phenanthrene	146	1,100	ppb
Phenol	146	100*	ppb
Pyrene	146	2,800	ppb
Tetrachloroethene	146	55*	ppb
Toluene	144	1.2	ppb
1,1,1-Trichloroethane	146	<1.2	ppb
Trichloroethene	146	<1.5	ppb
1,2,4-Trimethylbenzene	146	<1.9	ppb
1,3,5-Trimethylbenzene	146	<1.3	ppb
Vinyl Chloride	146	<1.1	ppb
m/*p*-Xylenes	146	2.7	ppb
o-Xylene	146	<2.1	ppb
Inorganic Analyte	***n***	**Screening Value**	**Units**
Arsenic	146	14.1	ppm
Barium	146	312	ppm
Beryllium	146	1.2	ppm
Cadmium	146	2.7	ppm
Chromium (Total)	146	22.0	ppm
Copper	146	61	ppm
Cyanide	146	<2.3	ppm
Lead	146	101	ppm
Manganese	146	1,760	ppm

Table 9.1-1. Screening Values for SCO Priority List Analytes (continued).

Inorganic Analyte (cont'd)	*n*	Screening Value	Units
Mercury	146	0.28	ppm
Nickel	146	29.5	ppm
Selenium	146	5.7	ppm
Silver	146	1.3	ppm
Zinc	146	180	ppm

Notes:

ppm = mcg/g; ppb = mcg/kg

Concentrations of SCO Priority List analytes 3-methylphenol (*m*-cresol), 4-methylphenol (*p*-cresol) and *n*-propylbenzene were not determined in rural survey samples.

Sample-specific Method Detection Limits were used for ranking purposes when analytes were not detected in a soil sample.

*Screening value may reflect laboratory or field contamination of some samples.

Table 9.1-2. Comparison of Screening Values to Health- and Groundwater Protection-Based SCOs.

Notes: Concentrations expressed in mcg/g (ppm). Bold SCOs were exceeded by screening values.

Analyte	Screening Value	Unrestricted	Residential	Restricted Residential	Commercial	Industrial	Ground-water
arsenic	14.1	**0.11**	**0.21**	**1.0**	**5.9**	**12**	15
barium	312	**72**	**140**	400(1)	400(1)	27,000	820
cadmium	2.7	**0.43**	**0.86**	4.3	9.3(1)	60	7.5
chromium (III)	22(2)	**18**	36	180	1,500	6,800	NS
chromium (VI)	22(2)	**11**	22	110	400	800	**19**
manganese	1,760	**180**	**360**	1,800	15,000	67,000	**390**
mercury (inorganic)	0.28(3)	**0.12**	1.2	5.8	47	220	NS
selenium	5.7	18	36	180	1500	6800	**1**
benz[a]anthracene	1.200	**0.052**	**0.1**	**0.39**	5.6	11	**0.52**
benzo[a]pyrene	1.100	**0.0011**	**0.01**	**0.039**	**0.56**	1.1	22
benzo[b]fluoranthene	1.200	**0.052**	**0.1**	**0.39**	5.6	11	1.7
benzo[k]fluoranthene	0.740	**0.52**	1	3.9	56	110	1.7
chrysene	0.850	**0.52**	1	3.9	56	110	**0.59**
dibenz[a,h]anthracene	0.047	**0.0052**	**0.01**	**0.039**	0.56	1.1	89,500
indeno[1,2,3-cd]pyrene	0.620	**0.052**	**0.1**	**0.39**	5.6	11	8.2
acetone	0.280	3,200	6,300	29,000	260,000	830,000	**0.05**
aldrin	<0.0048	**0.0019**	0.019	0.097	0.68	1.4	19
1,2-dichloroethane	<0.014	1.7	2.3	3.1	30	60	**0.01**
dieldrin	<0.0046	**0.0039**	0.039	0.2	1.4	2.8	0.1
1,4-dioxane	<0.091	7.3	9.8	13	130	250	**0.04**
2-methylphenol	<0.099	180	340	1,500	8,700	41,000	**0.05**
phenol	0.100	800(1)	800(1)	800(1)	800(1)	250,000	**0.02**

(1) Indicates SCO based on acute health effects.
(2) Indicates screening value for total chromium.
(3) Indicates screening value for total mercury.

Table 9.1-3. Summary Statistics for Five Elements in Selected Surveys of Rural New York State Soils.

Arsenic Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey (2005)[2]	265	119	5	<0.25 - 68.9	14.1
NYS DEC Region 3 (2003)	60	20	1	2.2 - 23.1	17.7
Al-Wardy (2002)	51	51	2	1.3 - 19.1	19.1[3]
Clarke *et al*. (1985)	11	5	3	3.4 - 19	19.0[4]
Shacklette and Boerngen (1984)	25	25	5	1.5 - 16.0	16.0[4]

Barium Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey (2005)[2]	265	119	5	4.5 - 743	278
NYS DEC Region 3 (2003)	60	20	1	38.5 - 1,060	187
Shacklette and Boerngen (1984)	25	25	5	200 - 500	500[4]

Cadmium Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey (2005)[2]	265	119	5	<0.05 - 4.15	2.7
NYS DEC Region 3 (2003)	60	20	1	<0.04 - 9.2	1.2
Al-Wardy (2002)	51	51	2	0.11 - 0.88	0.9[3]
Clarke *et al*. (1985)	11	5	3	<0.4 - 1.0	1.0[4]

Chromium Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey (2005)[2]	265	119	5	0.9 – 36.0	22.8
NYS DEC Region 3 (2003)	60	20	1	11.2 – 51.2	39.1
Al-Wardy (2002)	51	51	2	5.7 – 18.3	18.3[3]
Clarke *et al*. (1985)	11	5	3	13 – 72	72[4]
Shacklette and Boerngen (1984)	25	25	5	15 - 100	100[4]

Manganese Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey (2005)[2]	265	119	5	12.6 - 4,550	1,930
Al-Wardy (2002)	51	51	2	146 - 2,285	2,285[3]
Shacklette and Boerngen (1984)	25	25	5	70 - 2,000	2,000[4]

Table 9.1-3. Summary Statistics for Five Elements in Selected Surveys of Rural New York State Soils (continued).

Mercury Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey Non-Remote[2]	146	118	5	<0.01 – 0.34	0.28
NYS DEC Region 3 (2003)	60	20	1	0.04 – 0.92	0.69
Shacklette and Boerngen (1984)	25	25	5	0.03 – 0.60	0.60[4]

Selenium Data Set	Samples Collected	Lots Sampled	Regions Covered[1]	Range (ppm)	98th Percentile (ppm)
Rural Survey (2005)[2]	265	119	5	<0.36 - 6.5	4.4
NYS DEC Region 3 (2003)	60	20	1	0.04 – 2.9	2.6
Clarke *et al*. (1985)	11	5	3	<0.1 - <0.1	<0.1[4]
Shacklette and Boerngen (1984)	25	25	5	<0.1 – 0.64	0.64[4]

Notes:

(1) Number of Rural Soil Survey regions represented by survey samples (see Appendix D).

(2) Rural Survey summaries exclude analytical data for four samples collected at orchards.

(3) The maximum observed (100th percentile) concentration was employed because it was the nearest percentile to the 98th that was reported by the investigator.

(4) The 98th percentile is the maximum observed concentration because the investigators reported fewer than 50 observations.

Table 9.1-4. Statistical Comparison of Focus Metal Concentrations in Three Types of Rural Survey Samples.

Note: Concentrations expressed in mcg/g (ppm).

	As	Ba	Cd	Cr	Hg	Mn	Se
Near Source median	3	63	0.4	11	0.04	426	1.7
Source-Distant median	5	67	0.4	11	0.05	466	1.9
Remote median	5	61	0.4	11	0.06	467	2.0
Kruskal-Wallis *p**	*0.69*	*0.45*	*0.60*	*0.96*	*0.01*	*0.87*	*0.52*

* Adjusted for ties.

Table 9.1-5. Summary Statistics for PAHs in Rural Survey Soil Samples.

Note: Concentrations expressed in mcg/kg (ppb).

Benz[a]anthracene	*n*	Range (ppb)	98th Percentile
Blended[1]	118	<5.2 - 1,454	730
Combined	146	<5.2 - 2,900	1,200
Near Source	28	<5.8 - 2,900	2,900

Benzo[a]pyrene	*n*	Range	98th Percentile
Blended[1]	118	<5.9 - 1,704	554
Combined	146	<5.9 - 3,400	1,100
Near Source	28	<6.6 - 2,400	2,400

Benzo[b]fluoranthene	*n*	Range	98th Percentile
Blended[1]	118	<18 - 2,312	640
Combined	146	<18 - 4,600	1,200
Near Source	28	<20 - 3,300	3,300

Benzo[k]fluoranthene	*n*	Range	98th Percentile
Blended[1]	118	<12 - 856	420
Combined	146	<12 – 1,700	740
Near Source	28	<13 – 1,500	1,500

Chrysene	*n*	Range	98th Percentile
Blended[1]	118	<11 – 1,207	657
Combined	146	<11 – 2,400	850
Near Source	28	<12 – 1,300	1,300

Dibenz[a,h]anthracene	*n*	Range	98th Percentile
Blended[1]	118	<10 - 122	47
Combined	146	<10 - 230	47
Near Source	28	<11 - <110	<110[2]

Indeno[1,2,3-cd]pyrene	*n*	Range	98th Percentile
Blended[1]	118	<8.3 - 706	315
Combined	146	<8.3 - 1,400	620
Near Source	28	<9.2 - 660	660

[1] The “blended approach” substituted one-half the sample-specific Method Detection Limit when analytes were not detected in a soil sample.

[2] Analyte was not detected in near source samples (Method Detection Limit range: 11 to 110 ppb).

Table 9.1-6. Summary Statistics for Seneca-Babcock and Mineral Springs PAH Data.

Note: Concentrations expressed in mcg/kg (ppb). Observations below the reporting limit were assigned a value of zero ppb and were not used to calculate minima and averages.

Seneca-Babcock	B[a]A	B[a]P	B[b]F	B[k]F	Chr	D[a,h]A	IP
Maximum	7700	6900	7000	6300	8200	780	6300
Minimum Detection	290	320	330	340	460	38	250
Average Detection	2400	2300	2400	2300	2900	270	1900
# Detects	24	24	24	24	24	22	24
% Detects	100	100	100	100	100	92	100
Mineral Springs	**B[a]A**	**B[a]P**	**B[b]F**	**B[k]F**	**Chr**	**D[a,h]A**	**IP**
Maximum	3400	3100	3900	2400	3900	400	2800
Minimum Detection	90	92	150	97	150	190	92
Average Detection	1200	1100	1300	980	1400	300	970
# Detects	9	9	9	9	9	3	9
% Detects	100	100	100	100	100	33	100

B[a]A = Benz[a]anthracene
B[a]P = Benzo[a]pyrene
B[b]F = Benzo[b]fluoranthene
B[k]F = Benzo[k]fluoranthene
Chr = Chrysene
D[a,h]A = Dibenz[a,h]anthracene
IP = Indeno[1,2,3-cd]pyrene

Table 9.1-7. Summary Statistics for Hickory Woods PAH Data.

Note: Concentrations expressed in mcg/kg (ppb).

Pre-1931	**B[a]A**	**B[a]P**	**B[b]F**	**B[k]F**	**Chr**	**D[a,h]A**	**IP**
Maximum	3700	3800	3700	3800	4400	1300	2900
Minimum	170	190	20*	21*	210	64	180
Average	820	819	1130	749	1028	251	659
Geometric Mean	664	669	855	476	872	212	552
Standard Deviation	605	615	800	648	706	179	466
50^{th} Percentile	653	665	860	600	790	210	545
75^{th} Percentile	1000	1000	1425	900	1300	283	780
90^{th} Percentile	1490	1500	2300	1400	2000	416	1130
# Detects	68	68	66	55	61	68	68
% Detects	100	100	97.1	92.7	100	100	100
Post-1989	**B[a]A**	**B[a]P**	**B[b]F**	**B[k]F**	**Chr**	**D[a,h]A**	**IP**
Maximum	7800	5500	7200	3000	6000	710	2500
Minimum	15*	17*	20*	21*	21*	22*	22*
Average	467	420	560	634	537	104	276
Geometric Mean	166	157	225	158	232	50	118
Standard Deviation	987	802	1081	558	933	156	439
50^{th} Percentile	135	135	195	130	190	22*	89
75^{th} Percentile	405	388	560	390	550	118	264
90^{th} Percentile	1035	995	1550	796	1160	265	700
# Detects	97	93	103	72	77	45	84
% Detects	91.5	87.7	97.2	91.1	97.5	42	79.3

For all calculations, observations below the reporting limit were assigned a value of one-half an assigned chemical-specific detection limit (CSDL). The CSDL is indicated below (within parentheses).

B[a]A = Benz[a]anthracene (30 ppb)
B[a]P = Benzo[a]pyrene (33 ppb)
B[b]F = Benzo[b]fluoranthene (40 ppb)
B[k]F = Benzo[k]fluoranthene (42 ppb)
Chr = Chrysene (41 ppb)
D[a,h]A = Dibenz[a,h]anthracene (43 ppb)
IP = Indeno[1,2,3-cd]pyrene (44 ppb)

* Analyte was not detected in the soil sample. Indicated value is one-half the CSDL.

Table 9.1-8. Method Detection Limit Ranges and RSBCs for Organic Compounds Other Than Polycyclic Aromatic Hydrocarbons.

Note: Values are expressed in mcg/g (ppm).

Compound	MDL Range	RSBC
acetone	0.0078 - 0.036	0.04
aldrin	0.0011 - 0.0050	0.005
1,2-dichloroethane	0.0032 - 0.0015	0.002
dieldrin	0.0010 - 0.0048	0.005
1,4-dioxane	0.021 - 0.095	0.1
2-methylphenol	0.022 - 0.240	0.2
phenol	0.014 - 0.160	0.2

Table 9.1-9. Rural Soil Background Concentrations (RSBCs).

Note: Values are expressed in mcg/g (ppm).

Analyte	RSBC
arsenic	16
barium	350
cadmium	2.5
chromium (III)	30
chromium (VI)	NE
manganese	2,000
mercury (inorganic)	0.3
selenium	4
benz[a]anthracene	1.0
benzo[a]pyrene	1.0
benzo[b]fluoranthene	1.0
benzo[k]fluoranthene	0.8
chrysene	1.0
dibenz[a,h]anthracene	0.1
indeno[1,2,3-cd]pyrene	0.5
acetone	0.04
aldrin	0.005
1,2-dichloroethane	0.02
dieldrin	0.005
1,4-dioxane	0.01
2-methylphenol	0.2
phenol	0.2

NE = “Not established”

Table 9.1-10. SCOs After Consideration of Health Risk, Groundwater Protection and Rural Soil Background Concentrations.

Notes: Concentrations expressed in mcg/g (ppm). Bold SCOs are RSBCs.

Analyte	RSBC	Unrestricted	Residential	Restricted Residential	Commercial	Industrial	Ground-water
arsenic	16	**16**	**16**	**16**	**16**	**16**	**16**
barium	350	**350**	**350**	400(1)	400(1)	27,000	820
cadmium	2.5	**2.5**	**2.5**	4.3	9.3(1)	60	7.5
chromium (III)	30	**30**	36	180	1,500	6,800	NE
chromium (VI)	NE	11	22	110	400	800	19
manganese	2,000	**2,000**	**2,000**	**2,000**	15,000	67,000	**2,000**
mercury (inorganic)	0.3	**0.3**	1.2	5.8	47	220	NE
selenium	4.0	18	36	180	1500	6800	**4.0**
benz[a]anthracene	1.0	**1.0**	**1.0**	**1.0**	5.6	11	**1.0**
benzo[a]pyrene	1.0	**1.0**	**1.0**	**1.0**	**1.0**	1.1	22
benzo[b]fluoranthene	1.0	**1.0**	**1.0**	**1.0**	5.6	11	1.7
benzo[k]fluoranthene	0.8	**0.8**	1	3.9	56	110	1.7
chrysene	1.0	**1.0**	1	3.9	56	110	**1.0**
dibenz[a,h]anthracene	0.1	**0.1**	**0.1**	**0.1**	0.56	1.1	89,500
indeno[1,2,3-cd]pyrene	0.5	**0.5**	**0.5**	**0.5**	5.6	11	8.2
acetone	0.04	3,200	6,300	29,000	260,000	830,000	0.05
aldrin	0.005	**0.005**	0.019	0.097	0.68	1.4	19
1,2-dichloroethane	0.02	1.7	2.3	3.1	30	60	**0.02**
dieldrin	0.005	**0.005**	0.039	0.2	1.4	2.8	0.1
1,4-dioxane	0.01	7.3	9.8	13	130	250	0.04
2-methylphenol	0.2	180	340	1,500	8,700	41,000	**0.2**
phenol	0.2	800(1)	800(1)	800(1)	800(1)	250,000	**0.2**

(1) Indicates SCO based on acute health effects.
NE = "Not established"

9.2 Background Soil Concentrations (Ecological)

In establishing a Rural Soil Background Concentration for protection of ecological resources (RSBC-ER), only the data from the Rural Survey "habitat" or "remote" samples were used because the survey was specifically designed to sample habitat areas in proximity to human activity rather than pristine or wilderness habitat areas. Such a landscape condition approximates the likely scenario for habitat existing in proximity to a brownfield site. Data were available for 121 habitat area samples. Following a quality review of sampling locations and analytical data, the final habitat area data set consisted of data from 96 samples for estimation of the rural soil background concentrations for habitat areas. The protocol for the quality review of habitat area samples as well as descriptive statistics for the final 3 organic and 12 inorganic analytes is presented in Appendix D *Habitat Area Sampling Protocol, Quality Review, and Data Analysis* of the Summary Report on the Rural Survey (Appendix D).

9.2.1 Selecting the Rural Soil Background Concentration for Ecological Resources

The 95th percentile analyte concentrations from the Rural Survey habitat area data set were compared to the ecological soil cleanup objectives (ESCOs) to determine which rural soil background concentrations might exceed the risk-based cleanup objectives. Establishing a risk-based cleanup value inherently implies that contaminant concentrations above that value are toxic, and contaminant concentrations below it are not. Because the toxicity of soil-borne contaminants to organisms varies considerably with the characteristics of the substrate, the overall ecological condition of the sample location, and the variability of the exposed organisms themselves, it is generally not possible to explicitly define the line between toxic and non-toxic concentrations of contaminants. Most often, it is possible to identify a concentration below which the substrate is always non-toxic, and also to identify a concentration above which the substrate is always toxic. This leaves a middle range of concentrations between these two points that elicit a mix of toxic and non-toxic responses.

When deriving the ESCO values, the NYS DEC strove to minimize the likelihood of over-predicting soil toxicity by using LOELs rather than NOELs. The LOEL generally allows for a risk of about a 10 to 20% chance of a toxic response occurring at that particular soil

concentration. This allowance of risk reduces the likelihood of unnecessarily remediating a site that is not toxic; however, it increases the possibility that some soil that is toxic might not be remediated.

A similar approach was taken in establishing rural soil background concentrations to replace risk-based ESCOs. At the 95th percentile of the analyte distributions, there is some chance that a non-contaminated sample might be identified as contaminated, but that chance must be balanced against the risk of increasing toxicity above the 10 to 20% already inherent in the ESCO. The chance of either error occurring can be minimized by adequately sampling and characterizing the contaminated site.

Rural soil background concentrations for habitat areas are shown in Table 9.2-1.

9.2.2 Establishing Background-Based ESCOs

Based on the 95th percentile analyte concentrations of SCO Priority List compounds, rural soil background concentrations for six analytes were found to exceed the risk based ESCOs: arsenic, lead, manganese, mercury, selenium, and zinc (Table 9.2-2). Therefore, for these elements, the soil cleanup objectives for protection of ecological resources as shown in the draft proposed 6 NYCRR Part 375-6.8(a) and 375-6.8(b) are based on rural soil background concentrations rather than risk-based considerations.

Table 9.2-1. Rural Soil Background Concentrations for Habitat Areas.

Organic Analyte	*n*	RSBC	Units
Fluoranthene	95	87	ppb
Naphthalene	96	14	ppb
Pyrene	95	170	ppb
Inorganic Analyte	***n***	**RSBC**	**Units**
Arsenic	96	13.0	ppm
Barium	96	176	ppm
Beryllium	96	1.1	ppm
Cadmium	96	2.1	ppm
Chromium (Total)	96	19.1	ppm
Copper	96	33	ppm
Lead	96	63	ppm
Manganese	96	1600	ppm
Mercury	96	0.18	ppm
Nickel	96	25	ppm
Selenium	96	3.9	ppm
Silver	96	0.7	ppm
Zinc	96	109	ppm

Table 9.2-2. ESCOs Replaced by Rural Soil Background Values

SCO Priority List Analyte	Ecological Soil Cleanup Objective (ppm)	Rural Soil Background Concentration (ppm)
Arsenic	10	13
Lead	50	63
Manganese	500	1600
Mercury	0.1	0.18
Selenium	1	3.9
Zinc	50	109

Notes:

ppm = mcg/g; ppb = mcg/kg

9.3 Cap Approach and Values

Some of the factors that are not considered in the equations used to calculate SCOs may have impacts on protection of public health or ecological resources, or they may have impacts on other beneficial or favorable attributes of soil that people value. Some potential chemical impacts to soil that are not quantitatively accounted for in the SCO equations include impacts to soil appearance, texture, odor, and aerability. There may also be other factors that are not accounted for that are more directly related to protection of health and ecological resources.

The equations used to calculate chronic health-based SCOs are only as valid as the assumptions upon which they are based. Inherent assumptions relate to chemical species, contaminant availability, and sorptive limitations of the soil matrix. Some of these assumptions effectively impose boundary conditions upon the applicability of the equations. For example, calculations for the SCOs assume that the contaminant is part of the soil matrix, and this may not be true at soil concentrations that exceed the soil saturation level for the contaminant. In addition to the assumption that soil contaminants are part of the soil matrix, there may be other assumptions that may not be valid under all commonly encountered circumstances. Thus, while the SCO equations incorporate reasonable estimates of exposure and toxicity, they rely upon assumptions that are not universally applicable, and they might not account for all possible conditions or factors that could be important for determination of protective SCOs. In some instances, not accounting for these factors or relying on inappropriate assumptions may result in calculation of SCO values that are unreasonably high. For these reasons, maximum acceptable soil contaminant concentrations - or “caps” - were developed for each land-use category.

Section 27-1415.5 of the Environmental Conservation Law requires removal or control of contaminant sources, which include grossly contaminated soils, NAPLs, and free product, for every site in the Brownfield Cleanup Program. The law also provides a hierarchy of source removal and control measures from the most preferable to the least preferable, with the most preferable being removal and/or treatment. Since the calculated chronic health-based SCOs could allow extremely high levels of some soil contaminants (i.e., gross contamination), the final chronic health-based SCOs are to be limited to maximum acceptable values or caps.

Since the Environmental Conservation Law also provides for land use-based soil cleanup levels, the levels of contaminants that would be considered grossly contaminated will vary by use. Factors that were considered in the determination of these maximum levels include: visual considerations (appearance), olfactory impacts (odor), and saturation levels (C_{sat}), among others. Where calculated SCOs exceed the caps, these caps will be substituted in the Track 1 and 2 SCO tables in Section 3.8 (e) of 6 NYCRR 375-3. The caps are presented in the Table 9.3-1.

These maximum contaminant levels are not to be considered as cleanup levels for the total concentration (summation) of contaminants. For example, 1000 ppm (for industrial land use) is not to be considered an acceptable total concentration for all organic contaminants present at the site, unless other site- or contaminant-specific information supports such a decision. Rather they represent the maximum contaminant level of any individual chemical.

Table 9.3-1. Maximum SCOs for Individual Chemicals

Use of the Site	Maximum SCOs for Individual Organics	Maximum SCOs for Individual Inorganics
Unrestricted	100 ppm	10,000 ppm
Residential	100 ppm	10,000 ppm
Restricted Residential	100 ppm	10,000 ppm
Commercial	500 ppm	10,000 ppm
Industrial	1000 ppm	10,000 ppm
Groundwater	1000 ppm	10,000 ppm
Ecological Resources	100 ppm	10,000 ppm

9.4 Detection Limits

In some cases, the calculated SCOs are below levels at which laboratories can report the results with certainty. In these cases, the calculated values have been replaced with the Contract Required Quantitation Levels (CRQL) that is published in the NYS DEC Analytical Services Protocol (ASP). A CRQL is the reporting limit that an analytical laboratory performing work for NYS DEC must meet. The CRQL corresponds to the lowest concentration level on the analytical method calibration curve (this is described in detail in the ASP). Section 27-1415.6(c) of the Environmental Conservation Law requires that the tables of SCOs be updated every five years. These updates will incorporate improvements in detection and quantitation limits by the laboratories and include revised CRQLS as appropriate.

10.0 Other Considerations

10.1 Vapor Intrusion Pathway

10.1.1 Migration of Soil Contaminants into Indoor Air

Volatile contaminants (e.g., solvents, gasoline, elemental mercury) in subsurface soil may migrate into soil vapor and subsequently contaminate indoor air. Some of these contaminants may leach from soil into groundwater, and then migrate from groundwater into soil vapor and indoor air. In areas where the water table is elevated and in contact with buildings, contaminants in groundwater may volatilize directly into indoor air.

One or more of the aforementioned contaminant migration pathways may need to be considered when soil is contaminated with volatile substances or chemicals. Such pathways are less often a concern when soil is contaminated by SVOCs, although some SVOCs can migrate into indoor air from soil and groundwater. With few exceptions (e.g., elemental mercury), migration of metal contaminants into indoor air is not a concern because most metals are essentially non-volatile.

10.1.2 Existing Structures

Mathematical models have been developed in an attempt to estimate concentrations of contaminants in indoor air resulting from concentrations in soil and groundwater. Although rational, these models are imprecise and limited in the scope of their applications. They are therefore typically used only for screening purposes under suitable conditions.

For example, the US EPA's 2002 *Draft Guidance for Evaluating Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils* describes the use of the agency's vapor intrusion models in a tiered screening approach (US EPA 2002a). The US EPA models are based on those originally developed by Johnson and Ettinger (1991), and may be used to back-estimate levels of contaminants in soil or groundwater from levels in indoor air.

There are, however, several limitations to the US EPA models, which the agency sets forth in Appendix G of its 2002 draft vapor intrusion guidance document. For example, the US EPA cautions that its models may not be applicable when fractures, macropores, wells, tree roots, heterogeneous fill, bedded utilities or other preferential pathways are present in the subsurface. Some of these preferential pathways occur at contaminated sites, but the potential for contaminant transport along these pathways is neglected in the model.

In addition, the US EPA models assume that groundwater table fluctuations are small, with no contamination in the capillary fringe. At many sites in New York State, seasonal groundwater fluctuations are relatively large, creating a “smear zone” of contamination above the water table that increases migration of contaminants into soil vapor.

Furthermore, the US EPA models assume slab-on-grade or basement-type construction, although dirt floors and crawlspaces are common in some parts of New York State. Dirt floors and crawlspaces may be subject to greater infiltration of soil vapor.

Also, for the US EPA approach to yield valid estimates of chemical concentrations in indoor air, building air exchange rates must be sufficiently high (*e.g.*, >0.25/hr). In New York State, "tight buildings" with lower air exchange rates are possible.

Finally, the US EPA models assume that foundations of buildings remain dry (not wetted by groundwater) all year. This last limitation is potentially important in parts of New York State where structures were built in areas of shallow groundwater. Vapor migration models assume a reduction in contaminant levels as vapors move through the subsurface, but such calculations may underestimate indoor air contamination if a structure is in contact with groundwater.

10.1.3 Future Structures

When considering contaminant migration into future structures, the model limitations listed above again restrict applications, and the risk assessor cannot fully employ site-specific parameters that might increase confidence in the model results. For example, subsurface vapor

intrusion models require an estimate of the moisture content of soil under a structure. This is difficult to predict before a building is constructed. Soil under buildings is generally drier than soil around buildings, or soil samples collected before construction. As soil moisture is a highly influential model parameter, a high level of uncertainty concerning soil moisture content will increase uncertainty associated with model output.

10.1.4 US EPA Recommendations for Soil Screening

The US EPA developed generic Soil Screening Levels (SSLs) sometimes used to conduct risk-based screenings of data on contaminant concentrations in soil (US EPA 2002b). These screening levels typically consider exposures due to incidental ingestion, dermal contact, and inhalation. The vapor intrusion pathway was not considered in the development of generic SSLs, but the US EPA acknowledged the potential importance of the vapor intrusion pathway in its SSL guidance, advising:

> *"Because there is substantial variation in the values for the parameters used in the Johnson and Ettinger model, it is very difficult to identify suitable default values for inputs such as building dimensions and the distance between contamination and a building's foundation. As a result, EPA has not developed generic SSLs for this pathway. Instead, managers of sites contaminated with volatiles are encouraged to calculate site-specific SSLs for this pathway using the spreadsheets provided and site-specific values for key input parameters."*

We agree with the US EPA that generic soil values should not assume a completed soil vapor exposure pathway because conditions, and thus appropriate input values, vary greatly from site to site. We add that site-specific application of US EPA vapor intrusion models ("spreadsheets") may not necessarily solve the problem because conditions common in New York State, such as elevated and fluctuating water tables, violate key model assumptions. Furthermore, the substantial uncertainty inherent in model input parameters and estimates, especially when considering hypothetical (future) structures, argues against the use of mathematical models in developing generic SCOs.

10.1.5 Approach

As part of the approach to brownfields and other contaminated sites, the agencies have developed draft guidance on identifying and addressing current and potential human exposures to contaminated subsurface vapors associated with known or suspected volatile chemical contamination (NYS DOH, 2005). This draft guidance, and any subsequent guidance, is to be used to address the vapor intrusion pathway.

References

Johnson PC and Ettinger RA. 1991. Hueristic model for predicting the intrusion rate of contaminant vapors into buildings. Environ. Sci. Technol. 25: 1445-1452.

NYS DOH (New York State Department of Health). 2005. Guidance for Evaluating Soil Vapor Intrusion in the State of New York. Troy, NY: NYS DOH, Center for Environmental Health, Bureau of Environmental Exposure Investigation. Public Comment Draft. February 2005.

US EPA (US Environmental Protection Agency). 2002a. Draft Guidance for Evaluating Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils.

US EPA (US Environmental Protection Agency). 2002b. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, Office of Solid Waste and Emergency Response, OSWER 9355.4-24.

10.2 Protection of Adjacent Residential Uses

Legislation establishing New York State's Brownfield Cleanup Program (Article 27, Title14 of the Environmental Conservation Law) requires the NYS DEC, in consultation with the NYS DOH, to develop regulations that create an approach for the remediation of contamination at brownfield sites. The legislation states that in developing the tables of SCOs, the department shall consider "the protection of adjacent residential uses" (Section 27-1415.6(b)(iii)). As previously described, SCOs have been developed for five land-use categories: unrestricted, residential, restricted residential, commercial, and industrial. The SCOs for one of these categories (unrestricted) are based on consideration of the soil ingestion, dermal absorption, inhalation, and vegetable and animal product consumption exposure pathways in a residential setting. The unrestricted SCOs are inherently protective of adjacent residential uses. Therefore, protection of adjacent residential use only needs to be considered for the residential, restricted residential, commercial, and industrial categories.

The legislation did not define the term "adjacent residential use." The departments consider this term to mean types of land uses included in the unrestricted, residential and restricted residential land use categories that are geographically close enough to brownfield sites in any restricted land use category such that the residential sites could be impacted by the transport of soil contaminants from restricted use sites. For example, chemicals remaining in soil at an industrial site could be transported to an adjacent residential property in wind-blown dust, in rainwater runoff, or in the water of a stream that flows from the industrial property to a nearby residential property. The departments also consider the term "adjacent residential use" to mean land uses included such as day care facilities and schools that are close enough to commercial or industrial facilities at former brownfield sites to be potentially impacted by the transport of any soil contaminants remaining at those sites.

The NYS DEC will select a remedial program for each brownfield site. The legislative definition of remedial program states, in part, that a "remedial program shall mean all remedial activities or actions undertaken to eliminate, remove, treat, abate, control, manage, or monitor hazardous waste or petroleum at or emanating from a brownfield site." In designing/selecting a remedial program for planned residential, restricted residential, commercial, or industrial land

uses, the NYS DEC will include measures to mitigate the transport of soil contaminants from the sites to adjacent residential properties, during and after implementation of the remedial program. Since the selection of a remedial program will address the protection of adjacent residential uses, the SCOs for the residential, restricted residential, commercial, and industrial land-use categories do not need to address off-site exposures to contaminants emanating from those sites. A similar approach will be employed to deal with potential environmental and public health threats which may result from a brownfield site's impact to surface waters. Here also, any potential impact will be addressed in the remedy selection process rather than through modification of the SCOs in the tables.

10.3 Exposure to Residual Contaminants at a Site

The legislation (§ 27-1415.1) states that "In all cases, the target risk of residual contamination at a site shall not exceed an excess cancer risk of one-in-one million for carcinogenic endpoints and a hazard index of one for non-cancer endpoints." As required by § 27-1415.6 of the legislation, SCOs for individual chemicals reflect risk levels that do not exceed one-in-one million for carcinogenic endpoints and a hazard index of one for non-cancer endpoints. When remedial actions are taken to address elevated levels of individual chemicals, such actions will typically reduce the concentrations of those chemicals at the site to levels that are substantially lower than the SCO values (i.e., the remedial actions will reduce the concentrations to more than just below the SCO). Remedial actions to address elevated contaminant levels also will typically result in decreases in the concentrations of other site-related contaminants that did not exceed corresponding SCO values. Therefore, the residual risk (i.e., post-remediation) associated with exposure to residual contaminants at brownfield sites is expected to be below the target risk levels identified in the legislation.

10.4 Cleanup Levels Achieved at Other Sites

Environmental Conservation Law Section 27-1415 requires the NYS DEC to consider the "feasibility of achieving more stringent remedial action objectives, based on experience under the existing state remedial programs, particularly where toxicological, exposure, or other pertinent data are inadequate or nonexistent for a particular contaminant." First, it is noted that where the toxicological, exposure, or other pertinent data were inadequate or nonexistent for a particular contaminant, the Department elected not to develop an SCO for such contaminant. Decision documents contain either a numerical based cleanup objective (e.g., 10 ppm) or a performance based cleanup objective (excavate to a specified depth). In an effort to guide the cleanup objectives, the NYS DEC issued TAGM 4046 in 1992. Since the issuance of TAGM 4046, the majority of decision documents that included numerical cleanup objectives specified the SCOs in TAGM 4046 or higher. In other words, the TAGM 4046 cleanup values would be the lowest numbers contained in a decision document; however, the cleanup number may have been higher based upon site-specific considerations. For some sites, the cleanup number may have been lower to provide protection for ecological resources. It is believed that the ensuing remedial actions generally have achieved those cleanup levels. For purposes of this evaluation, the NYS DEC considered TAGM 4046 as the most stringent cleanup objectives contained in a cleanup decision document under the State Superfund, Environmental Restoration, or Voluntary Cleanup Programs. In addition, the NYS DEC also considered the public health and environmental impacts as well as the feasibility of more stringent alternatives, including the information set forth in this Technical Support Document and the various reference source documents. Based upon these considerations, the NYS DEC has determined that while it may be possible to achieve cleanup values which are more stringent than those set forth in the SCO tables, since both public health and the environment will be protected through the use of the SCOs and more stringent levels will not significantly increase this level of protection, the SCOs set forth in the tables in Section 375-3.8 achieve the objective of Article 27-1415 of the Environmental Conservation Law.

11.0 Final SCO Tables from Part 375

Tables 11-1 and 11-2 show the final SCOs as presented in 6 NYCRR Part 375-6.8. The Unrestricted use values shown in Table 11-1 were derived from the final human-health based SCOs (Table 5.6-1), the groundwater SCOs (Table 7-1) and the ecological SCOs (Table 8.6-1). The lowest of these values was selected as the final SCO, unless a corresponding rural soil background concentration (Tables 9.1-9 and 9.2-1) was higher, in which case the lowest rural soil background concentration was selected as the final SCO. If the final SCO was lower than the CRQL for a chemical (Section 9.4), the CRQL was substituted as the final SCO.

Table 11-1. Final Unrestricted Use SCOs as Presented in 6 NYCRR Part 375-6.8(a).

Unrestricted Use Soil Cleanup Objectives		
Contaminant	**CAS Number**	**Unrestricted Use**
Metals		
Arsenic	7440-38-2	13[c]
Barium	7440-39-3	350[c]
Beryllium	7440-41-7	7.2
Cadmium	7440-43-9	2.5[c]
Chromium, hexavalent[e]	18540-29-9	1[b]
Chromium, trivalent[e]	16065-83-1	30[c]
Copper	7440-50-8	50
Total Cyanide[e,f]		27
Lead	7439-92-1	63[c]
Manganese	7439-96-5	1600[c]
Total Mercury		0.18[c]
Nickel	7440-02-0	30
Selenium	7782-49-2	3.9[c]
Silver	7440-22-4	2
Zinc	7440-66-6	109[c]
PCBs/Pesticides		
2,4,5-TP Acid (Silvex)[f]	93-72-1	3.8
4,4’-DDE	72-55-9	0.0033[b]
4,4’-DDT	50-29-3	0.0033[b]
4,4'-DDD	72-54-8	0.0033[b]
Aldrin	309-00-2	0.005[c]
alpha-BHC	319-84-6	0.02
beta-BHC	319-85-7	0.036

Unrestricted Use Soil Cleanup Objectives		
Contaminant	**CAS Number**	**Unrestricted Use**
Chlordane (alpha)	5103-71-9	0.094
delta-BHC	319-86-8	0.04
Dibenzofuran[f]	132-64-9	7
Dieldrin	60-57-1	0.005[c]
Endosulfan I[d,f]	959-98-8	2.4
Endosulfan II[d,f]	33213-65-9	2.4
Endosulfan sulfate[d,f]	1031-07-8	2.4
Endrin	72-20-8	0.014
Heptachlor	76-44-8	0.042
Lindane	58-89-9	0.1
Polychlorinated biphenyls	1336-36-3	0.1
Semivolatile organic compounds		
Acenaphthene	83-32-9	20
Acenapthylene[f]	208-96-8	100[a]
Anthracene[f]	120-12-7	100[a]
Benz(a)anthracene[f]	56-55-3	1[c]
Benzo(a)pyrene	50-32-8	1[c]
Benzo(b)fluoranthene[f]	205-99-2	1[c]
Benzo(g,h,i)perylene[f]	191-24-2	100
Benzo(k)fluoranthene[f]	207-08-9	0.8[c]
Chrysene[f]	218-01-9	1[c]
Dibenz(a,h)anthracene[f]	53-70-3	0.33[b]
Fluoranthene[f]	206-44-0	100[a]
Fluorene	86-73-7	30
Indeno(1,2,3-cd)pyrene[f]	193-39-5	0.5[c]
m-Cresol[f]	108-39-4	0.33[b]

Unrestricted Use Soil Cleanup Objectives		
Contaminant	**CAS Number**	**Unrestricted Use**
Naphthalene[f]	91-20-3	12
o-Cresol[f]	95-48-7	0.33[b]
p-Cresol[f]	106-44-5	0.33[b]
Pentachlorophenol	87-86-5	0.8[b]
Phenanthrene[f]	85-01-8	100
Phenol	108-95-2	0.33[b]
Pyrene[f]	129-00-0	100
Volatile organic compounds		
1,1,1-Trichloroethane[f]	71-55-6	0.68
1,1-Dichloroethane[f]	75-34-3	0.27
1,1-Dichloroethene[f]	75-35-4	0.33
1,2-Dichlorobenzene[f]	95-50-1	1.1
1,2-Dichloroethane	107-06-2	0.02[c]
cis-1,2-Dichloroethene[f]	156-59-2	0.25
trans-1,2-Dichloroethene[f]	156-60-5	0.19
1,3-Dichlorobenzene[f]	541-73-1	2.4
1,4-Dichlorobenzene	106-46-7	1.8
1,4-Dioxane	123-91-1	0.1[b]
Acetone	67-64-1	0.05
Benzene	71-43-2	0.06
n-Butylbenzene[f]	104-51-8	12
Carbon tetrachloride[f]	56-23-5	0.76
Chlorobenzene	108-90-7	1.1
Chloroform	67-66-3	0.37
Ethylbenzene[f]	100-41-4	1
Hexachlorobenzene[f]	118-74-1	0.33[b]

Unrestricted Use Soil Cleanup Objectives		
Contaminant	**CAS Number**	**Unrestricted Use**
Methyl ethyl ketone	78-93-3	0.12
Methyl tert-butyl ether[f]	1634-04-4	0.93
Methylene chloride	75-09-2	0.05
n-Propylbenzene[f]	103-65-1	3.9
sec-Butylbenzene[f]	135-98-8	11
tert-Butylbenzene[f]	98-06-6	5.9
Tetrachloroethene	127-18-4	1.3
Toluene	108-88-3	0.7
Trichloroethene	79-01-6	0.47
1,2,4-Trimethylbenzene[f]	95-63-6	3.6
1,3,5-Trimethylbenzene[f]	108-67-8	8.4
Vinyl chloride[f]	75-01-4	0.02
Xylene (mixed)	1330-20-7	0.26

All Soil clean up objectives (SCOs) are in parts per million (ppm).
Footnotes:
[a] The SCOs for unrestricted use were capped at a maximum value of 100 ppm, as discussed in the TSD.
[b] For constituents where the calculated SCO was lower than the Contract Required Quantitation Limit (CRQL), the CRQL is used as the Track 1 SCO value.
[c] For constituents where the calculated SCO was lower than the rural soil background concentration as determined by the DEC/DOH rural soil survey, the rural soil background concentration is used as the Track 1 SCO value for this use of the site.
[d] SCO is the sum of Endosulfan I, Endosulfan II and Endosulfan Sulfate.
[e] The SCO for this specific compound (or family of compounds) is considered to be met if the analysis for the total species of this contaminant is below the specific SCO.
[f] Protection of ecological resources soil cleanup objectives were not developed for contaminants identified in Table 375-6.7(b) with "NS". Where such contaminants appear in Table 375-6.7(a), the applicant may be required by the Department to calculate a protection of ecological resources soil cleanup objective according to the Technical Support Document.

Table 11-2. Final Restricted Use SCOs as Presented in 6 NYCRR Part 375-6.8(b).

Restricted Use Soil Cleanup Objectives							
		Protection of Public Health				Protection of Ecological Resources	Protection of Ground-water
Contaminant	CAS Number	Residential	Restricted-Residential	Commercial	Industrial		
Metals							
Arsenic	7440-38-2	16[f]	16[f]	16[f]	16[f]	13[f]	16[f]
Barium	7440-39-3	350[f]	400	400	10,000[d]	433	820
Beryllium	7440-41-7	14	72	590	2,700	10	47
Cadmium	7440-43-9	2.5[f]	4.3	9.3	60	4	7.5
Chromium, hexavalent[h]	18540-29-9	22	110	400	800	1[e]	19
Chromium, trivalent[h]	16065-83-1	36	180	1,500	6,800	41	NS
Copper	7440-50-8	270	270	270	10,000[d]	50	1,720
Total Cyanide[h]		27	27	27	10,000[d]	NS	40
Lead	7439-92-1	400	400	1,000	3,900	63[f]	450
Manganese	7439-96-5	2,000[f]	2,000[f]	10,000[d]	10,000[d]	1600[f]	2,000[f]
Total Mercury		0.81[j]	0.81[j]	2.8[j]	5.7[j]	0.18[f]	0.73
Nickel	7440-02-0	140	310	310	10,000[d]	30	130
Selenium	7782-49-2	36	180	1,500	6,800	3.9[f]	4[f]
Silver	7440-22-4	36	180	1,500	6,800	2	8.3
Zinc	7440-66-6	2200	10,000[d]	10,000[d]	10,000[d]	109[f]	2,480
PCBs/Pesticides							
2,4,5-TP Acid (Silvex)	93-72-1	58	100[a]	500[b]	1,000[c]	NS	3.8
4,4’-DDE	72-55-9	1.8	8.9	62	120	0.0033 [e l]	17
4,4’-DDT	50-29-3	1.7	7.9	47	94	0.0033 [e l]	136
4,4'-DDD	72-54-8	2.6	13	92	180	0.0033 [e l]	14

Restricted Use Soil Cleanup Objectives							
		Protection of Public Health				Protection of Ecological Resources	Protection of Ground-water
Contaminant	CAS Number	Residential	Restricted-Residential	Commercial	Industrial		
Aldrin	309-00-2	0.019	0.097	0.68	1.4	0.14	0.19
alpha-BHC	319-84-6	0.097	0.48	3.4	6.8	0.04[k]	0.02
beta-BHC	319-85-7	0.072	0.36	3	14	0.6	0.09
Chlordane (alpha)	5103-71-9	0.91	4.2	24	47	1.3	2.9
delta-BHC	319-86-8	100[a]	100[a]	500[b]	1,000[c]	0.04[k]	0.25
Dibenzofuran	132-64-9	14	59	350	1,000[c]	NS	210
Dieldrin	60-57-1	0.039	0.2	1.4	2.8	0.006	0.1
Endosulfan I	959-98-8	4.8[i]	24[i]	200[i]	920[i]	NS	102
Endosulfan II	33213-65-9	4.8[i]	24[i]	200[i]	920[i]	NS	102
Endosulfan sulfate	1031-07-8	4.8[i]	24[i]	200[i]	920[i]	NS	1,000[c]
Endrin	72-20-8	2.2	11	89	410	0.014	0.06
Heptachlor	76-44-8	0.42	2.1	15	29	0.14	0.38
Lindane	58-89-9	0.28	1.3	9.2	23	6	0.1
Polychlorinated biphenyls	1336-36-3	1	1	1	25	1	3.2
Semivolatiles							
Acenaphthene	83-32-9	100[a]	100[a]	500[b]	1,000[c]	20	98
Acenapthylene	208-96-8	100[a]	100[a]	500[b]	1,000[c]	NS	107
Anthracene	120-12-7	100[a]	100[a]	500[b]	1,000[c]	NS	1,000[c]
Benz(a)anthracene	56-55-3	1[f]	1[f]	5.6	11	NS	1[f]
Benzo(a)pyrene	50-32-8	1[f]	1[f]	1[f]	1.1	2.6	22
Benzo(b)fluoranthene	205-99-2	1[f]	1[f]	5.6	11	NS	1.7
Benzo(g,h,i)perylene	191-24-2	100[a]	100[a]	500[b]	1,000[c]	NS	1,000[c]
Benzo(k)fluoranthene	207-08-9	1	3.9	56	110	NS	1.7

Restricted Use Soil Cleanup Objectives							
		Protection of Public Health				Protection of Ecological Resources	Protection of Ground-water
Contaminant	CAS Number	Residential	Restricted-Residential	Commercial	Industrial		
Chrysene	218-01-9	1[f]	3.9	56	110	NS	1[f]
Dibenz(a,h)anthracene	53-70-3	0.33[e]	0.33[e]	0.56	1.1	NS	1,000[c]
Fluoranthene	206-44-0	100[a]	100[a]	500[b]	1,000[c]	NS	1,000[c]
Fluorene	86-73-7	100[a]	100[a]	500[b]	1,000[c]	30	386
Indeno(1,2,3-cd)pyrene	193-39-5	0.5[f]	0.5[f]	5.6	11	NS	8.2
m-Cresol	108-39-4	100[a]	100[a]	500[b]	1,000[c]	NS	0.33[e]
Naphthalene	91-20-3	100[a]	100[a]	500[b]	1,000[c]	NS	12
o-Cresol	95-48-7	100[a]	100[a]	500[b]	1,000[c]	NS	0.33[e]
p-Cresol	106-44-5	34	100[a]	500[b]	1,000[c]	NS	0.33[e]
Pentachlorophenol	87-86-5	2.4	6.7	6.7	55	0.8[e]	0.8[e]
Phenanthrene	85-01-8	100[a]	100[a]	500[b]	1,000[c]	NS	1,000[c]
Phenol	108-95-2	100[a]	100[a]	500[b]	1,000[c]	30	0.33[e]
Pyrene	129-00-0	100[a]	100[a]	500[b]	1,000[c]	NS	1,000[c]
Volatiles							
1,1,1-Trichloroethane	71-55-6	100[a]	100[a]	500[b]	1,000[c]	NS	0.68
1,1-Dichloroethane	75-34-3	19	26	240	480	NS	0.27
1,1-Dichloroethene	75-35-4	100[a]	100[a]	500[b]	1,000[c]	NS	0.33
1,2-Dichlorobenzene	95-50-1	100[a]	100[a]	500[b]	1,000[c]	NS	1.1
1,2-Dichloroethane	107-06-2	2.3	3.1	30	60	10	0.02[f]
cis-1,2-Dichloroethene	156-59-2	59	100[a]	500[b]	1,000[c]	NS	0.25
trans-1,2-Dichloroethene	156-60-5	100[a]	100[a]	500[b]	1,000[c]	NS	0.19
1,3-Dichlorobenzene	541-73-1	17	49	280	560	NS	2.4

Restricted Use Soil Cleanup Objectives							
		Protection of Public Health				Protection of Ecological Resources	Protection of Ground-water
Contaminant	CAS Number	Residential	Restricted-Residential	Commercial	Industrial		
1,4-Dichlorobenzene	106-46-7	9.8	13	130	250	20	1.8
1,4-Dioxane	123-91-1	9.8	13	130	250	0.1[e]	0.1[e]
Acetone	67-64-1	100[a]	100[b]	500[b]	1,000[c]	2.2	0.05
Benzene	71-43-2	2.9	4.8	44	89	70	0.06
n-Butylbenzene	104-51-8	100[a]	100[a]	500[b]	1,000[c]	NS	12
Carbon tetrachloride	56-23-5	1.4	2.4	22	44	NS	0.76
Chlorobenzene	108-90-7	100[a]	100[a]	500[b]	1,000[c]	40	1.1
Chloroform	67-66-3	10	49	350	700	12	0.37
Ethylbenzene	100-41-4	30	41	390	780	NS	1
Hexachlorobenzene	118-74-1	0.33[e]	1.2	6	12	NS	3.2
Methyl ethyl ketone	78-93-3	100[a]	100[a]	500[b]	1,000[c]	100[a]	0.12
Methyl tert-butyl ether	1634-04-4	62	100[a]	500[b]	1,000[c]	NS	0.93
Methylene chloride	75-09-2	51	100[a]	500[b]	1,000[c]	12	0.05
n-Propylbenzene	103-65-1	100[a]	100[a]	500[b]	1,000[c]	NS	3.9
sec-Butylbenzene	135-98-8	100[a]	100[a]	500[b]	1,000[c]	NS	11
tert-Butylbenzene	98-06-6	100[a]	100[a]	500[b]	1,000[c]	NS	5.9
Tetrachloroethene	127-18-4	5.5	19	150	300	2	1.3
Toluene	108-88-3	100[a]	100[a]	500[b]	1,000[c]	36	0.7
Trichloroethene	79-01-6	10	21	200	400	2	0.47
1,2,4-Trimethylbenzene	95-63-6	47	52	190	380	NS	3.6
1,3,5-Trimethylbenzene	108-67-8	47	52	190	380	NS	8.4
Vinyl chloride	75-01-4	0.21	0.9	13	27	NS	0.02
Xylene (mixed)	1330-20-7	100[a]	100[a]	500[b]	1,000[c]	0.26	1.6

All Soil clean up objectives (SCOs) are in parts per million (ppm).
NS=Not specified. See Technical Support Document (TSD).
Footnotes:
[a] The SCOs for residential, restricted-residential and ecological resources use were capped at a maximum value of 100 ppm, see TSD Section 9.3.
[b] The SCOs for commercial use were capped at a maximum value of 500 ppm, see TSD Section 9.3.
[c] The SCOs for industrial use and the protection of groundwater were capped at a maximum value of 1000 ppm, see TSD Section 9.3.
[d] The SCOs for metals were capped at a maximum value of 10,000 ppm, see TSD Section 9.3.
[e] For constituents where the calculated SCO was lower than the Contract Required Quantitation Limit (CRQL), the CRQL is used as the SCO value.
[f] For constituents where the calculated SCO was lower than the rural soil background concentration as determined by the DEC/DOH rural soil survey, the rural soil background concentration is used as the Track 2 SCO value for this use of the site.
[g] SCO is the sum of DDD, DDE and DDT.
[h] The SCO for this specific compound (or family of compounds) is considered to be met if the analysis for the total species of this contaminant is below the specific SCO.
[i] This SCO is for the sum of Endosulfan I, Endosulfan II and Endosulfan Sulfate.
[j] This SCO is the lower of the values for mercury (elemental) or mercury (inorganic salts), see TSD table 5.6-1.
[k] This SCO is derived from data on mixed isomers of BHC.
[l] This SCO is for the sum of DDD, DDE and DDT.