

Child-Specific Exposure Scenarios Examples



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Child-Specific Exposure Scenarios Examples

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ABSTRACT

The purpose of the *Child-Specific Exposure Scenarios Examples* is to outline scenarios for various child-specific exposure pathways and to demonstrate how data from the *Exposure Factors Handbook* (hereinafter *EFH*) (U.S. EPA, 2011a) may be applied for estimating dose. Exposure scenarios are tools to help the assessor develop estimates of exposure and dose to assess potential health risks. An exposure scenario generally includes facts, data, assumptions, inferences, and sometimes professional judgment about how the exposure takes place. In 2004, EPA published the *Example Exposure Scenarios* (U.S. EPA, 2004a) using human physiological and behavioral data from the *Exposure Factors Handbook* (U.S. EPA, 1997a), which has been superseded by the *EFH* (U.S. EPA, 2011a). This document provides an update of the 2004 *Example Exposure Scenarios*, focusing specifically on scenarios involving children. The example scenarios presented here have been selected to best demonstrate the use of the various key data sets in the *EFH* (U.S. EPA, 2011a) and to represent commonly encountered exposure pathways. An exhaustive review of every possible exposure scenario for every possible receptor population would not be feasible and is not provided. Instead, readers may use the representative examples provided here to formulate scenarios that are appropriate to the assessment of interest and to apply the same or similar data sets and approaches as shown in the examples.

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ABBREVIATIONS AND ACRONYMS

ADAF	Age-dependent adjustment factors
ADD	Average daily dose
ADR	Acute dose rate
BW	Body weight
EPA	U.S. Environmental Protection Agency
HEC	Human equivalent concentration
IRIS	Integrated Risk Information System
IUR	Inhalation Unit Risk
LADD	Lifetime average daily dose
NCEA	National Center for Environmental Assessment
RfC	Reference concentration
RfD	Reference dose

PREFACE

The National Center for Environmental Assessment (NCEA) of EPA's Office of Research and Development has prepared the *Child-Specific Exposure Scenarios Examples* to outline scenarios for various exposure pathways and to demonstrate how data from the *EFH* (U.S. EPA, 2011a) may be applied for estimating exposures for children. A similar document entitled *Example Exposure Scenarios* was published by EPA in 2004 (U.S. EPA, 2004a). The *Child-Specific Exposure Scenarios Examples* updates the children's exposure scenarios included in U.S. EPA 2004a.

An exposure scenario considers the physical setting, potential uses of a contaminated resource (e.g., future residential land use or consumption of fish), the population that may be exposed (infant, child, or adolescent), fate and transport of contaminants, and how exposure may occur including ingestion, dermal contact, and inhalation. Consideration of frequency and duration of exposure as well as seasonal variations are part of the development of an exposure scenario. The *Child-Specific Exposure Scenarios Examples* is intended to be a companion document to the *EFH*. The example scenarios were compiled from questions and inquiries received from users of the earlier versions of the *EFH* on how to select data from the Handbook. The scenarios presented in this report promote the use of the standard set of age groups recommended by the EPA in the report entitled *Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants* (U.S. EPA, 2005a).

Each scenario examined in this report refers to a single-chemical exposure route and pathway. EPA recognizes that individuals may be exposed to mixtures of chemicals through more than one pathway and one route. In the past few years there has been an increased emphasis in cumulative risk assessments¹, aggregate exposures², and chemical mixtures (U.S. EPA, 2008a, 2003). Detailed and comprehensive guidance for evaluating cumulative risk is not currently available. The Agency has, however, developed a framework that lays out a broad outline of the assessment process and provides a basic structure for evaluating cumulative

¹Cumulative risk assessment—An analysis, characterization, and possible quantification of the combined risks to health or the environment from multiple agents or stressors.

²Aggregate exposures—The combined exposure of an individual (or defined population) to a specific agent or stressor via relevant routes, pathways, and sources.

risks. This basic structure is presented in the *Framework for Cumulative Risk Assessment* published in May 2003 (U.S. EPA, 2003). Additional guidance is available from EPA's *Concepts, Methods and Data Sources for Cumulative Health Risk Assessment of Multiple Chemicals, Exposures and Effects: A Resource Document* (U.S. EPA, 2007a). EPA encourages and supports the use of new and innovative approaches and tools to improve the quality of public health and environmental protection.

In general, the *Child-Specific Exposure Scenarios Examples* document provides examples using the point-estimate approach, but also includes an example of a simple probabilistic assessment for one scenario. In contrast to the point-estimate approach, probabilistic methods allow for a better characterization of variability and/or uncertainty in risk estimates. The use of probabilistic methods is contingent on the availability and quality of the data. Additional information on characterization of variability and uncertainty can be found in Chapter 2 of the *EFH* (U.S. EPA, 2011a).

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1. INTRODUCTION AND PURPOSE

Children's environmental exposures to contaminants can change significantly as they develop through infancy, childhood, and adolescence. These exposure differences are a result of both behavioral and rapid physiological changes as they grow. Children, therefore, may be physiologically susceptible to some environmental contaminants during certain life stages. Greater susceptibility due to greater exposure can lead to greater risk of adverse health effects for children relative to adults exposed to the same contaminants.

Since 1995, the U.S. Environmental Protection Agency (EPA) has specified that children must be explicitly considered when conducting risk assessments as part of a public health decision-making process. Subsequently, the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b) stressed the importance of considering life stage differences in both exposure and dose-response when assessing cancer risks from early life exposures. The guidance promotes the summing of doses or risks across all relevant life stages instead of averaging an age-specific dose over the entire lifetime. For carcinogens acting via a mutagenic mode of action, age-dependent adjustment factors (ADAFs) are used to account for susceptibility at various life stages (U.S. EPA, 2005b).

In 2002, EPA published the interim final version of the *Child-Specific Exposure Factors Handbook*, which was designed specifically to address the exposure factors related to children (U.S. EPA, 2008a). In 2008, the *Child-Specific Exposure Factors Handbook* was republished (U.S. EPA, 2008a), incorporating information from the *Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants* (U.S. EPA, 2005a). The 2008 version of the *Child-Specific Exposure Factors Handbook* rearranged the data and recommendations, to the extent possible, to be consistent with the standard set of childhood age groups provided in the 2005 guidance. The *Exposure Factors Handbook* published in 2011 retained the same age groupings for children. These childhood age groups are as follows:

- Less than 12-months old: birth to <1 month, 1 to <3 months, 3 to <6 months, and 6 to <12 months
- Greater than 12-months old: 1 to <2 years, 2 to <3 years, 3 to <6 years, 6 to <11 years, 11 to <16 years, and 16 to <21 years

1.1. PURPOSE

The purpose of the *Child-Specific Exposure Scenarios Examples* is to present childhood exposure scenarios using data from the *Child-Specific Exposure Factors Handbook* and updated children's data from the *Exposure Factors Handbook* (U.S. EPA, 2011a, 2008a; referred to throughout as *EFH*). These scenarios are not meant to be inclusive of every possibility, but they are intended to provide a range of scenarios that show how to apply exposure factors data to characterize childhood exposures. As such, these scenarios are not meant as templates for exposure assessors, but can be modified to meet specific needs.

1.2. RELATIONSHIP TO OTHER RELEVANT EPA REPORTS

In 2011, EPA published the *EFH*, which supersedes the *Child-Specific Exposure Factors Handbook* and previous versions of the *EFH* (U.S. EPA, 2011a, 2008). The *Child-Specific Exposure Scenarios Examples* supersedes the children's exposure scenarios included in U.S. EPA 2004a.

1.3. EXPOSURE ASSESSMENT GENERAL PRINCIPLES

Exposure assessment is a “process of estimating or measuring the magnitude, frequency, and duration of exposure to an agent, along with the number and characteristics of the population exposed” (Zartarian et al., 2007a). Exposure assessments are conducted for a variety of purposes including risk assessments, trend analyses, and epidemiological studies (U.S. EPA, 1992a). In the risk assessment context, the output of an exposure assessment is typically the estimation of the potential dose (U.S. EPA, 1992a). The potential dose is dependent on the concentration of the contaminant in a medium (e.g., soil, water, air) and the intake or contact rate of the population with the medium. This potential dose can be adjusted to include additional factors that further characterize the population being assessed and describe the exposure in terms of exposure duration and frequency.

The terms exposure and dose are closely related. Exposure is defined as the “contact of an organism with a chemical or physical agent, quantified as the amount of chemical available at the exchange boundaries of the organism and available for absorption” (IPCS, 2001). The dose refers to the amount of agent (e.g., chemical) that enters a target in a specified period of time after crossing a contact boundary. The units of dose are typically mg/kg-day. The example

scenarios provided in this report focus on the calculation of dose and not exposure. Often times, dose is calculated and combined with toxicity information to calculate risk. However, calculations of risk are outside the scope of this document. The principal focus of this document is on childhood doses from exposure to chemicals, but the concepts may apply to other agents.

Exposure can occur via ingestion, inhalation, or direct contact (i.e., dermal). Chemicals can be introduced into the gastrointestinal tract through dietary ingestion of foods and beverages or nondietary ingestion of foreign substances (e.g., soil). The outer contact boundary for ingestion exposures is the mouth. A chemical can enter the respiratory tract through the inhalation of particles, gases, vapors, and aerosols. For inhalation exposure, the outer contact boundary is the oral/nasal boundary. The characteristics of the inhaled agent affect its deposition, retention, translocation, and distribution within the respiratory system and other tissues in the body (U.S. EPA, 1994). Dermal absorption is governed by the characteristics of the skin (contact boundary) on the exposed part of the body, and the characteristics of the agent (e.g., physical-chemical properties) and the matrix in which it exists (e.g., water, soil); environmental conditions (e.g., temperature and humidity) can also play a role. The example scenarios presented in this document are organized according to these three routes of exposure (i.e., ingestion, inhalation, and dermal contact).

The population of interest in an exposure assessment, also known as the receptor population, may include children at various life stages to account for their rapidly changing physiology and behavior. In addition, the exposure assessment can evaluate only the population that is potentially exposed (i.e., “doers-only,” “consumers-only”) or it can assess the exposure over the entire population on a *per capita* basis. If one could sample everyone in the population, the “doers-only” or “consumers-only” will be those individuals who engage in the specific activity of interest. “Per capita” will be everyone in the population. Since not everyone in the population can be studied, in this report, “doers only” refers to only those individuals who reported doing the activity during the survey period. “Consumers-only” refers to only those individuals who reported food or water intake during the survey period. “Doers-only” or “consumers-only” contact rates are calculated by averaging activity rates or food or water intake rates across only the individuals in the survey who engaged in those activities or consumed those foods or beverages. Conversely, “per capita” contact rates are generated by averaging

consumer-only rates over the entire population, including those individuals that reported no activity or intake during the survey period. Generally, “per capita” contact rates are appropriate for use in exposure assessments for which average dose estimates are of interest. They are also useful for comparisons with other population groups. For example, for foods, they represent both individuals who ate the foods during the survey period and individuals who may eat the food items at some time, but did not consume them during the survey period. Per capita intake may underestimate consumption for the subset of the population that consumed the food in question (U.S. EPA, 2011a).

The equation used to express dose is based on the intensity, duration, and frequency of the exposure to the receptor population. The intensity typically is expressed as the concentration of the contaminant per unit mass or volume (i.e., $\mu\text{g/g}$, $\mu\text{g/L}$, mg/m^3 , ppm, etc.) in the medium multiplied by the contact rate (e.g., intake rate, inhalation rate). In the following examples, the concentration of a contaminant “ x ” is used as a generic term. Except for the concentration, the terms used in the dose equation are referred to as exposure factors. Exposure factors are factors related to human behavior and characteristics that help determine an individual's exposure to an agent. The concentration is based on site- and chemical-specific data that are not provided in the *EFH*.

Each of the main exposure routes has a range of exposure descriptors that explains the distribution of exposures occurring in the exposed population. The central tendency scenario is developed using means or 50th percentiles for contaminant concentration and exposure factors or by selecting the mean or median from the dose distribution. A high-end exposure scenario typically represents an individual in the upper end of the exposure distribution (i.e., over the 90th percentile, but less than the most exposed individual [U.S. EPA, 1992a]). High-end scenarios are developed using a combination of central and upper estimates for the contaminant concentration and/or exposure factors or by selecting an upper-percentile from the dose distribution. The choice of the parameters the assessor sets at a central tendency value versus an upper-percentile depends on judgement, the sensitivity of the parameters, and regulatory requirements.

A bounding scenario is defined as an exposure higher than any expected to occur in the actual population (U.S. EPA, 1992a). A theoretical upper bound is estimated by assuming limits

for all the variables used to calculate exposure and dose that, when combined, will result in the mathematically highest exposure or dose (highest concentration, highest intake rate, lowest body weight [BW]) (U.S. EPA, 1992a). However, it is generally not necessary to use the theoretical upper bound to assure that the exposure or dose calculated is above the actual distribution (U.S. EPA, 1992a). Thus, bounding estimates in the examples included in this report are estimated using upper-percentile estimates for most of the dose equation, but keeping some variables at the mean value (i.e., BW, surface area). An upper-percentile estimate of an exposure factor is defined as a value between the 90th and the 99.9th percentile in the exposure factor distribution (U.S. EPA, 2011a). In the *EFH*, the 95th percentile, if available, was used to represent the upper-percentile in the recommendations because it is the middle of the range between the 90th and 99.9th percentiles. For some factors, a specific upper-percentile could not be defined because the data were not available.

Another aspect of exposure assessment is the duration and the frequency over which the exposure occurred. An acute exposure is a one-time exposure to a contaminant by the oral, dermal, or inhalation route for 24 hours or less. Chronic exposure is defined as a repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species). A subchronic exposure is defined as a repeated exposure by the oral, dermal, or inhalation route for more than 30 days, up to approximately 10% of the life span in humans (U.S. EPA, 2011b).

The potential dose may be calculated as a potential average daily dose (*ADD*), lifetime average daily dose (*LADD*), or the acute dose rate (*ADR*). The *ADD*, calculated for a noncarcinogenic contaminant exposure, is a dose averaged over a specified timeframe. The general equation used for *ADD* is presented below (see eq 1). Equations used to calculate a dermal dose include some additional terms (e.g., surface area, dermal permeability coefficient) and are presented in Section 4. Historically, the *LADD* is calculated for contaminant exposure that is expressed over an adult lifetime (e.g., 70 years). *LADD* is generally used when assessing exposure to carcinogens. However, consistent with the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*, if exposure is less than lifetime, the dose is calculated by summing time weighted doses that occur during each life stage and

averaging across the total exposure period (U.S. EPA, 2005b). It is important to note that the average life expectancy has increased to an average of 78 years for males and females combined, as stated in the *EFH* (U.S. EPA, 2011a). The increase in life expectancy in humans is largely attributed to decreases in nonmalignant disease mortality, with little change in malignant disease mortality. The use of 70 years has been a policy decision due to the fact that there is no evidence to suggest that cancer risk per year of exposure has changed simply due to increased life expectancy. Dividing the *LADD* over a period of 78 years instead of 70 years will have the effect of lowering the cancer risk by approximately 10%. Although this is not a significant difference, for consistency, an average lifetime of 70 years is used as a reference value when calculating the *LADD*. For noncarcinogenic acute exposures, the *ADR* is used. To calculate the *ADR*, the exposure frequency, exposure duration, and averaging time are all set equal to 1 to adjust the calculation for a one-time exposure.

$$ADD = \frac{C \times CR \times EF \times ED}{BW \times AT} \quad (1)$$

where:

<i>ADD</i>	= potential average daily dose of the contaminant of interest (mg/kg-d);
<i>C</i>	= concentration of the contaminant within the media of interest (mg/g; mg/L; mg/cm ² ; mg/m ³);
<i>CR</i>	= average daily contact rate of the media of interest (g/d; L/d; cm ² /d; m ³ /d);
<i>EF</i>	= exposure frequency (d/yr);
<i>ED</i>	= exposure duration (yr);
<i>BW</i>	= body weight (kg); and
<i>AT</i>	= averaging time (d).

Note that, in some cases, contact rate may be expressed in units of less than a day (e.g., L/hr). When this occurs, an additional term (e.g., exposure time) may be included to account for the portion of a day spent engaging in the activity of interest (e.g., hours/day). Also, for some exposure pathways, contact rates are expressed as intake rates. In some cases, the contact rate is provided on a body-weight (*BW*) basis (e.g., g/kg-day or L/kg-day); therefore, *BW* is not needed in the denominator of the dose equation. Also note that other algorithms or approaches may be

used to calculate dose depending on available data, software capabilities, regulatory goals, and statutory requirements. EPA program offices may also have guidances specific to their programs. In addition, the assessor may need to consider the bioavailability of the contaminant in the specific medium. The bioavailability will vary depending on the physicochemical characteristics of the contaminant and the characteristics of the medium (e.g., particle size in soil).

In the risk assessment context, the dose calculations are often combined with toxicity information to estimate risk. Exposure assessors are encouraged to consult with toxicologists regarding the appropriate application of toxicity values with the exposure assessment. For example, it may not be appropriate to use toxicity values derived to reflect chronic exposures (e.g., Reference Dose, cancer slope factors) when characterizing acute or subchronic exposures. In addition, when assessing cancer risk from exposures to mutagenic carcinogens, the assessor needs to consider the ADAFs to account for early life sensitivities. The reader is referred to the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* for further information about ADAFs and their application (U.S. EPA, 2005b).

The EPA uses inhalation dosimetry methodology for estimating exposure through the inhalation pathway because the amount of chemical that reaches the target organ is not a simple function of the inhalation rate and *BW* (U.S. EPA, 2009). In contrast with the ingestion pathway, the inhalation dosimetry methodology only requires a derivation of a time weighted average concentration adjusted for the duration and frequency of exposure (U.S. EPA, 2009). To estimate risk, the adjusted concentration is then compared with a Reference Concentration (*RfC*) or an Inhalation Unit Risk (*IUR*), which are expressed in units of concentration and the inverse of concentration, respectively. There may be cases in which an inhalation dose is of interest (e.g., the estimation of aggregate and cumulative doses; an analysis of relative pathway contribution).

1.4. EXPOSURE SCENARIOS

This document is intended to present example exposure scenarios that reflect the possible ways that data reported in the *EFH* (U.S. EPA, 2011a) may be used in childhood exposure assessments and risk assessments. The scenarios are representative of generic applications and should be tailored to specific program needs and the population and life stages of interest. As

such, they are not intended to supplant specific guidance issued by EPA program offices. In developing risk assessments under specific EPA programs, the risk assessor should consult specific programmatic guidance and requirements. The selection of life stages used in each scenario is not meant to imply that those are the only life stages for which the scenario could apply.

This report shows multiple examples for each of the main exposure routes (ingestion, inhalation, and dermal penetration), a range of exposure descriptors (e.g., central tendency, high-end, or bounding exposure), exposure durations (i.e., acute, subchronic, or chronic), and receptor populations (e.g., single or multiple age ranges, only participants in an activity called “doers-only,” “consumers-only,” and *per capita* population). For demonstration purposes, only one exposure descriptor was chosen for each exposure scenario. This is not meant to imply that only those chosen descriptors will be of interest for that particular scenario. Throughout the report, high-end scenarios are constructed using a combination of central tendency estimates and high-end estimates for the various parameters of the dose equation. The intent of the report is not to provide prescriptive guidance on how to estimate a high-end dose, since subjective judgement is required.

Specific scenarios were selected based on inquiries received from users of the past version of the *EFH*. These are scenarios that exposure and risk assessors may frequently encounter. For example, food intake scenarios were selected to illustrate the use of per capita versus consumer-only data. Fish consumption was of particular interest because of the associations between contaminants that may be found in fish and children’s susceptibility to these chemicals. Human milk intake and nondietary exposures are pathways that are unique to children. Likewise, the inhalation and dermal scenarios were selected based on locations where children spend their time and activities in which they are engaged (e.g., schools, indoor environments, outdoor activities). Exposure durations and the corresponding dose metrics are presented in Table 1.

Table 1. Exposure durations and dose metrics

Exposure type	Exposure duration	Dose metric
Acute	≤24 hours	<i>ADR</i>
Subchronic	>30 days <10% life span in humans	<i>ADD_{subchronic}</i>
Chronic	>10% life span in humans	<i>ADD_{chronic}</i>
Lifetime	Life span	<i>LADD</i>

The example scenarios are presented according to exposure route. Each scenario assumes that the concentration of the chemical in the specific medium is known, either measured or modeled. Mean values for the exposure concentration are used throughout the report when calculating central tendency dose estimates. It should be noted that some exposure assessors use the upper confidence limit of the mean for a more conservative estimate. Since the concentration values are assumed to be known, this report does not address fate and transport considerations for the estimation of the concentration term. In addition, the scenarios do not address exposures via multiple routes. This is not meant to imply that exposures from other routes are negligible for a particular scenario. For example, exposure to contaminated homegrown vegetables implies contaminated soils, which may also result in exposures via the nondietary pathway. In addition, the scenarios described do not include potential exposures that may be experienced by the parents, care givers, or other workers present in the same microenvironments. Each example provides an introduction describing the scenario, the algorithm used for estimating dose, the suggested input values of exposure factors with the calculation of the estimated dose, and the uncertainties and limitations of the data and/or approach used in the example. Exposure factors input values were derived from the recommendations published in the *EFH* (U.S. EPA, 2011a).

Table 2 provides a summary of the example exposure scenarios presented in this document. The outcome for most of the scenarios included in this document is a point estimate for the described scenario. However, one scenario (2.6—Ingestion of Contaminated Drinking Water: Children <21 Years, Distribution of Chronic Daily Exposure) presents a simple example of the use of a probabilistic approach for estimating dose. Toxicity values needed to calculate risk from each exposure scenario described in Table 2 should match the exposure duration of interest (i.e., acute, subchronic, chronic). The exposure duration should also match the health

end point of interest (i.e., carcinogens and noncarcinogens). For additional information on exposure assessment, refer to Chapter 1 of the *EFH* (U.S. EPA, 2011a).

Table 2. Child-Specific Exposure Scenarios Examples roadmap

#	Scenario Title	Exposure media	Receptor population	Exposure distribution	Calculated dose or concentration	Age range (yr, unless stated otherwise)
Ingestion scenarios						
2.1	Per Capita Ingestion of Contaminated Homegrown Exposed Vegetables: Children Aged 1 to <11 Years, in Gardening Households, Central Tendency, Chronic Average Daily Dose	Homegrown exposed vegetables	Children, per capita	Central Tendency	ADD, chronic	1 to <11
2.2	Ingestion of Contaminated Soil and Dust in and Around the Home: Young Children Aged 1 to <6 Years, Central Tendency, Lifetime Average Daily Dose	Soil and dust	Young children	Central tendency	LADD ^a , chronic	1 to <6
2.3	Ingestion of Contaminated Indoor Dust: Children at School Aged 6 to <11 Years, Central Tendency, Subchronic Average Daily Dose	Indoor dust	School children	Central tendency	ADD, subchronic	6 to <11
2.4	Ingestion of an Environmental Contaminant by Nondietary Hand-to-mouth Behaviors: Infants and Toddlers 3 Months to <2 Years, Bounding, Acute Dose Rate	Nondietary hand-to-mouth activity	Infants and toddlers	Bounding	ADR, acute	3 mo to <2 yr
2.5	Exposure Time of Incidental Ingestion of Contaminated Pool Water to Reach a Reference Dose (RfD) Level of Exposure: Children Aged 6 to <11 Years, Bounding	Pool water	Children, doers-only	Bounding	RfD used to calculate ET	6 to <11
2.6	Ingestion of Contaminated Drinking Water: Children Aged <21 Years, Distribution of Chronic Average Daily Dose	Drinking water	Children, consumers-only	Distribution	ADD, chronic	Birth to <21 yr
2.7	Ingestion of Contaminated Human Milk: Infants Aged Birth to <12 Months, High-end, Subchronic Average Daily Dose	Human milk	Infants, consumers-only	High-end	ADD, subchronic	Birth to <12 mo
2.8	Ingestion of Contaminated Recreational Atlantic Marine Finfish: Children Aged 3 to <6 Years, Central Tendency, Subchronic Average Daily Dose	Recreational Marine fish	Children, consumers-only	Central tendency	ADD, subchronic	3 to <6

Table 2. Child-specific Exposure Scenarios Examples roadmap (continued)

#	Scenario Title	Exposure media	Receptor population	Exposure distribution	Calculated dose	Age range (years, unless stated otherwise)
Inhalation scenarios						
3.1	Inhalation of Contaminated Air while Playing in a School Yard: School Children Aged 6 to <11 Years, Central Tendency, Subchronic Adjusted Air Concentration	Outdoor air	School children, doers-only	Central tendency	<i>C air adjusted</i> , subchronic	6 to <11
3.2	Inhalation of Aerosolized Contaminants from Water During and After Showering: Children and Teens Aged 6 to <18 Years, Chronic Central Tendency, Lifetime Adjusted Air Concentration	Aerosolized water	Children and teens, doers-only	Central tendency	<i>C air adjusted</i> ^a , chronic	6 to <16
3.3	Inhalation of Contaminated Indoor Air: Residential Children Aged 3 to <11 Years, Bounding, Acute Dose Rate	Indoor air	Residential children, doers-only	Bounding	<i>ADR</i>	3 to <11
3.4	Inhalation of Contaminated Air During Bus Transportation: School Children and Teens Aged 6 to <16 Years, High-end, Chronic Average Daily Dose	Air on bus transportation	Children and teens, doers-only	High-end	<i>ADD</i> , chronic	6 to <16
Dermal scenarios						
4.1	Dermal Contact with Contaminated Soil: Teen Athletes Aged 11 to <16 Years, Central Tendency, Subchronic Average Daily Dose	Outdoor soil	Teen athletes, doers-only	Central tendency	<i>ADD</i> , subchronic	11 to <16
4.2	Dermal Contact with an Inorganic Contaminant while Wading in a Recreational Pond: Children Aged 6 to <16 Years, Bounding, Acute Dose Rate	Recreational water	Children, doers-only	Bounding	<i>ADR</i>	6 to <16
4.3	Dermal Contact with an Organic Contaminant in Water While Showering: Children and Teens Aged 6 to <16 Years, High-end, Lifetime Average Daily Dose	Potable water	Children and teens, doers-only	High-end	<i>LADD</i> ^a	6 to <16

^aThese scenarios assess exposure to a carcinogen; thus, the exposure is averaged over a lifetime value.

1.5. CUMULATIVE EXPOSURES

This report provides childhood exposure scenario examples, each relating to one route of exposure and one chemical. EPA recognizes that childhood exposure can occur from multiple routes and to one or multiple stressors. The *Framework for Cumulative Risk Assessment* (U.S. EPA, 2003) provides a simple and flexible structure for conducting and evaluating cumulative risk assessment. EPA (2003) defines cumulative risk as “the combined risks from aggregate exposures to multiple agents or stressors.” Agents or stressors are defined in a broader sense to include chemicals, as well as biological or physical agents (e.g., noise, nutritional status), or the change or loss of a necessity such as habitat.

Considerations regarding the cumulative evaluation of chemical stressors are discussed in EPA’s report entitled *A Framework for Assessing Health Risks of Environmental Exposures to Children* (U.S. EPA, 2006). The first step in quantifying exposure for a cumulative risk assessment is the characterization of the population and study area so that all existing and future pathways can be identified (U.S. EPA, 2007a). It is particularly important when assessing childhood exposures to identify the relevant and unique exposure factors that may be used to adjust the exposure estimate based on differential exposures (U.S. EPA, 2007a). The next step is to group the chemicals of concern according to the timing, medium, or exposure pathway (U.S. EPA, 2007a). This step typically requires the exposure analyst to consult with toxicologists to determine the types of chemical exposures that could be associated with a particular end point. Information about the potential chemicals’ co-occurrence in each compartment/medium and their potential interactions affecting transformation, fate, and transport are also useful (U.S. EPA, 2007a).

Concepts, methods, and data sources for cumulative health risk assessment are described in more detailed in EPA’s report entitled *Concepts, Methods, and Data Sources for Cumulative Health Risk Assessment of Multiple Chemicals, Exposures and Effects: A Resource Document* (U.S. EPA, 2007a). Cumulative exposure assessments can be complex and may require the use of models. Modeling tools have been developed that facilitate the evaluation of cumulative exposures (e.g., *U.S. EPA Stochastic Human Exposure and Dose Simulation Model for Multimedia, Multipathway Chemicals (SHEDS-Multimedia)*; http://www.epa.gov/heads/products/sheds_multimedia/sheds_mm.html) (U.S. EPA 2008b; Zartarian et al., 2007b).

1.6. OTHER SOURCES OF INFORMATION ON EXPOSURE ASSESSMENT

For additional information on exposure assessment resources, the reader is encouraged to refer to EPA-Expo-Box (a toolbox for exposure assessors) (available at <http://www.epa.gov/risk/expobox/>). Links to the following EPA resources included in EPA-Expo-Box may be useful:

- Methods for Assessing Exposure to Chemical Substances, Volumes 1–13 (U.S. EPA, 1983-1989);
- Pesticide Assessment Guidelines, Subdivisions K and U (U.S. EPA, 1984, 1986a);
- Standard Scenarios for Estimating Exposure to Chemical Substances During Use of Consumer Products (U.S. EPA, 1986b);
- Selection Criteria for Mathematical Models Used in Exposure Assessments: Surface Water Models (U.S. EPA, 1987);
- Selection Criteria for Mathematical Models Used in Exposure Assessments: Groundwater Models (U.S. EPA, 1988a);
- Superfund Exposure Assessment Manual (U.S. EPA, 1988b);
- Risk Assessment Guidance for Superfund, Volume I, Part A, Human Health Evaluation Manual (U.S. EPA, 1989);
- Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (U.S. EPA, 1990);
- Risk Assessment Guidance for Superfund, Volume I, Part B, Development of Preliminary Remediation Goals (U.S. EPA, 1991a);
- Risk Assessment Guidance for Superfund, Volume I, Part C, Risk Evaluation of Remedial Alternatives (U.S. EPA, 1991b);
- Guidelines for Exposure Assessment (U.S. EPA, 1992a);
- Dermal Exposure Assessment: Principles and Applications (U.S. EPA, 1992b);
- Soil Screening Guidance (U.S. EPA, 1996a);

- Occupational and Residential Exposure Test Guidelines: OPPTS 875.1000 Background for Application Exposure Monitoring Test Guidelines. Group A (U.S. EPA, 1996b);
- Occupational and Residential Exposure Test Guidelines: OPPTS 875.2000 Background for Postapplication Exposure Monitoring Test Guidelines. Group B. (U.S. EPA, 1996c);
- Guiding Principles for Monte Carlo Analysis (U.S. EPA, 1997b);
- Policy for Use of Probabilistic Analysis in Risk Assessment at the U.S. Environmental Protection Agency (U.S. EPA, 1997c);
- Sociodemographic Data Used for Identifying Potentially Highly Exposed Populations (U.S. EPA, 1999a);
- Report of the Workshop on Selecting Input Distributions for Probabilistic Assessments U.S. EPA, 1999b);
- Options for Development of Parametric Probability Distributions for Exposure Factors (U.S. EPA, 2000a);
- Revised Methodology for Deriving Health-Based Ambient Water Quality Criteria (U.S. EPA, 2000b);
- Risk Assessment Guidance for Superfund, Volume I, Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments (U.S. EPA, 2001a);
- Risk Assessment Guidance for Superfund Volume III, Part A, Process for Conducting Probabilistic Risk Assessments (U.S. EPA, 2001b);
- Framework for Cumulative Risk Assessment (U.S. EPA, 2003b);
- Risk Assessment Guidance for Superfund, Volume I, Part E, Supplemental Guidance for Dermal Risk Assessment (U.S. EPA, 2004b);
- Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants (U.S. EPA, 2005a);
- Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (U.S. EPA, 2005b);
- Cancer Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005c);

- Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (U.S. EPA, 2005d);
- A Framework for Assessing Health Risk of Environmental Exposures to Children from Environmental Exposures (U.S. EPA, 2006);
- Concepts, Methods, and Data Sources For Cumulative Health Risk Assessment of Multiple Chemicals, Exposures and Effects: A Resource Document (U.S. EPA, 2007a);
- Dermal Exposure Assessment: A Summary of EPA Approaches (U.S. EPA, 2007b);
- Child-Specific Exposure Factors Handbook (U.S. EPA, 2008a);
- Stochastic Human Exposure and Dose Simulation Model for Multimedia, Multipathway Chemicals (SHEDS-Multimedia) Dietary Model. Details of SHEDS-Multimedia Version 3: Technical Manual (U.S. EPA, 2008);
- Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Part F, Supplemental Guidance for Inhalation Risk Assessment (U.S. EPA, 2009);
- Exposure Factors Handbook: 2011 Edition (U.S. EPA 2011a);
- Highlights of the Exposure Factors Handbook (U.S. EPA 2011c);
- Recommended Use of Body Weight^{3/4} (BW^{3/4}) as the Default Method in Derivation of the Oral Reference Dose (U.S. EPA, 2011d);
- Standard Operating Procedures for Assessing Residential Pesticide Exposure Assessment (U.S. EPA, 2012a); and
- The Stochastic Human Exposure and Dose Simulation Model for Multimedia, Multipathway Chemicals (SHEDS-Multimedia): Dietary Module. SHEDS-Dietary version 1. Technical Manual. (U.S. EPA, 2012b).

1.7. COMMON CONVERSION FACTORS

Frequently, exposure assessments require the use of volume, mass or area conversion factors. Conversion factors may be used to convert these units of measure to those needed to calculate dose. These factors are used, for example, to ensure consistency between the units used to express exposure concentration and those used to express intake. Table 3 provides a list of common conversion factors that may be required in the exposure equations.

Table 3. Common conversion factors

To Convert	Multiply	To Obtain
Volume		
cubic centimeters (cm ³)	0.000001	cubic meters (m ³)
cubic centimeters (cm ³)	0.001	liters (L)
cubic meters (m ³)	1,000,000	cubic centimeters (cm ³)
gallons (gal)	3.785	liters (L)
liters (L)	0.264	gallons (gal)
liters (L) (water)	1,000	grams (g) (water)
liters (L)	1,000	milliliters (mL)
liters (L)	1,000	cubic centimeters (cm ³)
milliliters (mL)	0.001	liters (L)
milliliters (mL) (water)	1	grams (g) (water)
Mass		
grams (g)	0.0022	pound (lb)
grams (g) (water)	1	milliliters (mL) (water)
grams (g) (water)	0.001	liters (L) (water)
grams (g)	1,000	milligrams (mg)
grams (g)	0.001	kilograms (kg)
kilograms (kg)	1,000	grams (g)
micrograms (μg)	0.001	milligrams (mg)
milligrams (mg)	0.001	grams (g)
milligrams (mg)	1,000	micrograms (μg)
pounds (lb)	454	grams (g)
Area		
square centimeters (cm ²)	0.0001	square meters (m ²)
square meters (m ²)	10,000	square centimeters (cm ²)

2. EXAMPLE INGESTION SCENARIOS

2.1. PER CAPITA INGESTION OF CONTAMINATED HOMEGROWN EXPOSED VEGETABLES: CHILDREN AGED 1 TO <11 YEARS, IN GARDENING HOUSEHOLDS, CENTRAL TENDENCY, CHRONIC AVERAGE DAILY DOSE

2.1.1. Introduction

At sites with soil or water contamination, or where deposition of atmospheric contaminants has been observed or is expected based on modeling, the potential exists for locally grown exposed vegetables to become contaminated. Exposed vegetables are those that are grown above ground and do not have outer protective coatings that are removed before consumption. Thus, chronic exposure to these contaminants may exist among children who ingest exposed vegetables grown in gardens in the contaminated area. The dose via intake of contaminated exposed vegetables is a function of the concentrations of the contaminants in the vegetables, the rate at which children consume the food, and the frequency and duration of exposure.

This example assumes exposure via contaminated homegrown exposed vegetables. The example calculates the central tendency average daily dose from the ingestion of homegrown exposed vegetables for consumers consisting of children aged 1 to <11 years. This example uses intake data for four age ranges (1 to <2 years, 2 to <3 years, 3 to <6 years, and 6 to <11 years) derived from the *EFH* (U.S. EPA, 2011a). Values obtained from tables within the *EFH* (U.S. EPA, 2011a) are cited as *EFH* Table X-X. The mean consumer-only homegrown exposed vegetable intake rate, based on the population that gardens, from Table *EFH* 13-60 is converted to a mean per capita rate. This value is the per capita mean for the entire survey population that gardens (i.e., all ages combined). It is used with age-specific per capita intake data for all exposed vegetables (i.e., not just homegrown exposed vegetables, and not just gardening households) from *EFH* Table 9-20 to develop age-specific mean per capita intake rates for homegrown exposed vegetables in gardening households for the four age groups of children between the ages of 1 and <11 years. The information on homegrown intake originates from analyses performed by EPA on data from the 1987–1988 Nationwide Food Consumption Survey, which currently is the best source of data available to EPA on consumption of home-produced food. This example assumes that the quantity of homegrown exposed vegetables produced in the garden is sufficient to support intake at this rate. The intake rates used in this example represent per capita intake rates for households that participate in home gardening. In

addition, the intake rates are adjusted to account for losses during food preparation, as described in U.S. EPA (2011a).

This scenario assumes that the children live within the contaminated area over the duration of the exposure (and thus are continually exposed from 1 to <11 years of age) and that they eat homegrown exposed vegetables throughout each year. It is also assumed that all of the homegrown exposed vegetables are obtained from the contaminated area.

2.1.2. Dose Algorithm

For consumers, the dose of a specified contaminant via ingestion of homegrown exposed vegetables is expressed as an average daily dose ($ADD_{hg\ exp-veg\ ing}$) per BW and is calculated as follows:

$$ADD_{hg\ exp-veg\ ing} = \frac{C_{hg\ exp-veg} \times IR_{hg\ exp-veg\ per\ capita\ mean-adj} \times EF \times ED}{AT} \quad (2)$$

where:

- $ADD_{hg\ exp-veg\ ing}$ = potential average daily dose per kg BW of the contaminant from ingestion of homegrown exposed vegetables (mg/kg-d);
- $C_{hg\ exp-veg}$ = concentration of the contaminant in the homegrown exposed vegetables (mg/g);
- $IR_{hg\ exp-veg\ per\ capita\ mean-adj}$ = age-specific daily intake rate of homegrown exposed vegetables among children in gardening households; per capita mean, adjusted for preparation losses (g/kg-d);
- EF = exposure frequency (d/yr);
- ED = exposure duration (yr); and
- AT = averaging time (d).

As detailed in the following subsections, the $ADD_{hg\ exp-veg\ ing}$ values are calculated for each of the four age ranges of 1 to <2 years, 2 to <3 years, 3 to <6 years, and 6 to <11 years, using available data from the EFH). These estimates are then summed to obtain an $ADD_{hg\ exp-veg\ ing}$ estimate for the entire 1- to <11-year-old age range.

2.1.3. Input for Exposure Factor Variables

$C_{hg\ exp-veg}$ —The concentration of the contaminant in homegrown exposed vegetables is either the measured or predicted concentration (e.g., based on modeling). Since the scenario is

estimating central tendency dose, the mean or median contaminant concentration would be used. For the purposes of the example calculations shown below, it is assumed that the mean concentration of the contaminant in homegrown exposed vegetables is 1×10^{-3} mg/g for all consumers within the 1- to <11-year age range.

IR_{hg exp-veg per capita mean-adj}—The age-specific average intake rates of homegrown exposed vegetables for children in gardening households are estimated by first converting the consumer-only mean intake rate of homegrown exposed vegetables for the total population of gardening households (*IR_{hg exp-veg-consumer only mean}*) from *EFH* Table 13-60 to the mean per capita rate (*IR_{hg exp-veg-per capita mean}*) as follows:

$$IR_{hg\ exp-veg-per\ capita\ mean} = \frac{(IR_{hg\ exp-veg-consumer\ only\ mean} \times N_c)}{N_t} \quad (3)$$

where:

N_c = weighted number of gardening individuals who consumed homegrown exposed vegetables during the survey period (*EFH* Table 13-60); and

N_t = weighted total number of individuals who garden (*EFH* Table 13-4).

$$IR_{hg\ exp-veg-per\ capita\ mean} = \frac{1.57 \frac{g}{kg-d} \times 25,737,000}{68,152,000}$$

$$IR_{hg\ exp-veg-per\ capita\ mean} = 0.59 \frac{g}{kg-d}$$

This mean value (*IR_{hg exp-veg-per capita mean-unadj}*) represents the quantity of food brought into the house, and does not account for preparation or postcooking losses (i.e., unadjusted value).

The value can be adjusted to represent the quantity of food as-eaten (*IR_{hg exp-veg-per capita mean-adj}*) as follows:

$$IR_{hg\ exp-veg-per\ capita\ mean-adj} = IR_{hg\ exp-veg-per\ capita\ mean-unadj} \times \left(1 - \frac{\% \text{ preparation loss}}{100}\right) \times \left(1 - \frac{\% \text{ post cooking loss}}{100}\right) \quad (4)$$

where:

Preparation loss = 12% (see *EFH* Table 13-69); and

Postcooking loss = 22% (see *EFH* Table 13-69).

$$IR_{hg\ exp.veg.per\ capita\ mean.adj} = 0.59 \frac{g}{kg-d} \times \left(1 - \frac{12}{100}\right) \times \left(1 - \frac{22}{100}\right)$$

$$IR_{hg\ exp.veg.per\ capita\ mean.adj} = 0.41 \frac{g}{kg-d}$$

This mean per capita value represents the average rate of homegrown exposed vegetable intake across all age groups of the population that gardens; age-specific intake rates are not available in the *EFH* for homegrown exposed vegetable intake among gardening households. Thus, age-specific intake rates are estimated by assuming that the ratios of age-specific intake to total population intake for homegrown exposed vegetables for children in gardening households would be the same as the ratios for intake of all exposed vegetables (i.e., not just homegrown exposed vegetables, and not just gardening households), based on data from *EFH* Table 9-20 and presented here in Table 4.

Table 4. Estimation of age specific per capita mean homegrown exposed vegetable intake rates for children in gardening households (g/kg-day) for four age ranges among children aged 1 to <11 years

Age	Mean Per Capita Intake All Exposed Vegetables (g/kg-d)	Ratio of Age-Specific to Total Population Intakes of All Exposed Vegetables ¹	Mean Per Capita Intake Homegrown Exposed Vegetables; Gardening Households (g/kg-d) ²
Total Population	1.3	1.0	0.41
1 to <2 yr	2.0	1.54	0.63
2 to <3 yr	2.0	1.54	0.63
3 to <6 yr	1.6	1.23	0.50
6 to <11 yr	1.2	0.92	0.38

¹ Calculated as the age-specific intake rate for all exposed vegetables divided by the total population intake rate for all exposed vegetables (from *EFH* Table 9-20 [U.S. EPA, 2011a]).

² Calculated as the mean adjusted per capita intake of homegrown exposed vegetables for the total population of children in gardening households (0.41g/kg-d), times the ratios of age-specific to total population intakes of all exposed vegetables.

EF—Exposure frequency is 365 days per year for each age range because the intake rate used in this example represents a long-term average daily intake over the entire year. (It does not mean that contaminated homegrown exposed vegetables are consumed each day of the year; instead, any intake that occurs during the year is averaged over the year to yield an average daily rate.)

ED—Exposure duration is the length of time over which exposure occurs, in years. This example assumes that a child is exposed continuously from ages 1 to <11 years. Thus, exposure duration is assumed to be 1, 1, 3, and 5 years for the 1 to <2 year, 2 to <3 year, 3 to <6 year, and 6 to <11 year age ranges, respectively, for a total of 10 years.

AT—Because the chronic average daily dose is being calculated in this example, the averaging time is equivalent to the exposure duration expressed in days. To determine this value, 365 days/year is multiplied by the value of exposure duration for the given age range, and summed for all age ranges.

2.1.4. Calculations

Using the dose algorithm and exposure factors shown above, the potential $ADD_{hg\ exp-veg\ ing}$ for a child in a specific age range from consuming homegrown exposed vegetables is estimated by applying eq 2 to the exposure factor data for that age range. The value of $ADD_{hg\ exp-veg\ ing}$ depends on values established for $IR_{hg\ exp-veg\ per\ capita\ mean-adj}$, $C_{hg\ exp-veg}$, EF , ED , and AT . For children within the four age ranges, Table 5 presents mean point estimates of $ADD_{hg\ exp-veg\ ing}$.

Table 5. Summary of average daily dose of contaminant associated with consumption of homegrown exposed vegetables (mg/kg-day) for four age ranges among children in gardening households, aged 1 to <11 years

Dose Equation Parameters and Output	Age Ranges			
	1 to <2 yr	2 to <3 yr	3 to <6 yr	6 to <11 yr
$C_{hg\ exp-veg}$ (mg/g)	1.0×10^{-3}	1.0×10^{-3}	1.0×10^{-3}	1.0×10^{-3}
$IR_{hg\ exp-veg\ per\ capita\ mean-adj}$ (g/kg-d)	0.63	0.63	0.50	0.38
EF (d/yr)	365	365	365	365
ED (yr)	1	1	3	5
AT (d)	365	365	1,095	1,825
$ADD_{hg\ exp-veg\ ing}$ (mg/kg-d)	6.3×10^{-4}	6.3×10^{-4}	5.0×10^{-4}	3.8×10^{-4}

In the following calculation, the *ADDs* for each age group are averaged over the 10-year time period representing ages 1 to <11 years.

$$ADD_{hg \text{ exp-veg ing}} = \frac{\left[\left(6.3 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}} \times 1 \text{ yr} \right) + \left(6.3 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}} \times 1 \text{ yr} \right) + \left(5.0 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}} \times 3 \text{ yr} \right) + \left(3.8 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}} \times 5 \text{ yr} \right) \right]}{10 \text{ yr}}$$

$$ADD_{hg \text{ exp-veg ing}} = 4.7 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}}$$

2.1.5. Uncertainties

The example here presents results for the mean per capita dose via homegrown exposed vegetable ingestion for the population of children aged 1 to <11 years, in gardening households. High-end dose may be estimated by replacing the mean intake rates with upper-percentile values, or by using a high-end concentration with mean intake rates. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements. U.S. EPA (2011a) and Phillips and Moya (2012) provide information about converting upper-percentile consumer only intake rates to per capita upper-percentile rates. If a bounding estimate is desired, both the concentration in the homegrown exposed vegetables and the intake rates may be set to high-end or maximum values.

The estimate reflects per capita doses among children in households that garden. The per capita data for the population that gardens includes both individuals who ate homegrown exposed vegetables during the survey period as well as those that did not, but may eat homegrown exposed vegetables at some other time during the year. The uncertainties associated with this example include the source data for the intake rate and concentration data. The concentration of the chemical will vary depending on preparation and cooking methods. The intake data were collected more than 30 years ago and over a short period of 1 week for an estimated 3,000 children in 4,300 households across the U.S. The extrapolation of a short survey data over a long period adds to the uncertainty of the intake rate data. Therefore, these data may not reflect current eating patterns and long-term distributions. These data were considered to be collected using sound methodology and were considered to have a high degree of quality assurance. Although the data were adjusted to account for preparation losses, there is added

uncertainty in that adjustment factors based on a mixture of vegetables were used. This may not always be representative of the mixtures of vegetables eaten by the population of interest. There may also be uncertainties in the contaminant concentration as a result of sampling or analytical methods.

2.2. INGESTION OF CONTAMINATED SOIL AND DUST IN AND AROUND THE HOME: YOUNG CHILDREN AGED 1 TO <6 YEARS, CENTRAL TENDENCY, LIFETIME AVERAGE DAILY DOSE

2.2.1. Introduction

Exposure via ingestion of soil and dust can occur in areas where soil contamination exists. Indoor dust can also be contaminated with outdoor soil. Receptors could include all children, especially those who spend time playing both outdoors, and indoors on the floor. The dose via this exposure pathway is estimated based on the concentration of contaminants in outdoor soils or indoor dust at or near the child's residence, the intake rate, exposure frequency, and exposure duration. Young children are exposed to soil and dust primarily through hand-to-mouth and object-to-mouth activities. As defined in the *EFH* (U.S. EPA, 2011a), soil and dust are:

Soil. Particles of unconsolidated mineral and/or organic matter from the earth's surface that are located outdoors, or are used indoors to support plant growth. It includes particles that have settled onto outdoor objects and surfaces (outdoor settled dust).

Indoor Settled Dust. Particles in building interiors that have settled onto objects, surfaces, floors, and carpeting. These particles may include soil particles that have been tracked or blown into the indoor environment from outdoors as well as organic matter.

Outdoor Settled Dust. Particles that have settled onto outdoor objects and surfaces due to either wet or dry deposition. Note that it may not be possible to distinguish between soil and outdoor settled dust, since outdoor settled dust generally would be present on the uppermost surface layer of soil.

In this example, exposure via ingestion of soil and dust is assumed and the central tendency *LADD* from this pathway is evaluated for the population of young children who often play outdoors, and crawl and play on the floor indoors, and handle toys or other objects that may contain soil and/or dust (ages 1 to <6 years). The *LADD* is calculated because the contaminant in

this scenario is assumed to be a carcinogen and the carcinogen toxicity values are expressed as lifetime values; thus, the childhood exposure period must be spread over a lifetime. Furthermore, it is assumed that the receptor population is not exposed to this carcinogen after this exposure period.

2.2.2. Dose Algorithm

The *LADD* of a specified contaminant via ingestion of contaminated soil and dust is calculated as follows:

$$LADD_{soil + dust\ ing} = \frac{C_{soil + dust} \times CF \times IR_{soil + dust} \times EF \times ED}{BW \times LT} \quad (5)$$

where:

$LADD_{soil + dust\ ing}$	= potential lifetime average daily dose from ingestion of soil and dust (mg/kg-d);
$C_{soil + dust}$	= concentration of contaminant in soil and dust (mg/g);
CF	= conversion factor of 0.001 g/mg;
$IR_{soil + dust}$	= intake rate of soil and dust (mg/d);
EF	= exposure frequency (d/yr);
ED	= exposure duration (yr);
BW	= average body weight (kg); and
LT	= lifetime (d).

2.2.3. Input for Exposure Factor Variables

$C_{soil + dust}$ —The concentration of contaminants in soil and dust is either the measured level of the chemical of interest or predicted concentration, based on modeling. For estimating central tendency doses, the assessor typically uses an estimate of the mean or median concentration. For this example, the estimated mean concentration of chemical “x” in soil and dust is 1×10^{-3} mg/g.

CF —A conversion factor is required to convert between milligrams (mg) and grams (g), 0.001 g/mg to translate the intake rate to units of g/d.

$IR_{soil + dust}$ —The recommended central tendency intake rate of soil and dust for young children (1 to <6 years old) is 100 mg/d, (*EFH* Table 5-1).

EF—This example uses an exposure frequency of 350 days per year, assuming that young children are away from home (e.g., on vacation), the source of contamination, for two weeks per year. The home and surrounding yard are assumed to be the only sources of contamination.

ED—This example uses an exposure duration of 5 years (from age 1 to <6 years), based on the assumption that after 5 years of age, children no longer play in outdoor soil or crawl on the floor, and their soil and dust ingestion is limited compared to that of younger children.

BW—The average *BW* for children between the ages of 1 and <6 years can be estimated by calculating a time weighted average for children 1 to <2 years, 2 to <3 years, and 3 to <6 years. These *BWs* are provided in *EFH* Table 8-1. The average *BW* for 1 to <6 year old children is 16.2 kg using the calculation shown below. This *BW* was used in these example calculations.

$$BW = \frac{(11.4 \text{ kg} \times 1 \text{ yr}) + (13.8 \text{ kg} \times 1 \text{ yr}) + (18.6 \text{ kg} \times 3 \text{ yr})}{5 \text{ yr}}$$

$$BW = 16.2 \text{ kg}$$

LT—Because the contaminant used in this example is assumed to be a carcinogen, the dose is averaged over the lifetime (i.e., the *LADD* is calculated). A lifetime (*LT*) of 70 years for a member of the general population is used as a reference value. For use in the calculations, this value is converted to 25,550 days (i.e., 70 years × 365 days/year).

2.2.4. Calculations

Using the dose algorithm and exposure factors shown above, the *LADD_{soil + dust ing}* is estimated as follows for the population of young children:

$$LADD_{soil + dust \text{ ing}} = \frac{1 \times 10^{-3} \frac{\text{mg}}{\text{g}} \times 0.001 \frac{\text{g}}{\text{mg}} \times 100 \frac{\text{mg}}{\text{d}} \times 350 \frac{\text{d}}{\text{yr}} \times 5 \text{ yr}}{16.2 \text{ kg} \times 25,550 \text{ d}}$$

$$LADD_{soil + dust \text{ ing}} = 4.2 \times 10^{-7} \frac{\text{mg}}{\text{kg-d}}$$

2.2.5. Uncertainties

The example presented here is used to represent central tendency dose among a population of young children, ages 1 to <6 years, via ingestion of soil and indoor dust. The high end dose can be estimated by replacing the mean intake rate with a higher intake rate, or by using a high-end concentration with a mean intake rate. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements. If a bounding dose estimate is desired, the concentration of contaminants may also be set to the maximum measured or modeled concentration.

The uncertainties associated with this example scenario are mainly related to assumed activity patterns of the receptor population and the input parameters used. Soil ingestion rates are highly uncertain. Implicit in this scenario is that young children ages 1 to <6 years ingest soil and dust at the same intake rate specified in the *EFH* (U.S. EPA, 2011a). It should be noted that intake rate might decrease as activity patterns change with age. Also, the intake rate for children in specific age ranges may not represent long-term behaviors and day-to-day and seasonal variability. These input parameters are derived from data collected from a variety of studies focused on soil ingestion with limited data on dust ingestion. The uncertainties associated with this example are as follows: (1) the assumption is made that 100% of the soil and dust that the children ingest comes from their home environment (i.e., it does not consider any portion of soil intake that may come from time spent away from home such as at daycare or school, nor does it consider difference in contaminant concentration from sources other than the home environment); (2) the methodologies of the soil/dust intake studies are considered to have limitations with numerous sources of measurement error; (3) the studies have limited representativeness of the U.S. population; and (4) eight of the nine *EFH* supporting studies were focused on soil or combined soil and dust ingestion with no, or very limited, focus on dust ingestion. There may also be uncertainties in the contaminant concentration as a result of sampling or analytical methods. The assessor should also consider the bioavailability of the contaminant in soil and dust. The bioavailability will vary depending on the physicochemical characteristics of the contaminant and the characteristics of the soil and dust (e.g., particle size).

2.3. INGESTION OF CONTAMINATED INDOOR DUST: CHILDREN AT SCHOOL AGED 6 TO <11 YEARS, CENTRAL TENDENCY, SUBCHRONIC AVERAGE DAILY DOSE

2.3.1. Introduction

Indoor dust can become contaminated from a variety of sources (e.g., use of pesticides, building materials, particle-bound contaminants infiltrating indoors from outdoors). The exposure scenario for this example is ingestion of contaminated indoor dust at a school. Receptors could include all school children. This example assumes that the outdoor soil is not contaminated, and exposure occurs only to indoor dust at school. Dose via this pathway is estimated based on the concentration of contaminants in indoor dust, the intake rate of indoor dust, exposure frequency, exposure time, and exposure duration. In this example, exposure via ingestion of indoor dust at school is assumed and the central tendency subchronic (<7 years) average daily exposure from this pathway is evaluated for children ages 6 to <11 years. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

2.3.2. Dose Algorithm

The *ADD* of a specified contaminant via ingestion of contaminated dust by children at school is calculated as follows:

$$ADD_{dust\ ing} = \frac{C_{dust} \times CF \times IR_{dust} \times EF \times ET \times ED}{BW \times AT} \quad (6)$$

where:

$ADD_{dust\ ing}$	= potential subchronic average daily dose of the contaminant from ingestion of contaminated dust (mg/kg-d);
C_{dust}	= concentration of the contaminant in the ingested dust (mg/g dust);
CF	= conversion factor of 0.001 g/mg;
IR_{dust}	= average daily intake rate of dust for children aged 6 to <11 years (mg/d);
EF	= exposure frequency (d/yr);
ET	= exposure time (unitless fraction representing the portion of the day spent in school);
ED	= exposure duration (yr);
BW	= average body weight (kg); and
AT	= averaging time (d).

2.3.3. Input for Exposure Factor Variables

C_{dust} —The concentration of contaminants in indoor dust is either the measured level of the chemical of interest in the indoor dust or predicted concentration, based on modeling. For estimating central tendency doses, the mean or median values would be used. In this example, it is assumed that the mean concentration of chemical “x” in indoor dust is 1×10^{-3} mg/g.

CF —A conversion factor is required to convert between milligrams and grams, 0.001 g/mg.

IR_{dust} —The recommended central tendency intake rate of indoor dust for young children (6 to <11 years old) is 60 mg/day (*EFH* Table 5-1; U.S. EPA, 2011a).

EF —School is in session for approximately 37 weeks/year or 185 days/year. The school is assumed to be the only source of contamination.

ET —For “doers-only”, the average time spent in school for 6 to <11 year-old children is approximately 400 minutes/day (i.e., 6.7 hours/day) (*EFH* Table 16-17). Since dust ingestion only occurs during waking hours, the fraction of time spent in school should be based on waking hours and not on 24 hours. Children 6 to <11 years spend 613 minutes/day or 10 hours/day sleeping or napping (*EFH* Table 16-25; U.S. EPA, 2011a). Thus, these children are awake approximately 14 hours/day (24 hours/day – 10 hours/day). Assuming that 6.7 hours/day during the time they are awake is spent at school, 0.48 of the day (i.e., 6.7/14) is spent in school where they may be ingesting indoor dust. The school is assumed to be the only source of contamination.

ED —Exposure duration is the length of time over which the exposure occurs. In this example, an ED of 5 years (from age 6 to <11 years) is used. It is assumed that the children attend the contaminated school the entire 5 years.

BW — The average BW for children from 6- to <11-years old of 31.8 kg *EFH* Table 8-1 (U.S. EPA, 2011a).

AT —Because the subchronic ADD is being calculated in this example, the averaging time is equivalent to the exposure duration. The averaging time of 5 years is converted to 1,825 days for this calculation (i.e., 5 years \times 365 days/year).

2.3.4. Calculations

Using the dose algorithm and exposure factors shown above, the $ADD_{dust\ ing}$ is estimated as follows for the population of children:

$$ADD_{dust\ ing} = \frac{(1 \times 10^{-3}) \frac{\text{mg}}{\text{g}} \times 0.001 \frac{\text{g}}{\text{mg}} \times 60 \frac{\text{mg}}{\text{d}} \times 185 \frac{\text{d}}{\text{yr}} \times 0.48 \times 5 \text{ yr}}{31.8 \text{ kg} \times 1,825 \text{ d}}$$

$$ADD_{dust\ ing} = 4.6 \times 10^{-7} \frac{\text{mg}}{\text{kg-d}}$$

2.3.5. Uncertainties

The example presented here is used to represent central tendency dose among a population of school children, ages 6 to <11 years, via ingestion of indoor dust. For high-end estimates, a combination of upper-percentile and central tendency inputs would be used. The choice of which parameter(s) should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements. If a bounding dose estimate is desired, both the intake rate and the concentration of contaminant may be set to the upper-percentile values. It is important to note that the bounding estimate results in dose estimates that may be unreasonably high for the population of interest.

The uncertainties associated with this example scenario are mainly related to assumed activity patterns of the receptor population and the input parameters used. This scenario represents a central tendency dose; higher exposures may occur depending on activity patterns. Implicit in this scenario is that school children ages 6 to <11 years ingest indoor dust at the same intake rate specified in the *EFH* (U.S. EPA, 2011a). It should be noted that intake rates might decrease as activity patterns change with age. The periods studied may not represent long-term behaviors, and day-to-day and seasonal variability were not well characterized. These input parameters are derived from data collected from a variety of studies. The uncertainties associated with this example are as follows: (1) the methodologies of the dust intake studies are considered to have limitations with numerous sources of measurement error; (2) the dust intake studies have limited representativeness of the U.S. population; and (3) eight of the nine dust intake studies were focused on soil ingestion with no, or limited, focus on dust ingestion. These

facts make it difficult to characterize the individual contributions from soil and dust to the total intake.

The assessor may also consider the bioavailability of the contaminant in soil and dust. The bioavailability will vary depending on the physicochemical characteristics of the contaminant and the characteristics of the dust (e.g., particle size). Also, this scenario assumes that the contaminant is not present in outdoor soils and that children are not exposed to the contaminant in soil or dust outside the school environment. This may be true for some contaminants, however, indoor dust will often be a mixture of outdoor soils and indoor sources. Children who participate in after-school programs may spend more time at school. In those cases, it would be appropriate to use a higher number of hours spent at school to estimate the *ET*. It should be noted that the contaminant may be brought into other microenvironments (e.g., home) on the children's body, shoes, or clothing, in which case adjustments to the *ET* based on the fraction of time spent in school would not be appropriate. In addition, the approach used here assumes that dust ingestion occurs at a steady rate throughout the time that one is awake. The supporting studies are not detailed enough to show if this is true. For a more reliable dose estimate, a study would need to be conducted to specifically estimate the indoor dust intake rate for children. This scenario only considers exposure via dust ingestion, dermal exposure may also be a concern and would need to be evaluated.

2.4. INGESTION OF AN ENVIRONMENTAL CONTAMINANT BY NONDIETARY HAND-TO-MOUTH BEHAVIORS: INFANTS AND TODDLERS 3 MONTHS TO <2 YEARS, BOUNDING, ACUTE DOSE RATE

2.4.1. Introduction

Infants and toddlers exhibit a high frequency of hand-to-mouth and object-to-mouth behaviors. In instances where infants and toddlers are playing in a contaminated room, they may be exposed to contaminants through mouthing of contaminated surfaces and objects and/or transferring contaminants from surfaces to their hands and subsequently into their mouths. Potential exposure to contaminants by nondietary hand-to-mouth and object-to-mouth behaviors may occur, for example, after use of a sprayed biocide or household cleaning product. This example estimates bounding acute dose via ingestion of contaminants through hand-to-mouth behaviors for infants and toddlers ages 3 months to <2 years. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

2.4.2. Dose Algorithm

Dose via this pathway would be calculated as follows:

$$ADR_{hand-to-mouth\ ing} = \frac{[C_{surface} \times CR_{hand-to-mouth} \times CSA_{hands}] \times [TE_{SH} \times TE_{HM} \times ET \times EF \times ED]}{BW \times AT} \quad (7)$$

where:

$ADR_{hand-to-mouth\ ing}$	= acute potential dose rate from contaminated surface (mg/kg-d);
$C_{surface}$	= contaminant loading on surface (mg/cm ²);
$CR_{hand-to-mouth}$	= contact rate (contacts/hr);
CSA_{hands}	= contact surface area of hand (cm ² /contact);
TE_{SH}	= transfer efficiency from surface to hand (%);
TE_{HM}	= transfer efficiency from hand-to-mouth (%);
ET	= exposure frequency (hr/event);
EF	= exposure frequency (events/d);
ED	= exposure duration (d);
BW	= average body weight (kg); and
AT	= averaging time (d).

2.4.3. Input for Exposure Factor Variables

$C_{surface}$ —The contaminant loading on indoor surfaces can be measured or predicted based on modeling the chemical of interest on a floor surface or object. For estimating the acute dose in this example, the maximum value is used. For the purposes of the example calculations shown below, it is assumed that the modeled maximum loading of chemical “x” on a surface is 1×10^{-6} mg/cm².

$CR_{hand-to-mouth}$ —The upper-percentile contact rate for children is presented in *EFH* Table 4-1 (U.S. EPA, 2011a). In this example, the 95th percentile contact rates are used to calculate the acute bounding dose. For children ages 3 months to <6 months, 6 months to <12 months, and 1 year to <2 years, the 95th percentile contact rates are 65, 52, and 63 contacts per hour, respectively. The weighted average of these contact rates is 60 contacts per hour and is calculated as follows:

$$CR_{hand-to-mouth} = \frac{\left[\left(65 \frac{\text{contacts}}{\text{hr}} \times 3 \text{ mo} \right) + \left(52 \frac{\text{contacts}}{\text{hr}} \times 6 \text{ mo} \right) + \left(63 \frac{\text{contacts}}{\text{hr}} \times 12 \text{ mo} \right) \right]}{21 \text{ mo}}$$

$$CR_{hand-to-mouth} = 60 \frac{\text{contacts}}{\text{hr}}$$

CSA_{hands} —The surface area for body parts is presented in *EFH* Table 7-2 (U.S. EPA, 2011a). In this example, mean surface areas are used because surface area and *BW* are strongly correlated and the mean values are most representative of the surface area of individuals with an average *BW*. For children 3 months to <6 months, 6 months to <12 months, and 1 year to <2 years the mean contact surface areas (*CSA*) for hands are 0.020, 0.024, and 0.030 m² per contact, respectively. A square meter is converted to square centimeters using the conversion of 1 m² = 10,000 cm². For this example, the contact surface areas used to calculate the weighted average *CSA* are 200 cm² for 3 months to <6 months, 240 cm² for 6 months to <12 months, and 300 cm² for 1 year to <2 years. It is assumed that the contact surface area is 100% of the hands for children. The weighted *CSA_{hands}* is 269 cm² and is calculated as follows:

$$CSA_{hands} = \frac{\left[\left(200 \frac{\text{cm}^2}{\text{contact}} \times 3 \text{ mo} \right) + \left(240 \frac{\text{cm}^2}{\text{contact}} \times 6 \text{ mo} \right) + \left(300 \frac{\text{cm}^2}{\text{contact}} \times 12 \text{ mo} \right) \right]}{21 \text{ mo}}$$

$$CSA_{hands} = 269 \frac{\text{cm}^2}{\text{contact}}$$

TE_{SH} —The transfer efficiency (TE_{SH}) from surface to hand is 14%, as estimated in *EFH* Table 7-27 (U.S. EPA, 2011a). This is the highest transfer efficiency reported by Cohen-Hubal et al. (2005) from a study in which adult volunteers contacted surfaces treated with nontoxic fluorescent tracer material to estimate the percentage transferred from a variety of surface types by the hands under a variety of conditions. This maximum value represents the initial surface contact with “sticky” hands.

TE_{HM} —The transfer efficiency from hand-to-mouth (also referred to as the saliva transfer efficiency) is assumed to be 16%. This value is based on results reported by Kissel et al. (1998),

who estimated the geometric mean transfer efficiencies from hand-to-mouth from thumb sucking and finger mouthing to be 10.1% and 15.9%, respectively.

ET—The exposure time is assumed to be a one-time acute exposure. For the purpose of this example, a 4-hour visit to a contaminated indoor space is assumed.

EF—The exposure frequency is assumed to be one event for acute exposure. This means that residues are transferred to the hand during the day of exposure without washing and reloading.

ED—The exposure duration is the length of time over which exposure occurs. For the purposes of this example, the acute exposure duration is assumed to be 1 day.

BW—*EFH* Table 8-1 (U.S. EPA, 2011a) reports recommended values for *BWs* for children. Using the age-specific mean *BWs*, the average *BWs* of 7.4, 9.2, and 11.4 kg are used for the age ranges of 3 to <6 months, 6 to <12 months, and 1 year to <2 years, respectively. The weighted average *BW* for this example is as follows:

$$BW = \frac{(7.4 \text{ kg} \times 3 \text{ mo}) + (9.2 \text{ kg} \times 6 \text{ mo}) + (11.4 \text{ kg} \times 12 \text{ mo})}{21 \text{ mo}}$$

$$BW = 10.2 \text{ kg}$$

AT—Because the acute dose is being calculated, the averaging time is 1 day.

2.4.4. Calculations

Using the dose algorithm and exposure factors shown above, the potential *ADR* for hand-to-mouth ingestion of a contaminant, $ADR_{hand-to-mouth \text{ ing}}$, is estimated:

$$ADR_{hand-to-mouth \text{ ing}} = \frac{\left[(1.0 \times 10^{-6}) \frac{\text{mg}}{\text{cm}^2} \times 60 \frac{\text{contacts}}{\text{hr}} \times 269 \frac{\text{cm}^2}{\text{contact}} \times 0.14 \times 0.16 \times 4 \frac{\text{hr}}{\text{event}} \times 1 \frac{\text{event}}{\text{d}} \times 1 \text{ d} \right]}{10.2 \text{ kg} \times 1 \text{ d}}$$

$$ADR_{hand-to-mouth \text{ ing}} = 1.4 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}}$$

2.4.5. Uncertainties

The example demonstrates a bounding estimate of dose to infants and toddlers resulting from exposure to contaminated surfaces. Central tendency doses may be estimated by replacing the contact rates and concentrations with central tendency values (e.g., mean or median). The high-end dose may be estimated by using a combination of mean and upper-percentile values for the exposure factors. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements.

The uncertainties associated with this example scenario are related to assumed activity patterns and contact rates of the receptor populations. This is an acute scenario and the maximum concentration was used. However, for other measures of dose (e.g., central tendency), the variability in the chemical concentration in the different rooms of the house where the child spends his/her time may need to be considered. The studies that derived the recommended values for contact rates were conducted with very small sample sizes over short data collection periods and in a small number of locations in the U.S. The data may not be representative of long duration exposure or of large populations. In addition, the transfer efficiency studies were conducted using adults with organic fluorescent tracers, rather than children. Chemical-specific transfer efficiencies may differ from those assumed here. Transfer efficiency studies did not evaluate surface-to-hand and hand-to-mouth transfer efficiency for particles such as dust. Hand-to-mouth transfer efficiency was assumed to be 16%, and this value may vary depending on the chemical as well as other factors. The scenario also assumes that there is a constant loading of the contaminant into the hand after each mouthing event with no hand washing. In reality, there may not be a constant replenishment of the chemical into the hand before each mouthing event and the amount transferred to the mouth is a fraction of the loading that remains from the prior insertion. Özkaynak et al. (2011) developed a model to simulate frequent mouthing events without contaminant replenishment which estimates soil and dust ingestion that may be used to further refine this scenario example.

2.5. EXPOSURE TIME OF INCIDENTAL INGESTION OF CONTAMINATED POOL WATER TO REACH A REFERENCE DOSE LEVEL OF EXPOSURE: CHILDREN AGED 6 TO <11 YEARS, BOUNDING

2.5.1. Introduction

Children may incidentally ingest chemicals in the water when swimming. This scenario estimates how long it would take to exceed a reference dose (*RfD*) for children playing and/or swimming in a swimming pool containing the chemical of interest. For the purpose of this example, it is assumed that the chemical is well mixed in the pool water. Receptors in this example include swimmers in swimming pools (i.e., owned privately, municipally, or by schools). Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

This example is designed to derive a daily exposure time that one could have over a subchronic exposure duration, to a specified concentration, without exceeding the *RfD*. A bounding dose for acute incidental ingestion of pool water is evaluated here for children aged 6 to <11 years. As a bounding estimate, it is assumed that the children swim every day (i.e., 365 days/year) in the same pool, and the pool water is not drained or cleaned over the duration of the exposure. In other cases, the assessor may want to solve the dose equation for the maximum concentration to which the swimmer can be exposed without exceeding the *RfD*. In that case, the assessor needs to assume an exposure time.

2.5.2. Dose Algorithm

The time-to-exceed a given dose via ingestion of contaminated pool water would be calculated using the following dose equation (eq 8) rearranged to solve for exposure time and replacing $ADD_{water\ ing}$ with the *RfD* as shown in eq 9.

$$ADD_{water\ ing} = \frac{C_{pool\ water} \times IR_{pool\ water} \times ET \times EF \times ED}{BW \times AT} \quad (8)$$

$$ET = \frac{RfD_{water\ ing} \times BW \times AT}{C_{pool\ water} \times IR_{pool\ water} \times EF \times ED} \quad (9)$$

where:

$ADD_{water\ ing}$	= potential average daily dose of the contaminant from ingestion of contaminated pool water (mg/kg-d);
$RfD_{water\ ing}$	= reference dose (mg/kg-d);
$C_{pool\ water}$	= concentration of chemical in the pool water (mg/L);
$IR_{pool\ water}$	= water intake rate (L/hr);
ET	= exposure time (hr/d), length of time necessary to exceed the reference for the chemical;
EF	= exposure frequency (d/yr);
ED	= exposure duration (yrs);
BW	= average body weight (kg); and
AT	= averaging time (d).

2.5.3. Input for Exposure Factor Variables

$RfD_{water\ ing}$ —A reference dose is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (U.S. EPA, 2011b). For the purposes of the example calculations shown below, it is assumed that the RfD of chemical “x” is 1×10^{-3} mg/kg-day.

BW —*EFH* Table 8-1 (U.S. EPA, 2011a) reports recommended mean values for BW s for children. The age-specific mean BW for 6- to <11-year-old children of 31.8 kg BW is used in this example.

AT —Because the ADD is being calculated in this example, the averaging time is equivalent to the exposure duration. The averaging time of 5 years is converted to 1,825 days for this calculation (i.e., 5 years \times 365 days/year).

$C_{pool\ water}$ —The concentration in pool water at the site is either the measured or predicted concentration, based on modeling, of the chemical of interest in the pool water consumed. It also is assumed that the pool water is well mixed and the chemical is equally distributed in the water. For estimating bounding exposures, the maximum value would be used. For the purposes of the example calculations, it is assumed that the modeled maximum concentration of chemical “x” in pool water is 0.1 mg/L.

$IR_{pool\ water}$ —The upper-percentile intake rate for the swimmer is assumed to be 120 mL/hour or 0.12 L/hour from *EFH* Table 3-5 (U.S. EPA, 2011a).

EF —Exposure frequency is assumed to be 365 days/year for this bounding estimate.

ED—Exposure duration is assumed to be 5 years.

2.5.4. Calculations

Using the dose algorithm and exposure factor inputs shown above, the *ET* would be calculated as follows for children (ages 6 to <11 years):

$$ET = \frac{(1 \times 10^{-3}) \frac{\text{mg}}{\text{kg-d}} \times 31.8 \text{ kg} \times 1,825 \text{ d}}{0.1 \frac{\text{mg}}{\text{L}} \times 0.12 \frac{\text{L}}{\text{hr}} \times 365 \frac{\text{d}}{\text{yr}} \times 5 \text{ yr}}$$
$$= 2.7 \frac{\text{hr}}{\text{d}}$$

2.5.5. Uncertainties

The example presented is used to represent bounding doses among a specific population from the incidental ingestion of pool water. This scenario does not include dermal penetration or inhalation dose, both of which may contribute to total exposure to pool water. Central tendency doses may be estimated by replacing the intake rate and concentrations with mean or median values. Also, exposure frequency may be replaced by mean values obtained from *EFH* Table 16-1 (U.S. EPA, 2011a). High-end doses may be estimated by using a combination of central tendency and upper-percentile values for the intake rates, concentrations, and exposure frequency. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements.

The uncertainties associated with this example scenario are related to assumed activity patterns and intake rates of the receptor populations and include uncertainties such as ingestion differences due to playing or swimming behaviors. Implicit in this scenario is the assumption that the child swimmer or wader actually consumes the chemicals in the pool water at the rates specified. It also assumes that the child visits the same pool for all 5 years and that the same chemicals are used in the pool for those 5 years. These assumptions are appropriate for the bounding estimate, but may be unrealistic for other estimates (e.g., central tendency). Another uncertainty is that *RfDs* are developed to represent chronic doses and may not be applicable to subchronic doses. The intake rate of pool water has a high uncertainty, because the data available are limited for this factor. The intake rate used in this assessment is 0.12 L/hour as

reported from only one study (U.S. EPA, 2011a). This study does not break out age ranges and encompasses all children less than 16 years of age. It also is not considered representative of the U.S. population.

2.6. INGESTION OF CONTAMINATED DRINKING WATER: CHILDREN AGED <21 YEARS, DISTRIBUTION OF CHRONIC AVERAGE DAILY DOSE

2.6.1. Introduction

In areas where contaminated surface water or ground water is used as a source of drinking water, there is the potential for contaminant exposure via ingestion of tap water. The dose via ingestion of contaminated drinking water is estimated based on the concentration of contaminants in the drinking water, the intake rate of drinking water, exposure frequency, and exposure duration. In this example, exposure via ingestion of drinking water is assumed and a distribution of chronic average daily doses from this pathway is evaluated for the population of children, birth to <21 years. It is assumed that the children's community water is contaminated; therefore, both the home and school drinking water supplies are contaminated, and the children ingest contaminated water from birth (e.g., formula prepared with water) through childhood until 21 years of age. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

Instead of a point estimate, this example scenario uses a probabilistic method to estimate the distributions of contaminant intakes in the exposed population. This approach is used to demonstrate how a probabilistic method could be used to estimate a dose. It does not mean that this is the only, or most appropriate, method for this scenario. Distributions are used to represent the contaminant concentration and intake rates. All other input parameters are held constant at their central values. The final output distribution is developed using a Monte Carlo simulation with the Crystal Ball version 7 software.

2.6.2. Dose Algorithm

The $ADD_{drinking\ water\ ing}$ resulting from consumption of contaminated drinking water, is calculated for each age group as follows:

$$ADD_{drinking\ water} = \frac{C_{drinking\ water} \times IR_i \times EF \times ED_i}{AT} \quad (10)$$

where:

$ADD_{drinking\ water\ ing}$	= potential chronic average daily dose from ingestion of contaminated drinking water ($\mu\text{g}/\text{kg}\cdot\text{d}$);
$C_{drinking\ water}$	= concentration of contaminant in contaminated drinking water ($\mu\text{g}/\text{mL}$);
IR_i	= intake rate of drinking water for age group i ($\text{mL}/\text{kg}\cdot\text{d}$);
EF	= exposure frequency (d/yr);
ED_i	= exposure duration for age group i (yr); and
AT	= averaging time (d).

The $ADD_{drinking\ water\ ing}$ can also be calculated as the average over all of the age ranges as follows:

$$ADD_{drinking\ water} = \frac{C_{drinking\ water} \times EF \times \sum(IR_i \times ED_i)}{AT} \quad (11)$$

2.6.3. Input for Exposure Factor Variables

$C_{drinking\ water}$ —The concentration of contaminant in drinking water is either the measured or predicted concentration based on modeling. For the purposes of this example, it is assumed that a measurement survey of the contaminated drinking water produced a mean concentration of $1\ \mu\text{g}/\text{mL}$ with a standard deviation of $0.5\ \mu\text{g}/\text{mL}$. Contaminant concentrations in natural environments typically are log normally distributed (Cullen and Frey, 1999), and this was assumed for this example.

$IR_{drinking\ water}$ —The distribution of intake rate of drinking water for ages birth to <21 years are presented in *EFH* Table 3-19 (U.S. EPA, 2011a). These data are summarized in Table 6 for 11 age ranges of children <21 years of age. These data are for consumers-only of community water including both direct and indirect sources. Water consumption data are positively skewed and have been found to be well represented by log-normal distributions (Roseberry and Burmaster, 1992). The Crystal Ball version 7 software was used to fit the data to log-normal distributions based on the mean and 90th percentile values.

EF —Exposure frequency is assumed to be 350 days per year for each age range. This is equivalent to 50 weeks of living where the water is contaminated, and accounts for 2 weeks away from the contaminated area for vacations. This value was held constant in the simulation.

ED—Exposure duration is the length of time over which exposure occurs in each age group expressed in years. For example, the 1- to <2-year age group has an exposure duration of 1 year. This value was held constant in the simulation.

AT—Because the chronic *ADD* is being calculated in this example, the averaging time is equivalent to the exposure duration. This value was held constant in the simulation.

All input values are summarized in Table 7.

Table 6. Consumer-only estimates of direct and indirect water ingestion: community water (mL/kg-day)

Age range	Sample size	Mean	Percentiles						
			10	25	50	75	90	95	99
Birth to <1 mo	37	137 ^a	11 ^a	65 ^a	138 ^a	197 ^a	235 ^a	238 ^a	263 ^a
1 to <3 mo	108	119	12 ^a	71	107	151	228 ^a	285 ^a	345 ^a
3 to <6 mo	269	80	7	27	77	118	148	173 ^a	222 ^a
6 to <12 mo	534	53	5	12	47	81	112	129	186 ^a
1 to <2 yr	880	27	4	9	20	36	56	75	109 ^a
2 to <3 yr	879	26	4	9	21	36	52	62	121 ^a
3 to <6 yr	985	21	3	8	17	29	43	52	83
6 to <11 yr	1,410	17	2	6	13	23	35	47	78
11 to <16 yr	2,113	12	1	4	8	15	26	35	62
16 to <18 yr	944	10	1	4	8	15	23	30	47
18 to <21 yr	1,086	11	1	3	7	15	26	36	58

^aEstimates are less statistically reliable based on guidance published in the *Joint Policy on Variance Estimation and Statistical Reporting Standards on NHANES III and CSFII Reports: NHIS/NCHS Analytical Working Group Recommendations* (NCHS, 1993).

Source: U.S. EPA (2011a).

Table 7. Input values by age range for ingestion of contaminated drinking water

Age range	<i>C</i> ($\mu\text{g/mL}$) ^a	<i>IR</i> (mL/kg-d) ^a	<i>EF</i> (d/yr)	<i>ED</i> (yr)	<i>AT</i> (d)
0 to <1 mo	1.0	137	350	0.08	30
1 to <3 mo		119		0.17	60
3 to <6 mo		80		0.25	90
6 to <12 mo		53		0.5	180
1 to <2 yr		27		1	365
2 to <3 yr		26		1	365
3 to <6 yr		21		3	1,095
6 to <11 yr		17		5	1,825
11 to <16 yr		12		5	1,825
16 to <18 yr		10		2	730
18 to <21 yr		11		3	1,095

^aThis exposure factor is represented by a log-normal distribution with only the mean shown here.

2.6.4. Calculations

The distribution of potential $ADD_{drinking\ water\ ing}$, values in the exposed population was estimated by conducting a Monte Carlo analysis using the Crystal Ball version 7 software. A total of 10,000 trials were used in the simulation. For each trial, a set of parameter values was obtained by randomly sampling the parameter distributions described above and then computing the ADD . The $ADDs$ from each trial were combined to form the final distribution. The results are summarized in Table 8 and illustrated in Figure 1.

Table 8. Average daily doses (ADD; $\mu\text{g}/\text{kg}\text{-day}$) from birth to 21 years of age for ingestion of contaminated drinking water

Age range	Mean	SD	10%	50%	90%	95%
0 to <1 mo	145	103	52	119	266	334
1 to <3 mo	132	107	42	103	253	326
3 to <6 mo	87	65	29	69	165	210
6 to <12 mo	61	46	17	46	120	155
1 to <2 yr	31	26	9	23	61	77
2 to <3 yr	29	24	9	22	57	72
3 to <6 yr	24	20	7	18	47	61
6 to <11 yr	19	17	6	15	38	48
11 to <16 yr	14	13	4	10	28	36
16 to <18 yr	12	11	3	8	24	33
18 to <21 yr	13	13	3	10	27	38
Birth to <21 yr	21	12	9	18	37	42

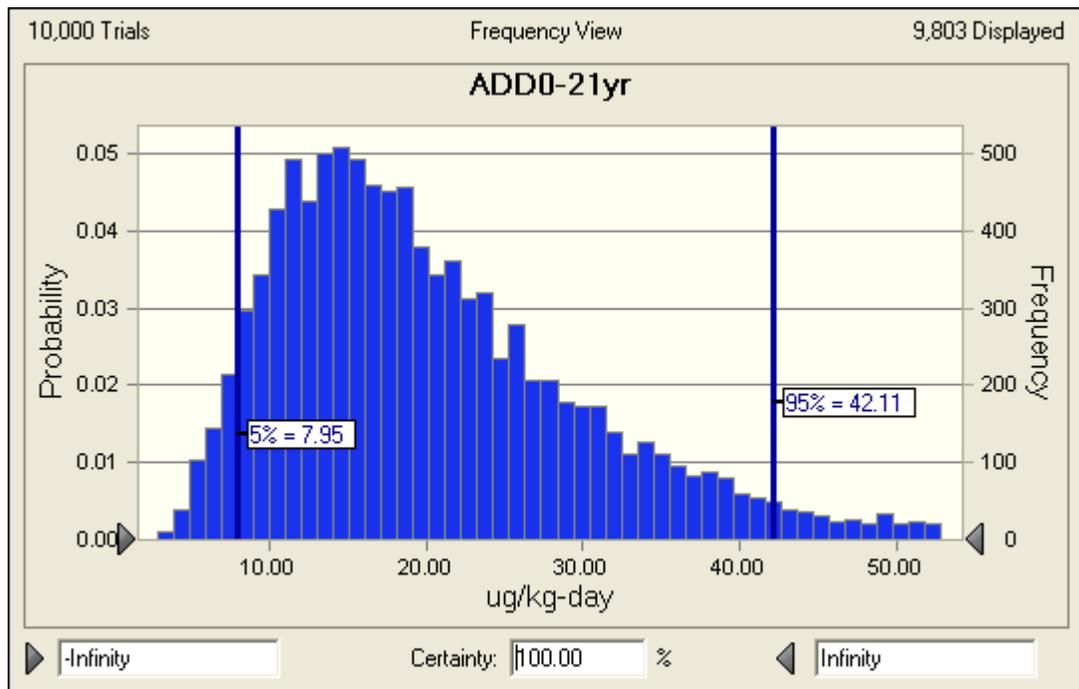


Figure 1. Distribution of average daily doses (ADD) from birth to 21 years of age.

2.6.5. Uncertainties

Log-normal distributions were used to represent drinking water intake rates for the purposes of this example because water consumption data have been found to be well represented by log-normal distributions (Roseberry and Burmaster, 1992). The fitted distributions were based on means and 90th percentiles; however, more accurate fits may be possible using more data. Alternatively, the raw data could be used to derive empirical distributions.

This scenario assumes that all of the ingested water is contaminated community tap water. However, children may ingest water from other sources that are either not contaminated or have different levels of contamination. The intake rates of drinking water in the *EFH* (U.S. EPA, 2011a) are derived from the data collected over a short period of time (2 days). The extrapolation to chronic intake in this example might introduce some degree of uncertainty and would not account for variation in children activity levels due to seasonal changes over a long timeframe of 21 years.

Information on uncertainty and variability and probabilistic risk analysis can be found in Chapters 1 and 2 of the *EFH* (U.S. EPA, 2011a). The Monte Carlo simulation conducted for this example scenario assumed that all variables were independent. However, drinking water intake rates for the age groups may be positively correlated. This is because generally a high water consumer at one age is likely to be a high water consumer at other ages. This positive correlation may not be too strong at the young ages included in this example because children's habits may change. This is a potential source of uncertainty in the simulation across all ages (i.e., birth to <21 years). The correlations between variables can be defined in the Crystal Ball version 7 program. To assess the possible uncertainty associated with the assumption of independence, a second simulation was run assuming a correlation coefficient of 0.8 between the drinking water intake rates for all age groups. The results did not change significantly.

The *ADD* could also be calculated using a deterministic approach. Assuming average inputs for all exposure parameters, the central tendency point estimate would be:

$$ADD_{drinking\ water} = \frac{1 \frac{\mu\text{g}}{\text{mL}} \times 350 \frac{\text{d}}{\text{yr}} \times [(137 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 0.08 \text{ yr}) + (119 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 0.17 \text{ yr}) + (80 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 0.25 \text{ yr}) + (53 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 0.5 \text{ yr}) + (27 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 1 \text{ yr}) + (26 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 1 \text{ yr}) + (21 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 3 \text{ yr}) + (17 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 5 \text{ yr}) + (12 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 5 \text{ yr}) + (10 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 2 \text{ yr}) + (11 \frac{\text{mL}}{\text{kg}\cdot\text{d}} \times 3 \text{ yr})]}{7,660 \text{ d}}$$

$$ADD_{drinking\ water} = 17.9 \mu\text{g/kg-d}$$

This *ADD* is similar to the central tendency estimate derived using the probabilistic approach as shown in Table 8.

2.7. INGESTION OF CONTAMINATED HUMAN MILK: INFANTS AGED BIRTH TO <12 MONTHS, HIGH-END, SUBCHRONIC AVERAGE DAILY DOSE

2.7.1. Introduction

For infants who are breastfed, the potential exists for intake of contaminants through nursing. The mother may be exposed to contaminants through ingestion, inhalation, and dermal absorption. In addition, the mother may have stores of contaminants in adipose tissue from past exposures. These contaminants may reenter the maternal circulation during pregnancy or nursing. Because of the high lipid content in human milk, contaminants that are lipophilic are likely to be passed to a nursing child via this exposure pathway. The aqueous portion of human milk also could allow transfer of hydrophilic contaminants to the infant. The potential receptors are infants who consume human milk with no other milk substitutes. In this example, exposure via contaminated human milk is assumed for infants who are breastfed from 0 to <12 months of age. Covered are four age ranges (0 to <1, 1 to <3, 3 to <6, and 6 to <12 months). Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X. According to the EFH (U.S. EPA, 2011a), the potential subchronic daily dose for all four of the age ranges should be calculated separately for each age range and then the weighted average calculated for the entire age range for this scenario. In this example, the high-end dose of a lipophilic contaminant is calculated using central estimates of the concentration of the contaminant in the lipid portion of human milk, the upper-percentile lipid intake rates of human milk, the exposure frequency, the exposure duration, and the averaging time.

2.7.2. Dose Algorithm

The *ADD* of a specified contaminant via ingestion of contaminated human milk is calculated as follows:

$$ADD_{human\ milk\ ing} = \frac{C_{lipid} \times IR_{lipid\ human\ milk} \times EF \times ED}{AT} \quad (12)$$

where:

$ADD_{human\ milk\ ing}$	= potential subchronic average daily dose from ingestion of contaminated human milk (mg/kg-d);
C_{lipid}	= concentration of contaminants in the lipid portion of contaminated human milk (mg/g lipid);
$IR_{lipid\ human\ milk}$	= lipid intake rate of human milk for infants who are fed human milk (g lipid/kg-d);
EF	= exposure frequency (d/mo);
ED	= exposure duration (mo); and
AT	= averaging time (d).

2.7.3. Input for Exposure Factor Variables

C_{lipid} —The concentration of a contaminant in the lipid portion of human milk is either the measured or predicted concentration based on modeling, of the contaminant in this medium. When estimating central tendency, the mean or median values would be used. For the purposes of the example calculations shown below, it is assumed that the central tendency concentration of chemical “x” in human milk is 1×10^{-3} mg/g lipid.

$IR_{lipid\ human\ milk}$ —The upper-percentile intake rate of lipids from human milk for infants from birth to <12 months of age can be estimated based on intake rate of lipids from human milk for infants provided in *EFH* Table 15-1 (U.S. EPA, 2011a). The intake rates presented in this table are normalized to *BW* and are calculated as the mean for each age range. It is assumed that in the general population, each age group is equally represented. The recommended intake rates are then converted from mL/kg-day to g/kg-day assuming the density of human milk is 1.03 g/mL (NAS, 1991). The lipid intake rates for human milk are summarized here in Table 9. For the purpose of this example, intake rates for exclusively breastfed infants are used. Exclusively breastfed infants are those whose sole source of milk comes from human milk, with no other milk substitutes (U.S. EPA, 2011a). Also, it is assumed that the contaminant is only present in the lipid portion of human milk.

Table 9. Upper-percentile lipid intake rates from human milk

Age range	Lipid intake rate (mL/kg-d)	Density-converted lipid intake rate (g/kg-d)
0 to <1 mo	8.7	9.0
1 to <3 mo	8.0	8.2
3 to <6 mo	6.1	6.3
6 to <12 mo	5.2	5.4

EF—Exposure frequency is 30 days/month.

ED—Exposure duration is the length of time over which the exposure occurs. For the purpose of this example, it is assumed that infants are breastfed for the first year of life. After that time, the infant’s diet is changed. Thus, the exposure duration is equal to the number of months within each age range.

AT—Because the subchronic *ADD* is being calculated in this example, the averaging time is equivalent to the exposure duration. This value is converted to days using 30 days/month for the purposes of this calculation and is different for each age range (Table 10).

2.7.4. Calculations

Using the dose algorithm (eq 12) and the input for the exposure factors shown above, the potential $ADD_{human\ milk\ ing}$ from human milk ingestion is estimated for the individual age ranges.

Table 10. Summary of *ADD* of contaminant associated with human milk for four age ranges among infants aged birth to <12 months

Equation 12 input and output	Age ranges			
	0 to <1 mo	1 to <3 mo	3 to <6 mo	6 to <12 mo
<i>C</i> (mg/g)	1×10^{-3}	1×10^{-3}	1×10^{-3}	1×10^{-3}
<i>IR</i> (g/kg-d)	9.0	8.2	6.3	5.4
<i>EF</i> (d/mo)	30	30	30	30
<i>ED</i> (mo)	1	2	3	6
<i>AT</i> (d)	30	60	90	180
<i>ADD</i> (mg/kg-d)	9.0×10^{-3}	8.2×10^{-3}	6.3×10^{-3}	5.4×10^{-3}

The weighted average *ADD* for the entire age range is calculated as follows:

$$ADD_{Human\ milk\ ing} = \frac{\left[\left((9.0 \times 10^{-3}) \frac{\text{mg}}{\text{kg-d}} \times 30\ \text{d} \right) + \left((8.2 \times 10^{-3}) \frac{\text{mg}}{\text{kg-d}} \times 60\ \text{d} \right) + \left((6.3 \times 10^{-3}) \frac{\text{mg}}{\text{kg-d}} \times 90\ \text{d} \right) + \left((5.4 \times 10^{-3}) \frac{\text{mg}}{\text{kg-d}} \times 180\ \text{d} \right) \right]}{360\ \text{d}}$$

$$ADD_{Human\ milk\ ing} = 6.4 \times 10^{-3} \frac{\text{mg}}{\text{kg-d}}$$

2.7.5. Uncertainties

The example presented here represents a high-end exposure scenario for the receptor population of infants 0 to <12 months of age who are breastfed. Central tendency and bounding dose estimates also can be derived from the data in Tables 9 and 10 using all central tendency or all upper-percentile input values, respectively.

This scenario specifies that a population of infants is breastfed the first year of life, using weight-averaged intake rates for human milk specified in the *EFH* (U.S. EPA, 2011a). It also is assumed here that the contaminant concentration is constant in human milk composition; however, studies have shown that contaminant concentrations may be affected by maternal nutrition (U.S. EPA, 2011a). In addition, the concentration of the chemical may decline as lactation continues and the mother's body burden declines.

This scenario also assumes a measured concentration. Models used to estimate human milk concentrations introduce additional uncertainties. Description of these models can be found in EPA's *Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities* (U.S. EPA, 2005d). Another assumption in this scenario is that the exposure is to the contaminant dissolved in the lipid portion of the human milk. However, some of the contaminant may also be present in the nonlipid portion of the human milk which is not considered here. The intake rate estimates are a source of uncertainty because they are based on studies from 1980–2000, where participants were white in the mid-to-upper socioeconomic classes. The intake rates are based on studies that have small sample sizes and cover short time periods. These may not be a good representation of intake rates over longer periods of time. Finally, there is uncertainty in the lipid content correction. It has been reported that the lipid

content of human milk varies with the length of time of nursing and increases from the beginning (foremilk) to the end (hindmilk) of the nursing session (NAS, 1991).

2.8. INGESTION OF CONTAMINATED RECREATIONAL ATLANTIC MARINE FINFISH: CHILDREN AGED 3 TO <6 YEARS, CENTRAL TENDENCY, SUBCHRONIC AVERAGE DAILY DOSE

2.8.1. Introduction

There is the potential for contamination of finfish and shellfish as a result of bioaccumulation of certain types of chemicals (e.g., methylmercury and lipophilic compounds such as dioxins and PCBs) in fish tissues. This may result in exposure among the general population via consumption of marine or freshwater fish. Receptors could include children of any age who consume contaminated fish. In this example, the dose via consumption of contaminated recreationally caught Atlantic marine finfish is estimated based on the concentration of chemicals in Atlantic marine finfish, intake rates of recreationally caught Atlantic marine finfish, exposure frequency, and exposure duration. In this example, central tendency subchronic average daily doses via ingestion of recreationally caught Atlantic marine finfish are evaluated for children (3 to <6 years of age) residing in a contaminated area. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

2.8.2. Dose Algorithm

The *ADD* of a specified contaminant via ingestion of contaminated marine finfish can be estimated as follows:

$$ADD_{fish\ ing} = \frac{C_{fish} \times IR_{fish} \times EF \times ED}{BW \times AT} \quad (13)$$

where:

- $ADD_{fish\ ing}$ = potential average daily dose from ingestion of fish caught at a contaminated site (mg/kg-d);
- C_{fish} = concentration of a contaminant in uncooked fish (mg/g fish);
- IR_{fish} = intake rate of uncooked recreational Atlantic marine finfish for the population of interest (g/d);
- EF = exposure frequency (d/yr);
- ED = exposure duration (yr);

BW = average body weight (kg); and
AT = averaging time (d).

2.8.3. Input for Exposure Factor Variables

C_{fish}—The concentration of a contaminant in Atlantic marine finfish is either the measured or predicted concentration, based on modeling. For estimating central tendency doses, the mean or median values would be used. For the purpose of the example calculations, it is assumed that the mean concentration of chemical “x” in uncooked fish is 1×10^{-3} mg/g.

IR_{fish}—Survey data for Atlantic marine finfish intake rates for children are relatively limited. However, a recommended mean consumption of recreationally caught Atlantic marine finfish for children 3 to < 6 years of age is 2.5 g/day, as measured uncooked, is provided in EFH Table 10-3. The value represents both survey anglers who ate recreational fish during the survey period and those that did not, but may eat recreationally caught fish during other periods. EFH Table 10-3 (U.S. EPA, 2011a) provides intake rates in terms of the wet weight mass of uncooked fish. The assessor should ensure that measurements of the contaminant concentration in fish also are on an uncooked, wet weight basis.

EF—Exposure frequency is 350 days/year because the data used in estimating *IR_{fish}* are assumed to represent average daily intake over a long term (i.e., over a year). However, it is assumed that the children are away from the contaminated source (i.e., on vacation) for 2 weeks during the year.

ED—Exposure duration is the length of time over which exposure occurs. This example assumes that children consume recreationally caught marine finfish for only the 3 years of life indicated.

BW—EFH Table 8-1 (U.S. EPA, 2011a), reports recommended values for *BWs* for children aged 3- to <6-years old. The mean *BW* for children in this age group is 18.6 kg.

AT—Because the subchronic *ADD* is calculated in this example, the averaging time is equivalent to the exposure duration. Thus, *AT* is 1,095 days (i.e., 3 years).

2.8.4. Calculations

$$ADD_{fish\ ing} = \frac{(1 \times 10^{-3}) \frac{mg}{g} \times 2.5 \frac{g}{d} \times 350 \frac{d}{yr} \times 3\ yr}{18.6\ kg \times 1,095\ d}$$

$$ADD_{fish\ ing} = 1.3 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}}$$

2.8.5. Uncertainties

The example presented here is used to represent central tendency doses among children, aged 3 to <6 years, via consumption of contaminated recreationally caught Atlantic marine finfish. High-end doses may be estimated by replacing the mean intake rate with an upper-percentile value or by using a high-end concentration and mean intake rate. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements. In addition, if a bounding dose estimate is desired, the concentration in fish also may be set to the maximum measured or modeled concentration. It is important to note that the bounding estimate may result in a dose estimate that is unreasonably high for the population of interest.

Uncertainties associated with this example scenario are related to assumed activity patterns of the receptor population and the input parameters used. Implicit in this scenario are the assumptions that children, aged 3 to <6 years, remain living near the site of contamination over that timeframe and consume only Atlantic marine recreationally caught finfish that were obtained from the contaminated site. As an alternative, a term denoting the fraction of fish assumed to be obtained from the source area could be included in the dose algorithm. Ideally, both concentration and intake rate data on a species-specific basis would be preferred. However, reliable estimates of species-specific intake rates are rarely available.

In this example, a single value for the average contaminant concentration in fish is used to estimate central tendency subchronic dose. This assumes that the average concentration of the fish consumed is equal to the sample/modeled average concentration. The variability in average contaminant concentration in fish might introduce some degree of uncertainty. In reality, the contaminant concentration may vary with the species of fish. Variability may result from differences in bioaccumulation of contaminants in different fish species, and in fish differing in size and placement in the food chain. Finally, the intake rate value associated with this scenario is derived from short-term consumption survey data used to estimate the distribution of the fish intake rate over a long period. There are no adjustments made for losses that may occur during preparation and cooking. The choice of using uncooked or as-consumed intake rate also depends

on how the contaminant concentration is reported. It is more conservative to use the uncooked intake rate values in this scenario; however, if the as-consumed information is desired, data are presented in *EFH* Table 13-69 (U.S. EPA, 2011a) to adjust the intake rate for moisture and fat loss during preparation.

In some instances, it also may be necessary to convert wet weight (either as-consumed or uncooked) intakes to dry weight intakes, or to convert wet weight into lipid weight intake rates (i.e., whichever state of preparation was used in the study reporting contaminant concentrations should also be used for the intake rates). The equations used in these conversions are presented in *EFH* Section 10.9 (U.S. EPA, 2011a). Below are the equations and sample calculations for converting the 2.5 g/day wet weight of uncooked fish used in this scenario to dry weight and to lipid weight. For these sample calculations, the average moisture and lipid contents for all marine fish and shellfish species from *EFH* Table 10-125 were calculated (U.S. EPA, 2011a). The average moisture and lipid content for all marine fish and shellfish species were 75.54% and 4.05%, respectively. The dry weight intake rate (g/day) is calculated by

$$IR_{dw} = IR_{ww} \left(\frac{100 - W}{100} \right) \quad (14)$$

where:

IR_{dw} = dry weight intake rate (g/d);
 IR_{ww} = wet weight intake rate (g/d); and
 W = percentage water content (%).

$$IR_{dw} = 2.5 \frac{\text{g}}{\text{d}} \left(\frac{100 - 75.54}{100} \right)$$

$$IR_{dw} = 0.61 \frac{\text{g}}{\text{d}}$$

The lipid weight intake rate (g/day) is similarly calculated by:

$$IR_{lw} = IR_{ww} \left(\frac{L}{100} \right) \quad (15)$$

where:

IR_{lw} = lipid weight intake rate (g/d);
 IR_{ww} = wet weight intake rate (g/d); and

L = percentage lipid (fat) content (%).

$$IR_{lw} = 2.5 \frac{\text{g}}{\text{d}} \left(\frac{4.05}{100} \right)$$

$$IR_{lw} = 1.0 \times 10^{-1} \frac{\text{g}}{\text{d}}$$

3. EXAMPLE INHALATION EXPOSURE SCENARIOS

Inhalation of a contaminant is not necessarily a simple function of inhalation rate and *BW*. The amount of chemical that reaches the target organ is highly dependent on the human respiratory anatomy and physiology and the physicochemical properties of the contaminant (U.S. EPA, 2009). Current EPA methodology uses the principles of inhalation dosimetry to determine the human equivalent concentration (HEC) for calculating an *RfC* or *IUR* (U.S. EPA, 2009, 1994). To apply the *RfC* methodology, it is unnecessary to calculate an inhaled dose when using toxicity values from the Integrated Risk Information System (IRIS) in a risk assessment. To estimate risk via inhalation, EPA recommends the use of the concentration of the chemical in air as the exposure metric (e.g., mg/m³), rather than inhalation intake of a contaminant in air based on intake rate and *BW* (e.g., mg/kg-d). Inhalation risk assessments require an air concentration adjusted to represent a continuous exposure. The risk calculations are different for noncarcinogens and carcinogens:

- For noncarcinogens, *RfCs* are used. These are expressed in concentration units. The *RfC* methodology uses the measured or modeled concentration of the chemical in the inspired air, adjusted to represent a continuous exposure.
- For carcinogens, IRIS uses unit risk values. These are expressed in inverse concentration units. In this approach, the unit risk is multiplied by the measured or modeled concentration of the chemical in the inspired air adjusted to represent a continuous exposure (U.S. EPA, 1994).

Advances in inhalation gas dosimetry show some evidence of higher inhaled doses in young children (i.e., 3 months) than in adults (U.S. EPA, 2012c). This life stage may warrant alternative modeling approaches or adjustments based on chemical-specific information. More information about advances in inhalation gas dosimetry and life stages can be found in EPA's *Advances in Inhalation Gas Dosimetry for Derivation of a Reference Concentration (RfC) and Use in Risk Assessment* (U.S. EPA, 2012c).

Example Exposure Scenarios 3.1 and 3.2 demonstrate how the adjusted air concentrations may be calculated. Although EPA recommends the use of the chemical concentration in air as the exposure metric, there may be cases where an inhalation dose is of interest. Estimations of cumulative doses or analyses of relative pathway contributions are examples where an inhaled

dose may be necessary. Example Exposure Scenarios 3.3 and 3.4 demonstrate the use of inhalation rates to fully calculate the *ADR* and *ADD*, respectively.

3.1. INHALATION OF CONTAMINATED AIR WHILE PLAYING IN A SCHOOL YARD: SCHOOL CHILDREN AGED 6 TO <11 YEARS, CENTRAL TENDENCY, SUBCHRONIC ADJUSTED AIR CONCENTRATION

3.1.1. Introduction

Contaminants may be released into the outdoor air from industrial sources or transportation. This may result in exposure via inhalation by residents, commercial/industrial workers, students, and recreational populations. The following is an example of an inhalation exposure estimate for elementary school-age children 6- to <11-years old exposed to contaminated outdoor air while playing in a school yard. Central tendency subchronic daily exposure from inhalation is evaluated for this population. This approach would be used in conjunction with subchronic toxicity values to characterize risk of adverse noncancer health effects. In this example, the outdoor air concentration is adjusted for the time children spent in school to reflect a continuous exposure. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

3.1.2. Exposure Algorithm

Adjusted outdoor air concentration via inhalation of contaminated air while playing in a school yard would be calculated as follows:

$$EC_{\text{outdoor air adjusted}} = \frac{C_{\text{outdoor air}} \times ET \times CF \times EF \times ED}{AT} \quad (16)$$

where:

$EC_{\text{outdoor air adjusted}}$ = adjusted exposure concentration of contaminant in the outdoor air (mg/m³);

$C_{\text{outdoor air}}$ = concentration of contaminant in the outdoor air (mg/m³);

ET = exposure time (min/d);

CF = conversion factor (d/min);

EF = exposure frequency (d/yr);

ED = exposure duration (yr); and

AT = averaging time (d).

3.1.3. Input for Exposure Factor Variables

$C_{outdoor\ air}$ —The concentration of contaminant in air at the site is either the measured or predicted concentration based on modeling, of the chemical of interest in the air at the site of interest. For estimating central tendency exposures, the mean or median values are used. For the purposes of this example, it is assumed that the mean measured concentration of chemical “x” in air is $1 \times 10^{-3} \text{ mg/m}^3$.

ET —*EFH* Table 16-19 (U.S. EPA, 2011a) reports the age-specific time spent on the playground at school for “doers-only.” The mean exposure time (ET) is 80 minutes/ day for children aged 6 to <11 years.

CF —The conversion factor needed to convert the exposure time of minutes to days is 1 day/1,440 minutes.

EF —For this example, exposure frequency is assumed to be 185 days/year. This is equivalent to 37 weeks of full-time school, and accounts for 15 weeks off for summer and winter vacation, or other school closings.

ED —Exposure duration is the length of time over which exposure occurs. For the purposes of this example, the ED for 6- to <11-year-old school children is assumed to be 5 years (i.e., first grade through fifth grade). This assumes that the exposure occurs in the same school yard where outdoor air contamination exists.

AT —The averaging time is equivalent to the exposure duration because this example assumes the assessor is evaluating risk of noncancer health effects. For the purposes of this example, the AT is converted to 1,825 days (i.e., 5 years \times 365 days/year).

3.1.4. Calculations

Using the exposure algorithm and the input for the exposure factor variables, the $EC_{outdoor\ air\ adjusted}$ for elementary school-age children would be as follows:

$$EC_{outdoor\ air\ adjusted} = \frac{1 \times 10^{-3} \frac{\text{mg}}{\text{m}^3} \times 80 \frac{\text{min}}{\text{d}} \times \frac{1 \text{ d}}{1,440 \text{ min}} \times 185 \frac{\text{d}}{\text{yr}} \times 5 \text{ yr}}{1,825 \text{ d}}$$

$$EC_{outdoor\ air\ adjusted} = 2.8 \times 10^{-5} \frac{\text{mg}}{\text{m}^3}$$

3.1.5. Uncertainties

The example presented here represents a central tendency adjusted air concentration of contaminant reflecting the inhalation exposures of the general population of elementary school children playing in a school yard. A high-end adjusted exposure concentration may be estimated by using a high-end concentration and also increasing exposure time or exposure frequency. Caution should be used, however, in setting all exposure factor inputs to upper-percentile values, as the resulting dose estimates may exceed reasonable maximum exposures for the population of interest. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements.

The uncertainties associated with this example scenario are related to the concentrations and assumed exposure time and exposure frequency. Uncertainty related to seasonal and weather variations (e.g., temperature, wind direction and strength) may lead to overestimates or underestimates of exposure. An underestimate of exposure concentration may also occur if the contaminant migrates from outdoors to indoors, resulting in additional exposures. This calculation does not account for these additional potential exposures, season or weather variations.

3.2. INHALATION OF AEROSOLIZED CONTAMINANTS FROM WATER DURING AND AFTER SHOWERING: CHILDREN AND TEENS AGED 6 TO <18 YEARS, CENTRAL TENDENCY, LIFETIME ADJUSTED AIR CONCENTRATION

3.2.1. Introduction

Because volatile contaminants can be released into the air from contaminated water, there is the potential for exposure among residents via inhalation during and after showering. In this example, the lifetime average adjusted exposure concentration from this pathway is evaluated for children and teens aged 6 to <18 years. It should be noted that exposures can also result from inhalation during other water uses around the household as well as dermal exposures. This scenario, however, focuses on the exposures via inhalation during and after showering. The approach below would be used for carcinogen exposure when using chronic toxicity values (e.g., from IRIS) in a risk assessment. It is assumed that children move away from the contaminated source after age 18 (e.g., moving away to attend college). Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X. If this is a mutagenic carcinogen,

appropriate ADAFs would need to be applied in accordance with *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b).

3.2.2. Exposure Algorithm

The adjusted exposure air concentration ($EC_{air\ adjusted}$) via inhalation would be calculated as follows:

$$EC_{air\ adjusted} = \frac{C_{air} \times ET \times CF \times EF \times ED}{LT} \quad (17)$$

where:

$EC_{air\ adjusted}$	= adjusted exposure concentration of contaminant in the air during and after showering (mg/m^3);
C_{air}	= concentration of contaminant in the air during and after showering (mg/m^3);
ET	= exposure time (min/d);
CF	= conversion factor (d/min);
EF	= exposure frequency (d/yr);
ED	= exposure duration (yr); and
LT	= lifetime (d).

3.2.3. Input for Exposure Factor Variables

C_{air} —The concentration of contaminants in air is either the measured or predicted concentration based on modeling, of the chemical of interest in the air of the shower room. For estimating central tendency exposures, mean or median values are used. For the purposes of this example, it is assumed that mean measured concentration of chemical “x” in air is $1 \times 10^{-3} \text{ mg}/\text{m}^3$.

ET —Exposure time is the estimated time spent showering and in the bathroom after showering occurs. *EFH* Table 16-29 (U.S. EPA, 2011a) presents the estimated time spent showering and time spent in the shower room immediately following showering. For the purpose of this example, the mean ET for children ages 6 to <11 years is 24 minutes/day and for teens ages 11 to <16 years is 26 minutes/day. Although the *Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants* (U.S. EPA, 2005a) recommends 16 to < 21 years as the next age group for children, for this scenario it was

assumed that children moved away from the contaminated source at age 18. Time spent in the shower and immediately following showering is not available for children 16 to < 18 years. Therefore, the value for 16 to < 21 years (i.e., 28 minutes/day) is used in this example. The average of the two 5-year and the 2-year age ranges is calculated as:

$$ET = \frac{\left(24 \frac{\text{min}}{\text{d}} \times 5 \text{ yr}\right) + \left(26 \frac{\text{min}}{\text{d}} \times 5 \text{ yr}\right) + \left(28 \frac{\text{min}}{\text{d}} \times 2 \text{ yr}\right)}{12 \text{ yr}}$$

$$ET = 25.5 \frac{\text{min}}{\text{d}}$$

CF—The conversion factor needed to convert the exposure time of minutes to days is 1 day/1,440 minutes.

EF—Exposure frequency is the number of times an exposure is expected to occur in a year and is expressed in days/year. For the purposes of this example, it is assumed that children shower 350 days of the year (i.e., once per day) assuming that they are away from the contaminated sources for approximately 2 weeks out of the year.

ED—Exposure duration is the length of time over which exposure occurs. For the purpose of this example, the *ED* is 5 years for the first two life stages and 2 years for the third life stage, i.e., 12 total years.

LT—The averaging time is equivalent to the lifetime of an individual in the receptor population because this example calculates the average exposure air concentration over a lifetime. For the purpose of this example, the average lifetime of 70 years for men and women is used because the exposure is assumed to reflect the general population. The value is converted to 25,550 days (i.e., 70 years × 365 days/year).

3.2.4. Calculations

Using the exposure algorithm and exposure factor inputs shown above, the $EC_{air\ adjusted}$ for children and teens during and after showering would be as follows:

$$EC_{air\ adjusted} = \frac{(1 \times 10^{-3}) \frac{\text{mg}}{\text{m}^3} \times 25.5 \frac{\text{min}}{\text{d}} \times \frac{1 \text{ d}}{1,440 \text{ min}} \times 350 \frac{\text{d}}{\text{yr}} \times 12 \text{ yr}}{25,550 \text{ d}}$$

$$EC_{air\ adjusted} = 2.9 \times 10^{-6} \frac{\text{mg}}{\text{m}^3}$$

3.2.5. Uncertainties

The example presented here is used to represent a central tendency adjusted air concentration for children and teens from exposure via inhalation to contaminated air during and after showering. An adjusted air concentration, based on high-end exposures may be estimated by using a combination of mean and upper-percentile values for the exposure factors. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements. If a bounding dose estimate is desired, the concentration of contaminants may be set to the maximum measured or modeled concentrations.

Uncertainties in this scenario relate to assumptions regarding the concentration variable used in the calculation. It was assumed that the concentration will be steady. It is unclear how well the average concentration of contaminant in indoor air represents the true time weighted average. This example assumes that the concentration was measured. Assessors typically estimate a different concentration in the shower stall than in the bathroom immediately after showering using models; however there are uncertainties with the models used to estimate these concentrations. The concentration will vary depending on various factors including for example: water temperature, volatility of the chemical, bathroom size, use of exhaust fan, water flow rate—just to name a few. This scenario also assumes that children are not exposed to the contaminant at other locations where they may spend their time. Another uncertainty relates to the amount of time spent showering and in the shower room immediately after. This example assumes that children take showers instead of baths. The time spent taking baths will be slightly higher than the time spent taking showers. It also assumes that children take one shower per day.

3.3. INHALATION OF CONTAMINATED INDOOR AIR: RESIDENTIAL CHILDREN AGED 3 TO <11 YEARS, BOUNDING, ACUTE DOSE RATE

3.3.1. Introduction

At sites where localized volatile contaminants intrude into residences either from the use of contaminated water in the household or from the use of commercial products or other materials results in indoor air contamination, there exists the potential for exposure among

residents via inhalation. In this example, a one-time exposure via inhalation of contaminated indoor air is assumed and the bounding acute dose from this pathway is evaluated for children aged 3 to <11 years playing at a residence. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X. An *ADR* is calculated in this example to be used as an input in a cumulative dose estimate or a relative pathway contribution analysis. This inhalation dose rate is not used to estimate risks. Risks from the inhalation pathway would be estimated by calculating a time weighted average exposure concentration or a peak exposure concentration to be used with an acute inhalation toxicity value. For chronic exposure scenarios, *RfCs* or *IURs* would be used, as appropriate.

3.3.2. Dose Algorithm

The *ADR* via inhalation of contaminated indoor air is calculated as follows:

$$ADR_{\text{indoor air inh}} = \frac{C_{\text{indoor air}} \times ET \times IR \times ED}{AT} \quad (18)$$

where:

- $ADR_{\text{indoor air inh}}$ = acute dose rate of contaminated indoor air (mg/kg-d);
- $C_{\text{indoor air}}$ = concentration of contaminants in the indoor air (mg/m³);
- ET = exposure time (min/d);
- IR = inhalation rate (m³/min-kg);
- ED = exposure duration (d); and
- AT = averaging time (d).

3.3.3. Input for Exposure Factor Variables

$C_{\text{indoor air}}$ —The concentration of a contaminant in air is either the measured or predicted concentration, based on modeling, of the chemical of interest in the air at the site of interest. For estimating bounding doses, the maximum values would be used. For the purpose of the example calculations, it is assumed that the modeled bounding concentration of chemical “x” in air from the breathing zone is 1×10^{-3} mg/m³.

ET —*EFH* Table 16-1 (U.S. EPA, 2011a) reports the age-specific time spent indoors at a residence over a 24-hour period. This scenario represents a one-time exposure for children at any age within the age range of 3 to <11 years. Therefore, a time weighted average exposure

time was estimated to represent a child within that age range. The upper-percentile times are 1,355 minutes/day (22.6 hours/day) for ages 3 to <6 years and 1,275 minutes/day (21.3 hours/day) for children ages 6 to <11 years. The weighted average of these is calculated as follows:

$$ET = \frac{\left(1,355 \frac{\text{min}}{\text{d}} \times 3 \text{ yr}\right) + \left(1,275 \frac{\text{min}}{\text{d}} \times 5 \text{ yr}\right)}{8 \text{ yr}}$$

$$ET = 1,305 \frac{\text{min}}{\text{d}}$$

IR—The recommended inhalation rates on a *BW* basis for children during short-term exposures are given in *EFH* Table 6-18 (U.S. EPA, 2011a). For the purpose of this example, it is assumed that the children spend some time sleeping and napping, some time playing at light intensity, and some time sedentary. A weighted average inhalation rate is calculated using inhalation rate data for each age range (i.e., 3 to <6 years and 6 to <11 years). Children age 3 to <6 years spend an average of 681 minutes/day (11.4 hours/day) sleeping or napping (*EFH* Table 16-25). Whereas children age 6 to <11 years spend 613 minutes/day (10.2 hours/day) sleeping or napping. In this example, the rest of the time is apportioned equally between sedentary and light activities for each age range (i.e., [(1,355 minutes/day – 681 minutes/day) ÷ 2 = 337 minutes/day] for children 3 to <6 years; and [(1,275 minutes/day – 613 minutes/day) ÷ 2 = 331 minutes/day] for children 6 to <11 years). The 95th percentile inhalation rate values for sleeping or napping are 3.5×10^{-4} m³/minute-kg for children from age 3 to <6 years and 2.1×10^{-4} m³/minute-kg for children from age 6 to <11 years (*EFH* Table 6-18). The 95th percentile sedentary inhalation rate values are 3.5×10^{-4} m³/minute-kg for children from age 3 to <6 years and 2.2×10^{-4} m³/minute-kg for children from age 6 to <11 years. The 95th percentile inhalation rate values for a light intensity activity are 8.7×10^{-4} m³/minute-kg for children from age 3 to <6 years and 5.3×10^{-4} m³/minute-kg for children from age 6 to <11 years. Using these values, the adjusted inhalation rate for activity intensity is calculated below for each age range.

$$IR_{3\ to < 6} = \frac{\left[\left((3.5 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 681 \frac{\text{min}}{\text{d}} \right) + \left((3.5 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 337 \frac{\text{min}}{\text{d}} \right) + \left((8.7 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 337 \frac{\text{min}}{\text{d}} \right) \right]}{1,355 \frac{\text{min}}{\text{d}}}$$

$$IR_{3\ to < 6} = 4.8 \times 10^{-4} \frac{\text{m}^3}{\text{min}\cdot\text{kg}}$$

$$IR_{6\ to < 11} = \frac{\left[\left((2.1 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 613 \frac{\text{min}}{\text{d}} \right) + \left((2.2 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 331 \frac{\text{min}}{\text{d}} \right) + \left((5.3 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 331 \frac{\text{min}}{\text{d}} \right) \right]}{1,275 \frac{\text{min}}{\text{d}}}$$

$$IR_{6\ to < 11} = 3.0 \times 10^{-4} \frac{\text{m}^3}{\text{min}\cdot\text{kg}}$$

The weighted average *IR* is calculated as follows:

$$IR_{3\ to < 11\text{yr}} = \frac{\left((4.8 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 3\ \text{yr} \right) + \left((3.0 \times 10^{-4}) \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 5\ \text{yr} \right)}{8\ \text{yr}}$$

$$IR_{3\ to < 11\text{yr}} = 3.7 \times 10^{-4} \frac{\text{m}^3}{\text{min}\cdot\text{kg}}$$

ED—Exposure duration is the length of time over which exposure occurs. For the purpose of this example, the exposure duration is assumed to be 1 day.

AT—Because the *ADR* is calculated in this example, the averaging time is equivalent to the exposure duration—1 day.

3.3.4. Calculations

Using the dose algorithm and exposure factors shown above, the $ADR_{indoor\ air\ inh}$ is estimated using the input for children ages 3- to <11-years old.

$$ADR_{indoor\ air\ inh} = \frac{1 \times 10^{-3} \frac{\text{mg}}{\text{m}^3} \times 1,305 \frac{\text{min}}{\text{d}} \times 3.7 \times 10^{-4} \frac{\text{m}^3}{\text{min}\cdot\text{kg}} \times 1 \text{ d}}{1 \text{ d}}$$

$$ADR_{indoor\ air\ inh} = 4.8 \times 10^{-4} \frac{\text{mg}}{\text{kg}\cdot\text{d}}$$

3.3.5. Uncertainties

The example presented here is used to represent bounding dose among residential children from inhalation of contaminated indoor air. Central tendency doses may be estimated by using mean values for contaminant concentration, exposure time, and inhalation rate. If a high-end dose estimate is desired, some high-end and some central tendency input values may be used. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements.

The uncertainties associated with this example scenario are related to the variation in activity level of the children during the day. Another uncertainty relates to the contaminant concentration. A contaminant will disperse and have varying concentrations throughout the residence. These variations may be modeled, but for the purpose of this example it is assumed that the concentration is constant throughout the home. In reality, the concentration will be affected by seasonality, temperature, and house characteristics. In addition, this scenario assumes that children are not exposed to the contaminant outside the residence.

3.4. INHALATION OF CONTAMINATED AIR DURING BUS TRANSPORTATION: SCHOOL CHILDREN AND TEENS AGED 6 TO <16 YEARS, HIGH-END, CHRONIC AVERAGE DAILY DOSE

3.4.1. Introduction

Air contamination on a bus may occur via (1) intrusion of volatile contaminants from outdoor ambient air, (2) volatilization from personal consumer products, and (3) volatilization from commercial materials in seats and flooring on the bus. This may result in exposure via inhalation. For the purposes of this example, exposure among school children and teens 6 to

<16 years old via inhalation of contaminated air occurs during bus transportation to and from school, which may include infiltration of the bus exhaust. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X. High-end chronic daily dose from inhalation is evaluated for doers-only of this population. An *ADD* is calculated in this example to be used as input in a cumulative dose estimate or a relative pathway contribution analysis. This inhalation dose estimate is not used to estimate risks. Risks from the inhalation pathway would be estimated by calculating a time weighted average concentration or a peak concentration to be used with a chronic toxicity value (e.g., *RfC* or *IUR*).

3.4.2. Dose Algorithm

Dose via this pathway would be calculated as follows:

$$ADD_{air\ inh} = \frac{C_{air} \times IR \times ET \times EF \times ED}{AT} \quad (19)$$

where:

<i>ADD_{air inh}</i>	= average daily dose of contaminated air on bus transportation (mg/kg-d);
<i>C_{air}</i>	= concentration of contaminant in the air on bus transportation (mg/m ³);
<i>IR</i>	= inhalation rate (m ³ /min-kg);
<i>ET</i>	= exposure time (min/event);
<i>EF</i>	= exposure frequency (events/yr);
<i>ED</i>	= exposure duration (yr); and
<i>AT</i>	= averaging time (d).

3.4.3. Input for Exposure Factor Variables

C_{air}—The concentration of contaminant in air at the site is either the measured or predicted concentration, based on modeling, of the chemical of interest in the air on bus transportation. For the purposes of this example, for a high-end dose, the 95th percentile concentration of chemical “x” in air is 1×10^{-3} mg/m³.

IR—The recommended inhalation rates for children during short-term exposures are given in *EFH* Table 6-2 (U.S. EPA, 2011a). For the purpose of this example, it is assumed that the children’s activity level is sedentary/passive. The mean inhalation rate values used for this example are 1.60×10^{-4} m³/minute-kg for children aged 6 to <11 years and 1.05×10^{-4} m³/minute-kg for children aged 11 to <16 years.

ET—The exposure time is estimated from *EFH* Table 16-23 (U.S. EPA, 2011a) for doers-only on bus transportation. The 95th percentiles for school children aged 6 to <11 years and teens aged 11 to <16 years are 107 minutes and 131 minutes per day, respectively.

EF—Exposure frequency is assumed to be 185 days/year for this example. This is equivalent to 37 weeks of full-time school, accounting for 15 weeks off for summer and winter vacation, or other school closings.

ED—Exposure duration is the length of time over which exposure occurs. For the purpose of this example, the *ED* is 5 years for each age range (10 years total). This assumes that all 10 years of the school commuting time are spent traveling through the same contaminated area and that the same contaminants are present in the bus exhaust.

AT—Because the chronic *ADD* is being calculated in this example, the averaging time is equivalent to the exposure duration. For the purposes of this example, the averaging time for each age group is converted to 1,825 days (i.e., 5 years × 365 days/year).

3.4.4. Calculations

Using the dose algorithm and input for the exposure factors, the *ADD* for each of the age ranges is calculated in Table 11.

Table 11. Summary of *ADD* for children and teens aged 6 to 16 years for inhalation of contaminated air on bus transportation

Equation 19 input and output	Age ranges	
	6 to <11 yr	11 to <16 yr
<i>C</i> (mg/m ³)	1 × 10 ⁻³	1 × 10 ⁻³
<i>IR</i> (m ³ /min-kg)	1.60 × 10 ⁻⁴	1.05 × 10 ⁻⁴
<i>ET</i> (min/d)	107	131
<i>EF</i> (d/yr)	185	185
<i>ED</i> (yr)	5	5
<i>AT</i> (d)	1,825	1,825
<i>ADD</i> (mg/kg-d)	8.7 × 10⁻⁶	7.0 × 10⁻⁶

The *ADD* for school children and teens averaged over the 10 years would be calculated as follows:

$$ADD_{air\ inh} = \frac{\left(8.7 \times 10^{-6} \frac{\text{mg}}{\text{kg-d}} \times 5 \text{ yr}\right) + \left(7.0 \times 10^{-6} \frac{\text{mg}}{\text{kg-d}} \times 5 \text{ yr}\right)}{10 \text{ yr}}$$

$$ADD_{air\ inh} = 7.9 \times 10^{-6} \frac{\text{mg}}{\text{kg-d}}$$

3.4.5. Uncertainties

The example presented here represents high-end doses among the general population of school children and teens from the inhalation of contaminated air during bus transportation and uses a high-end concentration with a mean inhalation rate. Alternatively, the high-end can be estimated by using a mean concentration with a high-end inhalation rate. The choice of which parameter should be set to the high-end would be dependent upon the sensitivities of the parameters, professional judgement, and regulatory requirements. Central tendency doses may be estimated by decreasing exposure time and using a mean concentration instead of a 95th percentile concentration.

The uncertainties associated with this example scenario are related to the contaminant concentration and assumed exposure time and exposure frequency. The concentration may be uncertain because it is typically obtained from outdoor measurements and using some penetration modeling factors or tools. The study used to derive the assumption for exposure time was conducted with small sample sizes and over short data collection period (24 hours) (U.S. EPA, 2011a). Therefore, the data may not be representative of long term behaviors. The exposure frequency may be higher if children attend summer school. This scenario also assumes that children are not exposed to the contaminant at other locations where they may spend time. There are also uncertainties associated with the inhalation rate data and assumptions regarding activity levels. These inhalation rates were derived from a wide range of groups within the U.S. population using indirect methods (U.S. EPA, 2011a).

4. EXAMPLE DERMAL EXPOSURE SCENARIOS

4.1. DERMAL CONTACT WITH CONTAMINATED SOIL: TEEN ATHLETES AGED 11 TO <16 YEARS, CENTRAL TENDENCY, SUBCHRONIC AVERAGE DAILY DOSE

4.1.1. Introduction

At sites where localized soil contamination exists, there is the potential for exposure via dermal contact with soil during outdoor activities. Exposure also may occur from soil that is tracked into the home or other buildings (i.e., schools, businesses, etc.). Therefore, receptors could include nearby residents, students, and recreational populations. Estimating the dose via dermal contact with the soil considers not only the concentrations of contaminants in the soil, but also the surface area of the skin that contacts the soil, the amount of soil that adheres to the skin per unit surface area, the fraction of contaminant in the soil that penetrates the skin, and the frequency and duration of exposure. For the purposes of this example, the dose to teen athletes via dermal contact with contaminated soil is calculated. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X. A subchronic dermal *ADD* from soil contact is evaluated for the teen athlete. For this example, a teen athlete (aged 11 to <16 years) playing soccer for one-half of the year is evaluated. It is assumed that the athlete is exposed to the same source of contaminated soil during the entire duration of exposure. An average soil concentration is used to account for the variability of soil concentration at the site.

4.1.2. Dose Algorithm

The *ADD* of a contaminant by dermal contact in a soil would be calculated as follows:

$$ADD_{ABS \text{ soil contact dermal}} = \frac{C_{soil} \times CF \times EF \times ED \times ABS \times \frac{SA}{BW} \times AF_{soil}}{AT} \quad (20)$$

where:

$ADD_{ABS \text{ soil contact dermal}}$ = absorbed average daily dose from dermal contact with contaminated soil (mg/kg-d);

C_{soil} = concentration of contaminant in the soil at the site (mg/kg);

CF = conversion factor (kg/mg);

EF = exposure frequency (events/yr);

<i>ED</i>	= exposure duration (yr);
<i>ABS</i>	= absorption fraction; this value is chemical specific (unitless);
<i>SA/BW</i>	= surface area of the skin that contacts the soil (cm ² /event-kg);
<i>AF_{soil}</i>	= adherence factor for soil (mg/cm ²); and
<i>AT</i>	= averaging time (d).

4.1.3. Input for Exposure Factor Variables

C_{soil}—The concentration of contaminant in soil at the site is either the measured or predicted concentration based on modeling, of the chemical of interest in the soil at the site of interest. For the purposes of the example calculations, it is assumed that the mean measured concentration of chemical “x” in soil to which the athlete is exposed is 1×10^{-3} mg/kg. Also assumed is that the concentration of contaminant in the soil that contacts the skin surface and is available to be absorbed.

CF—A conversion factor of 1×10^{-6} kg/mg is required to convert between mg/kg and kg/mg.

EF—Exposure frequency is the number of times that exposure is expected to occur in a year. Exposure frequency is assumed to be 130 events/year (i.e., 130 days/year). This assumes that individuals contact soil from athletic fields once per day for 5 days/week, 6 months/year (i.e., assumes no exposure associated with this athletic activity during the winter and summer months). It should be noted that this frequency assumption is used for illustrative purposes only. There may be cases where the exposure frequency is higher or lower. An implicit assumption of this scenario is that exposure (and absorption of the contaminants through the skin) occurs for each event in which soil contacts (and adheres to) a given surface area of the skin. This occurs without regard to the duration of the exposure event because a certain fraction of the contaminant in the soil on the skin is assumed to be absorbed for each event. Also assumed is that one ‘event’ occurs on each of the 130 days/year; on these occasions, soil adheres to the skin and remains there for the duration of the exposure event (i.e., until the fraction of contaminant specified in the absorption fraction (*ABS*) assumption below has been absorbed). However, in some cases alternate loading and removal mechanisms may affect the frequency of events (i.e., if hand washing and reloading of soil on the skin occurs).

ED—Exposure duration is the length of time over which exposure occurs. For the purposes of this example, the *ED* for 11- to <16-year-old teen is assumed to be 5 years. This assumes that 5 years are spent playing soccer on contaminated athletic fields.

ABS—This value is chemical specific. Information on absorption fractions can be obtained from EPA’s *Dermal Exposure Assessment: Principles and Applications* (U.S. EPA, 1992b). EPA also has developed the draft Part E Supplemental Guidance for Dermal Risk Assessment of the *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual* (U.S. EPA, 2004b). This document is a source of data on dermal absorption. For the purposes of the calculations provided below for this example, it is assumed that the absorption fraction for the chemical of interest (i.e., chemical “x”) is 0.1 (i.e., 10%). Thus, it is assumed that 10% of the chemical in the soil contacting the skin is absorbed.

SA/BW—*SA/BW* is the ratio of skin surface area to *BW* and is expressed as cm² of skin surface area per kg of *BW*. Assumptions have been made regarding the surface area of specific body parts that are expected to be exposed to soil per event. For this example scenario (i.e., teen athlete), it is assumed that an individual will wear short pants and a short-sleeved shirt, and that the hands, arms, and legs will come into contact with the soil. *EFH* Table 7-2 provides the mean recommended surface area contribution of hands, arms, and legs in m² needed to obtain a summed surface area value. Next, the summed surface area value is converted into cm² using the conversion factor of 1 m² = 10,000 cm². The total estimated surface area of the hands, arms, and legs is divided by the *BW* to yield the *SA/BW* ratio (cm²/kg). The mean *BW* for children in this age range, 56.8 kg, was obtained from *EFH* Table 8-1.

$$SA_{hands} = \frac{0.072 \text{ m}^2 \times 10,000 \frac{\text{cm}^2}{\text{m}^2}}{56.8 \text{ kg}} = 12.7 \frac{\text{cm}^2}{\text{kg}}$$

$$SA_{arms} = \frac{0.227 \text{ m}^2 \times 10,000 \frac{\text{cm}^2}{\text{m}^2}}{56.8 \text{ kg}} = 40.0 \frac{\text{cm}^2}{\text{kg}}$$

$$SA_{legs} = \frac{0.483 \text{ m}^2 \times 10,000 \frac{\text{cm}^2}{\text{m}^2}}{56.8 \text{ kg}} = 85.0 \frac{\text{cm}^2}{\text{kg}}$$

AF_{soil} —The assessor should use adherence factor data that most closely resemble the exposure scenario of concern. In this case, the mean AF_{soil} for outdoor activities were used. The soil adherence factors for this scenario were obtained from *EFH* Table 7-4 (U.S. EPA, 2011a) and are tabulated in Table 12.

Table 12. Soil adherence factors (AF_{soil}) for teen athletes playing soccer

Body parts	Mean adherence factors (mg/cm ²)
Hands	0.11
Arms	0.011
Legs	0.031

AT —Because the ADD is being calculated for a specific age range (e.g., 11- to <16-year-old teens), the averaging time is equivalent to the exposure duration, except that the duration is expressed in days. For use in the calculations, this value is converted to 1,825 days (i.e., 5 years \times 365 days/year).

4.1.4. Calculations

Using the dose algorithm and exposure factor inputs shown above, the $ADD_{ABS\ soil\ contact\ dermal}$ would be calculated for each body part exposed and summed across body parts as follows:

$$ADD_{ABS\ soil\ contact\ dermal} = \frac{\left((1 \times 10^{-3}) \frac{\text{mg}}{\text{kg}} \times (1 \times 10^{-6}) \frac{\text{kg}}{\text{mg}} \times 130 \frac{\text{events}}{\text{yr}} \times 5 \text{ yr} \times 0.1 \right) \times \left(\left(12.7 \frac{\text{cm}^2}{\text{event}\cdot\text{kg}} \times 0.11 \frac{\text{mg}}{\text{cm}^2} \right) + \left(40.0 \frac{\text{cm}^2}{\text{event}\cdot\text{kg}} \times 0.011 \frac{\text{mg}}{\text{cm}^2} \right) + \left(85.0 \frac{\text{cm}^2}{\text{event}\cdot\text{kg}} \times 0.031 \frac{\text{mg}}{\text{cm}^2} \right) \right)}{1,825 \text{ d}}$$

$$ADD_{ABS\ soil\ contact\ dermal} = 1.6 \times 10^{-10} \frac{\text{mg}}{\text{kg}\cdot\text{d}}$$

4.1.5. Uncertainties

The example presented here is used to represent central tendency doses among teen athletes, aged 11 to <16 years, from dermal contact with contaminated soil. High-end doses may be estimated by assuming a different clothing scenario and replacing the assumptions regarding areas of the body exposed (e.g., including the face and torso). Alternatively, exposure frequency

may be increased to estimate high-end doses. If a bounding dose estimate is desired, the concentration in soil may be set to the maximum measured or modeled concentration, the assumed frequency of exposure may be increased (e.g., 250 times/year; 5 days/week; 12 months/year), and the clothing scenario may be revised to increase the areas of the body exposed. It is important to note that the bounding estimate results in dose estimates that may be unreasonably high for the population of interest.

The uncertainties associated with this example scenario are related to the assumed activity patterns of the receptor population and the input parameters used. Implicit in this scenario is the assumption that the population of interest contacts the contaminated soil from the site, and that adherence occurs over the assumed surface area of the skin at the rates shown in the *EFH* (U.S. EPA, 2011a; see Table 12). Another implicit assumption is that the soil is on the skin for the entire exposure event, which is assumed to be a day. This means that each event (whether it consists of a few minutes or several hours) is assumed to be 1 day. Multiple soil contact events in a single day are still treated as one event. Use of a 1-day exposure event is consistent with absorption values, which typically are based on 24-hour exposure periods. The assumption that absorption from contaminants in soil adhering to the skin occurs over 24 hours contributes to the uncertainty of the resulting estimates because it is reasonable that individuals may shower or bathe after a sporting event.

Selection of the clothing scenario or percentage of the body exposed should be based on the assessor's knowledge of the populations/activities and should be designed to reflect, as closely as possible, the skin surface area exposed for the activity of interest. However, the assumptions used regarding the clothing worn and the surface area exposed result in uncertainty in the assessment. In addition, clothing is not 100% effective in blocking exposure as dust can penetrate through cloth or deposit under loose clothing. In the *EFH* (U.S. EPA, 2011a), EPA recognizes the uncertainty associated with the surface area and adherence data and concludes that although there may be some selection bias associated with the recommended surface areas, they are the best available data for use in exposure assessment. The uncertainties associated with the adherence data result from the limited size of the data set, and the fact that adherence may be influenced by the clothing worn by the study participants, and soil properties (e.g., moisture content, particle size) that are not entirely accounted for in the available data.

4.2. DERMAL CONTACT WITH INORGANIC CONTAMINANTS WHILE WADING IN A RECREATIONAL POND: CHILDREN AGED 6 TO <16 YEARS, BOUNDING, ACUTE DOSE RATE

4.2.1. Introduction

The potential for exposure to chemical substances exists at sites where local surface water bodies (i.e., streams, ponds, lakes, bays, or rivers) have become contaminated. Children may be exposed to chemicals in the water via dermal absorption as a result of swimming and/or wading. Acute dose via dermal contact considers not only chemical concentrations in contact with the skin, but also the surface area of the skin that contacts the water, the absorption of the chemical in contact with the skin, exposure frequency, and exposure time. For the purposes of this example, surface water exposure among wading children (aged 6 to <16 years) is evaluated. Dermal exposure is assessed based on bounding acute exposure to an inorganic chemical while wading in contaminated surface water. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X.

4.2.2. Dose Algorithm

The *ADR* of an inorganic contaminant via dermal contact while wading is calculated as follows:

$$ADR_{wading\ water\ dermal} = \frac{DA_{event} \times SA \times EF \times ED}{BW \times AT} \quad (21)$$

where:

$ADR_{wading\ water\ dermal}$	= acute potential dose from dermal contact in recreational surface water while wading (mg/kg-d);
DA_{event}	= absorbed dose per hour (mg/cm ² -event);
SA	= surface area of the skin (cm ²);
EF	= exposure frequency (events/d);
ED	= exposure duration (d);
BW	= body weight of child (kg); and
AT	= averaging time (d).

4.2.3. Input for Exposure Factor Variables

DA_{event} —The absorbed dose per event (DA_{event}) is estimated considering the following factors:

- The permeability coefficient from water;
- The chemical concentration in water; and
- The event duration.

The approach to estimate DA_{event} differs with respect to inorganic and organic chemicals. This is consistent with current EPA policy directives (U.S. EPA, 2004b, 1997a, 1992b). For a dermal exposure in water using organic chemicals, see the approach presented in Scenario 4.3—Dermal Contact with an Organic Contaminant in Water while Showering. For inorganic chemicals, EPA recommends using the steady-state approach to estimate dermally absorbed doses. In this approach,

$$DA_{event} = K_p \times ET \times C_w \quad (22)$$

where:

- DA_{event} = absorbed dose per event (mg/cm²-event);
- K_p = dermal permeability coefficient of compound in water (cm/hr);
- ET = event time (hr/event); and
- C_w = chemical concentration in water (mg/cm³).

For the purpose of this example, the inorganic chemical “x” is used. It is assumed that chemical “x,” a permeability coefficient of 1×10^{-3} cm/hour, and a maximum chemical concentration of 1×10^{-6} mg/cm³.

ET—*EFH* Table 16-19 (U.S. EPA, 2011a) reports time spent in selected outdoor locations for children from 6 to <16 years of age. The 95th percentile exposure time spent in pool, river, or lake (doers-only) for children aged 6 to <11 years is 359 minutes/day (6.0 hours/day) and 228 minutes/day (3.8 hours/day) for children aged 11- to <16-years old. Assuming that these exposure times represent an event occurring over the course of 1 day, the exposure time would be 6 hours/event for children aged 6 to <11 years and 3.8 hours/event for children aged 11 to <16 years (i.e., a child’s skin is immersed in water for a total of either 6 or 3.8 hours over the course of the daily event, depending on the age of the child).

This results in a DA_{event} calculation for children aged 6 to <11 years of:

$$DA_{event} = (1 \times 10^{-3}) \frac{\text{cm}}{\text{hr}} \times 6 \frac{\text{hr}}{\text{event}} \times (1 \times 10^{-6}) \frac{\text{mg}}{\text{cm}^3}$$

$$DA_{event} = 6 \times 10^{-9} \frac{\text{mg}}{\text{cm}^2\text{-event}}$$

This results in a DA_{event} calculation for children aged 11 to <16 years of:

$$DA_{event} = 1 \times (1 \times 10^{-3}) \frac{\text{cm}}{\text{hr}} \times 3.8 \frac{\text{hr}}{\text{event}} \times (1 \times 10^{-6}) \frac{\text{mg}}{\text{cm}^3}$$

$$DA_{event} = 3.8 \times 10^{-9} \frac{\text{mg}}{\text{cm}^2\text{-event}}$$

SA—The surface area of the skin of a child (aged 6 to <16 years) for various body parts can be found in *EFH* Table 7-2 (U.S. EPA, 2011a). In this scenario, wading is assumed; therefore, only the *SA* of the legs and feet will be used. In this example, mean surface areas of the legs and feet are used because surface area and *BW* are strongly correlated and the mean values are most representative of the surface area of individuals with an average *BW*. Because surface area is divided by the body weight in the dose equation, using an upper-percentile surface area with an upper-percentile *BW* would be similar to using a mean surface area with a mean *BW*. The estimates are given in m^2 and must be converted to cm^2 ($1 \text{ m}^2 = 10,000 \text{ cm}^2$). For this example, the surface area is 0.311 m^2 for legs and 0.073 m^2 for feet ($3,110 + 730 = 3,840 \text{ cm}^2$), respectively, for ages 6 to <11 years, and 0.483 m^2 and 0.105 m^2 ($4,830 + 1,050 = 5,880 \text{ cm}^2$) for legs and feet, respectively, for ages 11 to <16 years.

EF—Exposure frequency is assumed to be one event per day.

ED—Exposure duration is the number of days over which exposure occurs. For the purposes of this example, the acute *ED* is assumed to be 1 day.

BW—*EFH* Table 8-1 (U.S. EPA, 2011a) reports recommended *BWs* for children from 6 to <16 years of age. The *BW* for boys and girls are averaged for ages 6 to <16 years. This calculation, using the mean of this distribution, also is weight averaged according to the number of years in each age range. For children aged 6 to <11 years of age, the mean recommended *BW* is 31.8 kg. For children aged 11 to <16 years of age, the mean recommended *BW* is 56.8 kg.

AT—Because the potential acute bounding dose is being calculated, the averaging time is equivalent to the *ED* of 1 day.

4.2.4. Calculations

Using the dose algorithm and the input for the exposure factors, the $ADR_{\text{wading water dermal}}$ for children aged 6 to <11 and 11 to <16 is calculated in Table 13.

Table 13. Summary of ADR for children aged 6 to <16 years for acute potential dose from dermal contact in recreational surface water while wading

Equation 21 input and output	Age ranges	
	6 to <11 yr	11 to <16 yr
DA_{event} (mg/cm ² -event)	6×10^{-9}	3.8×10^{-9}
SA (cm ²)	3,840	5,880
EF (events/d)	1	1
ED (d)	1	1
AT (d)	1	1
BW (kg)	31.8	56.8
ADR (mg/kg-d)	7.2×10^{-7}	3.9×10^{-7}

The $ADR_{\text{wading water dermal}}$ averaged over the 10 years of exposure for children aged 6 to <11 and 11 to <16 is calculated as follows:

$$ADR_{\text{wading water dermal}} = \frac{\left[\left(5 \text{ yr} \times 7.2 \times 10^{-7} \frac{\text{mg}}{\text{kg-d}} \right) + \left(5 \text{ yr} \times 3.9 \times 10^{-7} \frac{\text{mg}}{\text{kg-d}} \right) \right]}{10 \text{ yr}}$$

$$ADR_{\text{wading water dermal}} = 5.6 \times 10^{-7} \frac{\text{mg}}{\text{kg-d}}$$

4.2.5. Uncertainties

The example presented here is used to represent bounding doses among children aged 6 to <16 years while wading in surface water. Note that central tendency doses may be calculated by using mean concentrations and exposure time, and a lower FA .

There are uncertainties related to calculation of the absorbed dose per surface water exposure event (e.g., DA_{event}). As noted in *Dermal Exposure Assessment: Principles and Applications*, “the dermal permeability estimates are probably the most uncertain of the parameters in the dermal dose equation. Accordingly, the final dose and risk estimates must be

considered highly uncertain” (U.S. EPA, 1992b). A lack of measured data for a variety of chemicals makes the validation of the model difficult. There are also uncertainties associated with the surface areas and exposure times used in this example scenario.

4.3. DERMAL CONTACT WITH AN ORGANIC CONTAMINANT IN WATER WHILE SHOWERING: CHILDREN AND TEENS, AGED 6 TO <16 YEARS HIGH-END, LIFETIME AVERAGE DAILY DOSE

4.3.1. Introduction

The potential for exposure to chemical substances exists if children and teens shower in water from a contaminated source or in water that has been treated with substances that convert into toxicants over time. Receptors could include any children and teens who shower using such water. The dose via dermal contact considers not only the chemical concentration in the water, but also the surface area of the skin in contact with the water, the behavior of the chemical when it comes in contact with the skin, exposure duration, exposure time, and exposure frequency. This example examines the high-end dermal dose via showering in contaminated water for children and teens aged 6 to <16 years (age ranges: 6 to <11 and 11 to <16 years) and calculates the *ADD* for both age ranges, then weights the average daily doses according to the length of time these exposures occur to calculate the *LADD*. Values obtained from tables within the EFH (U.S. EPA, 2011a) are cited as EFH Table X-X. In this example, it is assumed that the receptors are not exposed to the contaminant again for the remainder of their lives. The *LADD* is calculated because the contaminant in this scenario is assumed to be a carcinogen and the carcinogen toxicity values are expressed as a lifetime value.

4.3.2. Dose Algorithm

Dose via this pathway would be calculated as follows:

$$LADD_{shower\ water\ dermal} = \frac{DA_{event} \times SA \times EV \times EF \times ED}{BW \times AT} \quad (23)$$

where:

- $LADD_{shower\ water\ dermal}$ = absorbed lifetime average daily dose from dermal contact with water during showering (mg/kg-d);
- DA_{event} = absorbed dose per event (mg/cm²-event);
- SA = surface area of the skin (cm²);
- EV = event frequency (events/d);

<i>EF</i>	= exposure frequency (d/yr);
<i>ED</i>	= exposure duration (yr);
<i>BW</i>	= body weight of child (kg); and
<i>AT</i>	= averaging time (d).

4.3.3. Input for Exposure Factor Variables

DA_{event}—The absorbed dose per event (mg/cm²-event) is estimated considering the following factors:

- The permeability coefficient from water,
- The chemical concentration in water, and
- The event duration.

Current EPA policy directs differing approaches to estimate *DA_{event}* with respect to inorganic and organic chemicals (U.S. EPA, 2004b, 1992b). For inorganic chemicals, the EPA recommends using a steady-state approach to estimate dermal absorption doses. This approach is illustrated in Scenario 4.2—Dermal Contact with Inorganic Contaminants while Wading in a Recreational Pond.

For organic chemicals, EPA provides two equations. The choice of appropriate equation is based on the event duration versus the lag time per event. If the duration of the event (*t_{event}*) is less than the time to reach steady-state (2.4τ), then the following equation is used to estimate *DA_{event}* (U.S. EPA, 2004b, 1992b):

$$\text{If } t_{event} \leq t^*, \text{ then: } DA_{event} = 2 \times FA \times K_p \times C_w \sqrt{\frac{6 \times \tau \times t_{event}}{\pi}} \quad (24)$$

where:

<i>DA_{event}</i>	= absorbed dose per event (mg/cm ² -event);
<i>FA</i>	= fraction available for absorption (dimensionless);
<i>K_p</i>	= dermal permeability coefficient of compound in water (cm/hr);
<i>C_w</i>	= chemical concentration in water (mg/cm ³);
<i>τ</i>	= lag time per event (hr/event);
<i>t_{event}</i>	= event duration (hr/event); and
<i>t*</i>	= time to reach steady-state.

If the duration of the event (t_{event}) is greater than the time to reach steady-state (2.4τ), then the equation incorporates a new coefficient B , which is a dimensionless ratio of the permeability coefficient of a compound through the *stratum corneum* relative to its permeability across the epidermis. U.S. EPA (2004) indicates that the lag time is the time during which absorption continues after the exposure has ended; for chemicals that exhibit a long lag time, “some of the chemical dissolved into skin may be lost due to desquamation during that absorption period.” It should be noted that the fraction available for absorption (FA) is different than the absorption fraction used in Scenario 4.1—Dermal Contact with Soil (*ABS*). The variable FA is used to account for the loss of chemical due to the desquamation of the outer skin layer and a corresponding reduction in the absorbed dermal dose.

The following equation is used to estimate this DA_{event} (U.S. EPA, 2004b, 1992b):

$$\text{If } t_{event} > t^*, \text{ then: } DA_{event} = FA \times K_p \times C_w \left[\frac{t_{event}}{1+B} + 2\tau \left(\frac{1+3B+3B^2}{(1+B)^2} \right) \right] \quad (25)$$

where:

DA_{event}	= absorbed dose per event (mg/cm ² -event);
FA	= fraction absorbed (dimensionless);
K_p	= dermal permeability coefficient of compound in water (cm/hr);
C_w	= chemical concentration in water (mg/cm ³);
τ	= lag time per event (hr/event);
t_{event}	= event duration (hr/event);
B	= dimensionless ratio of the permeability coefficient of a compound through <i>stratum corneum</i> relative to its permeability coefficient across the viable epidermis; and
t^*	= time to reach steady-state.

Guidance for using these equations is detailed in *Dermal Exposure Assessment: Principles and Applications* (U.S. EPA, 1992b) and *Risk Assessment Guidance for Superfund, Part E* (U.S. EPA, 2004b). The organic chemical benzene is used for the purposes of this example. Benzene, estimates for which are given in Appendix B of *Risk Assessment Guidance for Superfund, Part E* (U.S. EPA, 2004b), has a molecular weight (MW) of 78.1 and a log K_{ow} of 2.13. The K_p for benzene is 1.5×10^{-2} cm/hour and the FA is 1.0. To determine which equation

must be used to calculate DA_{event} , the lag time per event (τ) must be calculated. The following equation can be used:

$$\tau = \frac{l_{sc}^2}{6 D_{sc}} \quad (26)$$

In this equation, l_{sc} (the thickness of the *stratum corneum*) is 10^{-3} cm. This is a default recommended for use by EPA's *Risk Assessment Guidance for Superfund, Part E* (U.S. EPA, 2004b). D_{sc} is the *stratum corneum* diffusion coefficient (cm^2/hour), which can be calculated as follows:

$$D_{sc} = 10^{(-2.80 - 0.0056 MW)} \times l_{sc} \quad (27)$$

Using eq 26, the lag time per event (τ) is 0.29 hour/event, as shown in Table B-3 of *Risk Assessment Guidance for Superfund, Part E* (U.S. EPA, 2004b). Because the time to reach steady-state (t^*) is defined as 2.4τ , the t^* actually would be 0.70 hour or 42 minutes (U.S. EPA, 2004b). Based on *EFH* Table 16-1 (U.S. EPA, 2011a), the 95th percentile recommended value for shower duration is 41 minutes for a child aged 6 to <11 years, and 40 minutes for a child aged 11 to <16 years. Using these values as the event duration (t_{event}), $t_{event} \leq t^*$; thus, eq 24 would be used for the calculation of DA_{event} . If t_{event} had been greater than t^* , eq 25 would be used, and variable B would need to be calculated, as shown in eq 28:

$$B = K_p \frac{\sqrt{MW}}{2.6} \quad (28)$$

Assuming the mean concentration in water (C_w) is 1×10^{-3} mg/cm^3 , an example dose calculation for DA_{event} is as follows, and is the same for both age ranges using eq 25:

$$DA_{event} = 2 \times 1.0 \times (1.5 \times 10^{-2}) \frac{\text{cm}}{\text{hr}} \times (1 \times 10^{-3}) \frac{\text{mg}}{\text{cm}^3} \sqrt{\frac{6 \times 0.29 \frac{\text{hr}}{\text{event}} \times 0.70 \frac{\text{hr}}{\text{event}}}{\pi}}$$

$$DA_{event} = 1.9 \times 10^{-5} \frac{\text{mg}}{\text{cm}^2\text{-event}}$$

SA—The total surface area of the skin of a child can be found in *EFH* Table 7-1 (U.S. EPA, 2011a) for all children according to the age increments in the *Guidance on Selecting*

Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants (U.S. EPA, 2005a) document. In this example, mean total surface areas are used because surface area and *BW* are strongly correlated and the mean values are most representative of the surface area of individuals with an average *BW*. Because surface area is divided by the *BW* in the dose equation, using an upper-percentile surface area with an upper-percentile *BW* would be similar to using a mean surface area with a mean *BW*. The estimates are given in m² and must be converted to cm² (1 m² = 10,000 cm²). For this example, the averages of 1.08 m² for 6 to <11 years of age and 1.59 m² for 11 to <16 years of age are converted and used as 10,800 cm² and 15,900 cm², respectively.

EV—Since the *LADD* accounts for daily exposure, the event frequency is the number of events per day. For this scenario, it is assumed that one shower is taken daily (i.e., 1 event/day).

EF—Exposure frequency is the number of times an exposure is expected to occur in a year and is expressed in days per year. For the purposes of this scenario, it is assumed that children shower at home 350 days/year. This assumes that young children are away from home (e.g., on vacation) for 2 weeks/year.

ED—Exposure duration is the length of time over which the exposure occurs. This scenario covers a total duration of 10 years: 5 years from age 6 to <11 years and 5 years from age 11 to <16 years.

BW—*EFH* Table 8-1 (U.S. EPA, 2011a) reports recommended *BWs* for children aged 6 to <16 years. The *BWs* for boys and girls are averaged from age 6 to <11 years old and 11 to <16 years old. These calculations, using the mean of this distribution, are 31.8 kg and 56.8 kg, respectively.

AT—The averaging time (*AT*) of 5 years is used in the individual calculations of DA_{event} for each age range. Note that this assumes that no further exposures to the water contaminant of interest occur via showering after or before the 10 years examined here (i.e., aged 6 to <16 years). This *AT* value is converted to 1,825 days (that is, 5 years × 365 days/year). Because the *LADD* dose is calculated, the overall averaging time is equivalent to the lifetime (*LT*) of an individual in the receptor population. For the purposes of this example, the average lifetime of 70 years (25,550 days) for men and women is used because the receptors exposures are assumed to reflect the general population (U.S. EPA, 2011a).

4.3.4. Calculations

Using the dose algorithm and input for the exposure factors variables, the dermal *ADD* for children and teens while showering, is estimated for each age range in Table 14.

Table 14. Summary of *ADD* for children aged 6 to <16 years for high-end potential dose from dermal contact while showering

Equation 23 inputs and output	Age ranges	
	6 to <11 yr	11 to <16 yr
DA_{event} (mg/cm ² -event)	1.9×10^{-5}	1.9×10^{-5}
SA (cm ²)	10,800	15,900
EV (events/d)	1	1
EF (d/yr)	350	350
ED (yr)	5	5
AT (d)	1,825	1,825
BW (kg)	31.8	56.8
<i>ADD</i> (mg/kg-d)	6.2×10^{-3}	5.1×10^{-3}

The *ADDs* shown in Table 14 are then used to estimate the $LADD_{showering\ water\ dermal}$. Assuming exposure for 5 years as a 6- to <11-year old, 5 years of exposure as an 11- to <16-year old, and no exposure for the rest of the life time (i.e., 60 years), the weighted average over a lifetime for the *LADD* is calculated as:

$$LADD_{showering\ water\ dermal} = \frac{\left(5\ \text{yr} \times 6.2 \times 10^{-3} \frac{\text{mg}}{\text{kg-d}}\right) + \left(5\ \text{yr} \times 5.1 \times 10^{-3} \frac{\text{mg}}{\text{kg-d}}\right) + \left(60\ \text{yr} \times 0 \frac{\text{mg}}{\text{kg-d}}\right)}{70\ \text{yr}}$$

$$LADD_{showering\ water\ dermal} = 8.1 \times 10^{-4} \frac{\text{mg}}{\text{kg-d}}$$

4.3.5. Uncertainties

The example presented here is used to represent high-end doses among children aged 6 to <16 years for showering in contaminated water. Note that central tendency doses may be

estimated by replacing the 95th percentile shower duration with a mean value. The bounding estimate can be estimated by setting all the input parameters to upper-percentile values.

There are uncertainties related to calculation of the DA_{event} . According to *Dermal Exposure Assessment: Principles and Applications*, “The dermal permeability estimates are probably the most uncertain of the parameters in the dermal dose equation. Accordingly, the final dose and risk estimates must be considered highly uncertain” (U.S. EPA, 1992b). Frequently, for organic chemicals, K_p values are predicted using K_{ow} . *Dermal Exposure Assessment: Principles and Applications* states that “the uncertainty in the predicted K_p s is judged to be within plus or minus one order of magnitude from the best fit value” (U.S. EPA, 1992b). A lack of measured data for a variety of chemicals makes the validation of the model difficult.

Because of these uncertainties, U.S. EPA (1992b) recommends that an assessor conduct a “reality check” by comparing the total amount of contaminant in the water to which an individual is exposed, to the total estimated dose. U.S. EPA, 1992b states that “As a preliminary guide, if the dermal dose exceeds 50% of the contaminant in the water, the assessor should question the validity of the dose estimate.” Assessors are cautioned to consider the various uncertainties associated with this scenario to ensure that dose estimates are adequately represented.

GLOSSARY¹

Absorbed dose— The amount of a substance penetrating across an absorption barrier (the exchange boundaries) of an organism, via either physical or biological processes. Sometimes called internal dose. See also *Absorption barrier* and *Dose*.

Absorption barrier—Any exposure surface that may retard the rate of penetration of an agent into a target. Examples include the skin, respiratory tract lining, and gastrointestinal tract wall. See also *Target* and *Agent*.

Activity pattern data—Information on human activities used in exposure assessments. These may include a description of the activity, frequency of activity, duration spent performing the activity, and the microenvironment in which the activity occurs.

Acute dose rate (ADR)—Dose per unit time received over a short period of time. Units may include mg/day.

Acute exposure—A single exposure to a toxic substance that may result in severe biological harm or death. Acute exposures are usually characterized as lasting no longer than a day, as compared to longer, continuing exposure over a period of time. The *ADR* is used for an acute, noncarcinogenic exposure.

Adherence factor—The amount of a material (e.g., soil) that adheres to the skin per unit of surface area.

Age-dependent adjustment factors (ADAFs)—In cases where age-related differences in toxicity occur, differences in both toxicity and exposure need to be integrated across all relevant age intervals, by the use of ADAFs. This is a departure from the way cancer risks have historically been calculated based upon the premise that risk is proportional to the daily average of the long-term adult dose. For example, for chemicals or compounds with a mutagenic mode of action for carcinogenesis, in the absence of chemical-specific data, the risk from exposures that occur at early life stages should be calculated by applying the following ADAFs to the non-

¹ Glossary entries are adapted from IPCS (2004) and EPA (U.S. EPA, 1992a, 2004a, 2005a, 2011a, 2011b) publications.

age-specific slope factor: a factor of 10 for ages 0 to < 2 years; a factor of 3 for ages 2 to < 16 years; and a factor of 1 for ages >16 years.

Agent—A chemical, biological, or physical entity that contacts a target. See also *Target*.

Aggregate exposure—The combined exposure of an individual (or defined population) to a specific agent or stressor via relevant routes, pathways, and sources. Aggregate exposure can include exposure through multiple routes (e.g., dermal, inhalation, and ingestion).

Assessment—A determination or appraisal of possible consequences resulting from an analysis of data.

Average daily dose (ADD)—The mean daily amount of an agent to which a person is exposed, often averaged over a long period of time. EPA is transitioning from average daily dose methodologies to more refined aggregate and cumulative approaches for estimating exposure across each life stage. See also *Lifetime average daily dose (LADD)* and *Time-averaged exposure*.

Bias—A systematic error inherent in a method or caused by some feature of the measurement system.

Bounding estimate—An estimate of exposure, dose, or risk that is higher or lower than that incurred by the person with the highest or lowest exposure, dose, or risk in the population being assessed. Bounding estimates are useful in developing statements that exposures, doses, or risks are "not greater than" or "less than" the estimated value, because assumptions are used which define the likely bounding conditions.

Carcinogen—A substance or agent capable of causing cancer.

Central tendency exposure/dose—A measure of the middle or the center of an exposure/dose distribution. The mean is the most commonly used measure of central tendency.

Childhood exposure—Contact between an agent and a child. See also *Agent*.

Children—Individuals under the age of 21 years are classified as youth or children. For children less than 12 months old, the following age groups are recommended: birth to <1 month,

1 to <3 months, 3 to <6 months, and 6 to <12 months. For children older than 12 months, the following age groups are recommended: 1 to <2 years, 2 to <3 years, 3 to <6 years, 6 to <11 years, 11 to <16 years, and 16 to <21 years.

Chronic exposure—Repeated exposure by the oral, dermal, or/and inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).

Chronic intake—The long term period over which a substance crosses the outer boundary of an organism without passing an absorption barrier.

Community water—Includes tap water ingested from community or municipal water supply.

Consumer only—Refers to only those individuals who reported food or water during the survey period.

Contaminant concentration—Contaminant concentration is the concentration of the contaminant in the medium (air, food, soil, etc.) contacting the body and has units of mass/volume or mass/mass.

Cumulative exposure—Exposure via mixtures of contaminants through more than one pathway. Risk assessments in EPA are placing more emphasis on total exposures via multiple pathways.

Cumulative risk assessment—An analysis, characterization, and possible quantification of the combined risks to health or the environment from multiple agents or stressors. See also ***Risk assessment***.

Deposition—The removal of airborne substances to available surfaces that occurs as a result of gravitational settling and diffusion, as well as electrophoresis and thermophoresis.

Dermal absorption—A route of exposure by which substances can enter the body through the skin (i.e., uptake across the skin barrier following dermal exposure).

Dermal permeability—Measure of the ability of the substance to penetrate and move through the skin.

Direct water ingestion—Consumption of tap water as a beverage. It does not include water used for preparing beverages such as coffee or tea.

Distribution—A set of values derived from a specific population or set of measurements that represents the range and array of data for the factor being studied.

Doers—Survey respondents who report participating in a specified activity.

Dose—The amount of an agent that enters a target after crossing an exposure surface. If the exposure surface is an absorption barrier, the dose is an *absorbed dose*. If the exposure surface is not an absorption barrier, the dose is an *intake dose*.

Dose rate—Dose per unit time, for example in mg/day, sometimes also called dosage. Dose rates are often expressed on a per-unit-body-weight basis, yielding units such as mg/kg-day. They are also often expressed as averages over some time period, for example a lifetime.

Drinking water—All water consumed by individuals to satisfy body needs for internal water.

Dry weight intake rates—Intake rates that are based on the weight of the food consumed after the moisture content has been removed.

Dust ingestion—Consumption of dust that results from various behaviors including, but not limited to, mouthing objects or hands, eating dropped food, consuming dust directly, or inhaling dust that passes from the respiratory system into the gastrointestinal tract.

Effect—Change in the state or dynamics of an organism, system, or population caused by exposure to an agent. See also *Agent*.

Exclusively breastfed—Infants whose sole source of milk comes from human milk with no other milk substitutes. See also *Infant*.

Exposure—Contact between an agent and a target. See also *Agent* and *Target*.

Exposure assessment—The process of estimating or measuring the magnitude, frequency, and duration of exposure to an agent, along with the number and characteristics of the population exposed. See also *Agent*, *Exposure duration*, and *Exposure frequency*.

Exposure concentration—The concentration of a chemical in its transport or carrier medium at the point of contact.

Exposure duration—Length of time over which contact with the contaminant lasts.

Exposure event—The occurrence of continuous contact between an agent and a target.

Exposure factor—Factors related to human behavior and characteristics that help determine an individual's exposure to an agent.

Exposure frequency—The number of exposure events in an exposure duration. See also *Exposure duration*.

Exposure time—The length of time an individual engages in an activity which results in exposure.

Exposure pathway—The physical course a chemical takes from the source to the organism exposed.

Exposure route—The way a chemical pollutant enters an organism after contact, e.g., by ingestion, inhalation, or dermal absorption.

Exposure scenario—A set of facts, assumptions, and inferences about how exposure takes place that aids the exposure assessor in evaluating, estimating, or quantifying exposures.

General population—All individuals inhabiting an area or making up a whole group.

High-end exposure/dose—An estimate of individual exposure or dose for those persons at the upper end of an exposure or dose distribution, conceptually above the 90th percentile, but not higher than the individual in the population who has the highest exposure or dose. See also *Bounding estimate*.

Human equivalent concentration or dose (HEC or HED)—The concentration (for inhalation exposure) or dose (for other routes of exposure) of an agent that is believed to induce the same magnitude of toxic effect in humans experienced by an experimental animal species. This adjustment may incorporate toxicokinetic information on the particular agent and/or target, if

available, or use a default procedure, such as assuming that daily oral doses experienced for a lifetime are proportional to *BW* raised to the 0.75 power.

Indirect water ingestion—Includes water added during food preparation, but not water intrinsic to purchased foods. Indirect water includes for example, water used to prepare baby formulas, cake mix, or concentrated orange juice.

Infant—A life stage that covers four age ranges, from birth to <1, 1 to <3, 3 to <6, and 6 to <12 months. See also *Children*.

Inhalation dosimetry—Process of measuring or estimating inhaled dose.

Inhalation unit risk (IUR)—The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu\text{g}/\text{m}^3$ in air for a lifetime.

Inhaled dose—The amount of an inhaled substance that is available for interaction with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism.

Intake—The process by which a substance crosses the outer boundary of an organism without passing an absorption barrier (e.g., through ingestion or inhalation).

Intake rate—Rate of inhalation, ingestion, and dermal contact depending on the route of exposure. For ingestion, the intake rate is simply the amount of food or drink containing the contaminant of interest that an individual ingests during some specific time period (units of mass/time). For inhalation, the intake rate is the rate at which contaminated air is inhaled. Factors that affect dermal exposure are the amount of material that comes into contact with the skin, and the rate at which the contaminant is absorbed.

IRIS—EPA's Integrated Risk Information System (IRIS) is a human health assessment program that evaluates information on health effects that may result from exposure to environmental contaminants. The IRIS database is an online database and contains information on more than 550 chemical substances.

Key study/data—A study that is useful for deriving exposure factors. Alternatively, studies may be classified as “relevant” and not “key” for one or more of the following: (1) they provide supporting data (e.g., older studies on food intake that may be useful for trend analysis); (2) they provide information related to the factor of interest (e.g., data on prevalence of breastfeeding); or (3) the study design or approach makes the data less applicable for exposure assessment purposes (e.g., data on mouthing behavior that was intended to be used in reducing the risk of choking hazards).

Life stage—A distinguishable time frame in an individual’s life characterized by unique and relatively stable behavioral and/or physiological characteristics that are associated with development and growth. See also *Infant* and *Children*.

Lifetime—The average lifetime of 70 years for men and women is assumed to reflect the general population for consistency by EPA when calculating cancer risks.

Lifetime average daily dose (LADD)—Dose rate averaged over a lifetime. The *LADD* is used for compounds with carcinogenic or chronic effects. The *LADD* is usually expressed in terms of mg/kg-day or other mass/mass-time units. Often used in carcinogen risk assessments that employ linear low-dose extrapolation methods. See also *Average daily dose* and *Time-averaged exposure*.

Mean value—Simple or arithmetic average of a range of values, computed by dividing the total of all values by the number of values.

Measurement error—A systematic error arising from inaccurate measurement (or classification) of subjects on the study variables.

Median value—The value in a data set such that half the measured values are greater and half are less.

Moisture content—The portion of food that is made up by water. The percentage of water is needed for converting food intake rates and residue concentrations between whole weight and dry weight values.

Monte Carlo technique—A repeated random sampling from the distribution of values for each of the parameters in a generic (exposure or dose) equation to derive an estimate of the distribution of (exposures or doses in) the population. A form of probabilistic exposure estimation. See also *Distribution*.

Mouthing behavior—Activities in which objects, including fingers, are touched by the mouth or put into the mouth except for eating and drinking, and includes licking, sucking, chewing, and biting (usually infants and children).

Octanol/water partition coefficient (K_{OW})—The ratio of the solubility of a compound in octanol to its solubility in water. The higher the K_{OW} , the more nonpolar the compound is. The Log K_{OW} of a compound may be used as an indication of how well the compound will adsorb to soil.

Per capita intake rate—The average quantity of food consumed per person in a population composed of both individuals who ate the food during a specified time period and those that did not.

Pica—Pica behavior is the repeated eating of nonnutritive substances, whereas soil-pica is a form of soil ingestion that is characterized by the recurrent ingestion of unusually high amounts of soil (i.e., on the order of 1,000–5,000 mg/day or more).

Plain tap water—Water obtained directly from the faucet or tap. It excludes water intrinsic in foods, juices, and other beverages.

Potential average daily dose (ADD)—is the amount of substance ingested, inhaled, or applied to skin per day, not all of which will be absorbed.

Potential dose—The amount of a chemical contained in material ingested, air breathed, or bulk material applied to the skin.

Preparation losses—Net cooking losses, which include dripping and volatile losses, postcooking losses, which involve losses from cutting, bones, excess fat, scraps and juices, and other preparation losses, which include losses from paring or coring.

Random samples—Samples selected from a statistical population such that each sample has an equal probability of being selected.

Range—The difference between the largest and smallest values in a measurement data set.

Reasonable maximum exposure—A semiquantitative term referring to the lower portion of the high end of the exposure, dose, or risk distribution. As a semiquantitative term, it should refer to a range that can conceptually be described as above the 90th percentile in the distribution, but below the 98th percentile.

Receptor/receptor population—The population of interest, also known as the receptor population, is described by specific age ranges. For children, these age ranges take into account their rapidly changing physiology. See also *Infant and Children*.

Reference Concentration (RfC)—An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive target groups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used. Generally used in EPA's noncancer health assessments. Durations include acute, short-term, subchronic, and chronic.

Reference Dose (RfD)—An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive target groups) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used. Generally used in EPA's noncancer health assessments. Durations include acute, short-term, subchronic, and chronic.

Representativeness—The degree to which a sample is, or samples are, characteristic of the whole medium, exposure, or dose for which the samples are being used to make inferences.

Risk—The probability of an adverse effect in an organism, system, or population caused under specified circumstances by exposure to an agent.

Risk assessment—A process intended to calculate or estimate the risk to a given target organism, system, or population, including the identification of attendant uncertainties, following exposure to a particular agent, taking into account the inherent characteristics of the agent of concern as well as the characteristics of the specific target system. The risk assessment process includes four steps: hazard identification, dose-response assessment, exposure assessment, and risk characterization.

Sample—A small part of something designed to show the nature or quality of the whole. Exposure-related measurements are usually samples of environmental or ambient medium, exposures of a small portion of a population for a short time, or biological samples, all for the purpose of inferring the nature and quality of parameters important to evaluating exposure.

Soil—Particles of unconsolidated mineral and/or organic matter from the earth's surface that are located outdoors, or are used indoors to support plant growth.

Soil adherence—The quantity of soil that adheres to the skin and from which chemical contaminants are available for uptake at the skin surface.

Soil ingestion—The intentional or unintentional consumption of soil, resulting from various behaviors including, but not limited to, mouthing, contacting dirty hands, eating dropped food, or consuming soil directly. Soil-pica is a form of soil ingestion that is characterized by the recurrent ingestion of unusually high amounts of soil (i.e., on the order of 1,000–5,000 mg/ day or more). Geophagy is also a form of soil ingestion defined as the intentional ingestion of earths and is usually associated with cultural practices.

Subchronic exposure—Repeated exposure by the oral, dermal, or inhalation route for more than 30 days, up to approximately 10% of the life span in humans (more than 30 days up to approximately 90 days in typically used studies with laboratory animal species).

Surface area—Coating, triangulation, and surface integration are direct measurement techniques that have been used to measure total body surface area and the surface area of specific body parts. Consideration has been given for differences due to age, gender, and race.

Target—Any physical, biological, or ecological object exposed to an agent. See also *Agent*.

Time-averaged exposure—The time-integrated exposure divided by the exposure duration. An example is the daily average exposure of an individual to carbon monoxide (also called time weighted average exposure).

Time use data—Information on activities in which various individuals engage, length of time spent performing various activities, locations in which individuals spend time, and length of time spent by individuals within those various environments.

Toddler—A child over the age of 12 months and generally younger than 36 months. See also *Infant* and *Children*.

Transfer efficiency—The percentage of a material that ends up as a coating on the desired surface.

Uncertainty—Uncertainty represents a lack of knowledge about factors affecting exposure/dose or risk and can lead to inaccurate or biased. The types of uncertainty include scenario, parameter, and model.

Unit risk—The quantitative estimate in terms of either risk per $\mu\text{g/L}$ drinking water (water unit risk) or risk per $\mu\text{g/m}^3$ air breathed (air unit risk).

Upper percentile—Values in the upper tail (i.e., between 90th and 99.9th percentile) of the distribution of values for a particular exposure factor. Values at the upper end of the distribution for a particular set of data.

Uptake—The process by which a substance crosses an absorption barrier and is absorbed into the body.

Variability—Variability arises from true heterogeneity across people, places, or time and can affect the precision of exposure/dose estimates and the degree to which they can be generalized. The types of variability include spatial, temporal, and inter-individual.

Wet-weight intake rates—Intake rates that are based on the wet (or whole) weight of the food consumed. This is in contrast to dry-weight intake rates.

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