# **GUIDANCE FOR USE OF PROBABILISTIC ANALYSIS IN HUMAN HEALTH RISK ASSESSMENTS**



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#### **INTRODUCTION** 1.



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#### *Oregon Department of Environmental Quality* **GUIDANCE FOR USE OF PROBABILISTIC ANALYSIS IN HUMAN HEALTH RISK ASSESSMENTS ACRONYMS**



**This guidance is not a substitute for a working familiarity with probabilistic techniques, their application, and the field of human health risk assessment, nor is it intended to be exhaustive on the subject of probabilistic risk assessment. Additional training and information, beyond that contained in this document, are a prerequisite for the successful performance of a probabilistic risk assessment. Risk assessors and responsible parties are reminded that there is a requirement, per OAR 340-122-084(5)(a), to consult with DEQ prior to initiating a probabilistic assessment.**

#### **1.1. Purpose**

 The purpose of this document is to present a typical set of exposure factors and equations for calculating exposures (intake) for various exposure routes applicable to human receptors; these factors and equations are not applicable to ecological receptors. Continuous distributions (with supporting descriptive statistics) are provided for exposure factors commonly utilized in simple human health risk assessments. These typical exposure factors, based on U.S. Environmental Protection Agency (EPA) guidance and peer-reviewed scientific literature, and consistent with recent changes in Oregon administrative rules (OAR 340-122-084(1)), may be used by responsible parties to perform probabilistic human health risk assessments at relatively simple sites. In general, the exposure factors and equations described in this document are most likely to be sufficient for calculating exposure, risks, or risk-based concentrations for typical remedies (as defined by OAR 340-122-047) at simple sites governed by the requirements of OAR 340- 122-084. The goal is a prospective risk assessment that addresses risks that might exist if no action is taken.

 It is critical to keep in mind, however, that these factors and equations are based on a number of simplifying assumptions that may or may not reflect site-specific conditions. Because these values and distributions do not consider any site-specific characteristics that could influence exposure, it is incumbent upon the responsible party to demonstrate to the satisfaction of the Department that the site in question is an appropriate candidate for the application of these typical exposure routes, factors, and equations. If such a demonstration cannot be made and site conditions deviate from the simplifying assumptions on which these typical equations are based, the guidance provided in this document is unlikely to apply. It will then be necessary to make site-specific modifications to these parameters and/or develop additional or completely different parameters, using the mechanism of a site-specific risk assessment (per OAR 340-122-084(2)).

#### **1.2. Regulatory Basis**

 Oregon's Revised Cleanup Law (ORS 465.315(2)(a); formerly HB 3352) allows a responsible party to propose conducting either a deterministic or a probabilistic risk assessment. The resulting administrative rules provide state-specific definitions for both CTE and RME values, require that both be reported for any deterministic risk assessment (OAR 340-122-084(1)(g)), limit risk estimates to the level of the individual for human health (OAR 340-122-084(h)(A)), and give specific requirements for the conduct and content of probabilistic risk assessments (OAR 340-122- 084(1)(b); OAR 340-122-084(5)).

The ultimate goal of regulatory action under the Revised Cleanup Law is to ensure protection of human health and the environment. This is done, in part, by demonstrating that acceptable risk levels have been achieved or will be achieved at a site. For probabilistic risk assessments, the acceptable risk level is defined as:

- For individual carcinogens, a lifetime excess cancer risk for each carcinogen of less than or equal to one per one million at the 90th percentile, and less than or equal to one per one hundred thousand at the 95th percentile, each based upon the same distribution of lifetime excess cancer risks for an exposed individual (OAR 340-122-115(2)(b)).
- For multiple carcinogens and multiple exposure pathways, a cumulative lifetime excess cancer risk of less than or equal to one per one hundred thousand at the 90th percentile and less than or equal to one per ten thousand at the 95th percentile, each based upon the same distribution of cumulative lifetime excess cancer risks for an exposed individual (OAR 340-122-115(3)(b)).
- For noncarcinogens, a hazard index less than or equal to one at the 90th percentile, and less than or equal to ten at the 95th percentile, each based upon the same distribution of hazard index numbers for an exposed individual (OAR 340-122-115(4)(b)).

# **1.3. Probabilistic Methods Guidance**

# **1.3.1. Basic Principles**

 The fourteen basic principles for practice of probabilistic risk assessment described by Burmaster & Anderson (1994) form the basis for many of the regulatory requirements stipulated by OAR 340-122-084(5). Risk assessors should familiarize themselves with these principles prior to seeking Departmental approval to conduct a probabilistic risk assessment. Available EPA guidance should also be consulted (EPA 1997) - see Appendix A.

# **1.3.2. Variability and Uncertainty**

 The basic goal of a probabilistic risk assessment is to quantitatively characterize the uncertainty and variability in estimates of exposure or risk. Uncertainty and variability arise from different processes, as follows:

• **Variability** refers to observed differences attributable to true heterogeneity or diversity in a

population or exposure parameter. It results from natural random processes and stems from environmental, lifestyle, and genetic differences among humans. Variability is a fundamental property of the exposed population and is usually not reducible by further measurement or study.

• **Uncertainty** represents partial ignorance, or a lack of perfect knowledge, about a phenomenon for a population as a whole or for an individual in a population. Specific sources of uncertainty (parameter, model, scenario, and decision-rule) all contribute to the overall uncertainty in a exposure or risk estimate. Uncertainty is a property of the current state of knowledge and, in principle, may be reduced by further study or additional measurement.

 Note that a qualitative or quantitative uncertainty analysis is required by rule for human health risk assessments (OAR 340-122-084(2)(f)) and probabilistic assessments (OAR 340-122-  $084(5)(d)(B)(iv)$  and OAR 340-122-084(5)(d)(E)). A complete discussion of variability and uncertainty may be found in NCRP (1996), Frey (1992), Morgan & Henrion (1990), or Hoffman & Hammonds (1992). New methods, involving second-order random variables, are currently being developed and used to isolate variability from uncertainty throughout a computation (NCRP 1996).

When preparing an uncertainty analysis to meet the requirements of OAR 340-122-084(2)(f), a narrative and qualitative discussion of all potential sources of uncertainty, beyond those embodied quantitatively in the model calculations, is expected as a minimum. Among other factors, there should be a recognition of the relative degree of uncertainty inherent in different exposure pathways, in that pathways which involve a number of uncertain steps will have a greater degree of uncertainty than those with fewer steps and more certain parameter estimates. The combined impact of multiple, highly uncertain parameter selections on the resulting risk estimates should be considered. These factors should be acknowledged as elements to consider in interpreting risk assessment results and using those results to support decision-making. However, the exact features of an uncertainty analysis are expected to be site- and assessmentspecific.

#### **1.3.3. Sensitivity Analysis**

 The goal of a sensitivity analysis is to rank the input variables or variates on the basis of their contribution to variance in the output. Such an analysis should be used, as described in Principles 3 and 11 of Burmaster & Anderson (1994), to: (a) limit the probabilistic analysis to those variables that dominate or "drive" the exposure or risk estimates and (b) interpret risk assessment results. Note that performance of a sensitivity analysis during conduct of a probabilistic risk assessment is explicitly required by rule  $(OAR 340-122-084(5)(d)(E))$ . Methods for performing sensitivity analyses are discussed in NCRP (1996) and are a feature of several commercially available Monte Carlo software programs.

#### **1.3.4. Goodness-of-Fit Tests**

 As noted in Principle 8 of Burmaster & Anderson (1994), goodness-of-fit statistics should be reported whenever measured data are fit to a particular distribution. Goodness-of-fit tests are formal statistical tests of the hypothesis that the set of sampled observations are an independent sample from the assumed distribution. Common tests include the chi-square test, the Kolmogorov-Smirnov test, and the Anderson-Darling test. Risk assessors should never depend solely on the results of these test to select the analytic form of the distribution as they have low discriminatory power and are generally best for rejecting poor distribution fits rather than identifying good fits (EPA 1997). For additional information, consult D'Agostino & Stephens (1986). Further advice on this issue is provided in EPA (1997), which is included as Appendix A of this guidance.

#### **1.3.5. Correlations**

 One of the key steps in setting-up a quantitative variability and uncertainty analysis is determining whether or not any of the variables are correlated with each other. Covariance among the input parameters can significantly affect the results of the analysis. It is particularly important to consider covariance when the covariance is among the exposure or risk model's most sensitive variables. For additional information, consult Morgan & Henrion (1990), Iman & Conover (1982), or Smith et al. (1992). Note that, as required by rule (OAR 340-122-084(5)(d)(C)), correlations that might significantly affect the outcome of a probabilistic risk assessment must be described and discussed, unless they have otherwise been accounted for in this guidance.

# **1.3.6. Regulatory Requirements**

 Per OAR 340-122-084(5), prior to initiating any probabilistic risk assessment the responsible party shall discuss with the Department and receive concurrence on issues that shall include but are not limited to: (a) human exposure routes, (b) contaminants of greatest potential importance to human receptors, (c) current and future land use scenarios that are reasonable and appropriate for the specific site, and (d) sources and characteristics of the distributions (if any) proposed for use in the human health toxicity estimation. The probabilistic assessment may include a combination of parameters expressed as either point estimates or distributions.

 Once these requirements have been met, the responsible party must prepare and receive approval from the Department for a work plan that documents the specific approaches, techniques, and information sources that will be used to meet the information criteria given below. Direct communication with the Department is required during this process. The intent of requiring a discussion between the Department and any responsible party desiring to conduct a probabilistic risk assessment is to provide, prior to actual performance of such an assessment, a forum within which to discuss such issues as the questions to be addressed by the assessment, as well as the contaminants, exposure pathways, and other factors that most influence the risk assessment results. Emphasis will be placed on ensuring that the probabilistic risk analyses performed at sites will support specific needs in remedial action decision-making.

The probabilistic risk assessment is to include, but is not limited to, information regarding:

- All formulae used to estimate exposure point concentrations, exposure doses, hazard indices, and incremental lifetime cancer risks.
- For each input parameter expressed as a distribution, the shape of the full distribution and, to the extent practicable, the mean, standard deviation, minimum, 5th percentile, median, 95th percentile, and maximum of the specified distribution.
- Any correlations or dependencies between or among input variables that are known or expected to have the practical effect of significantly affecting the risk assessment.
- Justification for the selection of each distribution that clearly explains the basis for its choice and the rejection of other relevant distributions.
- The extent to which input distributions and their parameters capture and separately represent both stochastic variability and knowledge uncertainty. This information comprises a portion of but is not a replacement for a comprehensive discussion in the body of the baseline risk assessment of the qualitative and quantitative sources of uncertainty.

 Justification for selection and use of each distribution shall be based on one or more of the following: (a) distributions presented in a refereed or peer-reviewed scientific publication, (b) distributions presented in U. S. Environmental Protection Agency or American Society for Testing and Materials (ASTM) documents or available DEQ documents, (c) expert or professional judgment as agreed to with the Department, or (d) parametric distributions of input variables, with the exception of chemical environmental concentrations, fit quantitatively to measured data.

 For parametric distributions of input variables, the responsible party shall include but is not limited to information regarding: (a) parametric fits and the data on the same axes; (b) appropriate goodness-of-fit statistics, (c) the implications of any important differences between the parametric fits and the data, and (d) how the statistical process or underlying mechanism creating the random variable influenced the choice of the distribution.

 For each output distribution resulting from the probabilistic risk assessment, the responsible party shall provide but is not limited to information regarding: (a) the shape of the full distribution, (b) location of the acceptable risk level as defined by ORS  $465.315(1)(b)(A)$ , and (c) to the extent practicable, the mean, standard deviation, minimum 5th percentile, median, 95th percentile, and maximum of the specified distribution. Also to be included with the output of the risk calculations is a probabilistic sensitivity analysis for all key input distributions conducted so as to distinguish, to the extent possible, the effects of variability from the effects of uncertainty in the input variables.

 By rule (c.f., OAR 340-122-084(5)(e)), the probabilistic approach can be applied to areas of risk assessment other than exposure analysis. Provided the criteria listed above are met, probabilistic methods may be applied to: (a) environmental media contaminant concentration data, (b) transport and fate modeling, (c) exposure estimation, (d) human toxicity estimation, or (e) risk characterization. However, this probabilistic guidance is meant to address primarily human exposure assessment and, to a lesser extent, risk characterization. Although the Department has no immediate plans to develop guidance specific to use of probabilistic methods in other areas, it may develop such guidance eventually, as available information and the state of practice in Oregon dictate.

# **1.4. Exposure and Risk Modeling**

 The exposure and risk estimation model outlined below (and described in detail in Section 4) is designed to determine probability of an individual, drawn at random from a population of individuals with characteristics defined by the exposure factor distributions, being exposed to risk in excess of the acceptable risk levels as described in Section 1.2. Because contact and intake rates can vary with age and gender, the model starts with selection of a specific age and gender from user defined distributions, then selects age- and gender-specific exposure factors for different randomly selected individuals in the population. It does not select different exposure factors for one individual with a fixed age. The years of exposure are determined by the individual's age and their exposure duration (selected at random from a distribution of agespecific exposure durations). A dose is calculated for each year of exposure and then summed and averaged to give the average daily dose received over all the years of exposure. In general, the model operates as follows:

- 1. Select an age at random (from a distribution of ages) for a random individual in the population;
- 2. Select a gender at random (from a distribution of gender ratios) for that individual;
- 3. Select age- and gender- specific exposure factors (from distributions of such factors) for

that individual;

- 4. Select an age-specific body weight (from a distribution of body weights) for that individual;
- 5. Select or calculate other exposure factors for that individual;
- 6. Calculate the age-specific average or absorbed daily dose (ADD) received by that individual for each exposure route;
- 7. Sum age-specific ADDs over the exposure interval (from a distribution of exposure durations) to calculate the average yearly dose (AYD) for that individual;
- 8. Multiply AYD by the exposure duration and divide by the noncarcinogen averaging time (i.e., = exposure duration) to calculate the noncarcinogen average daily dose (NADD) for that individual;
- 9. Multiply AYD by the exposure duration and divide by the carcinogen averaging time (i.e., = lifetime) to calculate the carcinogen average daily dose (CADD) for that individual;
- 10. Estimate the risk posed to that person by noncarcinogen and carcinogen doses received from each exposure route, then accumulate the risk estimates for that individual with those of all other randomly selected individuals as an estimate of risks to a population of individuals;
- 11. Return to Step 1, and repeat the process for the next person in the population, where the number of iterations (generally 10,000) of the model represents a sample of the population.

 Oregon rules (OAR 340-122-084(1)(f)) define reasonable maximum or high-end exposures as those at the  $90<sup>th</sup>$  percentile and it is these that the model is designed to estimate. The "maximum exposed individual" has been defined (EPA 1992) as an individual exposed above the 98<sup>th</sup> percentile but one that could ostensibly exist in the population. With this model, such an individual can be identified as one having the single highest dose/risk calculated in a population of size *n* and, by examining parameter values associated with this highest value, the specific characteristics (weight, age, ingestion rate, etc.) of this particular individual can be clearly stated. This model cannot, however, identify a "worst case exposure" or one that may or may not occur in the population and will usually not be observed in an actual population.

 In addition, doses and risks received by sensitive subpopulations (subsistence fishing, pregnant women, children, etc.) can be specifically modeled by restricting selection of exposure factor distributions to those uniquely characteristic of these subpopulations.

#### **1.5. Exposure Routes**

 Estimation of exposure begins with the identification of exposure pathways, routes, and points. An exposure pathway is the course a chemical or physical agent takes from a source to an exposed organism (EPA 1989, 1992). An exposure route is the way a chemical or physical agent comes in contact with a receptor (i.e., by ingestion, inhalation, dermal contact, etc.). The exposure scenario is a set of facts, assumptions, and inferences (including land and water uses) about how exposure takes place that aids in the evaluation, estimation, or quantification of exposure. The fundamental premise in establishing risk-based concentrations is that risk at a given site is a function of the receptors and exposure routes present, based on current or reasonably anticipated land and water uses in the locality of the site. Such land and water use designations are the basis for the risk assessment. Once risks have been determined for that set of land and water use designations, such designations cannot be changed unless risks are reassessed. To perform a risk assessment for exposure scenarios conditioned by one set of land and water use designations but then change to a different set of designations would invalidate the risk assessment and any remedy based on that assessment. The land and water uses that are allowable at a site are thus determined by showing that a set of exposure routes specific to each use does not produce unacceptable risks in human receptors.

 Following is a list of the primary exposure routes that might be considered when conducting a typical human health risk assessment:

- INGESTION / INGESTION
	- Incidental ingestion of contaminated soil
	- Incidental ingestion of contaminated house dust
	- Ingestion of contaminated water (ground or surface)
	- Ingestion of vegetables/fruits
	- Ingestion of animal products (meat/milk/eggs)
	- Ingestion of fish
- DERMAL CONTACT
	- Dermal contact with contaminated water
	- Dermal contact with contaminated soil
	- Dermal contact with contaminated house dust
- INHALATION
	- Inhalation of particulates (fugitive dust)
	- Inhalation of soil vapors (outdoors)
	- Inhalation of soil vapors (indoors)
	- Inhalation of vapors from water (indoors)

 Exposure estimation defines the relationship between a contaminant's environmental concentration and its exposure point concentration, or the quantity of that contaminant to which a receptor is ultimately exposed. Specific equations, described in detail in Section 2 of this guidance, are used to quantify doses received via these various exposure routes.

#### **1.6. Empirical and Simulated Distributions**

 The distributions proposed in this document are believed to be consistent with the best available data; primary sources of those data are cited in conjunction with the corresponding exposure factor. In some cases where an appropriate continuous frequency distribution for a given exposure factor was already available in the primary literature, it is presented here as the distribution for that factor. This type is labeled as an empirical distribution. In cases where only limited descriptive statistics were available for a given factor, selected statistics, in conjunction with Monte Carlo methods, were used to simulate the proposed distribution for that factor. This type is labeled as a simulated distribution. In all cases, site-specific data or the results of further research may permit better definition of exposure factor distributions. As with deterministic risk assessment, probabilistic modeling occasionally requires simplifying assumptions and the exercise of professional judgment.

 Simulated distributions were derived from a model constructed using Microsoft® Excel 5.0 for spreadsheet functions, Microsoft® Visual Basic for custom function creation, and Crystal Ball® (version 4.0c, 32 bit architecture; from Decisioneering, Inc., Denver, CO), with Latin Hypercube sampling, for Monte Carlo processing and data accumulation. The number of iterations (10,000) was selected somewhat arbitrarily; no tests were performed to ascertain whether this number resulted in stability of the mean or percentile values. Furthermore, no effort was made to test the adequacy of the pseudo-random number generator employed by Crystal Ball® (Barry 1996). Seed values for the various simulations were generated randomly within the software package.

#### **1.6.1. Distribution Notation**

 A uniform notation scheme was adopted for all of the distributions recommended in this guidance, as follows:

$$
X \sim \text{NamedDistribution} \text{Param 1, Param 2, LB, UB};
$$
 units

where X is the exposure factor; NamedDistribution is the type of distribution (Normal, Lognormal, etc.); Param 1 and Param 2 (and others depending on the distribution being defined) are the parameters (e.g., mean, standard deviation, etc.) that define the shape of the distribution; LB and UB are the lower and upper bounds (truncation points), respectively, of the distribution; units are the units of measure for X. This notation is intended primarily to allow any distribution to be easily implemented in one or more of the most popular commercially available Monte Carlo simulation software packages. As such, this notation may depart somewhat from mathematically correct formalisms.

#### **1.6.2. Truncation**

 Truncation refers to the process of setting lower and/or upper limits on the range of a parametric distribution representing a particular exposure factor. For example, it may be necessary to prevent sampling those portions of a distribution that would be expected to return unreasonable or nonsensical results (e.g., an adult body weight  $> 1000$  kg). Note, however, that truncation changes the characteristics of a probability distribution.

 With the exception of uniform and triangular distributions, explicit lower and upper bounds are given for each of the distributions recommended in this guidance. Truncation was accomplished in one of three ways:

- 1. Using limits governed by natural or physical processes;
- 2. With additional data from reliable sources or informed professional judgment;
- 3. In the absence of contradictory information, a lower bound at or near the  $0.1<sup>th</sup>$  percentile and an upper bound at or near the 99.9<sup>th</sup> percentile, to include ≈99.8% of the distribution.

 The exact method selected for truncation is described with the particular exposure factor to which it applies.

# **1.7. References**

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 This section presents exposure estimation equations cross-referenced to each exposure route listed in Section 1.5 and Table 2-1. Various scenario- and contaminant-specific exposure parameters are summarized in Table 2-2. These individual equations can be combined in different ways, to represent differing exposure scenarios as by determined current and future land and water uses. These site-specific exposure models estimate the average daily exposure associated with each contaminant within each medium (soil, water, air) for each receptor of *k*th age. The following equations treat exposure frequency and exposure duration differently and more explicitly than typical EPA default exposure equations (EPA 1991). Parameters may be expressed as either point estimates or distributions (Burmaster & von Stackelberg 1991).

#### **2.1. Average Daily Dose 2.1.1. Incidental Ingestion of Soil**

$$
ADD_1 \sim \frac{C_s \cdot IRS \cdot CF_{km}}{BW}
$$

**Equation 2. 1** 

Where:



Values for  $C_s$  can be derived primarily from onsite measurements or secondarily through the use of various intermedia transfer factors. Only when the exposure unit is very large or the contact behavior of individual members of the exposed population is such that contact with a given media may not be equal across the exposure unit, does EPA (1995) consider it reasonable to consider distributing any of these terms through the use of an appropriate probability density function (PDF). However, Oregon cleanup rules provide that any or all of these terms may be distributed regardless of the characteristics of the exposure unit, provided the requirements of OAR 340-122-084(5) are met. It is also important to recognize that the spatial distribution of contaminants should be taken into consideration when computing values for  $C_s$ . Refer to Burmaster & Thompson (1996), Clifford et al (1995), and Ginevan & Splitstone (1997) for examples of analysis of data within a spatial context.

 Absorption refers to the amount of a contaminant that is able to cross biological membranes and be taken up by the blood for subsequent distribution to target tissues. Here, and in all subsequent equations with the exception of those describing dermal contact exposures, the average daily dose (ADD) is expressed as the amount of contaminant at the exchange boundary and available for absorption; it is not equivalent to absorbed dose (EPA 1989). If the reference dose and or cancer slope factor for a given contaminant are expressed in terms of absorbed dose, they should be adjusted accordingly (see EPA 1989; Appendix A).

# **TABLE 2 - 1**

# **EXPOSURE ROUTE - EXPOSURE ESTIMATION EQUATION CROSS-REFERENCE MATRIX**



#### *Oregon Department of Environmental Quality* **GUIDANCE FOR USE OF PROBABILISTIC ANALYSIS IN HUMAN HEALTH RISK ASSESSMENTS SECTION 2 - EXPOSURE ESTIMATION EQUATIONS**



#### **2.1.2. Ingestion of Water (Tap, Surface, Ground)**

$$
ADD_2 \sim \frac{C_w \cdot IRW}{CF_{lm}}
$$

**Equation 2. 2** 

Where:



As with  $C_s$ , values for  $C_w$  can be derived primarily from onsite measurements or secondarily through the use of various intermedia transfer factors.

Age-specific values for  $IRW_k$  are indexed to the actual body weights of survey respondents and are expressed in units of ml of water consumed per kg body weight per day. Consequently, use of these data in estimating potential dose does not require the body weight factor in the denominator of the ADD calculation (EPA 1996c).

#### **2.1.3. Ingestion of Vegetables/Fruits**

$$
ADD_3 \sim C_v \cdot \left( IRV_v \cdot F_v + IRV_f \cdot F_{fr} \right) \cdot V_{cf} \cdot CF_{gg}
$$

**Equation 2. 3** 

$$
C_{v} = \left(\frac{C_{s}}{PEF} \cdot K_{ap}^{pt}\right) + \left(\frac{C_{s}}{VF_{s}} \cdot K_{ap}^{gs}\right) + \left(C_{s} \cdot K_{ps}\right) + \left(C_{s} \cdot K_{ps(roots)}\right)
$$

**Equation 2. 4** 

$$
K_{ps(roots)} = \frac{270 \cdot K_{ow}^{-0.58}}{K_{oc} \cdot f_{oc}}
$$

# **Equation 2. 5**

$$
K_{ap}^{gs} = \left[ f_{pa} + (f_{pw} + f_{pl} \times K_{ow}) \cdot \frac{RT}{H} \right] \cdot 10^{-3}
$$

#### **Equation 2. 6**

$$
K_{ps} = 0.784 \cdot \left\{ \exp \left[ \frac{(\log K_{ow} - 1.78)^2}{2.44} \right] \right\} \cdot \frac{1}{K_{oc} \cdot f_{oc}}
$$

#### **Equation 2. 7**

Where:





Note that the log value in Equation 2.7 is  $log_{10}$ . It should be noted that the age-specific values for IRV are indexed to the actual body weights of survey respondents and are expressed in units of grams of food consumed per kg body weight per day. Consequently, use of these data in estimating potential dose does not require the body weight factor in the denominator of the ADD calculation (EPA 1997).

The  $C_v$  term accounts for the potential for contaminants to reach vegetables or fruit through any or all of the following routes: (a) from soil to roots, (b) from soil to aboveground plant parts via root uptake (translocation), (c) from air as particulate deposition onto foliar surfaces, and (d) from air as vapors to aboveground plant parts. The equations for  $K_{ap}^{pt}$ ,  $K_{ap}^{gs}$ , and  $K_{ps(roots)}$  are further described in McKone (1993).

 Briggs et al. (1982) have developed a regression equation based on the octanol-water partition coefficient  $(K_{ow})$  for translocation of contaminants from roots to shoots. They noted that there appears to be an optimum lipophilicity for maximum translocation of contaminants to stems  $(K_{ps})$  in the range of  $log_{10}(K_{ow})$  -0.5 to 3.5. These factors represent the ratio of contaminant concentration in the plant tissue to contaminant concentration in soil solution. These relationships better represent the difficulty more highly lipophilic compounds (log  $K_{ow}$ ) 6) have in crossing root membranes and being translocated in plant tissues.

The vegetable correction factor  $(V_{cf})$  considers that most contaminants will not be evenly dispersed throughout a fruit or a vegetable but will remain on the surface and in a thin layer surrounding this surface. Activities such as washing or peeling, by removing this contaminated surface layer prior to ingestion, are thus anticipated to greatly reduce the level of contamination received through Ingestion. In the absence of information supporting a specific value for this parameter, its default value is 1.

The fraction of vegetables or fruit obtained from the site parameter ( $0 \le F_v \le 1$ ,  $0 \le F_f$   $\le 1$ ) is

an estimate of that fraction of total vegetables consumed which are grown within the contaminated site (exposure unit). To start, it may be assumed that all vegetables consumed are grown onsite in contaminated soils, so that  $F_v$  or  $F_{fr} = 1$ . If site-specific information is available on sources of vegetables, an appropriate distribution may be used to model a range of  $F_v$  or  $F_{fr}$ values.

#### **2.1.4. Ingestion of Animal Products**

$$
ADD_4 \sim (C_m \cdot IRM_m \cdot F_m + C_{dp} \cdot IRM_{dp} \cdot F_{dp} + C_e \cdot IRM_e \cdot F_e) \cdot CF_{gg}
$$
  
\n
$$
C_{dp} = C_s \cdot 7.9 \times 10^{-9} \cdot K_{ow}
$$
  
\nEquation 2. 8  
\nEquation 2.9  
\nEquation 2.9  
\nEquation 2.10

 $C_e = C_s \cdot 1.6 \times 10^{-6} \cdot K_{ow}$ 

#### **Equation 2. 11**

Where:



 $K_{ow}$  = *n*-Octanol-water partition coefficient (unitless)

 Note that the age-specific values for IRM are indexed to the actual body weights of survey respondents and are expressed in units of grams of food consumed per kg body weight per day. Consequently, use of these data in estimating potential dose does not require the body weight factor in the denominator of the ADD calculation (EPA 1997).

 McKone (1993) evaluated the steady-state contaminant concentration in meat (mg contaminant/kg fresh meat) divided by the animals' contaminant intake (mg contaminant/d) as  $2.5 \times 10^{-8} \times \text{K}_{\text{ow}}$  (see also Travis & Arms 1988). McKone (1993) evaluated the steady-state contaminant concentration in milk (mg contaminant/kg fresh milk) divided by the animals' contaminant intake (mg contaminant/d) as  $7.9 \times 10^{-9} \times K_{\text{ow}}$  (see also Travis & Arms 1988). McKone (1993) evaluated the steady-state contaminant concentration in chicken eggs (mg contaminant/kg fresh eggs) divided by the animals' contaminant intake (mg contaminant/d) as  $1.6 \times 10^{-6} \times K_{\text{ow}}$  (see also Travis & Arms 1988).

The fraction of meat, milk, or eggs from site parameter ( $0 \le F_{m,dn,e} \le 1$ ) is an estimate of that fraction of total meat, milk, or eggs consumed which are raised within the contaminated site (exposure unit). To start, it may be assumed that all meat, milk, or eggs consumed are produced onsite in contaminated soils, so that  $F_{m,dp,e} = 1$ . If site-specific information is available on the sources of the vegetables, an appropriate beta distribution may be used to model a range of  $F_{m,dp,e}$ values.

#### **2.1.5. Ingestion of Fish**

$$
ADD_5 \sim \frac{C_f \cdot IRF_f \cdot F_f \cdot CF_{gg}}{BW}
$$

**Equation 2. 12** 

$$
C_f = C_w \cdot BCF_f
$$

**Equation 2. 13** 

$$
\log BCF_f = 0.76 \cdot \log K_{\scriptscriptstyle \rm \scriptscriptstyle OW} -0.23
$$

**Equation 2. 14** 

Where:

ADD<sub>5</sub> = Average daily dose from ingestion of local fish (mg/[kg⋅d])  $C_f$  = Concentration of contaminant in finfish (mg/kg)

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Note that the log value in Equations 2.14 is log<sub>10</sub>. The derivation of the BCF<sub>f</sub> equation is given in Lyman et al. (1982), equation 5-2, page 5-5. The use of BCF accounts only for accumulation of contaminants directly from surface or pore waters. Additional equations must be applied to estimate: (a) contaminant partitioning from sediment to pore waters, at which point BCF may be used (EPA 1993) and (b) accumulation of contaminants in fish as a result of food ingestion.

The fraction of fish from site parameter ( $0 \le F_f \le 1$ ) is an estimate of that fraction of total fish consumed which are caught within the contaminated site (exposure unit). As a default, it may be assumed that all fish consumed originate from contaminated waters onsite, so that  $F_f = 1$ . If sitespecific information is available on the sources of the fish, an appropriate distribution may be used to model a range of  $F_f$  values.

#### **2.1.6. Dermal Contact with Soil**

$$
ADD_{6} \sim \frac{DA_{soil} \cdot SA \cdot EF_{evd} \cdot F_{b}}{BW}
$$

**Equation 2. 15** 

 $DA_{\text{solid}} = C_s \cdot AF \cdot DAF \cdot CF_{\text{km}}$ 

**Equation 2. 16** 

$$
SA = 0.102 \cdot BW^{0.682}
$$

**Equation 2. 17** 

Where:





 In general, EPA (1992a, 1998c) should be consulted for guidance on the evaluation and use of the absorption (DAF) parameter. Dermal uptake of contaminants is a function of the exposed skin surface area. In most cases only a portion of the total body surface is exposed to chemicals in contaminated media and estimates of the area of the affected body parts can be used to calculate a contact rate for the substance(s) of concern. It should be noted that clothing may not be a significant impediment to soil contact with skin.

 Dermal uptake of contaminants is a function of the exposed skin surface area. In most cases only a portion of the total body surface is exposed to chemicals in contaminated media and estimates of the area of the affected body parts can be used to calculate a contact rate for the substance(s) of concern. Direct measurement of body surface area involves techniques which are difficult and time-consuming. For this reason, several formulae have been developed which estimate total body surface area as a function of body height and weight (EPA 1989; Boyd, 1935; DuBois & DuBois 1916; Costeff 1966). Murray & Burmaster (1992) compared these formulae and also assessed the effect of the correlation between height and weight on the body surface area distribution. They concluded that body surface area distributions were similar for the four models (see EPA 1996c, Table 6-10). They also found that assuming correlation between height and body weight influenced the final distribution by less than one percent. Given these findings, the relationship (Equation 2.17) developed by Burmaster (1998), was taken as a reasonable method for calculating total surface area (SA) as function of body weight (BW).

The fraction of total skin area exposed  $(F_b)$  parameter represents the amount of skin surface area exposed under different scenarios and for different age groups. Because this is clearly a site- and scenario-specific parameter that is influenced by season, activity, and age, establishing an *a priori* single-point value or distribution is difficult. The recommended EPA default value for  $F_b$  (soil contact during outdoor activities) is 0.25 (EPA 1996b). This value has been applied when establishing preliminary remedial goals, presumably on the basis that clothing limits exposed skin surface to that on the head, hands, forearms, and lower legs (EPA 1989, 1996b). EPA (1992) also provides a range of point-estimates of  $F<sub>b</sub>$  influenced by clothing coverage by season: 5% exposed in winter, 10% in spring and fall, 25% in summer. This range from 5% to

25% also covers various combinations of head, hand, forearm, and lower leg exposures. It should be noted that clothing may not actually be a significant impediment to soil contact with skin and under some conditions, the value of  $F_b$  may approach 1 even for a clothed receptor.

#### **2.1.7. Dermal Contact with Water (Tap, Surface, Ground)**

$$
ADD_{7} \sim \frac{DA_{water} \cdot SA \cdot EF_{evd} \cdot F_{b}}{BW}
$$

**Equation 2. 18** 

 $DA_{water} = 10^{-3} \cdot (C_w \cdot CF_{cl}) \cdot t_{event}$  {inorganics}

#### **Equation 2. 19**

$$
DA_{\text{water}} = 2 \cdot K_p \cdot (C_w \cdot CF_{cl}) \cdot \sqrt{\frac{6 \cdot \tau \cdot t_{\text{event}}}{\pi}}, \text{ for } t_{\text{event}} < t^* \text{ {organics}}
$$

#### **Equation 2. 20**

$$
DA_{\text{water}} = K_p \cdot (C_w \cdot CF_{cl}) \cdot \left[ \frac{t_{\text{event}}}{1+B} + 2\tau \cdot \left( \frac{1+3B}{1+B} \right) \right], \text{ for } t_{\text{event}} > t^* \text{ {organics}}
$$

#### **Equation 2. 21**

$$
\log K_p (organics) = -2.72 + 0.71 \cdot \log K_{ow} - 0.0061 \cdot MW
$$

#### **Equation 2. 22**

Where:



 $K_{ow}$  = *n*-Octanol-water partition coefficient  $MW = Continant-specific molecular weight (g/mol)$ 

For selected contaminants, values for  $\tau$ ,  $t^*$ ,  $K_p$ , and B may be obtained from Table 5-8 of EPA (1992a). For contaminants not listed in Table 5-8, these parameters may be calculated using equations given in Section 5.3.2 of EPA (1992a).

#### **2.1.8. Inhalation of Vapors (Soil, Water)**

$$
ADD_8 \sim C_a \cdot IRA \cdot CF_{mm}
$$

**Equation 2. 23** 

$$
C_a = \left[ \left( \frac{C_s}{V F_s} \right) \cdot F_s \right] + \left[ C_w \cdot V F_w \cdot F_w \right]
$$

**Equation 2. 24** 

$$
VF_s = Q/C \cdot \left( \frac{\left(\pi \cdot D_a \cdot I_e\right)^{0.5} \cdot CF_{cm}}{2 \cdot \rho_b \cdot D_a} \right)
$$

**Equation 2. 25** 

$$
D_a = \frac{\left[\left(\theta_a^{10/3} \cdot D_i \cdot H' + \theta_w^{10/3} \cdot D_w\right)/n^2\right]}{\rho_b \cdot K_d + \theta_w + \theta_a \cdot H'}
$$

**Equation 2. 26** 

**Equation 2. 27** 

$$
n=1-(\rho_b/\rho_s)
$$

 $\theta_a = n - \theta_w$ 

**Equation 2. 28** 

$$
K_d = K_{oc} \cdot f_{oc}
$$

#### **Equation 2. 29**

Where:



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#### *Oregon Department of Environmental Quality* **GUIDANCE FOR USE OF PROBABILISTIC ANALYSIS IN HUMAN HEALTH RISK ASSESSMENTS SECTION 2 - EXPOSURE ESTIMATION EQUATIONS**



The value of  $C_a$  can be obtained either by direct measurement of air concentrations in the area of interest (indoors, outdoors, etc.) or with a transport and fate model. If soil is the source media, the concentration of soil vapors in air may be estimated by using Equation 2.24, where the  $VF<sub>s</sub>$  term is an estimate of the emission rate of contaminant vapors from soils. The derivation of the  $VF_s$  term and associated equations can be found in EPA (1996), Equation 8, page 26. The air concentration due to volatilization from water during household (indoor) uses other than showering is also obtained with Equation 2.24. The EPA suggested default point estimate for  $VF_w$  is 0.5 L/m<sup>3</sup> (EPA 1991b, 1996, 1998b).

 Volatile chemicals in water have been defined as those with a Henry's Law constant [atm⋅m<sup>3</sup>/mol] greater than  $10^{-5}$  and a molecular weight less than 200 g/mol (EPA 1996, 1998d). It may therefore be possible to avoid quantitative evaluation of this pathway if the physicochemical properties of the contaminants in question do not indicate they would be volatile. However, if the conceptual site model suggests the possibility that a volatiles pathway may be exploited, judicious practice suggests, owing to large uncertainties involved with screening criteria, that the risk from this pathway should be quantitatively assessed.

#### **2.1.9. Inhalation of Soil Particles**

$$
ADD_9 \sim PM_{10} \cdot IRA \cdot CF_{mm}
$$

**Equation 2. 30** 

$$
PM_{10} = \left(\frac{C_s}{PEF}\right) \cdot F_s
$$

**Equation 2. 31** 

$$
PEF = Q/C \cdot \frac{CF_{sh}}{R_f \cdot (1-G) \cdot (U_m/U_t)^3 \cdot F(x)}
$$

**Equation 2. 32** 

$$
U_t = U_f \cdot 2.5 \cdot (\ln H_e / Z_o)
$$

**Equation 2. 33** 

$$
F(x) = 0.886 \cdot (U_t/U_m)
$$

#### **Equation 2. 34**

Where:



÷.

The value of  $PM_{10}$  can be obtained either by direct measurement of air concentrations in the area of interest (indoors, outdoors, etc.) or with a transport and fate model. If soil is the source media, the respirable particulate concentration may be estimated by using Equation 2.32, where the PEF term is an estimate of the emission rate of particulates from soils. The derivation of the PEF term and associated equations can be found in EPA (1996), Equation 5, page 23. The EPA default value for PEF is  $1.32 \times 10^9$  m<sup>3</sup>/kg (EPA 1996).

#### **2.2. Exposure Frequency**

 As exposure to contamination presumably occurs during the course of a specific activity, making a reasonable estimate of the frequency and duration of that activity is key to achieving a reasonable estimate of dose. Exposure frequency provides an estimate of the fraction of time, over the course of a year, that an individual spends performing a specific activity. A key concept here is that each iteration of the probabilistic exposure model deals with only one, indivisible individual receptor. In probabilistic models, however, where exposure frequencies for different activities (such as residential and occupational) can be represented by separate distributions spanning an interval with a fixed physical upper limit (24 hours; 365 days), it would be possible for each distribution to simultaneously return values near or at this upper limit - causing a total time estimate in excess of this physical upper limit.

 The intermediate term "average yearly dose" (AYD) is introduced to emphasize the need to carefully account for time (as frequency and duration) in a probabilistic model, as follows:

$$
A Y D_i = \frac{\sum_{k=A_s}^{EI} A D D_{ik} \cdot (E F_{hd(i)} / 24 \ hr/d) \cdot E F_{dy(i)}}{EI}
$$

#### **Equation 2. 35**

Where:



 Exposure frequency expresses the time that an individual is in contact with contaminated media via a given exposure route. An individual's activity patterns strongly influence which exposure routes will be exploited and for how long. While it may be theoretically possible for an individual to engage in one activity or experience one exposure route for 24 hours per day (EF<sub>hd</sub>  $= 24$ ) and 365 days per year (EF<sub>dy</sub>  $= 365$ ), this is generally an unreasonable assumption. A meaningful exposure model should more realistically account for the time an individual spends contacting contaminated media as a function of their activity pattern. This can be done in different ways depending on exposure route. The brief discussions below introduce instances where risk assessors may have an opportunity to develop alternative estimates for exposure parameters based on published data and/or site-specific time-use surveys.

For incidental soil ingestion, exposure to a contaminant of concentration  $C_s$  in soil is assumed to occur at a rate of  $x$  mg per 24 hour day (IRS),  $EF_{dy}$  days per year. The worst case assumption is that soil contaminated at concentration  $C_s$  is ubiquitous in an individual's environment; i.e., all the soil that they contact on a daily basis is contaminated at a level of  $C_s$ , in which case  $EF_{hd} = 24$ . Alternatively, it is possible to consider that an individual has an opportunity to incidentally ingest soil contaminated at concentration  $C_s$  only at certain times during a 24 hour day; for example, while gardening or playing outdoors. In these instances, EF<sub>hd</sub>  $<$  24, but EF<sub>dy</sub> may remain the same.

For tap water ingestion, exposure to a contaminant of concentration  $C_w$  in water is assumed to occur at a rate of  $x L$  per 24 hour day (IRW),  $EF_{dy}$  days per year. Here again, the worst case assumption is that an individual obtains all of their drinking water, all contaminated at concentration  $C_w$ , from a single source, to which they are exposed 24 hours per day. This is a plausible scenario for an individual who remains at their residence all day or whose drinking water sources at home and at work contain the same contaminant at concentration  $C_w$ . Again, it is possible to consider that an individual has an opportunity to consume water of concentration  $C_w$  only at certain times during a 24 hour day; for example, while at home or at work, but not both. In this instance,  $EF_{hd} < 24$  (perhaps just time spent at home), but  $EF_{dy}$  may remain the same.

For ingestion of food items, exposure to a contaminant of concentration  $C_v$ ,  $C_m$ ,  $C_{dp}$ ,  $C_e$ , or  $C_f$ in food stuffs is assumed to occur at a rate of *x* g per kg body weight per 24 hour day (IRM) or *x* g per 24 hour day (IRF),  $EF_{dy}$  days per year;  $EF_{hd}$  is not a factor in this case. The fraction ( $F_{v}$ ,  $F_m$ ,  $F_{dp}$ ,  $F_e$ , or  $F_f$ ) of all the food they eat that is contaminated is the key factor in estimating exposure. Here again, the worst case assumption is that an individual obtains all of their food, all contaminated at concentration  $C_v$ ,  $C_m$ ,  $C_{dp}$ ,  $C_e$ , or  $C_f$  from a single source ( $F_v$ ,  $F_m$ ,  $F_{dp}$ ,  $F_e$ , or  $F_f$  = 1). Again, it is possible to consider that an individual has an opportunity to consume food from different sources, some contaminated and some not, so that  $F_v$ ,  $F_m$ ,  $F_{dp}$ ,  $F_e$ , or  $F_f < 1$ , with  $EF_{dy}$  remaining the same.

For dermal contact with soil, exposure to a contaminant of concentration  $C_s$  in soil is estimated to occur at a rate of *x* mg per cm<sup>2</sup> per event (DA<sub>soil</sub>),  $EF_{\text{evd}}$  events per day, for  $EF_{\text{dy}}$ days per year; EF<sub>hd</sub> is not a factor in this case. As was the case with incidental ingestion of soil, the idea that all the soil contacted by an individual on a daily basis is contaminated at a level of  $C_s$  may be unrealistic. Even allowing for at least one soil contact activity (such as gardening or excavation work) per day ( $EF_{\text{evd}} = 1$ ), it is unlikely that these activities occur every day of the year, so that  $EF_{dy} < 365$ .

For dermal contact with water, exposure to a contaminant of concentration  $C_w$  in water is estimated to occur at a rate of *x* mg per cm<sup>2</sup> per event (DA<sub>water</sub>), t<sub>event</sub> hours per event,  $EF_{\text{evd}}$ events per day, for  $EF_{dy}$  days per year;  $EF_{hd}$  is not a factor in this exposure route. As was the case with contact with soil, the idea that all the water contacted by an individual on a daily basis is contaminated at a level of  $C_w$  may be unrealistic. Even allowing for at least one water contact activity (such as showering) per day ( $EF_{\text{evd}} = 1$ ), it is unlikely that these activities occur every day of the year (thus  $EF_{dy} < 365$ ) or last all day on any day (thus t<sub>event</sub>  $\ll 24$ ).

For inhalation of vapors or particulates, exposure to a contaminant of concentration  $C_a$  or PM<sub>10</sub> in air is assumed to occur at a rate of *x* m<sup>3</sup> per 24 hour day (IRA), EH<sub>hd</sub> hours per day, EF<sub>dy</sub> days per year. Again, there may be site-specific instances where an individual breathes air contaminated at concentration  $C_a$  or  $PM_{10}$  all day (EF<sub>hd</sub> = 24) but such instances are not likely to be typical. Even for an individual remaining in one location (e.g., at home) for long periods, unless they were totally sedentary, any change in activity pattern would create a change in breathing rate, possibly necessitating adjustment of IRA (or expressing IRA on a  $m<sup>3</sup>$  per hour basis).

 Although doses may be received simultaneously via different exposure routes, the sum of the time spent within multiple activities (home, at work, and elsewhere (commuting, shopping, etc.) cannot exceed 24 hr/d or 365 d/yr for any individual.

#### **2.3. Noncarcinogen Average Daily Dose**

 For noncarcinogens, intakes are calculated by dividing the daily doses by the averaging time  $(AT_n)$ , which is set equal to an exposure duration of one year, as follows:

$$
NADD_i = \frac{AYD_i}{AT_n}
$$

**Equation 2. 36** 

Where:



#### **2.4. Carcinogen Average Daily Dose**

 For carcinogens, intakes are calculated by prorating the average yearly dose over the carcinogen averaging time  $(AT<sub>c</sub>)$ , with a default value of 75 years (27,375 days), the average duration of any lifetime (EPA 1989a, 1996c). If gender-specific analyses are performed, average male and female lifetime durations of 72.1 years (26,316.5 days) and 78.9 years (28,798.5 days) (EPA 1996c), respectively, may be substituted for  $AT_c$ .

$$
CADD_i = AND_i \cdot (ED/AT_c)
$$

**Equation 2. 37** 

Where:



#### **2.5. Risk Calculations**

#### **2.5.1. Hazard Quotient / Hazard Index**

 Note that toxicologically and by rule (OAR 340-122-115(28)), the hazard index should combine hazard quotients from systemic toxicants with similar toxic endpoints and that, per OAR 340-122-084(i), an initial assumption of additivity should be made. Although the HQ is calculated as part of the risk assessment, the acceptable risk level for non-carcinogens is evaluated (see OAR 340-122-115(4)) only on the basis of the HI, and not the HQ.

$$
HQ_{ij} = NADD_i / RfD_j
$$

**Equation 2. 38** 

$$
H I_i = \sum_{j=1}^h H Q_{ij}
$$

# **Equation 2. 39**

Where:



#### **2.5.2. Incremental Lifetime Cancer Risk**

$$
ILCR_{ij} = CADD_i \times CSF_j
$$

**Equation 2. 40** 

$$
ILCR_i = \sum_{j=1}^{h} ILCR_j
$$

**Equation 2. 41** 

Where:



#### **2.6. References**

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### **TABLE 2 - 2**

### **SUMMARY OF CONTAMINANT-SPECIFIC AND OTHER EXPOSURE FACTORS**









**\* NOTES:**Site-specific obtained through measurement or modeling

- <sup>b)</sup> Default value, may be replaced with a site-specific value
- <sup>c)</sup> Contaminant-specific physicochemical parameter

# **3.1. Ingestion Factors 3.1.1. Soil Ingestion Rate (IRS) 3.1.1.1. Explanation**

# Ingestion of soil is a potential source of human exposure to toxins. The potential is believed to be greater for children because they more frequently exhibit behaviors (*e.g.,* mouthing of objects or hands) which are conducive to inadvertent soil ingestion. Adults, however, may also ingest soil particles that adhere to food, cigarettes, or their hands. Deliberate soil ingestion by adults or children is known as pica or geophagia and is thought to be relatively uncommon, although the incidence is not well-defined. Here, the soil ingestion rate (IRS) pertains only to unintentional soil ingestion resulting from mouthing of objects or inadvertent hand-to-mouth activity.

 A number of early studies attempted to estimate the rate of soil ingestion by measuring the amount of dirt on the hands and then making assumptions about the transfer of that dirt to the mouth (Hawley 1985; Duggan & Williams 1977; Day et al 1975; Lepow et al 1974). More recently, researchers have tried to quantify soil ingestion using tracer methodology whereby the concentrations of certain trace elements are measured in soil and feces; these data are used to estimate the amount of soil ingested over a specific time period (Davis et al 1990; Van Wijnen et al 1990; Calabrese et al 1989; Clausing et al 1987; Binder et al 1986). A number of assumptions are inherent in studies employing tracers:

- Absorption and/or metabolism of tracer substances is assumed to be negligible;
- The tracer concentrations in the soil samples are assumed to reflect the concentrations in the soil ingested (*i.e.* Any ingested soil is assumed to originate from the yards or local areas from which composite samples are obtained, composite soil samples are assumed to adequately represent the heterogeneity of the soil with respect to tracer concentrations);
- Gastrointestinal transit time is assumed to be known and consistent among subjects.

 In addition to the stated assumptions, a number of limitations have contributed to the uncertainty associated with the measurement of soil ingestion:

- Sample populations are small and/or localized and, therefore, may not be representative of the general population of similarly aged individuals;
- The age range of the subjects is restricted (1-7 years old, except for a single study of six adults) and, therefore, may not be representative of the population at large;
- Studies have been short-term (3-8 days), due largely to the costly, labor-intensive and logistically complex nature of these studies;
- Seasonal variation in soil ingestion may bias results;
- Correction may or may not be made for tracer ingested from sources other than soil (*e.g.* food, medicines, toothpaste);
- Calculations of tracer quantities in food incorporate any error or uncertainty in the measurement of the amount of food consumed;
- Attempts to distinguish contributions from soil versus house dust have yielded conflicting results;
- Collection of input (food and nonfood tracer sources) and output (feces, urine) may be incomplete;
- The tracer methodology has not been validated using children as subjects.

 Failure to properly validate the tracer method for measurement of soil ingestion is, perhaps, the greatest limitation. A single attempt has been made to validate the technique in adults (Calabrese et al 1990). The authors conclude that with a four-day mass balance protocol it is possible to quantitatively estimate the amount of soil ingested by adults at a rate of 500 mg/d (Calabrese & Stanek 1991); however, their results suggest that the rate of adult soil ingestion is approximately 10% of this value. Furthermore, their subsequent analysis of the 1989 study of 64 children demonstrated that the reported values for soil ingestion were "far below their level of detection" for six of the eight tracers employed (Calabrese & Stanek 1991).

 Thompson & Burmaster (1991) were the first to present a parameterized distribution for soil ingestion which could be used for probabilistic analyses. They reanalyzed the data of Binder et al (1986), using the actual measured values for fecal weight (Binder and colleagues had originally estimated fecal weights after concluding that the measured values were too low), and demonstrated that the corrected daily soil ingestion values for children were lognormally distributed. Thompson & Burmaster (1991) attempted to apply similar analytical methods to the data of Calabrese et al (1989), but found that the published data were insufficient to determine a distribution. Stanek & Calabrese (1995a) reanalyzed the results of their 1989 Amherst study (Calabrese et al 1989) and constructed a distribution (assumed to be lognormal) of daily soil ingestion estimates by extrapolating the short-term 1989 study over 365 days. In a separate reanalysis of the 1989 data, in combination with data from Davis et al (1990), Stanek & Calabrese (1995b) developed another distribution of daily soil ingestion estimates for children. The distributions presented in these two papers (Stanek & Calabrese 1995ab) are very different and are also different than that constructed by Thompson & Burmaster (1991).

### **3.1.1.2. Distribution Definition**

 Clearly, the measurement of soil ingestion rates is complex and technically difficult and, due to the limitations detailed above, the level of confidence that can be placed in these studies at present is low. While the development of distributions for daily soil ingestion rates is highly problematic, primarily due to lack of substantial research in this area, there do appear to be enough data to provide a reasonable range of estimates for soil ingestion in children and adults, thus allowing the variability/uncertainty inherent in these data to enter exposure calculations. The distributions of soil ingestion rates described below are considered practical for use until further experimental data become available.

 **Soil ingestion rate, ages < 1 to 6 years** The Calabrese et al (1989) and Davis et al (1990) data are perhaps the best available on soil ingestion in children. When combined (CalEPA 1996), these data have the following percentile distribution:  $50^{th}$  - 37 mg/d,  $90^{th}$  - 156 mg/d,  $95^{th}$ - 217 mg/d,  $99^{th}$  - 535 mg/d, maximum - 11,415 mg/d. These data were used to define the parameters of a lognormal distribution, a distribution suggested by several other investigators (Burmaster and Thompson 1991; Stanek and Calabrese 1995a). The simulated cumulative distribution, whose properties are summarized in Tables 3-1 and 3-2, was generated using the following relationship:

$$
IRS_{1-6} \sim LogNormal[\mu = 3.61, \sigma = 1.15, LB = 0, UB = 400]; \quad mg/d
$$

**Equation 3. 1** 

Where:



Truncation of ages  $\lt 1$  to 6 years soil ingestion rate distribution was based on a physical lower bound of 0 mg/d and an upper bound equal to the upper bound reported by EPA (1997). Note that this truncation serves to specifically exclude the "pica" child; if this subpopulation is suspected of being present, an appropriate site-specific model should be developed. This distribution produces a mean value of  $\approx 60$  mg/d and a 90<sup>th</sup> percentile value of  $\approx 145$  mg/d. This  $90<sup>th</sup>$  percentile value is within the RME 100 mg/d to 200 mg/d range of recommended default values for children (ages 1 to 6 years) (EPA 1989, 1991, 1996).

 **Soil ingestion rate, ages 7+ years** The Calabrese et al (1990) preliminary tracer study involving 6 adults reported soil ingestion rates between 30 mg/d and 100 mg/d. These values are clearly not the limits of the range but merely points within a range. Given that a physical minimum value for this parameter is 0 mg/d, and considering the use of the lognormal distribution in describing soil ingestion by children, a lognormal distribution is also suggested for the 7+ age group. Following suggestions made by Seiler and Alvarez (1996), 30 mg/d and 100 mg/d were assumed to represent the  $2.5<sup>th</sup>$  percentile and 97.5<sup>th</sup> percentile, respectively, of this lognormal distribution. The simulated cumulative distribution, whose properties are summarized in Tables 3-1 and 3-2, was generated using the following relationship:

$$
IRS_{7+} \sim LogNormal[\mu = 4.00, \sigma = 0.31, LB = 0, UB = 480]; \quad mg/d
$$

**Equation 3. 2** 

Where:



 Truncation of age 7+ years soil ingestion rate distribution was based on a physical lower bound of 0 mg/d. The upper bound ingestion rate for adults is set at the modeled value developed by Hawley (1985). Empirical data suggest that it is highly unlikely for adult soil ingestion to exceed that of children. This distribution produces a mean value of approximately 57 mg/d and a 90<sup>th</sup> percentile value of  $\approx 81$  mg/d. This 90<sup>th</sup> percentile value approaches the 100 mg/d typically recommended as a single point RME default value for adults (EPA 1989, 1991, 1996).

 **Soil ingestion rate, occupational** There are currently no empirical data to support a distribution for an occupational soil ingestion rate  $(IRS<sub>o</sub>)$ . This factor is likely to be highly dependent upon the nature of the specific occupation(s) under consideration and may best be established on a site-specific basis. EPA (1996, 1997) both suggest 50 mg/d as a reasonable maximum estimate for this parameter; however, no estimates are given for most likely or minimum values. A physical lower boundary is 0 mg/d; a level that might be achieved if personal protective equipment and workplace hygiene are required. In the absence of either further experimental or empirically-derived site-specific data, DEQ recommends describing  $IRS<sub>o</sub>$ 

as with a single point value of 50 mg/d.

# **3.1.1.3. References**

- Binder S, Sokal D & Maughan D (1986) Estimating soil ingestion: the use of tracer elements in estimating the amount of soil ingested by young children. *Archives of Environmental Health* **41**, 341-345.
- Calabrese EJ & Stanek III EJ (1991) A guide to interpreting soil ingestion studies. II. Qualitative and quantitative evidence of soil ingestion. *Regulatory Toxicology and Pharmacology* **13**, 278-293.
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- EPA (1998) **Region 9 Preliminary Remediation Goals (PRGs) 1998**. Region IX, U.S. Environmental Protection Agency, May 1, 1998.
- EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. EPA/600/P-96/002Fabc, August 1997.
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- Thompson KM & Burmaster DE (1991) Parametric distributions for soil ingestion by children. *Risk Analysis* **11**, 339-343.
- Van Wijnen JH, Clausing P & Brunekreef B (1990) Estimated soil ingestion by children. *Environmental Research* **51**, 147-163.

# **TABLE 3 - 1**

## **SIMULATED SOIL INGESTION RATE DISTRIBUTIONS - STATISTICS**



### **TABLE 3 - 2**

## **SIMULATED SOIL INGESTION RATE DISTRIBUTIONS - PERCENTILES**



# **3.1.2. Tap Water Ingestion Rate (IRW)**

## **3.1.2.1. Explanation**

 Drinking water is a potential source of exposure to toxic materials through contamination of the drinking (tap) water source (ground water or surface water), the addition of substances to the water during treatment or leaching of contaminants from components of the water supply system. In order to quantify this exposure it is necessary to have an estimate of the amount of tap water consumed.

 Fluid consumption is typically reported as total tap water intake or total fluid intake. *Tap water intake* includes water which is directly consumed, plus tap water which is indirectly consumed in the form of foods and beverages prepared or reconstituted with tap water (*e.g.* coffee, tea, frozen juices, soups). Total *fluid intake* is the total tap water intake plus that water which is intrinsic (not added) to foods and beverages. The distributions recommended herein are for tap water intake specifically.

 Ershow and Cantor (1989) based their analysis on results of the Nationwide Food Consumption Survey (NFCS) conducted by the U.S. Department of Agriculture (USDA) in 1977-78. Participants in this study were interviewed concerning fluid consumption during the previous 24 hours and then asked to maintain a diary of intake for the next two days. The resultant data for total tap water intake (Table 3-3) are considered most representative of the general United States population due to the size of the sample (more than 26,000 individuals) and the balance that was achieved with regard to age, sex and geographic location of residence. Therefore, the Ershow and Cantor (1989) data were considered the most reliable and representative. Note that these data are normalized to body weight (ml/[kg·day]), thus obviating the need to account for a correlation between  $IRW_k$  and body weight.

## **3.1.2.2. Distribution Definition**

 Because Roseberry & Burmaster (1992) determined that the water intake data (in units of ml/d) of Ershow & Cantor (1989) fit lognormal distributions, it was assumed that these data in units of ml/[kg⋅d] would also assume a lognormal distribution. Log-transformed values (ln IRWk) from Table 3-3 were plotted against their corresponding *z*-scores and the method of least squares was used to fit the best straight line to the transformed data. The resulting values for μ, σ, LB, and UB are shown in Table 3-4. These are used, in conjunction with Equation 3.3, to define water intake rate distributions by age for both genders combined. The statistics and percentiles of these distributions are summarized in Tables 3-5 and 3-6, respectively.

 $IRW = LogNormal[\mu, \sigma, LB, UB]; \quad ml/[kg \cdot d]$ 

**Equation 3. 3** 



Truncation of the tap water ingestion rate distribution was based on a lower bound at the  $1<sup>st</sup>$ percentile and an upper bound at the  $99<sup>th</sup>$  percentile, to include 98% of the male and female population in each age group. Distribution parameters given by Ershow & Cantor (1989) were used to calculate, with the Excel® LOGINV function, values for LB and UB at the 1<sup>st</sup> percentile and the 99<sup>th</sup> percentile, respectively.

# **3.1.2.3. References**

EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. EPA/600/P-96/002Fabc, August 1997.

- Ershow AG & Cantor KP (1989) Total water and tap water intake in the United States: Population-based estimates of quantiles and sources. Life Sciences Research Office, FASEB, Bethesda MD.
- Roseberry AM & Burmaster DE (1992) Lognormal distributions for water intake by children and adults. *Risk Analysis* **12**, 99-104.

# **TABLE 3 - 3**

# **EMPIRICAL TOTAL TAP WATER INTAKE (ml/[kg**⋅**d]) DISTRIBUTIONS - PERCENTILES**



Source: Water intakes estimates from 1977-78 NFCS reported by Ershow and Cantor (1989), as listed in EPA (1997), Table 3- 7, page 3-7.

\* Value not reported due to insufficient number of observations.

## **TABLE 3 - 4**



# **SIMULATED TAP WATER INTAKE DISTRIBUTIONS - PARAMETERS**

Source: Calculation of best-fit parameters based on data provided in Ershow & Cantor (1989).

# **TABLE 3 - 5**

#### **SIMULATED TAP WATER INGESTION RATE DISTRIBUTIONS - STATISTICS**



#### **TABLE 3 - 6**

#### **SIMULATED TAP WATER INGESTION RATE DISTRIBUTIONS - PERCENTILES**



### **3.1.3. Vegetable/Fruit Ingestion Rate (IRV)**

#### **3.1.3.1. Explanation**

 Ingestion of contaminated food is a potential source of human exposure to toxic chemicals. Consumers of home-produced foods may be of particular concern because exposure resulting from local site contamination may be higher for this subpopulation (EPA 1997).

 The principal sources of data regarding consumption rates of various food items among the United States population is the Nationwide Food Consumption Survey (NFCS), which is conducted by the U.S. Department of Agriculture (USDA) approximately every 10 years, and the USDA Continuing Survey of Food Intakes by Individuals (CFSII).

 The most recent NFCS was carried out in 1987-88 and represents more than 10,000 individuals in approximately 4,300 households. Although this sample is smaller than the previous NFCS (more than 36,000 individuals in approximately 15,000 households were sampled in 1977-78), it is considered to be most representative of current eating patterns among the general population. The NFCS has two components. The household component collects data over a seven-day period regarding the socioeconomic and demographic characteristics of the households, as well as the types and sources of food consumed. The individual component gathers information on food consumption by individuals within each household over a three-day period. The data have been used to generate both consumer-only and per capita rates of intake for individual food items and for categories of foods.

 CFSII data from the 1989-1991 survey have been used by EPA to generate per capita intake rates for various food items and food groups. Approximately 15,000 individuals provided intake data over the three survey years. This is considered as the key study for the intake of fruits and vegetables (EPA 1997).

## **3.1.3.2. Distribution Definition**

Percentiles for the ingestion rate of total vegetables (Table 3-7) and total fruits (Table 3-8) were developed by the U.S. EPA from the CSFII data (all regions). As such, they are subject to the assumptions and professional judgment brought to the analysis by U.S. EPA. Note that these data are normalized to body weight  $(g/[kg \cdot day])$ , thus obviating the need to account for a correlation between IRV and body weight. Rather then assume a continuous distribution, a custom distribution was used to replicate the distribution percentiles as presented in the source publication. The properties of the simulated cumulative distribution, generated using the relationship shown below, are summarized in Tables 3-9, 3-10, 3-11, and 3-12.

 $IRV \sim$  *Custom*  $[DC, PC]$ ;  $g/[kg \cdot d]$ 

### **Equation 3. 4**

Where:



 As replicates of the original data, custom distributions do not have lower or upper bound truncations.

### **3.1.3.3. References**

EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. EPA/600/P-96/002Fabc, August 1997.

USDA (1988) Nationwide Food Consumption Survey 1987/88 Household Food Use. United States Department of Agriculture 1987/88 NFCS Database.

## **TABLE 3 - 7**

# **EMPIRICAL TOTAL VEGETABLE INTAKE RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



Source: Based on U.S. EPA's analyses of the 1989/91 CFSII, all regions; see EPA (1997), Table 9-4, page 9-12.

† These data are entered in the PC column of the custom distribution

§ These data are entered in the DC column of the custom distribution

## **TABLE 3 - 8**

## **EMPIRICAL TOTAL FRUIT INTAKE RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



Source: Based on U.S. EPA's analyses of the 1989/91 CFSII, all regions; see EPA (1997), Table 9-3, page 9-11.

† These data are entered in the PC column of the custom distribution

§ These data are entered in the DC column of the custom distribution

# **TABLE 3 - 9**

### **SIMULATED TOTAL VEGETABLE INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - STATISTICS**



# **TABLE 3 - 10**

### **SIMULATED TOTAL VEGETABLE INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



### **TABLE 3 - 11**

## **SIMULATED TOTAL FRUIT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - STATISTICS**



# **TABLE 3 - 12**

# **SIMULATED TOTAL FRUIT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



## **3.1.4. Fish Ingestion Rate (IRF)**

## **3.1.4.1. Explanation**

 Ingestion of contaminated fish is a potential source of human exposure to toxic chemicals; particularly bioaccumulating lipophilic organic compounds that are not otherwise metabolized or excreted. When evaluating fish consumption exposures, the key question is: What is the daily fish intake rate? For this guidance, this question is answered indirectly by combining a total daily fish consumption estimate (Ruffle et al 1994) with a site-specific estimate  $(F_f)$  of the fraction of total fish consumed that are obtained onsite (or in the locality of the facility). The Department would be open to receiving information from scientifically defensible surveys of recreational fishing activities in the locality of any given site.

 The fish ingestion distributions recommended in this guidance are unlikely to be representative of rates in populations that rely on subsistence fishing for a substantial portion of their diet. EPA guidance (EPA 1997) should consulted in these instances to develop distributions more appropriate for a subsistence fishing scenario. Depending on the frequency and duration of recreational fishing activity, these fish ingestion rates may not be sustainable from small bodies of water. The ability of a small lake or stream to sustain specific levels of fishing should be taken into consideration during development of the exposure model.

## **3.1.4.2. Distribution Definition**

 As recommended by EPA (1997), the lognormal distributions of Ruffle et al (1994) (Table 3- 13) were used to estimate daily long-term fish intake (g/d) distributions. Because the data on which the Ruffle et al (1994) distributions are based is now over twenty years out of date, EPA (1997) recommends that the distributions be shifted upward by 50% to estimate the current fish intake distribution. This is accomplished by adjusting the log mean ( $\mu$ ) by adding ln(1.5) = 0.4 to it so as to shift the distribution upward by 50%. Tables 3-14 and 3-15 summarize results obtained using the shifted Ruffle et al (1994) parameters (Table 3-13) in the following relationship:

$$
IRF \sim LogNormal[\mu, \sigma, LB, UB]; \quad g/d
$$

**Equation 3. 5** 





UB  $=$  Upper bound of daily fish ingestion rate  $(g/d)$ 

 Truncation of the daily fish ingestion rate distribution is based on a physical lower bound of 0 g/d. No upper bound is assumed.

## **3.1.4.3. References**

- EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. EPA/600/P-96/002Fabc, August 1997.
- Gephart LA, Tell JG & Triemer LR (1994) Exposure factors manual. *Journal of Soil Contamination* **3**, 47-117.
- Ruffle B, Burmaster DE, Anderson PD & Gordon HD (1994) Lognormal distributions for fish consumption by the general U.S. population. *Risk Analysis* **14**, 395-404.
- USDA (1988) Dataset: Nationwide Food Consumption Survey 1987/88 Household Food Use. United States Department of Agriculture 1987/88 NFCS Database.

# **TABLE 3 - 13**

# **BEST-FIT FISH INGESTION RATE (g/d) DISTRIBUTIONS - PARAMETERS**



Source: Ruffle et al (1994), log mean values shifted upward 50% per EPA (1996), page 10-50.

# **TABLE 3 - 14**

# **SIMULATED FISH INGESTION RATE (g/d) DISTRIBUTIONS - STATISTICS**



# **TABLE 3 - 15**

### **SIMULATED FISH INGESTION RATE (g/d) DISTRIBUTIONS - PERCENTILES**



### **3.1.5. Animal Product Ingestion Rate (IRM)**

### **3.1.5.1. Explanation**

 Ingestion of contaminated food is a potential source of human exposure to toxic chemicals. Consumers of home-produced foods may be of particular concern because exposure resulting from local site contamination may be higher for this subpopulation (EPA 1997). The principal sources of data regarding consumption rates of various food items among the United States population is the Nationwide Food Consumption Survey (NFCS), which is conducted by the U.S. Department of Agriculture (USDA) approximately every 10 years, and the USDA Continuing Survey of Food Intakes by Individuals (CFSII).

 The most recent NFCS was carried out in 1987-88 and represents more than 10,000 individuals in approximately 4,300 households. Although this sample is smaller than the previous NFCS (more than 36,000 individuals in approximately 15,000 households were sampled in 1977-78), it is considered to be most representative of current eating patterns among the general population. The NFCS has two components. The household component collects data over a seven-day period regarding the socioeconomic and demographic characteristics of the households, as well as the types and sources of food consumed. The individual component (CFSII) gathers information on food consumption by individuals within each household over a three-day period. The data have been used to generate both consumer-only and per capita rates of intake for individual food items and for categories of foods.

 CFSII data from the 1989-1991 survey have been used by EPA to generate per capita intake rates for various food items and food groups. Approximately 15,000 individuals provided intake data over the three survey years. This is considered as the key study for the intake of meat and dairy products (EPA 1997).

#### **3.1.5.2. Distribution Definition**

Percentiles for the ingestion rate of total meats (Table 3-16) and total dairy products (Table 3-17) were developed by the U.S. EPA from CSFII data (all regions). As such, they are subject to the assumptions and professional judgment brought to the analysis by U.S. EPA. Note that these data are normalized to body weight (g/[kg·day]), thus obviating the need to account for a correlation between  $IRM_k$  and body weight. Rather then assume a continuous distribution, a custom distribution was used to replicate the distribution percentiles as presented in the source publication. The properties of the simulated cumulative distribution, generated using the relationship shown below, are summarized in Tables 3-18, 3-19, 3-20, and 3-21.

 $\textit{IRM} \sim \textit{Custom}[DC, PC]; \quad g/[kg \cdot d]$ 

### **Equation 3. 6**

Where:



 As replicates of the original data, custom distributions do not have lower or upper bound truncations.

### **3.1.5.3. References**

EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. EPA/600/P-96/002Fabc, August 1997.

USDA (1988) Nationwide Food Consumption Survey 1987/88 Household Food Use. United States Department of Agriculture 1987/88 NFCS Database.

# **TABLE 3 - 16**

## **EMPIRICAL TOTAL MEAT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



Source: Based on U.S. EPA's analyses of the 1989/91 CFSII, all regions; see EPA (1997), Table 11-1, page 11-8.

† These data are entered in the PC column of the custom distribution

§ These data are entered in the DC column of the custom distribution
# **TABLE 3 - 17**

# **EMPIRICAL TOTAL DAIRY PRODUCT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



Source: Based on U.S. EPA's analyses of the 1989/91 CFSII, all regions; see EPA (1997), Table 11-2, page 11-9.

† These data are entered in the PC column of the custom distribution

§ These data are entered in the DC column of the custom distribution

# **TABLE 3 - 18**

# **SIMULATED TOTAL MEAT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - STATISTICS**



# **TABLE 3 - 19**

# **SIMULATED TOTAL MEAT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



# **TABLE 3 - 20**

# **SIMULATED TOTAL DAIRY PRODUCT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - STATISTICS**



# **TABLE 3 - 21**

## **SIMULATED TOTAL DAIRY PRODUCT INGESTION RATE (g/[kg·day]) DISTRIBUTIONS - PERCENTILES**



# **3.2. Dermal Factors 3.2.1. Soil Adherence Factor (AF) 3.2.1.1. Explanation**

 Chemical exposure via dermal contact with contaminated soil is a potentially important exposure pathway in humans. There is considerable uncertainty associated with this pathway due to our relative lack of knowledge concerning its critical components: the area of skin exposed during each contact event, the extent to which soil adheres to the skin, the degree to which contaminants move through the skin from the soil, the "effective thickness" of the soil, the duration of soil adherence to skin, and the frequency of skin contact with soil. Despite these complicating factors, dermal uptake of contaminants from soil is typically modeled as a function primarily of soil adherence and dermal absorption factor (EPA 1997). With the recognition that more sophisticated and perhaps more accurate techniques are under development, the adherence factor approach has been retained in this guidance for purposes of simplicity and conformance with current U.S. EPA practices.

 Most studies of the soil-to-skin adherence factor have measured adherence of soil to the hands (Kissel et al 1996a; Driver et al 1989; Duggan et al 1985; Que Hee at al 1985; Gallacher et al 1984; Charney et al 1980; Roels et al 1980; Lepow et al 1975). Finley et al (1994) have reviewed these studies and have proposed a soil-to-skin adherence probability density function (PDF) for use in probabilistic risk assessments. Kissel (1995), however, presents a credible argument that the composite PDF proposed by Finley et al (1994) is artificially and substantially shifted toward lower values due to misinterpretation of the study by Que Hee et al (1985). Furthermore, the claim by Finley et al (1994b) that soil type, particle size, and indoor versus outdoor activity have minimal influence on soil-to-skin adherence has been challenged (Kissel et al 1996ab; Kissel 1995; Driver et al 1989).

 EPA (1997; Tables 1-2 and 6-16) suggests the use of central estimates of AF based on body part and activity. Sedman (1989) indicates that  $0.5 \text{ mg/cm}^2$  event is potentially a maximum load value for soil adhering to skin. EPA (1998) has suggested, based on NCEA (!998), single-point default values of 0.08 mg/cm<sup>2</sup> event ("adult") and 0.3 mg/cm<sup>2</sup> event ("child") for this parameter. CalEPA (1996) suggests the following groupings, based on activity patterns, for single point soil dermal loading values: "background level"; i.e., activities not involving soil contact - 0.01 mg/cm<sup>2</sup>·event, "standard"; i.e., average soil contact in outdoor activities - 0.1 mg/cm<sup>2</sup>·event, "high"; i.e., repeated direct contact with soil - 1.0 mg/cm<sup>2</sup> event, and "very high"; i.e., caked soil on skin - 10 mg/cm<sup>2</sup> event. Kissel et al (1996b) recommend use of similar soil loading classes with identical values. These grouped soil loading values do not address the probability of

experiencing a given activity and several investigators (CalEPA 1996; EPA 1997; Kissel et al 1996b) currently suggest that insufficient data are available to develop a distribution or a probability function for soil loading.

# **3.2.1.2. Distribution Definition**

 NCEA (1998) analyzed adult body-part- and activity-specific adherence factor data provided by Kissel et al (1996b) and Holmes et al (1998) to generate weighted adherence factor estimates for a variety of activities. These data were used to define a lognormal distribution. Choice of the lognormal was based on its physical lower limit of 0 mg/cm<sup>2</sup>, ease of computation, and professional judgment. The simulated cumulative distribution, whose properties are summarized in Tables 3-22 and 3-23, was generated using the following relationship:

$$
AF_{7+} \sim LogNormal[\mu = -2.587, \sigma = 1.318, LB = 0, UB = 10]; \text{mg/cm}^2\text{.} event
$$

**Equation 3. 7** 

Where:



 Truncation of the soil adherence factor distribution was based on a physical lower bound of 0  $mg/cm<sup>2</sup>$  and an upper bound at the "very high" contact rate proposed by CalEPA (1996). The mean value (0.17 mg/cm<sup>2</sup>-event) of this distribution approaches the previous EPA (1997) default central tendency (CTE) value of 0.2 mg/cm<sup>2</sup>·event, while the median value (0.08 mg/cm<sup>2</sup>·event) is at the NCEA (1998) default value. The 90<sup>th</sup> percentile (0.41 mg/cm<sup>2</sup>-event) is between the "standard" and "high" loadings proposed by CalEPA (1996).

 NCEA (1998) also analyzed child body-part-specific adherence factor data to generate adherence factor estimates for dry and wet soils of 0.3 mg/cm<sup>2</sup>·event and 1.0 mg/cm<sup>2</sup>·event, respectively. These data were used to define a lognormal distribution with a geometric mean of 0.3 mg/cm<sup>2</sup> event and a 95<sup>th</sup> percentile value of 1.0 mg/cm<sup>2</sup> event. Choice of the lognormal was based on its physical lower limit of 0 mg/cm<sup>2</sup> event, ease of computation, and professional judgment. The simulated cumulative distribution, whose properties are summarized in Tables 3- 22 and 3-23, was generated using the following relationship:

 $AF_{1-6} \sim LogNormal[\mu = -1.20, \sigma = 0.73, LB = 0, UB = 10]; \quad mg/cm^2 \cdot event$ 

**Equation 3. 8** 

### Where:



 Truncation of the soil adherence factor distribution was based on a physical lower bound of 0  $mg/cm<sup>2</sup>$  and an upper bound at the "very high" contact rate proposed by CalEPA (1996). The mean value (0.39 mg/cm<sup>2</sup>-event) of this distribution is near the NCEA (1998) value for dry soils, while the 90<sup>th</sup> percentile (0.76 mg/cm<sup>2</sup>-event) is near the value for wet soils. Previous EPA (1997) default central tendency (CTE) and RME values of  $0.2 \text{ mg/cm}^2$ -event and  $1.0 \text{ m}$  $mg/cm^2$ -event fall at approximately the 30<sup>th</sup> and 95<sup>th</sup> percentiles, respectively.

# **3.2.1.3. References**

- CalEPA (1996) **Air Toxics Hot Spots Program Risk Assessment Guidelines Part IV, Technical Support Document, Exposure Assessment and Stochastic Analysis**. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. (Public Review Draft, December 1996).
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- Roels HA, Buchet JP, Lauwerys RR, Bruaux P, Claeys-Thoreau F, Lafontaine A & Verduyn G (1980) Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. *Environmental Research* **22**, 81-94.
- Sedman RM (1989) The development of applied action levels for soil contact: A scenario for the exposure of humans to soil in a residential setting. *Environmental Health Perspectives* **79**, 291-313.

## **TABLE 3 - 22**

### **SIMULATED SOIL ADHERENCE FACTOR DISTRIBUTIONS - STATISTICS**



## **TABLE 3 - 23**

# **SIMULATED SOIL ADHERENCE FACTOR DISTRIBUTIONS - PERCENTILES**



# **3.3. Inhalation Factors 3.3.1. Inhalation Rate (IRA) 3.3.1.1. Explanation**

 The human health risk associated with exposure to airborne contaminants is a function of contaminant concentration, duration of exposure and the inhalation rate (IRA). The inhalation rate, also referred to as the ventilation rate or breathing rate, is typically measured as the minute volume (*i.e.*, the total volume of air leaving the lung each minute). The minute volume ( $V<sub>E</sub>$ ) is the product of the volume of air exhaled with each breath (the tidal volume,  $V_T$ ) and the respiratory rate (breaths/min). The volume of air *entering* the lung is actually slightly greater because the amount of oxygen taken in is slightly more than the amount of carbon dioxide released. In the short term, the principal factors which influence inhalation rate are the level of physical activity, body temperature, and ambient temperature. In the longer term, inhalation rate is affected by age, gender, body weight and health status.

 The traditional approach used to estimate inhalation rate for a specified time period has been to calculate a time-weighted average of ventilation rates associated with periods of physical activity of varying duration. Layton (1993) has developed a technique permitting calculation of an energy-dependent inhalation rate, but it requires estimation of metabolic rate (based upon body weight) and the appropriate activity factor. Finley et al (1994) present a distribution for inhalation rate based upon the work of Layton (1993), but the authors do not address the potential correlation between body weight and inhalation rate. The uncertainty related to the unspecified correlation between body weight and inhalation rate may be avoided by use of an allometric equation (IRA<sub>k</sub> =  $0.5458 \times BW_k^{0.80}$ , n = 691, r<sup>2</sup> = 0.98) developed by Stahl (1967, cited in EPA 1993), where IRA is the inhalation rate of the *k*th age  $(m^3/d)$  and BW<sub>k</sub> is the body weight of the *k*th age (kg).

 Breathing rate may be more strongly influenced by activity than by body weight, in that, beyond the physiological minimum rate of air intake, an individual may engage in a variety of activities that generate a variety of breathing rates irrespective of body weight. The California Air Resources Board (CARB) sponsored two activity pattern studies in which activities of 2900 adults and children were recorded retrospectively for the previous 24 hours via telephone interview. CalEPA (1996) used the CARB data to construct distributions of daily breathing rates per kg body weight for children (ages 0-12 years) and adolescents/adults (ages > 12 years).

# **3.3.1.2. Distribution Definition**

For children (ages 0 to 12 years), the CalEPA daily breathing rates per kg body weight

distributions (Table 3-24) were used to construct distributions for  $IRA<sub>0-12</sub>$ . These data are used, in conjunction with Equation 3.8, to define air intake rate distributions for both genders combined. The statistics and percentiles of these distributions are summarized in Tables 3-25 and 3-26, respectively.

$$
IRA_{0-12} \sim LogNormal[\mu = 6.10, \sigma = 0.15, LB = 342.5, UB = 747.5]; \quad L/[kg \cdot d]
$$

**Equation 3. 9** 

Where:



Truncation of the daily inhalation rate distribution at the lower and upper bound is at the  $1<sup>th</sup>$ percentile and maximum values, respectively, as reported by CalEPA (1996).

 For adults (ages greater than 12 years), the CalEPA daily breathing rates per kg body weight distributions (Table 3-24) were used to construct distributions for  $IRA_{>12}$ . These data are used, in conjunction with Equation 3.8, to define air intake rate distributions for both genders combined. The statistics and percentiles of these distributions are summarized in Tables 3-25 and 3-26, respectively.

$$
IRA_{>12} \sim LogNormal[\mu = 5.38, \sigma = 0.28, LB = 112.8, UB = 638.8]; \quad L/[kg \cdot d]
$$

**Equation 3. 10** 

Where:



Truncation of the daily inhalation rate distribution at the lower and upper bound is at the  $1<sup>th</sup>$ 

percentile and maximum values, respectively, as reported by CalEPA (1996).

# **3.3.1.3 References**

- CalEPA (1996) **Air Toxics Hot Spots Program Risk Assessment Guidelines Part IV, Technical Support Document, Exposure Assessment and Stochastic Analysis**. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. (Public Review Draft, December 1996).
- EPA (1993) **Wildlife Exposure Factors Handbook, Vol. I.** Office of Research and Development, Washington, DC. EPA/600/R-93/187a.
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- Layton DW (1993) Metabolically consistent breathing rates for use in dose assessments. *Health Physics* **64**, 23-36.
- Stahl WR (1967) Scaling of respiratory variables in mammals. *Journal of Applied Physiology* **22**, 453-460.

# **TABLE 3 - 24**

# **EMPIRICAL DAILY BREATHING RATE DISTRIBUTIONS - STATISTICS & PERCENTILES**



Source: CalEPA (1996), Tables 3.19 and 3.20, pages 3-31 & 3-32.

† Value calculated from CalEPA (1996) data.

 $\ddagger$  Value equal to the 1<sup>th</sup> percentile value reported by CalEPA (1996).

§ Value equal to the maximum value reported by CalEPA (1996).

## **TABLE 3 - 25**

# **SIMULATED DAILY BREATHING RATE DISTRIBUTIONS - STATISTICS**



### **TABLE 3 - 26**

# **SIMULATED DAILY BREATHING RATE DISTRIBUTIONS - PERCENTILES**



## **3.4. Temporal Factors**

 Dose is a function of length of exposure. Exposure frequency expresses the time that an individual is in contact with contaminated media via a given exposure route. An individual's activity patterns strongly influence which exposure routes will be exploited and for how long. While it may be theoretically possible for an individual to engage in one activity or experience one exposure route for 24 hours per day ( $EF_{hd} = 24$ ) and 365 days per year ( $EF_{dv} = 365$ ), this is generally an unreasonable assumption. A meaningful exposure model should more realistically account for the time an individual spends contacting contaminated media as a function of their activity pattern. This can be done in different ways depending on exposure route.

 Although exposure frequencies and durations are likely to be highly site- and scenariospecific, as well as strongly influenced by season, activity, and age, some EPA central tendency and upper bound single point estimates are available (EPA 1992, 1996ab). These single point values can serve as a "point-of-departure" for establishing default distributions for these various exposure frequency parameters. The brief discussions below introduce instances where risk assessors may have an opportunity to develop alternative estimates for exposure parameters based on published data and/or site-specific time-use surveys.

For incidental soil ingestion, exposure to a contaminant of concentration  $C_s$  in soil is assumed to occur at a rate of  $x$  mg per 24 hour day (IRS),  $EF_{dy}$  days per year. The worst case assumption is that soil contaminated at concentration  $C_s$  is ubiquitous in an individual's environment; i.e., all the soil that they contact on a daily basis is contaminated at a level of  $C_s$ , in which case  $EF_{hd} = 24$ . Alternatively, it is possible to consider that an individual has an opportunity to incidentally ingest soil contaminated at concentration  $C_s$  only at certain times during a 24 hour day; for example, while gardening or playing outdoors. In these instances,  $EF_{hd}$  $<$  24, but EF<sub>dy</sub> may remain the same.

For tap water ingestion, exposure to a contaminant of concentration  $C_w$  in water is assumed to occur at a rate of  $x \perp$  per 24 hour day (IRW),  $EF_{dy}$  days per year. Here again, the worst case assumption is that an individual obtains all of their drinking water, all contaminated at concentration  $C_w$ , from a single source, to which they are exposed 24 hours per day. This is a plausible scenario for an individual who remains at their residence all day or whose drinking water sources at all locations contain the same contaminant at concentration  $C_w$ . Again, it is possible to consider that an individual has an opportunity to consume water of concentration  $C_w$ only at certain times during a 24 hour day; for example, while at home or at work, but not both. In this instance,  $EF_{hd} < 24$  (perhaps just time spent at home), but  $EF_{dy}$  may remain the same.

For ingestion of food items, exposure to a contaminant of concentration  $C_v$ ,  $C_m$ ,  $C_{dp}$ ,  $C_e$ , or  $C_f$ in food stuffs is assumed to occur at a rate of *x* g per kg body weight per 24 hour day (IRM) or *x* g per 24 hour day (IRF),  $EF_{dy}$  days per year;  $EF_{hd}$  is not a factor in this case. The fraction ( $F_{v}$ ,  $F_m$ ,  $F_{dp}$ ,  $F_e$ , or  $F_f$ ) of all the food they eat that is contaminated is the key factor in estimating exposure. Here again, the worst case assumption is that an individual obtains all of their food, all contaminated at concentration  $C_v$ ,  $C_m$ ,  $C_{dp}$ ,  $C_e$ , or  $C_f$  from a single source ( $F_v$ ,  $F_m$ ,  $F_{dp}$ ,  $F_e$ , or  $F_f$ = 1). Again, it is possible to consider that an individual has an opportunity to consume food from different sources, some contaminated and some not, so that  $F_v$ ,  $F_m$ ,  $F_{dp}$ ,  $F_e$ , or  $F_f$  < 1, with  $EF_{\text{dy}}$  remaining the same.

For dermal contact with soil, exposure to a contaminant of concentration  $C_s$  in soil is estimated to occur at a rate of *x* mg per cm<sup>2</sup> per event (DA<sub>soil</sub>),  $EF_{\text{evd}}$  events per day, for  $EF_{\text{dy}}$ days per year; EF<sub>hd</sub> is not a factor in this case. As was the case with incidental ingestion of soil, the idea that all the soil contacted by an individual on a daily basis is contaminated at a level of  $C_s$  may be unrealistic. Even allowing for at least one soil contact activity (such as gardening or excavation work) per day ( $EF_{\text{evd}} = 1$ ), it is unlikely that these activities occur every day of the year, so that  $EF_{dy} < 365$ .

For dermal contact with water, exposure to a contaminant of concentration  $C_w$  in water is estimated to occur at a rate of *x* mg per cm<sup>2</sup> per event (DA<sub>water</sub>), t<sub>event</sub> hours per event,  $EF_{\text{evd}}$ events per day, for  $EF_{dy}$  days per year;  $EF_{hd}$  is not a factor in this exposure route. As was the case with contact with soil, the idea that all the water contacted by an individual on a daily basis is contaminated at a level of  $C_w$  may be unrealistic. Even allowing for at least one water contact activity (such as showering) per day ( $EF_{\text{evd}} = 1$ ), it is unlikely that these activities occur every day of the year (thus  $EF_{dy} < 365$ ) or last all day on any day (thus t<sub>event</sub> « 24).

For inhalation of vapors or particulates, exposure to a contaminant of concentration  $C_a$  or PM<sub>10</sub> in air is assumed to occur at a rate of *x* m<sup>3</sup> per 24 hour day (IRA), EH<sub>hd</sub> hours per day, EF<sub>dy</sub> days per year. Again, there may be site-specific instances where an individual breathes air contaminated at concentration  $C_a$  or  $PM_{10}$  all day (EF<sub>hd</sub> = 24) but such instances are not likely to be typical. Even for an individual remaining in one location (e.g., at home) for long periods, unless they were totally sedentary, any change in activity pattern would create a change in breathing rate, possibly necessitating adjustment of IRA (or expressing IRA on a  $m<sup>3</sup>$  per hour basis).

### **3.4.1 Event Duration (tevent) 3.4.1.1. Explanation**

 Central tendency and upper bound point estimates for the duration of exposure for a bathing event  $(t_{event}^{bath})$  are 0.20 and 0.25, respectively (EPA 1992). For a swimming event, point estimates of central tendency and upper bound exposures  $(t_{event}^{swin})$  are 0.5 and 1, respectively (EPA 1992).

### *3.4.1.2. Distribution Definitions*

For t<sub>event</sub> bath, it is assumed that bathing time for an individual is a minimum of 0 (i.e., individual does not bath), most likely 0.20 (i.e., EPA CTE), and a maximum of 0.25 (i.e., EPA RME). For t<sub>event</sub> swim, it is assumed that swimming time for an individual is a minimum 0 (i.e., individual does not swim), most likely 0.5 (i.e., EPA CTE), and a maximum of 1.0 (i.e., EPA RME). Because the regions of zero probability are defined in this case by absolute physical limitations (the 24-hour length of a day) and the breakpoint (most likely case) can be reasonably determined from existing time survey data, a triangular distribution was adopted (Seiler & Alvarez 1996), as follows:

$$
t_{event}^{bath} \sim Triangular[\text{min} = 0, \text{mod} = 0.20, \text{max} = 0.25]; \quad h \cdot / event
$$

**Equation 3. 11** 

$$
t_{event}^{swim}
$$
 ~ Triangular $[\text{min} = 0, \text{mod} = 0.5, \text{max} = 1.0];$  *hr/event*

**Equation 3. 12** 

### Where:



 Properties of the simulated cumulative distributions, generated using the above relationships, are summarized in Tables 3-27 and 3-28.

# **3.4.1.3. References**

- EPA (1992) **Dermal Exposure Assessment: Principles and Applications**. Office of Research and Development, U.S. Environmental Protection Agency. (EPA/600/8-91/011B, January 1992, Interim Report)
- Seiler FA & Alvarez JL (1996) On the selection of distributions for stochastic variables. *Risk Analysis* **16**, 5-18.

# **TABLE 3 - 27**

# **SIMULATED EVENT DURATION (hr/event) DISTRIBUTIONS - STATISTICS**



### **TABLE 3 - 28**

## **SIMULATED EVENT DURATION (hr/event) DISTRIBUTIONS - PERCENTILES**



# **3.4.2. Event Frequency (EFevd)**

# **3.4.2.1. Explanation**

 EPA (1992) gives 1 as the central tendency and upper bound point estimate for daily event frequency  $(EF_{\text{evd}})$  dermal water contact while bathing and swimming.

## **3.4.2.2. Distribution Definitions**

For EF<sub>evd</sub>, it is assumed that events involving dermal contact with water for an individual occur a minimum of 0 times per day (i.e., no bathing or swimming), most likely 1 time per day (i.e., EPA CTE/RME), and a maximum of 2 times per day (i.e., two baths per day or one bath and one swim per day).

$$
EF_{\text{evd}}^{\text{bath}} \sim Triangular[\text{min} = 0, \text{mod} = 1, \text{max} = 2]; \quad \text{events}/\text{d}
$$

**Equation 3. 13** 

$$
EF_{evd}^{swim} \sim Triangular[\min = 0, \mod = 1, \max = 2]; \quad events/d
$$

**Equation 3. 14** 

Where:



 Properties of the simulated cumulative distributions, generated using the above relationship, are summarized in Tables 3-29 and 3-30.

# **3.4.2.3. References**

EPA (1992) **Dermal Exposure Assessment: Principles and Applications**. Office of Research and Development, U.S. Environmental Protection Agency. (EPA/600/8-91/011B, January 1992, Interim Report)

# **TABLE 3 - 29**

# **SIMULATED EVENT FREQUENCY DISTRIBUTION - STATISTICS**



### **TABLE 3 - 30**

# **SIMULATED EVENT FREQUENCY DISTRIBUTIONS - PERCENTILES**



# **3.4.3. Exposure Frequency, Daily (EFhd)**

# **3.4.3.1. Explanation**

 Because this site- and scenario-specific parameters is strongly influenced by season, activity, and age, establishment of an *a priori* distribution is difficult. These are, however, the results of time-use surveys that are available (EPA 1997) to starting point for establishing distributions for the following typical exposure scenarios: (a) time spent indoors at home, (b) time spent outdoors at home, (c) time spent away from home at work, (d) time away from home on vacation, and (e) time away from home for other reasons (school, shopping, daycare, etc.). With this approach, all of a receptor's time (on an hours/year basis) is accounted for, then the total apportioned across all exposure scenarios.

 The mean time spent indoors by children (ages 3-11 years) is 17-19 hr/d and time outdoors is 5-7 hr/d (EPA 1997). Gephart et al (1994) calculated a "home/away from home" value of 138/30 hr/wk for children ages 3-11 years. For the child indoor residential "most likely" exposure,  $EF_{hk}$  values were those derived by Gephart et al (1994). For a "minimum" case, it is assumed that a child would at least sleep indoors at home for 8 hr/d, with 2 hr/d spent outdoors. For a "maximum" case, a child not attending school or day care outside the home could potentially spend 18 hr/d at home, with 6 hr/d spent outdoors. A yearly two-week family vacation (24 hr/d away from the residence) is also assumed, based on cultural norms. Time away from the residence for "other" reasons includes such activities as pre-school, daycare, doctor visits, etc., and is calculated as the remainder of the total time otherwise not accounted for in another activity.

The mean time spent indoors for adults (men and women,  $> 12$  years of age) is given as 16.4 hr/d (21 hr/d total) and time outdoors for adults (men and women, 12 years and older) as 2.0 hr/d (residential) (EPA 1997). Gephart et al (1994) calculated the weighted mean hours per week (7 day week) for "home/away from home" as 98/70 hr/wk for men, 116/52 hr/wk for women, and 108/60 hr/wk for men and women combined. For the adult indoor residential "most likely" exposure values were those derived by Gephart et al (1994). For a "minimum" case, it is assumed that a adult would at least sleep indoors at home for 7 hr/d, with 1 hr/d spent outdoors. For a "maximum" case, a adult not working outside the home could potentially spend 20 hr/d at home, with 4 hr/d spent outdoors. A yearly two-week vacation (24 hr/d away from the residence) is also assumed, based on cultural norms. Time away from the residence for "other" reasons includes such activities as shopping, visiting, etc., and is calculated as the remainder of the total time otherwise not accounted for in another activity.

 Gephart et al (1994), based on information in EPA (1989), report a distribution of time spent at work for adults (males and females, ages 18 to 64) with a median value of 4.5 hr/d (5-day week; includes lunch, breaks, travel). They also report the average time spent at work (5-day week) by 20 to 44-year-old women and men as 3.6 hr/d and 8.2 hr/d, respectively. Thus, for the adult occupational "most likely" exposure, a value of 9 hr/d was selected (to include travel time). For a "minimum" case, it was assumed that an individual spends no time at work during the year or 0 d/yr. For a "maximum" case, a worker could potentially spend 16 hr/d at work.

# **3.4.3.2. Distribution Definition**

 Because the regions of zero probability are defined in this case by absolute physical limitations (the 24-hour length of a day) and the breakpoint (most likely case) can be reasonably determined from existing time survey data, a triangular distribution was adopted (Seiler & Alvarez 1996); the parameters for which are summarized in Table 3-31. The simulated cumulative distribution, whose properties are summarized in Tables 3-32 and 3-33, was generated using the following relationship:

$$
EF_{hd}^a \sim Triangular[\min, \mod, \max]; \quad hr/d
$$

**Equation 3. 15** 

Where:



# **3.4.3.3. References**

- EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. (Update to EPA/600/P-96/002Fabc, August 1997)
- Gephart LA, Tell JG & Triemer LR (1994) Exposure factors manual. *Journal of Soil Contamination* 3, 47-117.

Seiler FA & Alvarez JL (1996) On the selection of distributions for stochastic variables. *Risk Analysis* **16**, 5-18.

# **TABLE 3 - 31**

# **BEST-FIT HOURLY EXPOSURE FREQUENCY (hr/d) DISTRIBUTIONS -PARAMETERS**



# **TABLE 3 - 32**

# **SIMULATED HOURLY EXPOSURE FREQUENCY (hr/d) DISTRIBUTIONS - STATISTICS**



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# **TABLE 3 - 33**

### **SIMULATED HOURLY EXPOSURE FREQUENCY (hr/d) DISTRIBUTIONS - PERCENTILES**



### **3.4.4. Exposure Frequency, Yearly (EF<sub>dy</sub>)**

### **3.4.4.1. Explanation**

The EPA residential RME default value for  $EF_{dy}$  of 350 d/yr, assumes that an individual is "in residence" for some number of hours per day at least 7 days per week and 50 weeks per year (EPA 1998). The EPA RME default value for  $EF_{dy}$  of 250 d/yr for occupational exposures assumes that an individual is "at work" for 8 hours per day 5 days per week and 50 weeks per year (EPA 1998). For dermal contact with water (swimming exposures), CTE and RME values for  $EF_{dy}$  are 5 d/yr and 150 d/yr, respectively (EPA 1992). For dermal contact with soil, CTE and RME values for  $EF_{dy}$  are 40 d/yr and 350 d/yr, respectively (EPA 1992).

#### **3.4.4.2. Distribution Definition**

For  $EF_{dy}$ <sup>home</sup>, it is assumed that an individual could be at home a minimum of 350 d/yr (i.e., two week annual vacation) and a maximum of 365 d/yr (i.e., no two week annual vacation or vacation spent at home). For  $EF_{dy}$ <sup>work</sup>, it is assumed (based on the above cited information and professional judgment) that an individual could be at work between a minimum of 0 d/yr (i.e., doesn't work) to a maximum of 350 d/yr (two week annual vacation). For  $EF_{dy}^{sol}$ , it is assumed that exposure could occur a minimum of 0 d/yr (i.e., an unlikely but plausible case of no soil contact), most likely 40 d/yr (i.e., EPA CTE), and at a maximum of 350 d/yr (i.e., EPA RME). For  $EF_{dy}^{swin}$ , it is assumed that exposure could occur a minimum of 0 d/yr (i.e., no swimming), most likely 5 d/yr (i.e., EPA CTE), and at a maximum of 150 d/yr (i.e., EPA RME).

 Because, in all these cases, the regions of zero probability are defined by absolute physical limits (length of a day, week, year) and the breakpoint (most likely case) can be reasonably determined from existing time survey data (as described above), uniform and triangular distributions are adopted (Seiler & Alvarez 1996), as follows:

$$
EF_{dy}^{\text{home}} \sim Uniform[\min = 350, \max = 365]; \quad d/yr
$$

**Equation 3. 16** 

$$
EF_{dy}^{work} \sim Uniform[\min = 0, \max = 365]; \quad d/yr
$$

**Equation 3. 17** 

$$
EF_{dy}^{soil} \sim Triangular[\min = 0, \mod = 40, \max = 350]; \quad d / yr
$$

### **Equation 3. 18**

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 $EF_{dy}^{swim} \sim Triangular[\text{min} = 0, \text{mod} = 5, \text{max} = 150]; \quad d / yr$ 

### **Equation 3. 19**

Where:



### **3.4.4.3. References**

- EPA (1998) **Region 9 Preliminary Remediation Goals (PRGs) 1998**. Region IX, U.S. Environmental Protection Agency. (May 1, 1998)
- EPA (1997) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. (Update to EPA/600/P-96/002Fabc, August 1997
- EPA (1992) **Dermal Exposure Assessment: Principles and Applications**. Office of Research and Development, U.S. Environmental Protection Agency. (EPA/600/8-91/011B, January 1992, Interim Report)

# **TABLE 3 - 34**

# **SIMULATED EXPOSURE FREQUENCY DISTRIBUTIONS - STATISTICS**



## **TABLE 3 - 35**

# **SIMULATED EXPOSURE FREQUENCY DISTRIBUTIONS - PERCENTILES**



# **3.4.5. Exposure Duration (ED) 3.4.5.1. Explanation**

### *Residential*

 Due to the mobility of the population at large, the exposure duration for an individual selected at random from the population is often considerably less that the expected lifetime of that individual. In the context of a residential scenario, exposure duration  $(ED_r)$  is the number of years that an individual is expected to stay at a given residence. This is equivalent to: (a) the *residential occupancy period* (ROP) or the number of years between the time an individual moves into a new residence and the time that individual dies or moves out of the residence (also called the average total residence time). However, few studies, including those of the U.S. Bureau of the Census, have evaluated ROP. Rather, most studies measure *current residence time*; that is, the time since moving into the current residence. Because good data are lacking for ROP, values for current residence time are often substituted in exposure assessments.

 Israeli & Nelson (1992) used average current residence time data from 1985 and 1987 American Housing Surveys to estimate the distribution of the ROP (average total residence time) for different types of dwellings. Johnson & Capel (1992) developed a model which provides an approximation of the ROP based on residential distribution for different age categories. Finley et al (1994; Table XI) developed, based on the age-specific probability of moving at a rate described by the U.S. Census, a cumulative distribution of duration of time in a residence since birth; i.e., the probability of an individual staying in his/her residence of birth for *t* years. Finley et al (1994) recommended that the Israeli & Nelson (1992) distributions be used to evaluate population exposures in terms of residence type and that the Johnson & Capel (1992) distributions be used to evaluate populations defined as individuals of specific ages.

### *Occupational*

 The exposure duration for an occupational scenario (ED) represents the number of years that an individual is expected to remain at a given occupation. Measurement of occupational exposure duration is subject to the same difficulties as measurement of residential exposure duration in that most surveys assess *current* tenure, rather than *total* tenure (i.e., average projected job tenure), and current occupational tenure is frequently substituted for total tenure in exposure assessments. Furthermore, occupational tenure should not be confused with employer tenure. Occupational tenure generally refers to the cumulative number of years a person has worked at his or her occupation, regardless of the number of employers, interruptions in employment, or time spent in other occupations (Carey 1988, 1990). Employer tenure, on the other hand, is the amount of time worked for the same employer.
Carey (1988) presents median occupational tenure values for 277 occupations and discusses their relationship to age, sex, race, education and other demographic characteristics. However, this summary does not present sufficient information to support a probability distribution. Finley et al (1994) have developed a distribution of current occupational tenure which is based upon data from the U.S. Department of Labor, Bureau of Labor Statistics (USDL 1992). Shaw & Burmaster (1997) used U.S. Census data on length of employment and a Gompertz model to infer projected job tenure distributions for male and female U.S. workers in selected industries and occupations.

## **3.4.5.2. Distribution Definition**

### *Residential*

 Because the exposure model described in this guidance (Section 4) evaluates populations defined as individuals of specific ages, the approach to average total residence time developed by Johnson & Capel (1992) was selected to define distributions of  $ED<sub>r</sub>$ . Rather then assume a continuous distribution, a custom distribution was used to replicate the distribution percentiles a presented in the source publication (Table 3-36). The properties of the simulated cumulative distribution, generated using the relationship shown below, are summarized in Tables 3-37 and 3-38.

$$
ED_r \sim Customer[DC, PC]
$$

**Equation 3. 20** 

Where:



 As replicates of the original data, custom distributions do not have lower or upper bound truncations.

### **Occupational**

Shaw & Burmaster (1996) provide percentiles for projected job tenure time distributions for both men and women in four industry groups (construction; manufacturing; transportation, communication, other public utilities; wholesale and retail trade). Rather then assume a continuous distribution, a custom distribution was used to replicate the distribution percentiles as presented in the source publication (Table 3-39). The properties of the simulated cumulative distribution, generated using the relationship shown below, are summarized in Tables 3-40 and 3-41.

$$
ED_o \sim Customer[DC, PC]; yr
$$

**Equation 3. 21** 

Where:



 It should be noted that while these distributions are gender-specific, they are not age-specific. Practitioners may therefore choose one industry type as representative for all members of the potentially exposed population or construct a site-specific custom distribution to represent the mix of industry types in which the occupational population is employed.

# **3.4.5.3. References**

- Carey M (1990) Occupational tenure, employer tenure, and occupational mobility. *Occupational Outlook Quarterly* **Summer 1990**, 55-60.
- Carey M (1988) Occupational tenure in 1987: many workers have remained in their fields. *Monthly Labor Review* **11**, 3-12 (October, 1988).
- Finley B, Proctor D, Scott P, Harrington N, Paustenbach D & Price P (1994) Recommended distributions for exposure factors frequently used in health risk assessment. *Risk Analysis* **14**, 533-553.
- Israeli M & Nelson CB (1992) Distribution and expected time of residence for U.S. households. Risk Analysis 12, 65-72.
- Johnson J & Capel J (1992) **Monte Carlo Approach to Simulating Residential Occupancy Periods and Its Application to the General U.S. Population**. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency (EPA 450/3-92-011).
- Shaw CD & Burmaster DE (1996) Distribution of job tenure for U.S. workers in selected industries and occupations. *Human and Ecological Risk Assessment*.

USDL (1992) Employee tenure and occupational mobility in the early 1990s. Bureau of Labor

Statistics, Washington, DC. USDL 92-386.

### **TABLE 3 - 36**

# **BEST-FIT RESIDENTIAL EXPOSURE DURATION DISTRIBUTIONS - PARAMETERS**



Source: Johnson & Capel (1992); data for age class < 3 years from Finley et al (1994)

† These data are entered in the PC column of the custom distribution

§ These data are entered in the DC column of the custom distribution

## **TABLE 3 - 37**

### **SIMULATED RESIDENTIAL EXPOSURE DURATION DISTRIBUTIONS - STATISTICS**



### **TABLE 3 - 38**

### **SIMULATED RESIDENTIAL EXPOSURE DURATION DISTRIBUTIONS - PERCENTILES**



## **TABLE 3 - 39**

## **EMPIRICAL OCCUPATIONAL EXPOSURE DURATION (yr) DISTRIBUTIONS - PARAMETERS**



\* Source: Shaw & Burmaster (1997), Table 5, Projected Job Tenure by Industry Sector ( $C =$  construction; M = manufacturing;  $TCPU = transportation, communication, other public utilities; WRT = whole sale & retail trade).$ 

† These data are entered in the PC column of the custom distribution

§ These data are entered in the DC column of the custom distribution

# **TABLE 3 - 40**

## **SIMULATED OCCUPATIONAL EXPOSURE DURATION (yr) DISTRIBUTIONS - STATISTICS**



# **TABLE 3 - 41**

### **SIMULATED OCCUPATIONAL EXPOSURE DURATION (yr) DISTRIBUTIONS - PERCENTILES**



# **3.5. General Factors 3.5.1. Body Weight (BW) 3.5.1.1. Explanation**

 The second National Health and Nutrition Survey (NHANES II), conducted under the auspices of the National Center for Health Statistics (NCHS) from February, 1976 to February, 1980 is the source of one of the largest anthropometric databases for the United States (NCHS 1987). NHANES II was designed to collect information on the nutritional status and prevalence of overweight among the civilian, non-institutionalized population. Measurements, including body weight and height, were obtained for 20,322 individuals aged 6 months to 74 years, representing males and females of several races. Certain high-risk subpopulations were intentionally over-sampled, but the data were statistically adjusted to reflect the entire U.S. population based on age, sex, and race. The size of this data set, combined with the fact that measurements were taken at various times of the day and during different seasons of the year from geographic regions across the country, makes this the most comprehensive and reliable database for determining body weight distribution in the United States.

 Through visual inspection and linear regression analysis of the NHANES II data, Brainard and Burmaster (1992) confirmed that height and the natural logarithm of body weight of adults (18-74 years) follow a Gaussian (normal) distribution for both males and females. In subsequent work, Burmaster and Couch (1997) used exploratory data analysis methods to fit lognormal distributions to percentiles of body weight for males and females as a function of age from six months through 74 years.

## **3.5.1.2. Distribution Definitions**

Values for  $\mu$  and  $\sigma$  reported by Burmaster and Couch (1997; Table II) were used, in conjunction with Equation 3.22, to define body weight distributions by age and gender. Values for μ, σ, LB, and UB are summarized in Table 3-42. The properties of the simulated cumulative distributions, generated using the relationship shown below, are summarized in Tables 3-43, 3- 44, 3-45, 3-46, 3-47, 3-48, 3-49, and 3-50.

$$
BW_{kg} \sim LogNormal[\mu, \sigma, LB, UB]kg
$$

**Equation 3. 22** 

Where:





Truncation of the adult body weight distribution was based on a lower bound at the  $0.1<sup>th</sup>$ percentile and an upper bound at the  $99.9<sup>th</sup>$  percentile, to include 99.8% of the male and female population in each age class. Distribution parameters given by Burmaster and Couch (1997) were used to calculate, with the Excel® LOGINV function, values for LB and UB at the  $0.1<sup>th</sup>$ percentile and the  $99.9<sup>th</sup>$  percentile, respectively.

## **3.5.1.3. References**

- Brainard J & Burmaster DE (1992) Bivariate distributions for height and weight of men and women in the United States. *Risk Analysis* **12**, 267-275.
- Burmaster DE & Crouch EAC (1997) Lognormal distributions of body weight as a function of age for males and females in the United States, 1976-1980. *Risk Analysis* **17**, 499-505.
- NCHS (1987) Anthropometric reference data and prevalence of overweight, United States, 1976-80. (Najjar MF & Rowland M) *Vital & Health Statistics* Series 11, No. 238. National Center for Health Statistics, Public Health Service, Department of Health and Human Services. (DHHS Pub. No. (PHS)87-1688, October 1987).

## **TABLE 3 - 42**

# **BEST-FIT BODY WEIGHT (kg) DISTRIBUTIONS - PARAMETERS**



Source: Burmaster and Couch (1997; Table II for males and females) to age 74.

† For purposes of this guidance, distributions for ages 75 to 79 are set equal to the distribution for age 74

# **TABLE 3 - 43**

## **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Males) - STATISTICS**



# **TABLE 3 - 44**

## **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Males) - STATISTICS**



## **TABLE 3 - 45**

## **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Females) - STATISTICS**



# **TABLE 3 - 46**

## **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Females) - STATISTICS**



### **TABLE 3 - 47**

### **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Males) - PERCENTILES**



# **TABLE 3 - 48**

### **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Males) - PERCENTILES**



# **TABLE 3 - 49**

## **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Females) - PERCENTILES**



### **TABLE 3 - 50**

### **SIMULATED BODY WEIGHT (kg) DISTRIBUTIONS (Females) - PERCENTILES**



 This section illustrates how the equations presented in Section 2 and the distributions given in Section 3 are combined, within the framework of the age- and gender-specific model described in Section 1.4, to calculate probabilistic estimates of exposure and risk. Using a hypothetical example, exposure and risk estimates obtained from this probabilistic model are compared and contrasted with those calculated using a typical U.S. EPA deterministic exposure model (EPA 1991, 1996a). This example considers three different exposure routes: (1) incidental ingestion of contaminated soil (Section 2.1.1),  $(2)$  dermal contact with contaminated soil (Section 2.1.6), and (3) inhalation of soil vapors (Section 2.1.8); all assumed to occur while engaging in activities outdoors at a residence. Risks associated with both carcinogens and noncarcinogens are considered in this example.

### **4.1. Deterministic Approach**

### **4.1.1. Exposure Equations**

 Risk of adverse effects due to incidental ingestion of contaminated soils is calculated by combining intake from soil (dose) with the an appropriate oral reference dose or slope factor, as follows (EPA 1991, 1996a):

$$
ADD_{soil(c)} = \frac{C_s \times CF_{km} \times EF \times IFS_{adj}}{AT_c}
$$

**Equation 4. 1** 

$$
ADD_{sol(n)} = \frac{C_s \times CF_{km} \times EF \times IFS_{adj}}{AT_n}
$$

**Equation 4. 2** 

$$
IFS_{adj} = \frac{IRS_c \times ED_c}{BW_c} + \frac{IRS_a \times ED_a}{BW_a}
$$

**Equation 4. 3** 

$$
HQ_{soil} = \frac{ADD_{soil(n)}}{RfD_o}
$$

**Equation 4. 4** 

 $ILCR<sub>soil</sub> = ADD<sub>soil(c)</sub> \times CFS<sub>o</sub>$ 

### **Equation 4. 5**

Where:



 For this example, the concentration of the hypothetical contaminant - Hypothene™ - in soil is assumed to be lognormally distributed, with  $n = 15$ , a mean of 1.25 mg/kg, and a standard deviation of 0.64. The one-sided upper 90% confidence limit on the lognormal mean is typically taken as the value of  $C_s$ . Thus, using Land's method (Gilbert 1987), the value of  $C_s$  is estimated to be 3.78 mg/kg. The oral cancer slope factor for Hypothene™ is 2 (mg/[kg⋅d])-1 and its reference dose is  $7 \times 10^{-5}$  mg/[kg⋅d]. Single point U.S. EPA standard default values for the other exposure factors were taken from EPA (1996a), as follows:





 Risk of adverse effects due to dermal contact with contaminated soils is calculated by combining intake from soil (dose) with the an appropriate oral reference dose or slope factor, as follows (EPA 1996a):

$$
ADD_{\text{derm}(c)} = \frac{C_s \times CF_{km} \times EF \times SFS_{\text{adj}} \times ABS}{AT_c}
$$

**Equation 4. 6** 

$$
ADD_{\text{derm}(n)} = \frac{C_s \times CF_{km} \times EF \times SFS_{\text{adj}} \times ABS}{AT_n}
$$

**Equation 4. 7** 

$$
SFS_{adj} = \frac{AF \times SA_c \times ED_c}{BW_c} + \frac{AF \times SA_a \times ED_a}{BW_a}
$$

**Equation 4. 8** 

$$
HQ_{\text{derm}} = \frac{ADD_{\text{derm}(n)}}{RfD_o}
$$

**Equation 4. 9** 

 $ILCR_{\text{derm}} = ADD_{\text{derm}(c)} \times CFS_o$ 

### **Equation 4. 10**

Where:



Values for C<sub>s</sub> RfD<sub>o</sub>, and CSF<sub>o</sub> remain the same. Single point U.S. EPA standard default values for the other exposure factors were taken from EPA (1996a), as follows:





 Risk of adverse effects due to inhalation of vapors released from contaminated soils is calculated by combining intake from soil (dose) with the an appropriate inhalation reference dose or slope factor, as follows (EPA 1996a):

$$
ADD_{air(c)} = \frac{C_s \times (1/VF_s) \times EF \times InhF_{adj}}{AT_c}
$$

**Equation 4. 11** 

$$
ADD_{air(n)} = \frac{C_s \times (1/VF_s) \times EF \times InhF_{adj}}{AT_n}
$$

**Equation 4. 12** 

$$
InhF_{adj} = \frac{IRA_c \times ED_c}{BW_c} + \frac{IRA_a \times ED_a}{BW_a}
$$

**Equation 4. 13** 

$$
HQ_{air} = \frac{ADD_{air(n)}}{RfD_i}
$$

**Equation 4. 14** 

$$
ILCR_{air} = ADD_{air(c)} \times CFS_i
$$

**Equation 4. 15** 

Where:

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Values for  $C_s$  remain the same. The soil-to-air volatilization factor is calculated as 2.03  $\times$  $10^9$  m<sup>3</sup>/kg. The inhalation cancer slope factor for Hypothene<sup>™</sup> is 0.4 (mg/[kg⋅d])<sup>-1</sup> and its inhalation reference dose is  $7 \times 10^{-5}$  mg/[kg⋅d]. Single point U.S. EPA standard default values for the other exposure factors were taken from EPA (1996a), as follows:





### **4.1.2. Results**

A deterministic model with U.S. EPA default exposure factors and a  $90<sup>th</sup>$  percentile upper confidence limit estimate of the soil contaminant concentration gives results as shown in the table below. In accord with OAR 340-122-084 $(1)(g)$ , both central tendency (mean, CTE) and reasonable maximum  $(90<sup>th</sup>$  percentile, RME) values are reported for HQ and ILCR. Both LCLR<sub>soil</sub> and ILCR<sub>derm</sub> RME values are greater than  $1 \times 10^{-6}$  indicating that, for this hypothetical example, an upper bound soil concentration of 3.78 mg/kg would pose an unacceptable cancer risk to individuals exposed via these exposure routes. The LCLR<sub>air</sub> RME value is substantially less than  $1 \times 10^{-6}$  indicating that exposure via inhalation does not pose an unacceptable cancer risk. All CTE and RME hazard quotients are less than 1, indicating that adverse non-cancer effects are not expected.



## **4.2. Probabilistic Approach**

## **4.2.1. Exposure and Risk Model Overview**

 This guidance uses an age-specific model that estimates exposures and risks to a population of potentially exposed human receptors. A flowchart of this model appears in Figure 4-1 (numbers enclosed in brackets [1] in the model description below are keyed to identical bracketed numbers in the figure). Each iteration of the model represents a statistical model of one individual drawn from this population. The range of characteristics of individuals within the population is represented by probability distributions for the various exposure factors.

 The modeling process starts [0] by generating [1] a distribution of ages representative of the population, then randomly selecting [2] an age *x* for the first person from that distribution. Ages can range from < 1 to 79 years and can be represented by a variety of distributions, from uniform (a typical default distribution) to a custom distribution conditioned by site-specific data. A distribution of gender ratios is then generated [3] and a gender *g* is randomly selected [4] from this distribution. The exposure duration represents the number of years an individual receptor is likely to be exposed to contamination in the future. Exposure duration distributions [5] are available for a variety of residential (by age class) and occupational scenarios. Estimates of exposure duration drawn at random from one of these distributions are added [6] to the age *x* of the individual receptor to determine the exposure interval (EI), the number of years between the start and end of exposure.

 For the exposure interval, exposure factor distributions are generated [7] and exposure factor values selected [8] from these for inclusion in the exposure algorithms. Note that any of these factors can be represented either as a single point value or as a distribution. Following selection of the various exposure factor distributions (or point estimates), the average or absorbed daily dose (ADD) that an individual receptor of age *x* and gender *g* receives via a given exposure route can be calculated [10], using the exposure equations [9] described in Section 2. The ADD is then multiplied by estimated of daily and weekly exposure frequency values drawn from appropriate distributions [11] to generate an estimate of total dose (AYD) via a given exposure route averaged over one year [12].

 For noncarcinogens, the average yearly dose (AYD) is divided by the noncarcinogen averaging time  $(AT_n)$  to yield the noncarcinogen average daily dose (NADD) [13]. Dividing NADD by a reference dose [14] value yields an estimate of the hazard quotient (HQ) [15] for a given receptor-contaminant-exposure route combination. This estimate of the hazard quotient is accumulated, along with all other estimates of individual hazard quotient values, to build a distribution [16] of non-cancer 'risk' values.

 For carcinogens, AYD is multiplied by the exposure duration divided by the carcinogen averaging time  $(AT_c)$  to yield the carcinogen average daily dose  $(CADD)$  [17]. Multiplying CADD by a cancer slope factor [18] gives an estimate of the incremental lifetime cancer risk (ILCR) [19] for a given receptor-contaminant-exposure route combination. This estimate of the ILCR is accumulated, along with all other estimates of ILCR values, to build a distribution [20] of cancer risk values.

 Following adding a value to the hazard quotient and ILCR distributions, the model checks [21] whether the required number of iterations (samples) has been achieved (10,000 for this example); if not, the process repeats, beginning with random selection of another individual of age *x*; otherwise, modeling stops [22]. The ILCR and HQ distributions represent the range of risks to a sample of 10,000 randomly selected individuals from a population of indeterminate size. The characteristics of this population are defined by the various distributions and point estimates.

## **4.2.2. Exposure and Risk Model Detail**

This particular model was constructed using Microsoft® Excel 5.0 for spreadsheet functions, Microsoft® Visual Basic for custom function generation, and Crystal Ball® (version 4.0c, 32 bit architecture), with Latin Hypercube sampling, for Monte Carlo processing and data accumulation. The notation "=CB." indicates an exposure factor distribution; exposure factors not so marked are single point values.

## **4.2.2.1. Age and Gender Exposure Factors**

 The current age of an individual receptor is selected at random from a distribution of ages. This distribution may be either site-specific, as determined by surveys of surrounding populations, or taken from the published literature. For example, 1990 U.S. Census data indicate that, in general, 10% of the U.S. population is comprised of individuals between the ages of 0 and 6 years, 20% between 6 and 18 years, and 70% older than 18 years. For this example, the distribution of ages within the population was described by a custom distribution that is then rounded-down to a whole integer, to give the value of As, the year at which exposure is assumed to begin, as follows:

$$
A_s \sim \text{ROUNDDOWN}(\text{CB}.\text{Customer}[\text{DC1}, \text{DC2}, \text{PC}]\text{0})
$$

**Equation 4. 16** 

Where:



The value for  $A_s$  resulting from the distribution is rounded-down to a whole integer with the Excel® ROUNDDOWN function. Exposure is assessed from  $(A<sub>s</sub>)$  forward; how far forward is determined by the exposure duration (ED). The distribution chosen to express ED depends on the scenario being evaluated. For non-occupational exposures, projected future residence time is a function of age. An age sensitive case function ("exp\_dur") is used to select the appropriate value for ED, as follows:

$$
ED \sim ROUNDUP\big( ED(A_s), 0\big)
$$

**Equation 4. 17** 

Where:

 $ED = Distribution of exposure durations (yr)$  $A_s$  = Distribution of year at which exposure starts (yr)  $ED(A<sub>s</sub>)$  = Age-sensitive exposure duration selection function (shown below) Function  $ED(A_s)$ Select Case  $(A_s)$ Case Is  $<$  3  $ED = Workshop(1).Cells(5, 5)$  Case 3 To 11  $ED = Work sheets(1).Cells(5, 6)$  Case 12 To 20  $ED = Work sheets(1).Cells(5, 7)$  Case 21 To 30  $ED = Work sheets(1).Cells(5, 8)$  Case 31 To 60  $ED = Work sheets(1).Cells(5, 9)$ Case Is  $> 60$  $ED = Work sheets(1).Cells(5, 10)$  End Select End Function

 Six age-specific distributions for ED are stored in row 5, columns 5 - 10 of this worksheet. The value for ED resulting from the distribution is rounded-up to a whole integer with the Excel® ROUNDUP function. This value, in combination with  $A_s$ , is used to estimate  $A_e$ , the year at which exposure is assumed to end. Rounding-down As and rounding-up ED gives the largest possible estimate of the difference between  $A_s$  and  $A_e$ , i.e., the number of years over which exposure occurs. A logical IF statement is used to prevent *t* from assuming values greater than 79 years, so that:

$$
A_e \sim IF\big( ED + A_s > 79,79, ED + A_s\big)
$$

**Equation 4. 18** 

Where:



 With this condition in place, it would be possible for the value of ED to exceed the number of years between  $A_s$  and  $A_e$ . For example, if  $A_s$  were 69 years and ED 20 years,  $A_e$  would be limited to 79 years by Equation 4.13. Here the actual number of years over which exposure is averaged is 11 years (79 - 69 + 1; adding one makes the time span inclusive), not 20 years as indicated by ED. Thus the exposure interval (EI) value used in place of ED in all subsequent calculations is determined by an additional conditional IF statement, as follows:

$$
EI \sim IF(A_e - A_s < ED, A_e - A_s + 1, ED + 1)
$$

**Equation 4. 19** 

Where:



 The determination of gender (*g*) is based on a gender ratio distribution that may be either site-specific, as determined by surveys of surrounding populations, or taken from the published literature. For this example, the distribution of gender ratios within the hypothetical population is assumed to be 45% male and 55% female. These ratios can be described by a custom distribution as follows; when  $g = 0$ , the individual receptor is classed as a "male"; when  $g = 1$ as a "female":

$$
g \sim CB
$$
.*Customer*(*DC*, *PC*)

**Equation 4. 20** 

Where:



column (0, 1, unitless)

### **4.2.2.2. Age- and Gender-Dependent Exposure Factors**

 Age- and gender-specific distributions are required for body weight (BW), while only agespecific distributions are required for soil ingestion rate (IRS). A total of 160 BW distributions are defined for male and female receptors ranging in age from < 1 to 79 years (see Section 3.5.1 for details). A logical IF statement is then implemented to select the value of BW on the basis of gender, so that:

$$
BW_{_{kg}} = IF\left(g=0, BW_{_{k-male}}, BW_{_{k-female}}\right)
$$

**Equation 4. 21** 

Where:

 $BW_{kg}$  = Distribution of body weights by age and gender (kg)  $g =$  Gender identifier (unitless)  $BW_{k-male}$  = Distribution of male body weights by age (kg)  $BW_{k\text{-female}} = \text{Distribution of female body weights by age (kg)}$ 

 Beyond 17 years, only one BW distribution is used to represent each age class, i.e., the distribution for the 18-24 year class is implemented in the 18 year column, then each succeeding column (year) from 19 to 24 is set equal to the 18 year column. For IRS, one distribution represents receptors of both genders from ages 0 to 6 years and another distribution represents receptors greater than 6 years of age (see Section 3.1.1 for details). Per Oregon rules, contaminant concentrations may be represented with a distribution; however, in this example, the value for the contaminant concentration in soil  $(C_s)$  is fixed at its 90<sup>th</sup> percentile UCL value of 3.78 mg/kg.

## **4.2.2.3. Correlations**

 Body weight generally increases with age for the first 18 years of life, then remains approximately the same for the remaining years of life. If the body weight distribution in one age class were independent of that in the next higher age class, unnaturally large changes in body weight might occur yearly for a given individual receptor. To minimize this phenomenon, distributions between age classes are positively correlated  $(r^2 \approx 1)$  using the Crystal Ball® correlation feature.

## **4.2.2.4. Exposure Frequency**

For this example, it was assumed that exposure occurs while engaging in activities outdoors





 In this example for simplicity, exposure frequencies are entered as point values. Representativeness of the model could be increased by assigning age/gender-dependent distributions to these parameters, provided that steps are taken to ensure that the sum of the time spent within multiple activities (home, at work, and elsewhere (commuting, shopping, etc.) does not exceed 24 hr/d or 365 d/yr for any individual.

Equation 2.1 (from Section 2) is used to estimate the average daily dose  $(ADD<sub>1</sub>)$  received in any given year *k* through incidental ingestion of contaminated soil. As shown in Equation 2.35, these ADD values are then multiplied by  $EF_{hd}$  (hr/d) and  $EF_{dy}$  (d/yr) exposure frequencies, as appropriate, to give an estimate of average or absorbed yearly dose  $(AYD_1)$ .

Equation 2.35 is evaluated with a custom Visual Basic function ("ayd1") that sums  $ADD_1$ values over the interval from  $A_s$  to  $A_e$ . This function adds together all values for ADD<sub>1</sub> (stored in row 27 of this spreadsheet) for each year *k* (in columns) over the exposure interval between As and Ae, as follows:

```
Function ayd1(A_s, A_e)For counter = A_s To A_eage\_dose = Work sheets(1).Cells(27, counter + 5)ayd1 = ayd1 + age\_dose Next counter 
End Function
```
The results from this function are multiplied by  $EF_{hd}$  and  $EF_{dy}$  to obtain a value for  $AYD_1$ . For soil ingestion, it was assumed that soil could be incidentally ingested at any time while in residence, so that  $EF_{hd} = 12$  hr/d and  $EF_{dy} = 350$  d/yr.

Equation 2.15 (from Section 2) is used to estimate the absorbed daily dose  $(ADD_6)$  received in any given year *k* through dermal contact with contaminated soil. Here again, Equation 2.35 is evaluated with a Visual Basic function ("ayd6") that adds together all values for  $ADD_6$  (stored in row 44 of this spreadsheet) for each year *k* (in columns) over the exposure interval between As and Ae, as follows:

```
Function ayd6(A_s, A_e)For counter = A_s To A_eage\_dose = Work sheets(1).Cells(44, counter + 5)ayd6 = ayd6 + age\_dose Next counter 
End Function
```
 For dermal contact with soil, it was assumed that could only occur when an individual was outside at their residence, so that  $EF_{hd} = 2$  hr/d and  $EF_{dy} = 350$  d/yr.

Equation 2.23 (from Section 2) is used to estimate the average daily dose  $(ADD_8)$  received in any given year *k* through inhalation of vapors released from contaminated soil. Here again, Equation 2.35 is evaluated with a Visual Basic function ("ayd8") that adds together all values for ADD7 (stored in row 70 of this spreadsheet) for each year *k* (in columns) over the exposure interval between  $A_s$  and  $A_e$ , as follows:

```
Function ayd8(A_s, A_e)For counter = A_s To A_eage\_dose = Work sheets(1).Cells(70, counter + 5)ayd8 = ayd8 + age\_dose Next counter 
End Function
```
 For inhalation of soil vapors, it was assumed that could only occur when an individual was outside at their residence, so that  $EF_{hd} = 2$  hr/d and  $EF_{dy} = 350$  d/yr.

## **4.2.2.5. Dose Estimates**

The noncarcinogen average daily dose (NADD<sub>i</sub>) is computed using Equation 2.36. The carcinogen average daily dose (CADD<sub>i</sub>), over the exposure interval defined by ED and averaging time  $AT_c$ , is computed using Equation 2.37. For carcinogens, averaging time  $(AT_c)$  is equal to an average lifetime, where 75 years is the default value. There are, however, gender differences in average lifetime, with men and women having average lifetimes of 72.1 years and 78.9 years, respectively (EPA 1996b). A logical IF statement is implemented to set the value of  $AT_c$  on the basis of gender (*g*), as shown below.

$$
AT_c = IF\big(g = 0.72.1,78.9\big)
$$

#### **Equation 4. 22**

### **4.2.2.6. Risk Estimates**

As shown in Equation 2.40, the dose estimates for noncarcinogens  $(NADD_1, NADD_6,$  $NADD_7$ ) are then divided by an appropriate oral reference dose  $(RfD_0)$  to calculate a hazard quotient for the soil ingestion and dermal contact exposure routes. Using Equation 2.42, the dose estimates for carcinogens (CADD<sub>1</sub>, CADD<sub>6</sub>, CADD<sub>7</sub>) are multiplied by the contaminantspecific cancer slope factor to give incremental lifetime cancer risk (ILCR) estimates for the soil ingestion and dermal contact exposure routes. The dose and risk calculations are repeated 2,500 times, with HQ and ILCR values being accumulated in a forecast, which, upon completion of the model run, represents a distribution of HQ and ILCR estimates for the population.

 Sensitivity analyses were performed for the soil ingestion, dermal contact, and inhalation ILCR and HQ estimates. Results, expressed as an input parameter's percentage contribution to variance in either the HQ or ILCR, are as follows:



 For the soil ingestion, variation in body weight is the largest total contributor to variation in HQ and ILCR. In other words, the 160 body weight distributions contribute approximately 0.5% each to HQ and ILCR variation, for a total contribution of approximately 77%. For dermal contact, the dominant source of variation to both HQ and ILCR is the soil-to-skin adherence rate (AF). For the inhalation exposure route, the principle source of variation to HQ is the daily breathing rate (IRA), while for the inhalation ILCR it is body weight, followed closely by IRA and one of the exposure duration distributions.

## **4.2.3. Results**

 Statistics and percentiles describing the resulting distributions are shown in Tables 4-2 and 4- 3, respectively. Distributions are displayed graphically (with deterministic results as an overlay) in Figures 4-2, 4-3, 4-4, 4-5, 4-6, and 4-7. As shown in the table below, the soil ingestion exposure route (Figure 4-2) 90<sup>th</sup> percentile ILCR value exceeds  $1 \times 10^{-6}$  and the 95<sup>th</sup> percentile ILCR exceeds  $1 \times 10^{-5}$ , causing this route to fail both acceptable risk criteria. The dermal contact exposure route (Figure 4-3)  $90^{th}$  percentile ILCR value exceeds  $1 \times 10^{-6}$  and its  $95^{th}$ percentile value exceeds  $1 \times 10^{-5}$ , indicating an unacceptable level of risk for both criteria. The inhalation exposure route (Figure 4-4) 90<sup>th</sup> percentile ILCR value is less than  $1 \times 10^{-6}$  and its 95<sup>th</sup> percentile value is less than  $1 \times 10^{-5}$ , indicating an acceptable level of risk using both criteria.

 The ingestion, dermal contact, and inhalation hazard quotients (Figures 4-5, 4-6, and 4-7) are all less than 1 at the  $90<sup>th</sup>$  percentile and less than 10 at the  $95<sup>th</sup>$  percentile, indicating an acceptable level of risk for both routes under both criteria. The "maximally exposed individual" (i.e., the randomly selected individual out of 2,500 sampled receiving the highest dose) has soil and dermal maximum ILCR values of  $> 10^{-6}$ , as well as soil and dermal maximum HQ values of 8.65 and 1.74, respectively; a further indication that the 3.78 mg/kg RME soil concentration would pose an unacceptable risk.


Thus, for this exposed population, a RME soil concentration of 3.78 mg/kg would pose unacceptable cancer risks via soil ingestion and dermal contact.

# **4.3. Model Comparisons**

 For soil ingestion, the deterministic CTE and RME ILCR estimates (Figure 4-2) are at approximately the  $63<sup>rd</sup>$  and  $87<sup>th</sup>$  percentile of the distribution, respectively. For the soil ingestion HO estimate (Figure 4-5), the deterministic RME is at approximately the  $50<sup>th</sup>$  percentile of the distribution, while the CTE estimate is at approximately the  $5<sup>th</sup>$  percentile.



 For the soil dermal contact exposures, the deterministic HQ (Figure 4-6) CTE and RME values are approximately the  $5<sup>th</sup>$  and  $50<sup>th</sup>$  percentiles of the distribution, respectively. The RME ILCR point value (Figure 4-3) is at the  $75<sup>th</sup>$  percentile, while the CTE ILCR estimate is at the  $50<sup>th</sup>$  percentile of the distribution. For inhalation exposures, the deterministic HQ (Figure 4-7) CTE and RME values are approximately the  $25<sup>th</sup>$  and  $95<sup>th</sup>$  percentiles of the distribution, respectively. The RME ILCR point value (Figure 4-4) is at the  $70<sup>th</sup>$  percentile, while the CTE ILCR estimate is at the  $40<sup>th</sup>$  percentile of the distribution.

 In comparison to the deterministic method, the probabilistic approach produces lower HQ estimates of RME risk (HQ 0.08 versus 0.51) but the same RME ILCR estimate  $(1 \times 10^{-5})$  for the soil ingestion exposure route. For the dermal contact route, the probabilistic method produces a higher estimate of RME risk (HQ 0.04 versus 0.44; ILCR  $5 \times 10^{-6}$  versus  $1 \times 10^{-5}$ ) than does the deterministic. For the inhalation route, the probabilistic method produces basically the same estimate of RME risk (HQ 9  $\times$  10<sup>-6</sup> versus 8  $\times$  10<sup>-6</sup>; ILCR 1  $\times$  10<sup>-10</sup> versus 2  $\times$  10<sup>-10</sup>) than does the deterministic.

 These results suggest that correspondence of deterministic and probabilistic results will be strongly influenced by exposure route and associated key exposure parameters. However, deterministic and probabilistic results are not orders of magnitude apart. Thus, if the regulatory goal is in the  $90<sup>th</sup>$  to  $95<sup>th</sup>$  percentile range with consideration of reasonableness, as it is in Oregon, both methods appear to produce similarly protective results.

# **4.4 References**

- EPA (1996a) **Region 9 Preliminary Remediation Goals (PRGs) 1996**. Region IX, U.S. Environmental Protection Agency. (August 1, 1996)
- EPA (1996b) **Exposure Factors Handbook**. Office of Research and Development, U.S. Environmental Protection Agency. (Update to EPA/600/P-96/002Babc, August 1996, External Review Draft)
- Gilbert RO (1987) **Statistical Methods for Environmental Pollution Monitoring**. Van Nostrand Reinhold, New York.. pp.169-171







# **TABLE 4 - 2**

# **EXAMPLE SIMULATION - STATISTICS OF THE DISTRIBUTIONS**



# **TABLE 4 - 3**

## **EXAMPLE SIMULATION - PERCENTILES OF THE DISTRIBUTIONS**



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# **FIGURE 4 - 1**

# **PROBABILISTIC EXPOSURE AND RISK ESTIMATION MODEL FLOWCHART**



# **FIGURE 4 - 2**

## **EXAMPLE SIMULATION - ILCR FROM SOIL INGESTION**



# **FIGURE 4 - 3 EXAMPLE SIMULATION - ILCR FROM SOIL DERMAL CONTACT**  1.00.9dermal ILCR0.8CTERME0.7 0.6 Probability **Probability** 0.5 0.4 0.3 0.2 0.1 0.0 1E-09 1E-08 1E-07 1E-06 1E-05 1E-04 1E-03 **Risk**

# **FIGURE 4 - 4**

# **EXAMPLE SIMULATION - ILCR FROM INHALATION**





# **FIGURE 4 - 6 EXAMPLE SIMULATION - HQ FROM SOIL DERMAL CONTACT**  1.00.9dermal HQ0.8**CTE** RME0.7 0.6 Probability **Probability** 0.5 0.4 0.3 0.2 0.1 0.0 1.E-03 1.E-02 1.E-01 1.E+00 1.E+01 **HQ**

## **FIGURE 4 - 7**

# **EXAMPLE SIMULATION - HQ FROM INHALATION**

