Human Health

Risk Assessment Guidance

Oregon Department of Environmental Quality
Environmental Cleanup Program
811 SW Sixth Avenue
Portland, OR 97204

October 2010

10-LQ-023
10/18/2010

I. ACKNOWLEDGEMENTS

The primary author of DEQ's December 1998 human health risk assessment guidance was Bruce Hope. This document was updated by the following DEQ Environmental Cleanup Program staff:

Jim Anderson, Northwest Region
Ann Levine, Land Quality Division
Jennifer Peterson, Northwest Region
Mike Poulsen, Northwest Region
Paul Seidel, Northwest Region
Susan Turnblom, Western Region

Additional review was provided by DEQ cleanup program managers and staff. Members of the public were invited to review and comment on the May 12, 2010, draft of this document. Their suggestions were considered and incorporated where appropriate into this guidance document.

II. DISCLAIMER

This document provides information and technical assistance to the public and employees of the Department of Environmental Quality regarding the Department's cleanup program. This information should be interpreted and used in a manner that is fully consistent with the state's environmental cleanup laws and implementing rules. This document does not constitute rulemaking by the Environmental Quality Commission, and may not be relied upon to create a right or benefit, substantive or procedural, enforceable at law or in equity, by any person including the Department. The Department may take action at variance with this guidance.

III. APPROVAL

This guidance document has been approved for use by the Department of Environmental Quality Land Quality Division.

[Wendy Wiles]	[date]
Wendy Wiles, Division Administrator	Date

TABLE OF CONTENTS

TABLES

FIGURES

APPENDICES

LIST OF ABBREBVIATIONS

ABS	Absorption
ADC	Average Daily Concentration
ADD	Average Daily Dose
AF	Adherence Factor
AT	Averaging Time
B	Relative contribution of permeability coefficients
BW	Body Weight
C	Chemical Concentration
CF	Conversion factor
COC	Chemicals of Concern
COI	Chemicals of Interest
COPC	Chemicals of Potential Concern
CTE	Central Tendency Exposure
DA	Dose Absorbed
DAF	Dermal Absorption Factor
DCE	Dichloroethene (Dichloroethylene)
DEQ	Department of Environmental Quality
DQO	Data Quality Objective
ELCR	Excess Lifetime Cancer Risk
ED	Exposure Duration
EF	Exposure Frequency
EPA	US Environmental Protection Agency
EPC	Exposure Point Concentration
ET	Exposure Time
F	Fraction of media contaminated
GI	Gastrointestinal
HEAST	Health Effects Assessment Summary Tables
HI	Hazard Index
HQ	Hazard Quotient
INF	Inhalation Factor
IRA	Inhalation Rate
IRAF	Infant Risk Adjustment Factor
IRF	Ingestion Rate Fish
IRIS	Integrated Risk Information System
IRM	Ingestion Rate Total Meat, Dairy Product, Egg ingestion
IRQ	Ingestion Rate Food
IRS	Ingestion Rate Soil
IRV	Ingestion Rate Total Vegetable and Fruit
IRW	Ingestion Rate Water
IUR	Inhalation Unit Risk
K_p	Permeability Coefficient
K_{ow}	*n*-Octanol-Water Partition Coefficient
MDC	Maximum Detected Concentration
MW	Molecular Weight
OAR	Oregon Administrative Rule
ORS	Oregon Revised Statute
PAH	Polycyclic Aromatic Hydrocarbon
PBDE	Polybrominated Diphenyl Ether

PCB	Polychlorinated Biphenyl
PCE	Tetrachloroethene (Perchloroethylene, PERC)
PEF	Particulate Emission Factor
PPRTV	Provisional Peer-Reviewed Toxicity Value
RBCs	Risk-Based Concentrations
RBDM	Risk-Based Decision Making
RfC	Reference Concentration
RfD	Reference Dose
RME	Reasonable Maximum Exposure
SA	Skin Surface Area
SAS	Water Dermal Contact Factor
SF	Carcinogenic Slope Factor
SFS	Soil Dermal Contact Factor
TCE	Trichloroethene (Trichloroethylene)
TEF	Toxicity Equivalency Factor
UCL	Upper Confidence Limit
WDOE	Washington Department of Ecology

1. INTRODUCTION

1.1. PURPOSE AND ORGANIZATION

This document describes methods that may be used to perform human health risk assessments at cleanup sites in Oregon. These methods are based primarily on U.S. Environmental Protection Agency (EPA) guidance, and are consistent with Oregon Revised Statute (ORS 465.315(2)(a)). In general, the exposure factors and equations described in this document are sufficient for calculating exposure, risks, or risk-based concentrations for typical remedies at most cleanup sites. The goal of this guidance document is to expedite completion of a prospective, site-specific risk assessment that evaluates human health risks that might exist if no action is taken.

The required elements of a baseline human health risk assessment are described in Oregon Administrative Rule (OAR) 340-122-0084. Risk assessments should include both reasonable maximum estimates (RME) and central tendency estimates (CTE) of exposure and risk as specified in ORS/OAR. The definitions of acceptable risk level are provided in OAR 340-122-0115(2), (3), and (4). A baseline risk assessment may use either deterministic or probabilistic methods, and can be based on DEQ's published Risk-Based Concentrations (RBCs) (DEQ 2003). This guidance document is limited to the elements in deterministic risk assessments. DEQ has a separate draft guidance document for conducting probabilistic risk assessments (DEQ 1998d).

This section provides an overview of the risk assessment process. Section 2 discusses problem formulation, including development of a conceptual site model and screening procedures. We discuss the main risk assessment elements in Section 3. The major tables and figures are provided at the end of the main text. We present additional details on the following topics in the appendices: exposure assessment equations (Appendix A), incorporating early-life exposure (Appendix B), incorporating inhalation exposure (Appendix C), and evaluating potential risks to infants from consuming human milk (Appendix D).

1.2. PROCESS OVERVIEW

The overall deterministic human health risk assessment process involves the general steps shown schematically on Figure 1 at the end of the main text. Information on existing (and historical) site conditions, land and water uses, and the nature and extent of contamination are key prerequisites for a baseline risk assessment. Develop data quality objectives for the risk assessment prior to data collection to guide the nature and extent of contamination investigations, and also the collection of useful data for the risk assessment.

Land and water use information, along with that on existing and historical site conditions, helps identify potentially exposed human receptors, including any sensitive groups. For example, pregnant women and infants would be a sensitive group for mercury exposure. Information on the nature and extent of contamination within the locality of the facility (as defined by OAR 340-122-0115(35)) and potentially exposed populations allows for the identification of site-specific exposure scenarios and exposure routes. Combine information on contaminants, receptors, and exposure pathways to develop a conceptual site model that summarizes relevant site information and sets the stage for the baseline risk assessment. Use site data to develop exposure point concentrations (EPCs) which may subsequently be used for screening procedures to identify contaminants of potential concern (COPCs). The results of a screening evaluation will determine whether the site may present an unacceptable risk or not. If a site passes the screening step, DEQ will conclude that it does not present an unacceptable risk, and further action is not required

(although there are rare exceptions that we will not elaborate on in this guidance). If a site fails the screening step, either perform a DEQ-approved removal or remedial action, or complete a baseline risk assessment evaluating site-specific conditions to determine if the risk is unacceptable. The baseline risk assessment can be conducted using the risk-based decision making (RBDM) approach (DEQ 2003) or follow the traditional method (EPA 1989). DEQ recommends using the RBDM approach, if appropriate, given the relative simplicity of the method and DEQ's confidence in the appropriateness of the default values and equations; as long as conditions at the site are consistent with the assumptions used to develop the RBCs or site-specific RBCs are calculated using the methodology described in DEQ 2003.

With a traditional baseline assessment, it is your choice to conduct a deterministic or probabilistic risk assessment (PRA). Please consult with DEQ before initiating a PRA. DEQ recommends that any formal probabilistic risk assessment be preceded by some initial site and risk screening to determine if it is worthwhile to invest resources in a PRA.

A baseline risk assessment is described in OAR 340-122-0084(2) and includes:

- an exposure analysis, which involves calculating exposure point concentrations, selecting exposure model equations, and selecting exposure factor values
- a toxicity analysis evaluating the inherent toxicity of chemicals
- a risk characterization combining the results of the exposure and toxicity analyses to evaluate risk
- a quantitative and/or qualitative uncertainty analysis covering all aspects of the risk assessment.

To receive a quicker review of the risk assessment report, document the risk assessment results in a clear and consistent manner. To further expedite review of the risk assessment, provide DEQ with electronic copies of spreadsheets of data and calculations with working (unlocked) formulas as part of the documentation. If acceptable risk levels have been exceeded at the site, or if beneficial uses of water have been impaired, you will need to evaluate the presence of potential hot spots. Final determination of hot spots (DEQ 1998c) is made during the feasibility study.

2. PROBLEM FORMULATION

2.1. PROBLEM FORMULATION ELEMENTS

The problem formulation step follows the initial planning stage, and ends with the development of a conceptual site model describing the chemical sources, exposure pathways, and human receptors. In the problem formulation stage, use existing and historical site information to identify site-specific chemicals of interest (COIs). In this section, we discuss other problem formulation elements such as establishing data quality objectives, screening of COIs, determining land and water use, determining the nature and extent of contamination, determining the locality of the facility (the area where humans are reasonably likely to contact chemicals from the site), identifying potentially exposed populations, and developing a conceptual site model.

2.2. NATURE AND EXTENT OF CONTAMINATION DETERMINATION

Prior to initiating a risk assessment, you must determine the full nature and extent of contamination. This includes looking at the types of contaminants, their horizontal and vertical distribution, and their potential for movement. Information obtained during this task, along with that obtained from the previous task of identifying receptors, allows you to determine the locality of the facility (defined by OAR 340-122-0115(35); also see EPA 1989, Section 4, for further information). Develop data quality objectives (DQOs) for determining the nature and extent of contamination that allows the use of data throughout the investigation and risk assessment.

2.3. LAND AND WATER USE DETERMINATIONS

In Oregon, land and water use determinations are one of the key prerequisites for a baseline risk assessment regardless of the method used to complete the risk assessment. The fundamental premise is that risk at a given site is a function of the receptors and exposure routes present, which, in turn, are determined by the current and reasonably likely land and water uses in the locality of the facility. The land and water uses for human receptors at a site are protected by showing that exposure scenarios specific to each use do not produce unacceptable risks (DEQ 1998a and 1998b). A specific combination of receptors, exposure routes, and land and water uses can be described as an exposure scenario. Once you have determined potential risks for the set of land and water use designations appropriate to the site, such designations cannot be changed unless risks are reassessed in some manner. The degree of effort required to reassess the risks and any subsequent remedy will necessarily be site-specific and thus could span a broad range. The key point is that if a site's land and water uses are changed without a reassessment of risk appropriate for that site, the risk assessment and any remedy based on that assessment may no longer be protective.

2.4. DATA QUALITY OBJECTIVES FOR RISK ASSESSMENT

Before you conduct sampling to provide data for a baseline risk assessment, you need to determine the data quality objectives for the risk assessment. DEQ recommends that you develop DQOs for evaluating the nature and extent of contamination and the risk assessment before collecting any samples, to minimize data collection costs. During development of all DQOs, whether for determining the nature and extent of contamination or for performing the risk assessment, DEQ strongly recommends the use of US EPA's 7-step DQO development process (US EPA 2000c). A full discussion of the DQO process is beyond the scope of this document, but is covered in detail in US EPA 2000c. The DQO process is not limited to analytical quality assurance and reporting limits, but must include systematic project planning to

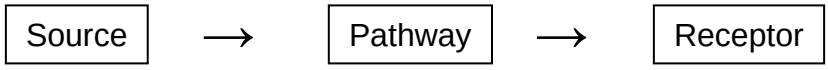
ensure that data collected will meet project objectives (US EPA 2006a). Without systematic planning, your risk analysis may be ambiguous or inconclusive, possibly leading to additional sampling, increased cost and project delays.

In addition to ensuring that collected data are matched to overall project objectives, DQOs will ensure that all environmental data collected in support of the risk assessment are of known and documented quality, have adequate detection limits, have been collected at locations and in media that are relevant to the quantitative risk assessment process, and are appropriate for any planned statistical methods. You will need to plan the number and type of samples, and determine the correct number of samples for statistical evaluations. For naturally occurring chemicals such as arsenic, it will be important to develop a plan for assessing background concentrations. For anthropogenic chemicals that are widespread in the environment, it is also important to determine the ambient concentrations unrelated to the release at the site.

It may be helpful at this point to prepare a preliminary conceptual site model to guide the sampling design process (see EPA 1989, Section 4.1.4 and EPA 1992c for further details). You should document the DQO process in your Sampling and Analysis Plan (SAP) and Quality Assurance Project Plan (QAPP). DEQ requirements for QAPPs are consistent with those of US EPA (see EPA 2001a).

2.5. CONCEPTUAL SITE MODEL

Before you proceed to the risk assessment, it is important to have a clear conceptual understanding of the various chemical sources, exposure pathways, routes of exposure, and types of receptors at your site. The key elements are:



A good way of presenting a conceptual site model is in a chart. An example conceptual site model is shown on Figure 2. The example conceptual site model covers exposure pathways and receptors for which default RBCs have been developed using RBDM guidance.

2.5.1. EXPOSURE SCENARIOS

Estimation of exposure involves the identification of exposure pathways, scenarios, and routes. An exposure pathway is the course a chemical or physical agent takes from a source to an exposed organism (EPA 1989). Exposure scenarios (designated “residential”, “industrial”, etc.) are comprised of one or more exposure routes appropriate to the potentially exposed population. 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.).

EPA (1991a, 1997) has defined four default exposure scenarios (residential, commercial/industrial, agricultural, recreational), and associated exposure factors, for use in human health risk assessments. DEQ has added additional exposure scenarios that are commonly found at sites in Oregon: limited duration excavation worker, longer-term construction worker, and urban resident. By using default scenarios, you should be able to limit the time and cost involved in preparing a site-specific risk assessment. Note that Oregon cleanup rules allow you to develop supportable site-specific scenarios in place of these default scenarios. Such site-specific scenarios allow consideration of unique site conditions, thus providing a more representative estimate of site-related risk.

2.5.2. EXPOSURE ROUTES

Consider the following list of primary exposure routes when conducting a typical human health risk assessment:

- SOIL
  - Incidental ingestion of contaminated soil or sediment
  - Dermal contact with contaminated soil or sediment
  - Inhalation of particulates (fugitive dust)
  - Inhalation of soil vapors (indoor and outdoor)
- WATER
  - Incidental ingestion of contaminated water
  - Dermal contact with contaminated water
  - Inhalation of vapors from the use of water
- AIR (indoor and outdoor)
  - Inhalation of vapors volatilized from soil
  - Inhalation of vapors volatilized from groundwater
- FOOD
  - Ingestion of fish
  - Ingestion of vegetables and fruits
  - Ingestion of animal products (meat, dairy products, eggs)
  - Infant Ingestion of maternal milk

You will not likely encounter all of the above exposure routes at every site. Conversely, unique site-specific conditions may require you to describe and quantify additional exposure routes.

2.5.3. POTENTIALLY EXPOSED POPULATIONS

You should discuss the characteristics (age, gender, etc.) of the human population in the locality of the facility. This is necessary to ensure that the selected exposure factors (Section 3.3) best represent the characteristics of the potentially exposed population. Generally, children and adults are the populations typically evaluated. However, you must ensure that specific sensitive groups, if any, are identified and included in the risk assessment as appropriate.

You should generally not perform a risk assessment that combines an overall population and a number of smaller, more sensitive groups, within the same model. If these groups can be initially identified, or are identified in the course of modeling an overall population, they should be modeled separately and not combined with other groups or the overall population. Doses and risks received by sensitive groups (e.g., subsistence fishers) can be specifically modeled by selecting exposure factors or toxicity values unique to these groups.

Infants are a sensitive group that should be included in all long-term exposure scenarios with exposure to bioaccumulating chemicals. This is because people will build up a body burden of chemicals that is passed from mothers to their infants through breastfeeding. Note that this pathway for bioaccumulation is based on exposure to female children and adults who will become mothers in the future; it is not based on identifying current breastfeeding of infants.

We simplified the incorporation of this pathway by providing a process for converting risk and hazard quotient calculations for typical exposure scenarios and receptors (*e.g*., adult occupational exposure, residential child and adult exposure) to risk and hazard quotient values

for breastfeeding infants (Section 3.4.2.3). For sites with occupational or residential exposure to PCBs, infants will be the receptors with the highest unacceptable risk. Other bioaccumulating chemicals considered are chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans, and DDT (and metabolites DDE and DDD).

2.5.4. CURRENT AND FUTURE EXPOSURE

It is important to consider both current and potential future exposure when developing exposure routes at your site. DEQ will acknowledge if current exposure is not actually occurring, for example, if there is asphalt paving over contaminated soil. However, DEQ will base risk decisions on both current and potential future exposure. That means we will want to see an evaluation of risk from the contaminated soil assuming that the asphalt cover does not remain in place, or that subsurface soil is brought to the surface in the future, to determine if engineering or institutional controls are required to maintain protectiveness.

2.6. SCREENING PROCEDURES

Screening of chemicals is allowed by OAR 340-122-0080(5). Chemicals detected at the site which have not been screened should be designated as “Chemicals of Interest” (COIs), while those that have been screened-in should be designated as “Chemicals of Potential Concern” (COPCs). Following the baseline risk assessment, chemicals that do not meet acceptable risk levels should be designated as “Chemicals of Concern” (COCs). COIs are screened on the basis of frequency of detection, background concentrations, and chemical concentrations relative to risk-based screening levels, as described below, to determine whether COIs should be retained as COPCs to be carried forward in the risk assessment.

Use of Qualified Data DEQ generally follows EPA guidance on the use of qualified data in risk assessments. The most commonly encountered data qualifier is J, indicating an estimated value. J-qualified data should be used in the risk assessment the same as unqualified data. Similarly, estimated maximum possible concentration (EMPC) qualified data should also be used in risk assessments. You may discuss uncertainties associated with qualified data in the Uncertainty section.

Frequency of Detection COIs that are infrequently detected may be artifacts in the data due to sampling, analytical, or other errors. COIs detected in less than five percent of the samples site-wide for a given media may not need to be selected as COPCs if there is sufficient reason to believe that they are artifacts. This assumes that detection limits were low enough to evaluate both ecological and human health risks, and that adequate sampling has occurred. If there are infrequent detections, but concentrations could be of concern, DEQ may still require that chemicals released at the site be evaluated in the risk assessment.

Background Concentration If the maximum detected concentration (MDC) of a naturally occurring COI is not greater than the concentration selected as a background value (derived either from the appropriate literature or from site-specific sampling), it need not be selected as a COPC (EPA 1994). The concept of background applies only to naturally-occurring chemicals. DEQ recommends that you develop site-specific background concentrations. However, in some circumstances, it may be appropriate to use regional background values. Table 1 provides a summary of regional background concentrations for common naturally-occurring chemicals.

When selecting inorganic background levels for a specific site, the preference for a source of such values is, in order:

1) Values calculated from site-specific data, assuming the sampling was performed according to a DQO appropriate for determining background.[1]
2) Local default values (e.g., those for southwestern Oregon), and
3) The regional default values for the Pacific Northwest listed in Table 1.

The regional default values can be used (a) to make an initial assessment of a site (before site-specific data are available), (b) if local default values are unavailable, or (c) to check the credibility of site-specific values. Regional values are to be used at the discretion of the cleanup project manager, and should not be seen as constituting a background "standard" or "criterion".

In addition to a simple comparison of maximum concentrations with background values, DEQ will consider an evaluation of site data compared with a background dataset. This can be done using the hypothesis testing feature of EPA's ProUCL program or other commercially available software (U.S. EPA 2010a). EPA provides other methods in ProUCL for comparing site data with background data. You should discuss your plans with DEQ before attempting these more complex evaluations of background data. In particular, it is important that the site data match the required assumptions for the hypothesis test planned for use. Most environmental data do not meet assumptions of equal variances, sample independence and distributional form that are required for common hypothesis tests, and can often yield incorrect results. Therefore, use of nonparametric procedures or a means to ensure assumptions are met should be made in consultation with DEQ.

Essential Nutrients According to EPA, chemicals that are essential human nutrients, are present at low concentrations (slightly above naturally-occurring levels), and are toxic only at high doses (relative to site levels) may be screened out (EPA 1989). Examples of essential nutrients that may qualify are iron, magnesium, calcium, potassium, and sodium. You must justify screening out essential nutrients that are regulated as hazardous materials. Arsenic is considered a potential essential nutrient, but because of its toxicity, arsenic should not be screened out without a comparison of site concentrations with background levels and RBCs.

Concentration-Risk Screen Any screening must take into consideration the potential for risk posed by exposure to: (a) individual COIs, (b) multiple COIs simultaneously within a given medium, and (c) individual or multiple COIs within different media. An individual COI in any given medium must be retained as a COPC if the concentration exceeds the RBC:

$$\frac{C}{RBC} > 1$$

Where C is the concentration of the chemical in a given medium, either the maximum concentration; or when sufficient data adequately representing the sampled area is available, the 90 percent upper confidence limit on the arithmetic mean (90% UCL). For vapor intrusion pathways, screening is typically limited to individual sample locations because site-wide estimates of concentrations may not be representative of concentrations beneath buildings. The Risk-Based Concentration (RBC) is either the default RBC calculated by DEQ, or the value you calculated using default or site-specific exposure values (DEQ 2003).

Select the RBC for the exposure scenarios appropriate for your site as presented in the

[1] If determination of background is a project need, it should be considered in project planning as part of the DQO process, so that resulting data will be adequate to determine an appropriate background concentration.

conceptual site model (Section 2.5). In most cases, DEQ's pre-calculated RBCs are the preferred screening values. They represent DEQ's current decisions regarding exposure factors and toxicity values. Also, EPA regional screening values have not been developed to cover all exposure pathways that could be relevant at a site in Oregon. For example, there are no EPA screening levels for soil vapor, or for volatile chemical transport from subsurface soil or groundwater to indoor air. If these are relevant pathways at your site, you should use the appropriate RBC for screening. If DEQ RBCs are not available for the COI, you can use EPA regional screening levels (EPA 2010c).

DEQ's Risk-Based Decision Making (RBDM) guidance explains how RBCs are derived (DEQ 2003)[2]. The RBC tables have been expanded beyond petroleum hydrocarbons, and now include most chemicals likely to be present at a site in Oregon (DEQ 2010c). In addition to the default RBCs available in the table, you can also use the spreadsheet available on our web site to calculate site- or chemical-specific RBCs (http://www.deq.state.or.us/lq/rbdm.htm).

DEQ will update the RBC tables periodically. EPA updates their regional screening table every half year. Toxicity values are periodically updated, which will result in revised RBCs or EPA screening levels. In addition, some exposure factors may be revised as new information becomes available. Please make sure that you are using the most current DEQ RBCs or EPA screening levels by checking the appropriate web sites (links available at http://www.deq.state.or.us/lq/rbdm.htm).

For carcinogens, individual RBCs are based on an excess lifetime cancer risk of 10^{-6}. It is highly unlikely that chemicals would exceed the cumulative standard of 10^{-5} with all concentrations below their RBCs. Therefore, DEQ does not require a cumulative risk screen for carcinogens that are below RBCs.

For non-carcinogens, the RBCs are based on a hazard quotient of 1. A cumulative hazard index of 1 also applies. It is possible that a few chemicals that would be screened out with concentrations just below their RBCs could exceed a cumulative hazard index of 1. For this reason, an additional condition is used to screen non-carcinogens. A non-carcinogenic chemical is screened in as a COPC if:

$$\frac{C}{RBC} > 0.1 \quad \text{and} \quad SUM(\frac{C}{RBC}) > 1$$

The following examples show how the screening steps are applied. **Example 1A** shows a simple case where all chemicals pass the screen.

[2] The equations used to calculate RBCs as shown in DEQ 2003 have been updated for consistency with EPA's inhalation risk assessment methodology (EPA 2009). Accordingly, Appendix B of DEQ 2003 is now outdated and does not represent current methodology as described in EPA 2009, and as implemented in the most recent version of DEQs spreadsheets. The equations used by DEQ to calculate RBCs are presented in Appendix C of this document.

Example 1A

All individual C/RBC values for carcinogens (ca) are less than 1, and the cumulative value (SUM(C/RBC)) is less than 1 for non-carcinogens (nc). Because SUM(C/RBC) is less than 1, none of the COIs, including those non-carcinogens with C/RBC > 0.1, would be retained as COPCs.

			C / RBC				
COI	**C (mg/kg)**	**RBC (mg/kg)**	**ca**	**nc**	**>1? (ca)**	**>0.1? (nc)**	**COPC?**
Chemical A (ca)	20	60	0.33	-	No	-	No
Chemical B (nc)	0.5	3	-	0.17	-	Yes	No
Chemical C (nc)	1	15	-	0.07	-	No	No
Chemical D (ca)	1	20	0.05	-	No	-	No
Chemical E (nc)	0.1	1	-	0.10	-	No	No
		Sum(C/RBC)		0.34		**Sum nc < 1**	

Example 1B shows another simple example where the concentrations of some chemicals exceed screening values.

Example 1B

Two individual C/RBC values (C and D) are greater than 1. The cumulative value (SUM(C/RBC)) is greater than 1 for non-carcinogens. However, none of the chemicals other than C and D have C/RBC values greater than 0.1. The two COPCs are chemicals C and D.

			C / RBC				
COI	**C (mg/kg)**	**RBC (mg/kg)**	**ca**	**nc**	**>1? (ca)**	**>0.1? (nc)**	**COPC?**
Chemical A (ca)	1	60	0.02	-	No	-	No
Chemical B (nc)	0.1	3	-	0.03	-	No	No
Chemical C (nc)	20	15	**-**	**1.3**	**-**	**Yes**	**Yes**
Chemical D (ca)	40	20	**2.0**	**-**	**Yes**	**-**	**Yes**
Chemical E (nc)	0.05	1	-	0.05	-	No	No
		Sum(C/RBC)		**1.4**		**Sum nc > 1**	

Example 1C gives an example of retaining a COI as a COPC if:

$$\frac{C}{RBC} > 0.1 \quad \text{and} \quad SUM(\frac{C}{RBC}) > 1$$

Example 1C

Two individual C/RBC values are greater than 1, and the non-carcinogenic cumulative (SUM(C/RBC)) value is greater than 1. An additional two non-carcinogenic C/RBC values exceed 0.1. Therefore, four COIs are retained as COPCs, two (C & D) based on individual screen, and two (B & E) based on cumulative screen

			C / RBC				
COI	**C (mg/kg)**	**RBC (mg/kg)**	**ca**	**nc**	**>1? (ca)**	**>0.1? (nc)**	**COPC?**
Chemical A (ca)	1	60	0.02	-	No	-	No
Chemical B (nc)	1	3	-	0.33	-	**Yes**	**Yes**
Chemical C (nc)	20	15	**-**	**1.3**	**-**	**Yes**	**Yes**
Chemical D (ca)	40	20	**2.0**	**-**	**Yes**	**-**	**Yes**
Chemical E (nc)	0.5	1	-	0.50	-	**Yes**	**Yes**
		Sum(C/RBC)		**2.1**		**Sum nc > 1**	

Example 1D demonstrates an example of a COI detected in multiple media (e.g., in both surface water and soil). The COI must be retained as a COPC if:

$$SUM(\frac{C}{RBC}) > 1$$

The evaluation in multiple media applies to both carcinogens and non-carcinogens.

You should conduct screening for multiple media if it is reasonable for a receptor to contact more than one medium, as reflected in the conceptual site model. For example, a child may contact soil, sediment, and surface water at a park along a river. Or an outside worker may be exposed to surface soil and vapors from groundwater volatilization (but not vapor intrusion to indoor air).

Example 1D

COI in multiple media. On the left, aggregate score (SUM(C/RBC)) for Chemical A is less than 1, indicating that it should not be retained as a COPC due to its presence in multiple media. On the right, Chemical B should be retained because its aggregate score exceeds 1.

Chemical A	C/RBC	COPC	Chemical B	C/RBC	COPC
Soil	0.25		Soil	0.37	
Sediment	0.14		Sediment	0.21	
Surface Water	0.11		Surface Water	0.75	
SUM(C/RBC)	0.50	No	**SUM(C/RBC)**	**1.33**	**Yes**

Prior to screening, adjust concentrations of chlorinated dibenzo-*p*-dioxins, chlorinated dibenzofurans, polycyclic aromatic hydrocarbons (PAHs), and polychlorinated biphenyls (PCBs) by applying the appropriate toxicity equivalency factors (TEF) (see DEQ (1997b) and EPA (1993) for further information). If an RBC is not available and cannot be calculated for a given COI, that COI must be identified as a COPC and retained for further discussion as a potential data gap in the risk assessment. The COPC may be addressed qualitatively or semi-quantitatively in the baseline risk assessment and discussed in the uncertainty evaluation.

In some cases where increasing chemical concentrations are possible due to contaminant degradation, it is inappropriate to screen out chemicals even though they may pass the initial concentration risk screen. A common example is a site with a solvent such as tetrachloroethene (PCE) that under certain circumstances will degrade to other, and in some cases more toxic chemicals. At a site where PCE is a COPC, the degradation products trichloroethene (TCE), dichloroethene (DCE), and vinyl chloride should also be retained as COPCs. The monitoring program may establish that these chemicals are not of potential concern, but there is a danger that TCE or vinyl chloride concentrations may increase over time, possibly leading to unacceptable risk levels. Collect data over time to establish degradation trends for the parent compound and the degradation products.

Disparities between DEQ's RBCs and EPA Screening Levels

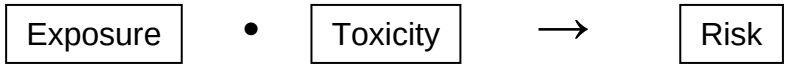
For most chemicals, DEQ's RBCs and EPA's screening levels should be very similar, given the similarity in exposure assumptions. However, in cases involving volatilization of carcinogens from soil, there may be a disparity in values. This is because DEQ includes a mass-limiting element in the volatilization equations, and EPA does not. EPA assumes that a volatile organic chemical can continually volatilize from surface soil for 25 or 30 years, and they calculate a screening level accordingly. In contrast, DEQ's RBC equations use reasonable assumptions regarding the mass of chemical present in soil. If, for example, the calculations show that the entire estimated mass of chemical in soil would volatilize in 3 years, the shorter exposure period is used to calculate the RBC based on long-term exposure. This will result in an order of magnitude difference between the DEQ RBC (based on 3 years of volatilization and 30 years of exposure) and the EPA screening level (based on 30 years of volatilization and 30 years of exposure).

3. BASELINE RISK ASSESSMENT

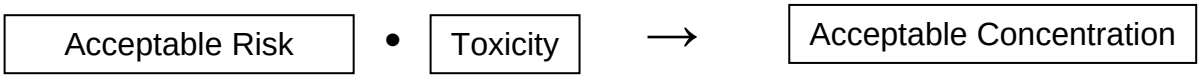
3.1. BASELINE RISK ASSESSMENT OPTIONS

When chemicals of potential concern (COPCs) are identified during the screening portion of the problem formulation stage, it does not necessarily mean that the chemicals at your site pose unacceptable risks. If concentrations at the site exceed RBCs, you may conduct a site-specific baseline risk assessment to quantify the potential risks posed by chemicals at your site, and determine if these risks are indeed unacceptable. In other cases, you may decide to remediate the site, or perform removal actions, without conducting further risk assessment. Prior to selecting a remedial action at a site, you will need to document in a residual risk assessment (Section 3.9) that any remaining chemicals at the site will result in acceptable risk levels.

There are two general approaches for a baseline risk assessment. The traditional approach follows EPA guidance for conducting an exposure assessment, toxicity assessment, and risk characterization. These elements are discussed below in Sections 3.2, 3.3, and 3.4. The basic concept is that risk is the combination of exposure and toxicity:



An alternative to the traditional approach is the Risk-Based Decision Making (RBDM) approach (DEQ 2003). Screening values, whether Risk-Based Concentrations (RBCs) or EPA screening values, are developed starting from an acceptable risk level, and then, using standard exposure and toxicity assumptions, calculating an acceptable concentration. This is essentially the inverse of the traditional approach.



Given the similarities between the approaches, the RBDM approach can be modified to produce a baseline risk assessment. The RBDM approach has many benefits in terms of time and effort to prepare a risk assessment; we therefore present this as the preferred method in this guidance document (Section 3.4.1). Guidance for the standard risk assessment approach is also presented (Section 3.4.2).

3.1.1. USE OF PROBABILISTIC METHODS

The information requirements of a probabilistic risk assessment are described in OAR 340-122-0084(5). Please consult with DEQ prior to starting if you wish to perform a probabilistic risk assessment. For further guidance, refer to DEQ (1998d). In addition, the generic remedy for PCBs at transformer sites (DEQ 1997a) illustrates the use of probabilistic methods to set screening levels and cleanup goals. However, the PCB generic remedy guidance does not include infant exposure (Appendix D). For this reason, DEQ is currently not applying the PCB generic remedy at sites.

3.2. EXPOSURE ASSESSMENT

In an exposure assessment, exposure factors and their associated numerical values (Appendix

A) are combined to quantify doses received by potentially exposed populations of receptors via all potentially complete exposure routes.

3.2.1. EXPOSURE POINT CONCENTRATIONS

The exposure point concentration is the concentration of a given chemical in a given medium at a location of potential contact with a specified receptor. This concentration term is typically the arithmetic mean of the concentration that is contacted over the exposure period. Oregon rule requires that risk assessments consider plausible upper-bound or high-end exposure [OAR 340-122-0084(1)(f)]. To represent reasonable maximum exposure (RME), Oregon rules specify using the 90 percent upper confidence limit on the arithmetic mean for environmental concentrations, unless a different estimate is acceptable to DEQ. For example, DEQ may accept the use of incremental sampling, involving the collection of large numbers of sub-samples composited into a small number of samples for laboratory analysis. In this case, the estimated mean value may be used without a UCL calculation. Alternatively, a 90 percent UCL may be calculated using the incremental sampling approach using field replicates as described in Alaska State soil sampling guidance (ADEC 2009). DEQ will typically require use of field replicates with this method. Because the use of incremental sampling in environmental applications is an emerging methodology at the time of this writing, consult DEQ before using it at a site in Oregon.

For common discrete sampling approaches, EPA guidance provides methods for calculating the 90 percent UCL term even if the data are not normally or log-normally distributed (EPA 2010b). The statistical methods available in EPA's ProUCL program also allow for incorporation of non-detect values in determining exposure point concentrations. We recommend using ProUCL for most statistical evaluations (http://www.epa.gov/nerlesd1/tsc/software.htm). EPA only provides recommendations for which estimation method to use for calculating 95 percent UCLs, not 90 percent UCLs. DEQ generally uses the method recommended by EPA for the 95 percent UCL, but calculated at the 90 percent level. In some cases, this may involve using a 95 percent UCL value to approximate a 90 percent UCL, because sometimes the Chebyshev inequality approach does not provide sufficient coverage.

Consistent with EPA recommendations, DEQ recommends a minimum of 8 to10 samples to calculate reliable UCL estimates. However, this will vary depenedent on site conditions. In cases where too few samples are available to adequately represent the site, DEQ may not accept risk assessment conclusions

DEQ has discretion in accepting other methods (area weighted-averaging, concentration distributions, bootstrapping, etc.) for computing the exposure point concentration. However, consult DEQ before employing any of these alternative statistical methods. The main sources for statistical methods for calculating exposure point concentrations are EPA (2002a) and EPA (2010a, b). When performing a vapor intrusion investigation, refer to DEQ's 2010 vapor intrusion guidance document (DEQ 2010). Due to the high variability in air and uncertainties in modeling from soil and groundwater, vapor intrusion investigations will be based on sub-slab or soil vapor samples as a primary line of evidence. Determination of exposure point concentrations in subsurface soil vapor is presented in Section 4 of DEQ 2010..

Oregon rules also require that central tendency exposure (CTE) be considered in the risk assessment. This includes using the arithmetic mean to represent environmental concentrations, and using mean estimates of exposure factors. Risks calculated using CTE values are rarely used in decision making, but they can provide perspective to decision makers,

which is particularly helpful in cases where it is infeasible to remediate to acceptable risk levels based on RME values.

Soil

For developing EPCs for soil, we generally use depth ranges of 0 to 3 feet for surface soil, and 0 to 15 feet for subsurface soil (for example, construction or excavation exposure). However, it will be important to understand where the highest chemical concentrations are present. For instance, if a spill results in high chemical concentrations in the top few inches, an EPC calculated using data over a range of 3 feet will underestimate the current risk from exposure to surface soil. You should therefore evaluate the distribution of chemicals in soil to decide on appropriate soil ranges for your site. If volatilization of chemicals from soil to indoor air is a relevant pathway, the EPC should be calculated using data representative of conditions at existing (or future) buildings.

Groundwater

A site-wide EPC for groundwater is generally not appropriate for most exposure scenarios. If drinking water use or industrial use of groundwater is occurring, the appropriate range of influence of the current or future extraction well should be considered. If volatilization of chemicals from groundwater to indoor air is a relevant pathway, the EPC should be calculated using data representative of conditions at existing (or future) buildings. In most cases, this will be data from one or two wells.

3.2.2. EXPOSURE ESTIMATION EQUATIONS

Appendix A of this guidance presents equations for all exposure routes likely to be encountered at a site. Additional information may be obtained from EPA (1989, Section 6) and EPA (2004).

3.2.3. EXPOSURE FACTOR VALUES

For convenience, default exposure factor values, acceptable to DEQ for use with default exposure scenarios, are listed in Table A-1 (RME) and Table A-2 (CTE) of Appendix A. Use of these default factors can lessen the time and effort involved in preparing a risk assessment. Oregon cleanup rules do, however, allow you to select those exposure factor values (obtained either from EPA guidance or from other sources) that most accurately reflect the exposed population. Selecting site-specific exposure factor values may provide a more representative estimate of site-related risk, but it is often difficult to document the appropriateness of site-specific values. Also, in most cases site-specific values will only apply to current conditions; you will likely need to use default values for potential future conditions.

Exposure factor values listed and described in EPA (1997) and other guidance documents should be given primary consideration for use in determining exposure to receptors. Note that substantial scientific evidence must be presented to support selection of site-specific exposure scenarios and factors which differ from EPA guidance or precedence. Using the expression “professional judgment” alone as justification for selection of a particular value, without any other supporting information or documentation, will generally not be acceptable. Exceptions include cases where EPA does not provide an exposure factor value and no scientific information to support determining a numerical value can reasonably be obtained. In these cases, a value may be determined in consultation with DEQ. DEQ (1997a) illustrates the type and extent of scenario-specific information required to establish alternative exposure factor values.

3.3. TOXICITY ANALYSIS

The purpose of the toxicity assessment is to compile toxicity data for the COPCs identified in the study area and to estimate the relationship between the amount of exposure or dose level of a COPC and the likelihood of adverse effects. You should also provide qualitative descriptions of the potential toxic properties of the COPCs.

3.3.1. SOURCES OF TOXICITY VALUES

The preferred sources of toxicity values are, in order:

1. EPA Integrated Risk Information System (IRIS) database (www.epa.gov/iris)
2. EPA Provisional Peer-Reviewed Toxicity Value (PPRTV) database
3. EPA Health Effects Assessment Summary Tables (HEAST)
4. EPA National Center for Environmental Assessment, Superfund Health Risk Technical Support Center
5. Other U.S. EPA documents or databases
6. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles
7. Other refereed technical publications

EPA's IRIS is the preferred source of toxicity information (reference doses, reference concentrations, cancer slope factors, or inhalation unit risks) (see also EPA 1989, Section 7). You can get a summary of standard toxicity values from EPA's regional screening table (EPA 2010c). EPA has updated their hierarchy to include provisional peer-reviewed toxicity values (PPRTVs), second only to IRIS values. PPRTVs can also be used as a source of toxicity values. If toxicity values are not available for a chemical, selection or derivation of alternative toxicity values will be required if the chemical is expected to contribute to unacceptable risks. This evaluation will be performed on a chemical-specific basis. One possible method for selection of alternative toxicity values is the use of reference doses and slope factors of structurally similar compounds as surrogates. For example, the following surrogates have been accepted by DEQ:

Chemical	**Surrogate**
Phenanthrene	Fluoranthene
4-Chlorophenol	2-Chlorophenol
Trans-Nonachlor	Chlordane

3.3.2. ASSESSMENT OF CARCINOGENS

EPA provides oral slope factors (SFs) to evaluate cancer risks, which are expressed as risk per milligram per kilogram body weight per day $(mg/kg/day)^{-1}$. For air, EPA provides inhalation unit risk (IUR) factors in units of risk per microgram per cubic meter $(\mu g/m^3)^{-1}$.

Early-Life Exposure

Carcinogens that act by a mutagenic mode of action can have greater toxicity during early-life stages (EPA 2005b). In these cases, you cannot use a single slope factor without modification. Currently, for sites in the DEQ Cleanup Program, the chemicals of primary interest for consideration of early-life exposure are vinyl chloride and the carcinogenic polycyclic aromatic hydrocarbons (cPAHs). If you have these chemicals at your site, and exposure to children is reasonably anticipated, use the procedures presented in Appendix B. DEQ RBCs for residential exposure to vinyl chloride and cPAHs incorporate early-life exposure. In the future as more

information becomes available, EPA may determine additional chemicals where it is necessary to include early-life evaluations.

3.3.3. ASSESSMENT OF NONCARCINOGENS

The potential for adverse health effects associated with noncarcinogens, such as organ damage, immunological effects, birth defects, or skin irritation, is assessed by comparing the estimated average daily intake (exposure dose) to a reference dose (RfD). RfDs typically are expressed in units of mg/kg/day. The RfD is an estimate of the daily human intake, including sensitive subgroups, which should not result in an appreciable risk of harmful effects. The uncertainty in these estimates may span an order of magnitude or more. RfDs may be derived for chronic and subchronic exposures. Chronic RfDs should be employed to evaluate all potential noncancer health effects for most exposure scenarios, except for scenarios with an exposure duration of less than two years. For scenarios with relatively short-term exposure, subchronic RfDs may be used, although they are less readily available.

For air, EPA provides an inhalation reference concentration (RfC) in units of $\mu g/m^3$ instead of an inhalation RfD.

3.3.4. ORAL-TO-DERMAL EXTRAPOLATION

Because EPA has not promulgated dermal route toxicity values, oral route RfDs and carcinogenic slope factors (SFs) will be used to evaluate exposures to substances by the dermal route. Such route-to-route extrapolation has a scientific basis because the distribution, metabolism, and elimination patterns (biokinetics) of chemicals are usually similar once they are absorbed, regardless of the exposure route. However, dermal toxicity values are typically based on an absorbed dose, whereas oral exposures usually are expressed in terms of an administered dose. If adequate data concerning the gastrointestinal (GI) absorption of a COPC is available, then dermal RfDs and SFs may be derived by applying a GI factor to the oral toxicity value.

EPA recommends an adjustment in oral toxicity factor (to account for absorbed dose in the dermal exposure pathway) if scientifically defensible data demonstrate that the GI absorption of the chemical, from a medium similar to the one studied (e.g., water, food), is significantly less than 100 percent. EPA uses a value of 50 percent as the cutoff to represent GI absorption low enough to require an adjustment in the toxicity factor (EPA 2004). No adjustment is necessary for GI absorption greater than 50 percent. The choice of this value considers intrinsic variability in the analysis of absorption studies, and removes the need to make comparatively small adjustments in the toxicity value. Also, making an adjustment for a higher GI absorption rate of 75%, for example, would otherwise imply a level of accuracy that is not supported by the science.

An absorption rate of 50 percent means that the observed toxic response was due to the half of the administered dose that was absorbed. Therefore, the toxicity of the absorbed dose is twice that of the administered dose.

Dermal toxicity factors are calculated using the following equations:

$$\text{Dermal RfD} = \text{Oral RfD} \times \text{ABS}$$

$$\text{Dermal SF} = \text{Oral SF} / \text{ABS}$$

Where:
ABS = Gastrointestinal absorption factor (fraction)

Absorption (ABS) values are available in EPA (2004). For chemicals without GI absorption values, EPA recommends using an ABS value of 100 percent. This assumption is more appropriate for organic chemicals, which are generally well absorbed across the GI tract. For inorganic compounds, there will be an associated underestimation of risk if actual absorption is less than the assumed 100 percent.

3.3.5. TOXICITY OF CDDS/CDFS AND DIOXIN-LIKE PCBS

Consistent with EPA, DEQ allows use of toxicity equivalency factors (TEFs) to evaluate toxic effects of polychlorinated dibenzo-*p*-dioxins (CDDs), polychlorinated dibenzofurans (CDFs), and co-planar (dioxin-like) PCBs congeners relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). Concentrations of congeners are multiplied by their TEFs to estimate the toxicity of these congeners relative to 2,3,7,8-TCDD; the resulting concentrations may be summed into a total 2,3,7,8-TCDD toxic equivalent (TEQ) concentration. World Health Organization TEFs for humans are listed in Table 2. (Van den Berg 2006).

DEQ applies the individual chemical acceptable risk level of 1×10^{-6} to individual CDD/CDF congeners and dioxin-like PCB congeners, and the 1×10^{-5} acceptable cumulative risk level to multiple congeners. If the analysis is only for total PCBs (*e.g.*, Aroclors), DEQ will apply the 1×10^{-6} acceptable risk level to the total PCB concentration. This is because the majority of the risk posed by the mixture may be due to one congener.

3.3.6. TOXICITY OF POLYCYCLIC AROMATIC HYDROCARBONS

DEQ accepts use of TEFs to evaluate toxic effects of polycyclic aromatic hydrocarbons (PAHs) relative to benzo(a)pyrene. Concentrations of other PAHs are multiplied by their TEFs to estimate their toxicity relative to benzo(a)pyrene; the resulting concentrations may be summed into a total benzo(a)pyrene toxic equivalent concentration. TEFs for humans are listed in Table 3 (ATSDR 1995; p. 168). Typically, however, slope factors based on the TEFs are available from EPA for the individual PAHs, so an evaluation using TEFs is not required.

DEQ applies the individual chemical acceptable risk level of 1×10^{-6} to individual carcinogenic PAHs. The acceptable risk level of 1×10^{-5} for cumulative exposure applies to all carcinogenic chemicals, which could include multiple cPAHs.

3.4. RISK CHARACTERIZATION

3.4.1. RISK-BASED DECISION MAKING APPROACH

The results of a risk-based decision making (RBDM) evaluation can be used to complete a risk assessment in accordance with DEQ's 2003 guidance document *Risk-Based Decision-Making for the Remediation of Petroleum-Contaminated Sites* (DEQ 2003, http://www.deq.state.or.us/lq/rbdm.htm). Comparing site concentrations with risk-based concentrations (RBCs) is an efficient means of determining if there is unacceptable risk at a site. The assumptions and calculations are similar to those used in traditional risk assessments. RBCs can be developed using default site values and exposure assumptions, or they can be made site-specific by using actual site characteristics (e.g., depth to groundwater, carbon content of soil) and exposure assumptions. As discussed in the Appendix H of the RBDM

guidance document, the basic RBDM approach does not fully meet the requirements of a risk assessment under DEQ's Cleanup program rules. To allow the RBDM approach to be used for conducting a risk assessment for a site, the following considerations need to be incorporated:

- Calculation of central tendency exposure in addition to reasonable maximum exposure
- Explicit calculation of risk
- Evaluation of cumulative risk
- Evaluation of uncertainty

These are relatively minor modifications. If you are using the RBDM approach at a site, Appendix H of the RBDM document provides guidance on how to complete the required risk assessment. At sites with less typical exposure scenarios (such as those involving sediment contact or ingestion of contaminated biota), you will need to either more closely follow traditional risk assessment guidance or provide additional supplemental risk assessment (Section 3.4.2 and Appendix A).

3.4.1.1. CENTRAL TENDENCY EXPOSURE RBCS

Most decisions regarding the acceptability of soil and groundwater concentrations at a site will be made using comparisons with reasonable maximum exposure RBCs as required by rule. However, RBCs based on central tendency exposure should also be calculated to meet cleanup program risk assessment requirements. The central tendency exposure input parameters (Appendix A, Table A-2), are generally based on averages. The central tendency information will also assist in the uncertainty analysis.

The appropriate concentration value for comparison with central tendency exposure RBCs is the arithmetic mean. Although decisions regarding cleanup goals will be made using RME values, during the feasibility study stage it may be valuable to consider central tendency exposure. This will be particularly important if it is not feasible to remediate the site to reasonable maximum exposure RBC levels.

3.4.1.2. PRESENTATION OF CALCULATED RISK

The direct comparison of concentrations with RBCs is the most straightforward means of determining whether there are acceptable concentrations at the site. As discussed earlier, this approach is essentially the inverse of that used in a traditional risk assessment. It is a simple matter to relate the two approaches. Presenting results in terms of risk will meet the rule requirements for conducting risk assessments.

For carcinogens, the RBC is calculated using an acceptable risk level of 10^{-6}. Because risk is proportional to concentration, the conversion equation is:

$$ELCR = \frac{Concentration}{RBC} \times 10^{-6}$$

Where:

ELCR	=	Excess lifetime cancer risk
Concentration	=	Chemical concentration (mg/kg, mg/L or µg/m^3)
RBC	=	Risk-Based Concentration (mg/kg, mg/L or µg/m^3)
10^{-6}	=	Acceptable excess lifetime cancer risk for individual carcinogens

For non-carcinogens, using an acceptable hazard quotient of 1, the conversion equation is

$$HQ = \frac{\text{Concentration}}{\text{RBC}} \times 1$$

Where:

HQ = Hazard quotient
Concentration = Chemical concentration (mg/kg, mg/L or mg/m^3)
RBC = Risk-Based Concentration (mg/kg, mg/L or mg/m^3)
1 = Acceptable HQ

3.4.1.3. CALCULATION OF CUMULATIVE RISK

At cleanup sites, it will be necessary to estimate the overall site risk. We suggest the following approach for evaluating cumulative cancer and noncancer risks from default RBC values, or from site-specific calculated RBCs.

Locate each of the site contaminants in the Table of RBCs and record its RBC for the appropriate media and pathways. Note whether the contaminant is considered a carcinogen (indicated by "c") or a noncarcinogen (indicated by "nc").

For each carcinogen, take the site-specific concentration and divide that value by the RBC. Add the results for all of the carcinogens and multiply this sum by 10^{-6} to estimate the cumulative risk from exposure to carcinogens. A risk of greater than 10^{-6} for any individual carcinogen or greater than 10^{-5} for the sum of all of the carcinogens may require further evaluation and should be discussed with DEQ.

$$\text{Risk} = \left[\left(\frac{\text{Conc}_{\text{ChemicalX}}}{\text{RBC}_{\text{ChemicalX}}}\right) + \left(\frac{\text{Conc}_{\text{ChemicalY}}}{\text{RBC}_{\text{ChemicalY}}}\right) + \left(\frac{\text{Conc}_{\text{ChemicalZ}}}{\text{RBC}_{\text{ChemicalZ}}}\right)\right] \cdot 10^{-6}$$

Present cancer risks to only one significant digit to avoid implying unwarranted precision. For instance, an ELCR of 1.3×10^{-6} should be presented as 1×10^{-6}, and would be considered an acceptable risk from exposure to an individual carcinogen. A cumulative ELCR of 1.5×10^{-5} should be presented as 2×10^{-5}, and would be considered an unacceptable risk from exposure to multiple carcinogens.

For each noncarcinogen, take the site-specific concentration and divide that value by the RBC. Add the results for all of the noncarcinogens to obtain the hazard index for the site. A hazard index (HI) of 1 or less is generally considered acceptable. A hazard index value greater than 1 may require further evaluation and should be discussed with DEQ (see Section 3.7).

$$\text{Hazard Index} = \left[\left(\frac{\text{Conc}_{\text{ChemicalX}}}{\text{RBC}_{\text{ChemicalX}}}\right) + \left(\frac{\text{Conc}_{\text{ChemicalY}}}{\text{RBC}_{\text{ChemicalY}}}\right) + \left(\frac{\text{Conc}_{\text{ChemicalZ}}}{\text{RBC}_{\text{ChemicalZ}}}\right)\right]$$

The HIs can be further refined by summing only chemicals with effects on the same organ system. Note, however, that many chemicals cause adverse effects on more than one organ. You need to consider all relevant effects when summing. Present hazard index and hazard quotient results to two significant digits. For example, an HQ of 1.69 should be reported as 1.7.

For carcinogens that also have an RfD for noncancer effects, a noncancer RBC should be calculated and included in the risk assessment. In the DEQ RBC spreadsheet, ToxData sheet, change the risk type from "c" to "nc", and recalculate the RBCs.

An example summary risk table is shown in Example 2.

Example 2
Summary Risk Table

			Calculated Risk	
Chemical	**UCL[1] or Maximum Concentration (mg/Kg)**	**RBC_{ss} Residential Soil[2] (mg/Kg)**	**Excess Lifetime Cancer Risk**	**Hazard Quotient**
Benzene	110	7.3 (c) 140 (nc)	2×10^{-5}	0.79
Benzo[a]pyrene	0.25	0.015 (c)	2×10^{-5}	NA
Toluene	30	5,800 (nc)	NA	0.0052
Naphthalene	55	4.6 (c) 34 (nc)	1×10^{-5}	1.6
TOTAL			4×10^{-5}	2.4

Notes:
[1] 90 percent upper confidence limit. As described in OAR 340-122-0084, other estimates may be used when acceptable to the Department. [2] Risk-based concentration for surface soil exposure. For benzene and naphthalene, the RBCs shown are for carcinogenic (c) and noncarcinogenic (nc) effects.

The conclusion of the example risk assessment is the same as that obtained from comparing soil concentrations with RBCs: the soil concentrations of benzene, benzo[a]pyrene and naphthalene are unacceptable. In this example, the excess lifetime cancer risk from residential soil exposure to benzene, benzo[a]pyrene, and naphthalene all exceed the regulatory limit of 1×10^{-6}. In addition, the cumulative risk exceeds the limit of 1×10^{-5}. Also, the calculated hazard quotient for naphthalene exceeds 1.

For the pathways involving transport, in many cases RBCs will not be available because the soil saturation limit or the water solubility limit is exceeded. Appendix H of the RBDM guidance document provides a method of using the soil saturation limit or water solubility limit in the risk calculations (DEQ 2003).

3.4.1.4. HUMAN MILK INGESTION

The RBC calculations do not currently include the breastfeeding infant pathway for bioaccumulating chemicals. Until the RBCs are updated, use the method presented in Section 3.4.2.3 and the values in Table 4 to convert RBCs for occupational and residential exposure to RBCs that will also protect breastfeeding infants. Use all relevant RBCs in your risk assessment.

3.4.1.5. UNCERTAINTY EVALUATION

A section on uncertainty should be included in the report. In this section, uncertainty in the exposure analysis (e.g., the conceptual site model), toxicity analysis, and risk characterization results should be evaluated qualitatively and quantitatively (if possible). This evaluation will allow managers to consider the uncertainty associated with the risk assessment.

3.4.2. STANDARD RISK CHARACTERIZATION APPROACH

For sites where all the exposure scenarios are not included in the RBDM guidance, it will be necessary to prepare a standard risk assessment for at least the scenarios that lack RBCs. You can also choose to use the standard approach for other reasons. Details regarding the exposure assessment are presented in Appendix A. The remainder of this section presents the risk characterization approach.

3.4.2.1. CANCER RISK

For ingestion and dermal exposure, potential excess lifetime cancer risk (ELCR) is assessed by multiplying the estimated dose (chronic daily intake, CDI) of a carcinogen by its slope factor (SF).

$$ELCR = CDI\ (mg/kg/day) \times SF\ (mg/kg/day)^{-1}$$

For inhalation exposure, ELCR is calculated by multiplying the long-term concentration by the inhalation unit risk factor (IUR).

$$ELCR = Conc.\ (\mu g/m^3) \times IUR\ (\mu g/m^3)^{-1}$$

Thes equations are adequate approximations for excess lifetime cancer risks given the magnitude of risks typically calculated at hazardous waste sites[3]. The calculated risk is expressed as the probability of an individual developing cancer over a lifetime and is an estimated upper-bound, incremental probability. Potential cancer risks should initially be calculated separately for exposure to each chemical for each exposure pathway. The separate potential cancer risks should be summed across all exposure pathways to obtain the total potential excess lifetime cancer risk to the potentially exposed population for comparison with the acceptable risk level for individual chemicals. Site-wide risks for all carcinogens should also be calculated. Calculated excess lifetime cancer risks should be compared to DEQ's acceptable risk levels of 1×10^{-6} for exposure to an individual carcinogen, and 1×10^{-5} for exposure to multiple carcinogens.

Present cancer risks to only one significant digit to avoid implying unwarranted precision. For instance, an ELCR of 1.3×10^{-6} should be presented as 1×10^{-6}, and would be considered an acceptable risk from exposure to an individual carcinogen. A cumulative ELCR of 1.5×10^{-5} should be presented as 2×10^{-5}, and would be considered an unacceptable risk from exposure to multiple carcinogens.

For classes of compounds consisting of multiple congeners (e.g., chlorinated dibenzo-*p*-dioxins and chlorinated dibenzofurans, or PCBs), the calculated risk for each congener should be compared with the acceptable risk level for individual carcinogens. Use the TEFs discussed in

[3] The precise equations are $ELCR = 1 - e^{-(CDI \times SF)}$ and $ELCR = 1 - e^{-(Conc \times IUR)}$.

Section 3.3.5. If analysis for PCB congeners is not performed, the calculated risk from total PCBs (as summed Aroclors or summed homologs) should be compared to the acceptable risk level for individual carcinogens. This is because the risk from the total PCBs may be primarily due to one congener.

3.4.2.2. NONCANCER EFFECTS

The potential for adverse effects resulting from exposure to noncarcinogens will in most cases be assessed by comparing the chemical-specific CDI or absorbed dose to its RfD. This comparison will be made by calculating the ratio of the estimated CDI (or absorbed dose) to the corresponding RfD to yield a hazard quotient (HQ):

$$HQ = \frac{CDI(mg/kg/day)}{RfD(mg/kg/day)}$$

For some exposure scenarios (e.g., construction worker), it is appropriate to use subchronic doses and subchronic RfD values, if available.

For inhalation exposure, the corresponding equation is:

$$HQ = \frac{Concentration\,(mg/m^3)}{RfC\,(mg/m^3)}$$

You should present HQs separately for each receptor evaluated, such as residents, occupational workers, etc. The receptor-specific HQs should then be summed across chemical and exposure pathways to generate a hazard index (HI). Separate HIs for different types of adverse health effects should be calculated only if the overall HIs exceed 1. Separate HIs should not be calculated if most of the HI value is attributable to a single chemical. Compare estimated hazard indices to DEQ's acceptable hazard index of 1. HQs and HIs should be presented to two significant digits. For example, an HQ of 1.69 should be reported as 1.7.

3.4.2.3. HUMAN MILK INGESTION

People who are exposed to bioaccumulative compounds such as PCBs, polybrominated biphenylethers (PBDEs), dioxins/furans, and DDTs, accumulate chemical concentrations in lipid tissue. These bioaccumulating chemicals will be present in human milk and may pose a threat to breastfeeding infants. EPA guidance is available to evaluate this exposure pathway (EPA 1998b and EPA 2005a). Because occupational and residential exposure includes exposure to women who may became mothers in the future, human milk ingestion is a relevant pathway for all sites with bioaccumulating compounds. For PCBs, exposure to the mother prior to breastfeeding will be the most important exposure pathway. Appendix D provides detailed information on the breastfeeding pathway and how to address it in a risk assessment. Use the infant risk adjustment factors in Table 4 to calculate risks to infants based on risks to occupational and residential receptors.

Note that an evaluation of breastfeeding risks is complicated by the known substantial health benefits of breastfeeding, as discussed in Appendix D. Including the breastfeeding exposure pathway in risk assessments is important to ensure that our environment is protective of infants. However, it is critical to understand that calculated risks are not intended to advise women about whether or not to breastfeed their infants. Rather, the purpose is to inform site clean-up managers so that they can make decisions that will lead to decreased exposure to women, and

ultimately lower concentrations of contaminants in the milk women produce for their infants. Calculated risks to infants support public health actions that encourage women to limit their own exposure to environmental contaminants so that their infants can receive the optimal health benefits from breastfeeding.

3.4.3. UNCERTAINTY ANALYSIS

Uncertainty in the exposure analysis, toxicity analysis, and risk characterization results should be evaluated quantitatively and or qualitatively (see EPA 1989, Sections 6.8, 7.6, and 8.4 for details).

3.5. DOCUMENTATION OF RESULTS

It is important that the results of the risk assessment, from problem formulation through identification of COPCs, to calculation of potential risk, be presented in a clear and transparent manner. You can use the report format presented in EPA 2001b, or another format acceptable to DEQ.

3.6. ACCEPTABLE RISK LEVEL DETERMINATION

Individual and cumulative acceptable risk levels for carcinogens and noncarcinogens are defined by OAR 340-122-0115(2)(a), (3)(a), and (4)(a). The acceptable ELCR for individual carcinogens (including congeners of chemical groups such as PCBs or chlorinated dioxin/furans) is 1×10^{-6}. The acceptable ELCR for exposure to multiple carcinogens is 1×10^{-5}. Calculated carcinogenic risk should be rounded to one significant digit (for example, 3×10^{-6}).

The acceptable noncarcinogenic hazard for exposure to individual and multiple chemicals is 1.0. To address the greater concern associated with exceeding a threshold concentration, calculated hazard quotients and hazard indices should be rounded to two significant digits (for example, 1.7).

3.7. POTENTIAL HOT SPOT DETERMINATION

Oregon rules require the identification of potential hot spots as defined in OAR 340-122-0115(32), which should be preliminarily identified in the risk assessment, and a final determination made in the feasibility study. DEQ has a guidance document for determining potential hot spots (DEQ 1998c). Hot spots are areas of unacceptable risk, where the concentrations are high enough that there is a preference for treatment. Contamination in groundwater or surface water is a hot spot if there is or will be a significant adverse effect on the beneficial use of the resource, and treatment is likely to restore or protect the beneficial use. For other media, there is a hot spot if:

A) Chemicals are present in concentrations exceeding risk-based concentrations corresponding to:
    a. 100 times the acceptable risk level for human exposure to each individual carcinogen;
    b. 10 times the acceptable risk level for human exposure to each individual noncarcinogen; or
    c. 10 times the acceptable risk level for exposure of individual ecological receptors or populations of ecological receptors to each individual hazardous substance;
B) Chemicals are reasonably likely to migrate, creating the conditions specified above; or
C) Chemicals are not reliably containable.

Potential hot spots are identified on the basis of individual sample locations. You should not base hot spots on EPCs calculated over larger areas.

3.8. RESIDUAL RISK ASSESSMENT

If unacceptable risks are determined at a site, a feasibility study will need to be conducted and remedial actions will likely be taken. Prior to the selection of a remedial action, a residual risk assessment is required (OAR 340-122-0084(4)). A residual risk assessment consists of two elements: 1) a quantitative assessment of the risk resulting from concentrations of chemicals remaining on the site at the conclusion of any treatment or removal; and 2) a qualitative or quantitative assessment of the adequacy and reliability of any institutional or engineering controls to be used to control chemicals remaining on the site.

In most cases, the residual risk assessment can be a simple evaluation. For example, if all chemical concentrations above acceptable risk levels are excavated from the site and disposed offsite, the residual risk assessment can consist of statements that this is a reliable remedial action, and that remaining chemical concentrations onsite will result in acceptable risk levels. If soil areas with unacceptable concentrations are to be capped, the residual risk assessment can state that capping is a reliable engineering control, and that preventing access to soil will reduce the risk to acceptable levels. If the cap should fail, the potential (short-term) risks would be those identified in the baseline risk assessment. One circumstance where the residual risk assessment will need to involve more than a reference to the baseline risk assessment is if the planned remedial action removes only a portion of the unacceptable chemical concentrations onsite. For example, if soil hot spots are removed, and the remaining unacceptable soil areas are capped, the residual risk assessment would need to document the reduced risk (relative to baseline conditions) that would occur if the soil cap were to fail. Monitoring and maintenance programs are typically required if elevated chemical concentrations are left on site, and the site remains listed on the Inventory.

4. REFERENCES

ADEC (2009) Alaska Department of Environmental Conservation. Division of Spill Response, Contaminated Sites Program, **Draft Guidance on Multi-Increment Soil Sampling**. March, 2009

ATSDR (1995). **Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs) (Update)**. Agency for Toxic Substances and Disease Registry, U.S. Department of Health & Human Services, Atlanta, Georgia (August 1995).

ATSDR (1998). **Toxicological Profile for Chlorinated Dibenzo-p-Dioxins (Update)**. Agency for Toxic Substances and Disease Registry, U.S. Department of Health & Human Services, Atlanta, Georgia (December 1998).

Birak P, Yurk J, Adeshina F, Lorber M, Pollard K, Choudury H and S. Kroner (2001). **Travis and Arms revisited: a second look at a widely used bioconcentration algorithm.** *Toxicology and Industrial Health* 17, 163-175.

Briggs GG, Bromilow RH & Evans AA (1982**). Relationship between lipophilicity and root uptake and translocation of non-ionized chemicals by barley.** *Pesticide Science* 13, 495-504.

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).

DEQ (1997a). **Generic Remedies for Soils Contaminated with Polychlorinated Biphenyls (PCBs)**. Waste Management and Cleanup Division, Oregon Department of Environmental Quality. (December 1997).

DEQ (1997b). **Policy on Toxicity Equivalency Factors**. Waste Management and Cleanup Division, Oregon Department of Environmental Quality. (October 23, 1997).

DEQ (1998a). **Guidance for Consideration of Land Use**. Waste Management and Cleanup Division, Oregon Department of Environmental Quality. (July 1, 1998)

DEQ (1998b). **Guidance for Conducting Beneficial Water Use Determinations at Environmental Cleanup Sites**. Waste Management and Cleanup Division, Oregon Department of Environmental Quality. (July 1, 1998)

DEQ (1998c). **Guidance for Identification of Hot Spots**. Waste Management and Cleanup Division, Oregon Department of Environmental Quality. (April 23, 1998).

DEQ (1998d). **Guidance for Use of Probabilistic Analysis in Human Health Risk Assessments**. Waste Management and Cleanup Division, Oregon Department of Environmental Quality. (Interim Final, January 1998).

DEQ (2003). **Risk-Based Decision-Making for the Remediation of Petroleum-Contaminated Sites**. Land Quality Division, Environmental Cleanup and Tanks Program, Oregon

Department of Environmental Quality. (September 22, 2003).

DEQ (2007). **Guidance for assessing Bioaccumulative Chemicals of Concern in Sediment. Oregon** Department of Environmental Quality; Environmental Cleanup Program. (April 3rd 2007).

DEQ (2009). **Revised table of Risk-Based Concentrations (RBCs)** http://www.deq.state.or.us/lq/rbdm.htm

DEQ (2010). **Guidance for Assessing and Remediating Vapor Intrusion in Buildings***.* Land Quality Division, Environmental Cleanup and Tanks Program, Department of Environmental Quality. (March 25, 2010).

EPA (1988). **Superfund Exposure Assessment Manual.** Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. [OSWER Directive 9285.5-1. EPA/540/1-88/001. NTIS PB89-135859], Washington, DC. (April 1988).

EPA (1989). **Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part A)**. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. (EPA/540/1-89/002, October 1989, Interim Final).

EPA (1991a). **Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors"**. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency. (OSWER Directive 9285.6-03, March 25, 1991).

EPA (1991b). **Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals)**. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. (EPA/540/R-92/003, December 1991, Interim).

EPA (1992b). **Supplemental Guidance to RAGS: Calculating the Concentration Term**. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency. (Publication Directive 9285.7-08I, May 1992).

EPA (1992c). **Guidance for Data Usability in Risk Assessment (Part A**). Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. (Publication 9285.7-09A, PB92-963356, April 1992, Final).

EPA (1993). **Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons**. Office of Research and Development, U.S. Environmental Protection Agency. (EPA/600/R-93/089, July 1993).

EPA (1994). **Statistical Methods for Evaluating the Attainment of Cleanup Standards, Volume 3, Reference-based Standards for Soils and Solid Media**. Office of Policy, Planning, and Evaluation, U. S. Environmental Protection Agency. (EPA 230-R-94-004, June 1994).

EPA (1996). **Soil Screening Guidance: Users Guide**. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency. (EPA/540/R-96/018, April 1996).

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

EPA (1998a). **Interim Final Guidance: Developing Risk-Based Cleanup Levels at Resource Conservation and Recovery Act Sites in Region 10.** Region 10, Office of Waste and Chemicals Management, U.S. Environmental Protection Agency. (EPA910/R-98-001, January 1998).

EPA (1998b). **Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions***.* EPA 600/R-98/137, December 1998.

EPA (2000a). **Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)**. U.S. Environmental Protection Agency, Office of Water. EPA-822-B-00-004, October 2000.

EPA (2000b). **Toxicological Review of Vinyl Chloride In Support of Summary Information on the Integrated Risk information System (IRIS)**. EPA/635R-00-004. U.S. Environmental Protection Agency, May 2000.

EPA (2000c). **Data Quality Objectives Process for Hazardous Waste Site Investigations**. (EPA QA/G-4HW): EPA/ 600/R-00/007. US Environmental Protection Agency, Washington D.C. January 2000. http://www.epa.gov/quality/qs-docs/g4hw-final.pdf

EPA (2001a). **EPA requirements for quality assurance project plans.** EPA QA-R5. EPA/240/B-01/005. U.S. Environmental Protection Agency. Office of Environmental Information. March 2001.

EPA (2001b). **Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments)**. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency. (EPA 540-R-97-003, Publication 9285.7-47, December 2001).

EPA (2002a). **Calculating upper confidence limits for exposure point concentrations at hazardous waste sites**. U.S. Environmental Protection Agency. Office of Emergency and Remedial Response. (OSWER 9285.6-10, December, 2002)

EPA (2002b). **Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites**. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. (OSWER 9355.4-24 December 2002).

EPA (2004). **Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)**. EPA/540/R/99/005. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency. (July 2004).

EPA (2005a). **Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities**. U.S. Environmental Protection Agency. EPA 530-R-05-006. September 2005.

EPA (2005b). **Guidelines for Carcinogenic Risk Assessment**. U.S. Environmental Protection Agency, EPA/630/P-03/001B. March 2005.

EPA (2005c). **Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens**. U.S. Environmental Protection Agency. EPA 630-R-03-003F. March 2005.

EPA. 2006a. **Systematic Planning: A Case Study for Hazardous Waste Site Investigations**. US EPA Office of Environmental Information. EPA/240/B-06/004. February 2006. http://www.epa.gov/quality/qs-docs/casestudy-final.pdf

EPA (2006b). **Implementation of the Cancer Guidelines and Accompanying Supplemental Guidance – Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Performing Risk Assessments that Include Carcinogens Described in the Supplemental Guidance as Having a Mutagenic Mode of Action**. Memorandum dated 14 June 2006.

EPA (2008). **Child-Specific Exposure Factors Handbook**. EPA/600/R-06/096F. National Center for Environmental Assessment. September 2008.

EPA (2009). **Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual** (Part F, Supplemental Guidance for Inhalation Risk Assessment). EPA-540-R-070-002. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency. (OSWER 9285.7-82, January 2009).

EPA. (2010a). **ProUCL Version 4.00.05 User Guide (Draft)**. EPA/600/R-07/038. U.S. Environmental Protection Agency. Office of Research and Development, Washington DC, May, 2010. http://www.epa.gov/nerlesd1/tsc/software.htm

EPA (2010b). **ProUCL Version 4.00.05 Technical Guide (Draft)**. EPA/600/R-07/041. U.S. Environmental Protection Agency. Office of Research and Development, Washington DC. May 2010. www.epa.gov/nerlesd1/tsc/software.htm.

EPA (2010c). **U.S. Environmental Protection Agency Regional Screening Levels**. http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm

McKone TE (1993). **The Precision of QSAR methods for estimating intermedia transfer factors in exposure assessments.** *SAR and QSAR in environmental research* 1, 41-51.

Travis CC, and AD. Arms AD (1988). **Bioconcentration of organics on beef, milk and vegetation**. Environmental Science and Technology. 22, 271-274.

USACE (2008). U**.S. Army Corps of Engineers. BSAF Database**. http://el.erdc.usace.army.mil/bsaf/.

Van den Berg, Martin, et al. (2006). **The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-like Compounds**. Published by Oxford University Press on behalf of the Society of Toxicology. ToxSci Advance Access, 7 July 2006.

WDOH 1995. **Development of Sediment Quality Criteria for the Protection of Human Health. Tier 1 Report**. Office of Toxic Substances, Washington Department of Health.

Table 1.

Oregon Default Background Concentrations for Inorganic Chemicals

NOTE: This table will likely be replaced by early 2011 following completion of a DEQ study of background concentrations in Oregon.

CHEMICAL	Soil (mg/kg, dw)	Freshwater		Marine	
		Water (μg/L)	Sediment (mg/kg, dw)	Water (μg/L)	Sediment (mg/kg, dw)
Antimony	4 (x)	<1 (h)	1 (w)	0.2 (u)	1 (v)
Arsenic	7 (s)	2 (i)	7.9 (w)	$0.005^{As(III)}$ (u) $1^{As(V)}$ (u)	9 (v)
Cadmium	1 (g)	<1 (h)	<0.5 (m)	0.1 (u)	0.9 (v)
Chromium	42 (e)	1 (h)	30 (n)	$0.002^{Cr(III)}$ (u) $0.2^{Cr(IV)}$ (u)	140 (v)
Copper	36 (g)	9 (j)	12 (o)	0.2 (u)	26 (v)
Lead	17 (f)	13.3 (k)	2 (p)	0.003 (u)	22 (v)
Mercury	0.07 (g)	<0.1 (h)	0.2 (q)	0.001 (u)	0.3 (v)
Nickel	38 (g)	5.5 (l)	20 (r)	0.5 (u)	59 (v)
Silver	1 (x)	<1 (h)	0.4 (w)	0.002 (u)	0.4 (v)
Selenium	2 (x)	0.2 (c)	0.4 (t)	$0.1^{Se(Vi)}$ (u) $0.05^{Se(IV)}$ (u)	0.5 (v)
Zinc	86 (a)	38 (b)	53 (d)	0.4 (u)	130 (v)

Notes:

(a) State-wide 90th percentile value from WDOE (1994). United States geometric mean value is 44 mg/kg (Fuhrer, 1986; Table 7). Zinc range in Oregon soils reported from <25 to 159 mg/kg (Fuhrer, 1989; Table 8).

(b) 90th percentile value of Lower Columbia River Basin data (1951 - 1993) (Fuhrer et al., 1996; Table 27). Zinc worldwide inland water background concentration reported as 10 μg/L.

(c) North American streams background concentration as reported in Fuhrer et al., 1996; Table 27.

(d) Concentration in Lower Columbia River sediment at >400 cm depth (Fuhrer and Horowitz, 1989; Table 10). Breakpoint for zinc between natural and anthropogenically-affected sediment reported as 145 mg/kg (Rickert et al., 1977). McCoy and Black (1998) report a freshwater reference value of 88 - 110 mg/kg.

(e) State-wide 90th percentile value from WDOE (1994). United States geometric mean value for Cr is 37 mg/kg (Fuhrer, 1986; Table 7).

(f) State-wide 90th percentile value for Washington (WDOE, 1994). United States geometric mean value is 16 mg/kg (Fuhrer, 1986; Table 7). Lead range in Oregon soils reported as 1.2 to 18 mg/kg (Fuhrer, 1989; Table 8).

(g) State-wide 90th percentile value from WDOE (1994).

(h) 90th percentile value of Lower Columbia River Basin data (1994) as reported in Fuhrer et al., 1996; Table 27.

(i) 90th percentile value of Lower Columbia River Basin data (1951 - 1993) as reported in Fuhrer et al., 1996; Table 27. Arsenic worldwide inland water background concentration reported as 2 μg/L.

(j) 90th percentile value of Lower Columbia River Basin data (1951 - 1993) as reported in Fuhrer et al.,

1996; Table 27. Copper worldwide inland water background concentration reported as 1.8 μg/L.

(k) 90th percentile value of Lower Columbia River Basin data (1951 - 1993) as reported in Fuhrer et al., 1996; Table 27. Lead worldwide inland water background concentration reported as 0.2 μg/L.

(l) 90th percentile value of Lower Columbia River Basin data (1951 - 1993) as reported in Fuhrer et al., 1996; Table 27. Nickel worldwide inland water background concentration reported as 0.3 μg/L.

(m) Concentration in Lower Columbia River sediment at >400 cm depth (Fuhrer and Horowitz, 1989; Table 10). McCoy and Black (1998) report a freshwater sediment reference value of 0.2 - 0.7 mg/kg. Cadmium concentrations in Portland Harbor bottom material reported to range from 0.4 to 1.2 mg/kg (Fuhrer, 1989; Table 8).

(n) Concentration in Lower Columbia River sediment at >400 cm depth (Fuhrer and Horowitz, 1989; Table 10). Breakpoint for chromium between natural and anthropogenically-affected sediment reported as 60 mg/kg (Rickert et al., 1977). McCoy and Black (1998) report a freshwater sediment reference value of 54 - 110 mg/kg.

(o) Concentration in Lower Columbia River sediment at >400 cm depth (Fuhrer and Horowitz, 1989; Table 10). Breakpoint for copper between natural and anthropogenically-affected sediment reported as 43 mg/kg (Rickert et al., 1977). McCoy and Black (1998) report a freshwater sediment reference value of 42 - 48 mg/kg.

(p) Concentration in Lower Columbia River sediment at >400 cm depth (Fuhrer and Horowitz, 1989; Table 10). Breakpoint for lead between natural and anthropogenically-affected sediment reported as 43 mg/kg (Rickert et al., 1977). McCoy and Black (1998) report a freshwater reference value of 13 - 23 mg/kg.

(q) Average concentration in "unpolluted" Willamette River Basin samples (Rickert et al., 1977; Table 8). McCoy and Black (1998) report a freshwater sediment reference value of 0.1 - 0.11 mg/kg.

(r) Concentration in Lower Columbia River sediment at >400 cm depth (Fuhrer and Horowitz, 1989; Table 10). McCoy and Black (1998) report a freshwater sediment reference value of 23 - 54 mg/kg.

(s) State-wide 90th percentile value from WDOE (1994). 95th percentile British Columbia regional soil background estimate for As is 10 mg/kg (BCE, 1999).

(t) Highest background value reported in Nagpal and Howell (2001). McCoy and Black (1998) report a freshwater sediment reference value of 1.1 - 4.6 mg/kg.

(u) Mean concentration values from Nozaki (1997). See also Quinby-Hunt and Turekian (1983) and Quinby-Hunt, and Wilde (1986/87).

(v) Maximum reference site values from Meador et al. (1994).

(w) Highest freshwater sediment reference site value from McCoy and Black (1998).

(x) 95th percentile British Columbia regional soil background value (BCE, 1999).

References for Table 1

BCE (1999). **Protocol for Contaminated Sites 4 - Determining Background Soil Quality**. British Columbia Ministry of Water, Land, and Air Protection. Victoria, British Columbia, Canada (September, 1999).

Fuhrer, G.J. (1986). **Extractable Cadmium, Mercury, Copper, Lead, and Zinc in the Lower Columbia River Estuary, Oregon and Washington.** Water Resources Investigations Report 86-4088. U.S. Geological Survey, Portland, Oregon.

Fuhrer, G.J. (1989). **Quality of Bottom Material and Elutriates in the Lower Willamette River, Portland Harbor, Oregon.** Water Resources Investigations Report 89-4005. U.S. Geological Survey, Portland, Oregon.

Fuhrer, G.J. and Horowitz, A.J. (1989). **The Vertical Distribution of Selected Trace Metals and Organic Compounds in Bottom Materials of the Proposed Lower Columbia River Export Channel.** Water Resources Investigations Report 95-4294. U.S. Geological Survey, Portland, Oregon.

Fuhrer, G.J., Tanner, D.O., Morace, J.L., McKenzie, S.W., and Skach, K.A. (1996). **Water Quality of the Lower Columbia River Basin: Analysis of Current and Historical Water-Quality Data through 1994.**

MacCoy, D.E. and Black, R.W. (1998). **Organic Compounds and Trace Elements in Freshwater**

Streambed Sediment and Fish from the Puget Sound Basin. USGS Fact Sheet 105-98. Puget Sound Basin NAWQA Study, U.S. Geological Survey, Seattle, Washington (September, 1998). {wa.water.usgs.gov/pugt/fs.105-98.html}

Meador, J.P., Clark Jr., R.C., Robisch, P.A., Ernest, D.W., Landahl, J.T., Varanasi, U., Chan, S-L., and McCain, B.B. (1994). **National Benthic Surveillance Project: Pacific Coast. Analyses of Elements in Sediment and Tissue Cycles I to V (1984-88).** NOAA Technical Memorandum NMFS-NWFSC-16. National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, Washington (August, 1994).

Nagpal, N. K. and Howell, K. (2001). **Water Quality Guidelines for Selenium, Technical Appendix**. British Columbia Ministry of Water, Land, and Air Protection. Victoria, British Columbia, Canada (September, 2001).

Nozaki, Y. A **fresh look at element distribution in the North Pacific.** EOS electronic supplement, posted May 27, 1997. {www.agu.org/eos_elec/}

Quinby-Hunt, M.S. and Turekian, K.K. (1983). **Distribution of elements in sea water**. EOS, 64: 130-131.

Quinby-Hunt, M.S. and Wilde, P. (1986/87). **Modeling of dissolved elements in sea water**. Ocean Science and Engineering, v, 11, no. 3,4, p. 153-251.

Rickert, D.A., Kennedy, V.C., McKenzie, S.W., and Hines, W.G. (1977). **A Synoptic Survey of Trace Metals in Bottom Sediments of the Willamette River, Oregon**. Geological Survey Circular 715-F. U.S. Geological Survey, Arlington, Virginia.

WDOE. (1994). **Natural Background Soil Metal Concentrations in Washington State**. Publication #94-115. Washington Department of Ecology, Olympia, WA (October, 1994).

Table 2
Toxic Equivalency Factors for Dioxins and PCBs

Chemical	Toxic Equivalency Factor (TEF)
Chlorinated Dibenzo-*p*-dioxins	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0003
Chlorinated Dibenzofurans	
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.03
2,3,4,7,8-PeCDF	0.3
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0003
Polychlorinated Biphenyls (PCBs)	
PCB 77 (3,3',4,4'-TeCB)	0.0001
PCB 81 (3,4,4',5-TeCB)	0.0003
PCB 105 (2,3,3',4,4'-PeCB)	0.00003
PCB 114 (2,3,4,4',5-PeCB)	0.00003
PCB 118 (2,3',4,4',5-PeCB)	0.00003
PCB 123 (2',3,4,4',5-PeCB)	0.00003
PCB 126 (3,3',4,4',5-PeCB)	0.1
PCB 156 (2,3,3',4,4',5-HxCB)	0.00003
PCB 157 (2,3,3',4,4',5-HxCB)	0.00003
PCB 167 (2,3',4,4',5,5'-HxCB)	0.00003
PCB 169 (3,3',4,4',5,5'-HxCB)	0.03
PCB 189 (2,3',4,4',5,5'-HxCB)	0.00003

Notes
(a) Source: Van den Berg et al., 2006.

Table 3
Toxic Equivalency Factors for Carcinogenic PAHs

COMPOUND	CASRN	TEF
Benzo[a]anthracene	56-55-3	0.1
Benzo[a]pyrene	50-32-8	1
Benzo[b]fluoranthene	205-99-2	0.1
Benzo[g,h,i]perylene	191-24-2	0.01
Benzo[k]fluoranthene	207-08-9	0.01
Chrysene	218-01-9	0.001
Dibenz[a,h]anthracene	53-70-3	1
Indeno[1,2,3-cd]pyrene	193-39-5	0.1

Notes:

PAHs = polycyclic aromatic hydrocarbons
CASRN = Chemical Abstract Services registration number
TEF = toxic equivalency factor
Source: EPA 1993.

Table 4
Default Infant Risk Adjustment Factors (IRAFs)
For Calculating Human Milk Consumption Risks
Based on Risks Calculated for Exposure to the Mother

Chemical	IRAF to Convert Chronic HQ for Mother to Subchronic HQ for Infant[a,e]		IRAF to Convert ELCR for Mother to ELCR for Infant[b]	
	Adult Exposure Pathways[c]	Residential Soil Exposure Pathway[d]	Adult Exposure Pathways[c]	Residential Soil Exposure Pathway[d]
CDDs/CDFs	2	0.3	1	0.7
DDT/DDE/DDD	2	0.3	0.007	0.004
Total PCB	25	4	1	0.6
PCB TEQ	2	0.3	1	0.7

Notes:

a) $HQ_{infant} = HQ_{mother} \times IRAF_{nc}$
b) $ELCR_{infant} = ELCR_{mother} \times IRAF_{ca}$

$IRAF_{nc}$ = Infant risk adjustment factor for noncancer effects
$IRAF_{ca}$ = Infant risk adjustment factor for cancer effects
HQ = hazard quotient
ELCR = excess lifetime cancer risk
CDD = chlorinated dibenzo-*p*-dioxin
CDF = chlorinated dibenzofuran
DDT = dichlorodiphenyltrichloroethane
DDE = dichlorodiphenyldichloroethene
DDD = dichlorodiphenyldichloroethane
PCB = polychlorinated biphenyl
TEQ = 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxicity equivalent

c) Adult pathways include occupational exposure pathways and exposure pathways such as residential groundwater ingestion and food ingestion that are often evaluated using adult exposure parameter values for noncancer risk. Residential exposure pathways for cancer effects use time-integrated exposure, which generally reflects adult exposure, with the exception of residential soil exposure.
d) Residential soil exposure for noncancer effects is typically evaluated using child exposure parameter values. Residential soil exposure for cancer effects uses time-integrated exposure.
e) IRAFs reflect differences in child/adult exposure and infant expoure, except for total PCBs, where the IRAFs include both differences in exposure and differences in chronic and subchronic RfDs. Considering exposure alone, the infant exposure adjustment factors (IEAFs) for total PCBs are:
Noncancer adult exposure pathways, IEAF = 38
Noncancer residential soil exposure pathways, IEAF = 5

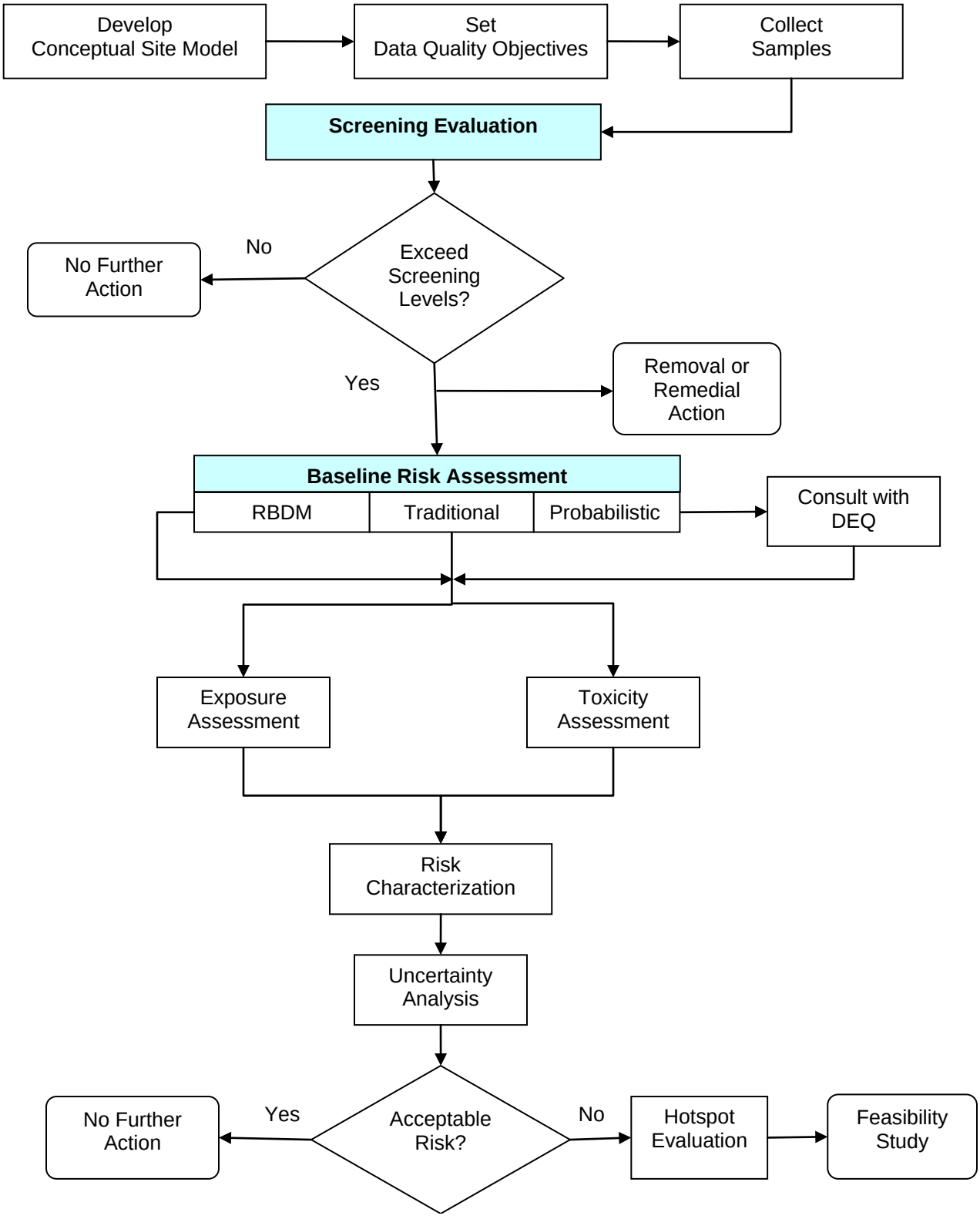
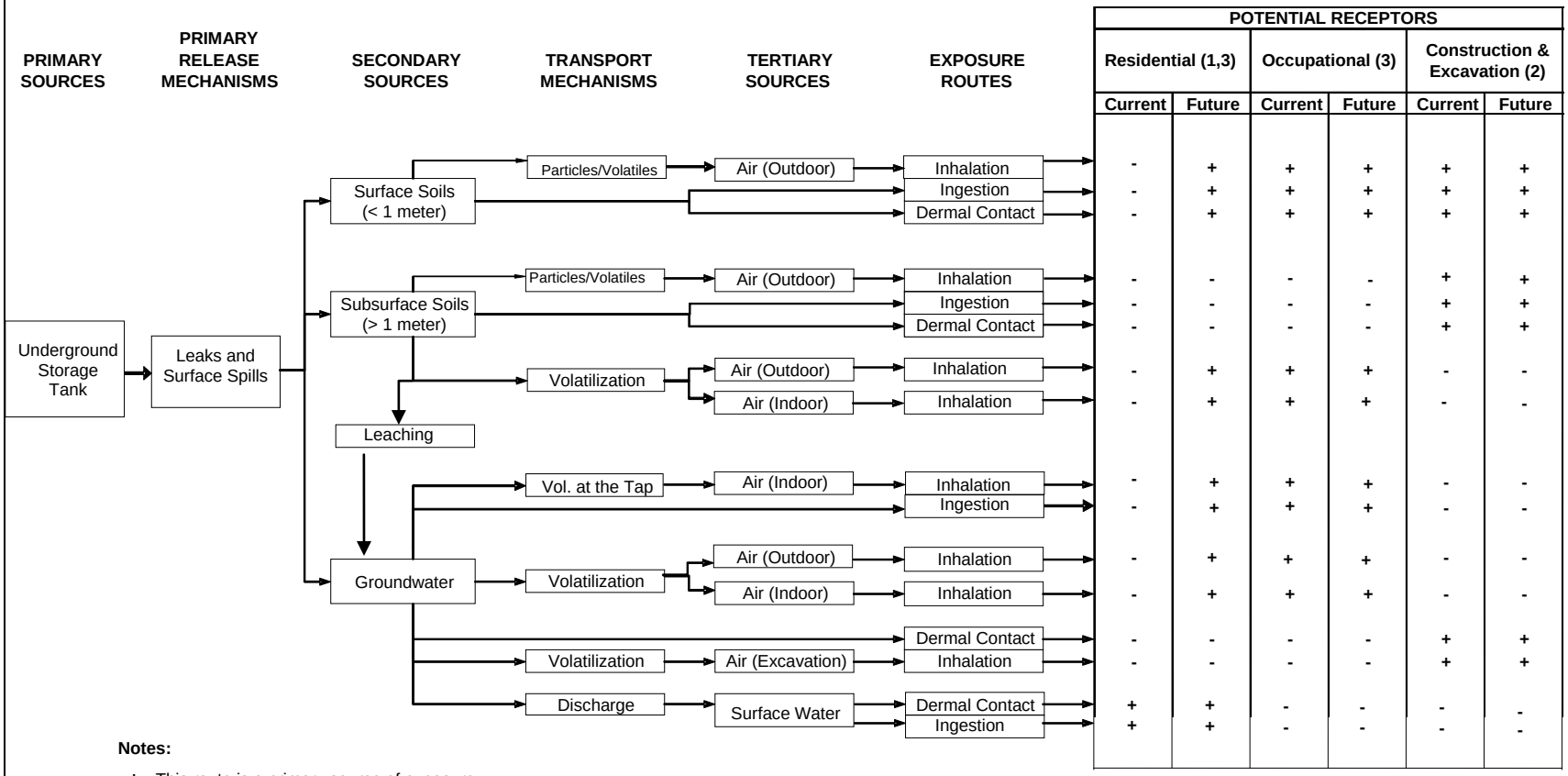


Figure 1. Overview of Human Health Risk Assessment Process



Notes:

+ This route is a primary source of exposure.
- There is no exposure by this route.
(1) Specify if urban residential or single-family residential. Include separate columns for these exposure scenarios if necessary.
(2) Include separate columns for these two exposure scenarios if necessary.
(3) Includes infant breastfeeding pathway for bioaccumulating chemicals.

Figure 2 Example Conceptual Site Model

APPENDIX A
Exposure Assessment Equations

Exposure elements are incorporated directly into the RBC equations provided in Appendix B of DEQ's RBDM guidance (DEQ 2003). The equations were updated to include early-life exposure and use of toxicity factors based on inhalation concentrations instead of doses. The updated equations are provided in this guidance document (Appendices B and C). The default assumptions for the RBC exposure factors are provided in the RBDM guidance document and also here in Tables A-1, A-2, and A-3. If you do not use RBCs in your risk assessment, you should use exposure equations appropriate for your exposure scenarios, most of which should be covered by the scenarios presented in this section.

This appendix presents exposure estimation equations for each exposure route listed in Section 2.5.2. These individual equations can be combined in different ways to represent differing exposure scenarios as determined by current and future land and water uses. The site-specific exposure models can be used to estimate the average daily exposure associated with each chemical within each medium (soil, water, air) for human receptors. The exposure factors and other terms used in these equations are summarized in Tables A-1, A-2, and A-3.

A.1 Age-Adjusted Intake Factors

A.1.1 Carcinogens

Residential exposures to carcinogens are calculated using age-adjusted factors because contact rates are different for children and adults. Use of these factors is important for soil ingestion exposures, which are higher during childhood and decrease with age, as well as for groundwater ingestion exposures due to the difference in water ingestion rates and body weights between children and adults. For the purposes of combining exposures across all pathways, age-adjusted factors are also used for residential inhalation, soil contact, water contact, and food item exposures. These factors approximate the integrated exposure from birth until age 30, combining contact rates, body weights, and exposure duration for two age groups: small children (age $\leq$ 6 years) and adults. Age-adjusted factors are described in RAGS Part B (EPA 1991b) and EPA Regional screening level documentation (EPA 2010c).

For carcinogens acting by a mutagenic mode of action, you need to evaluate early-life exposure. Special equations for this type of evaluation are presented in Appendix B.

Soil ingestion

$$IRS_{adj} = \frac{ED_c \cdot IRS_c}{BW_c} + \frac{(ED_r - ED_c) \cdot IRS_a}{BW_a}$$

Soil dermal contact

$$SFS_{adj} = \frac{ED_c \cdot AF_c \cdot SA_c}{BW_c} + \frac{(ED_r - ED_c) \cdot AF_a \cdot SA_a}{BW_a}$$

Water ingestion

$$IRW_{adj} = \frac{ED_c \cdot IRW_c}{BW_c} + \frac{(ED_r - ED_c) \cdot IRW_a}{BW_a}$$

Water dermal contact

$$SAS_{adj} = \frac{ED_c \cdot SA_c}{BW_c} + \frac{(ED_r - ED_c) \cdot SA_a}{BW_a}$$

Fish ingestion

$$IRF_{adj} = \frac{ED_c \cdot IRF_c}{BW_c} + \frac{(ED_r - ED_c) \cdot IRF_a}{BW_a}$$

Food ingestion

$$IRQ_{adj} = (ED_c \cdot \{IRV_c, IRM_c\}) + ((ED_r - ED_c) \cdot \{IRV_a, IRM_a\})$$

where:

IRS_{adj}	=	Age-adjusted incidental soil ingestion factor ([mg·yr]/[kg·d])
IRQ_{adj}	=	Age-adjusted food ingestion factor ([g·yr]/[kg·d])
IRF_{adj}	=	Age-adjusted fish ingestion factor ([g·yr]/[kg·d])
IRW_{adj}	=	Age-adjusted water ingestion factor ([L·yr]/[kg·d])
SFS_{adj}	=	Age-adjusted soil dermal contact factor ([mg·yr]/[kg·event])
SAS_{adj}	=	Age-adjusted water dermal contact factor ([cm^2·yr]/kg)
ED_c	=	Exposure duration, child (yr)
ED_r	=	Exposure duration, residential (yr)
BW_a	=	Body weight, adult (kg)
BW_c	=	Body weight, child (kg)
IRS_a	=	Soil ingestion rate, adult (mg/d)
IRS_c	=	Soil ingestion rate, child (mg/d)
IRM_c	=	Total meat, dairy product, egg ingestion rate, child (g/[kg·d])
IRM_a	=	Total meat, dairy product, egg ingestion rate, adult (g/[kg·d])
IRV_c	=	Total vegetable and fruit ingestion rate, child (g/[kg·d])
IRV_a	=	Total vegetable and fruit ingestion rate, adult (g/[kg·d])
IRF_c	=	Fish ingestion rate, child (g/d)
IRF_a	=	Fish ingestion rate, adult (g/d)
IRW_a	=	Water ingestion rate, adult (L/d)
IRW_c	=	Water ingestion rate, child (L/d)
AF_a	=	Adherence factor, adult (mg/cm^2·event)
AF_c	=	Adherence factor, child (mg/cm^2·event)
SA_a	=	Exposed skin surface area, adult (cm^2)
SA_c	=	Exposed skin surface area, child (cm^2)
IRA_a	=	Inhalation rate, adult (m^3/d)
IRA_c	=	Inhalation rate, child (m^3/d)

Because we use concentration-based toxicity factors for the inhalation pathway, an inhalation age-adjusted factor is not needed.

A.1.2 Non-Carcinogens

Age-adjusted factors are not necessary for exposure to non-carcinogens. The equation for residential non-carcinogen exposure can be applied to either children or adults using age-appropriate exposure factors. Typically, it is more important to evaluate non-carcinogenic exposure to children given their larger exposure rates (such as incidental soil ingestion) and lower body weight. Therefore, non-carcinogenic residential risks should be calculated for children rather than adults. For occupational exposure, assume that exposure will be limited to adults, and use appropriate adult exposure factors.

A.2 Soil

A.2.1 Incidental Ingestion

Carcinogens, residential

$$ADD_{si} = \frac{C_s \cdot IRS_{adj} \cdot CF_{km} \cdot EF_{dy}}{AT_c}$$

Carcinogens, occupational

$$ADD_{si} = \frac{C_s \cdot IRS \cdot CF_{km} \cdot EF_{dy} \cdot ED}{BW \cdot AT_c}$$

Noncarcinogens

$$ADD_{si} = \frac{C_s \cdot IRS \cdot CF_{km} \cdot EF_{dy} \cdot ED}{BW \cdot AT_n}$$

where:

ADD_{si}	=	Average daily dose from incidental soil ingestion (mg/[kg·d])
C_s	=	Chemical concentration in soil (mg/kg)
IRS_{adj}	=	Age-adjusted incidental soil ingestion factor ([mg·yr]/[kg·d])
IRS	=	Incidental soil ingestion rate (mg/d) [child or adult]
CF_{km}	=	Conversion factor (10^{-6} kg/mg)
BW	=	Body weight (kg) [child or adult]
EF_{dy}	=	Exposure frequency (d/yr) [child or adult]
ED	=	Exposure duration (yr) [child or adult]
AT_n	=	Averaging time, noncarcinogens (d) [= ED]
AT_c	=	Averaging time, carcinogens (d)

Average daily dose is generally presented as an administered dose, not an absorbed dose. Absorption refers to the amount of a chemical 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 chemical at the exchange boundary and available for absorption; it is not equivalent to absorbed dose (EPA 1989). In the unusual case where the reference dose and/or cancer slope factor for a chemical are expressed in terms of absorbed dose, the toxicity factors should be adjusted accordingly (see EPA 1989; Section 4). Values for C_s can be derived primarily from onsite measurements or secondarily through the use of various intermedia transfer factors. For noncarcinogens, intakes are calculated by averaging the daily doses over the averaging time (AT_n), which is set equal to the exposure duration (ED). For carcinogens, intakes are averaged over AT_c, which is typically 70 years (25,550 days).

A.2.2 Dermal

Carcinogens, residential

$$ADD_{sd} = \frac{C_s \cdot DAF \cdot SFS_{adj} \cdot CF_{km} \cdot EF_{evd} \cdot EF_{dy}}{AT_c}$$

Carcinogens, occupational

$$ADD_{sd} = \frac{DA_{soil} \cdot SA \cdot EF_{evd} \cdot EF_{dy} \cdot ED}{BW \cdot AT_c}$$

Noncarcinogens

$$ADD_{sd} = \frac{DA_{soil} \cdot SA \cdot EF_{evd} \cdot EF_{dy} \cdot ED}{BW \cdot AT_n}$$

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

where:

ADD_{sd}	=	Absorbed daily dose from contact with soil (mg/[kg·d])
SA	=	Exposed skin surface area (cm^2)
SFS_{adj}	=	Age-adjusted soil dermal contact factor ([mg·yr]/[kg·event])
EF_{dy}	=	Exposure frequency (d/yr) [child or adult]
EF_{evd}	=	Event frequency (events/d) [child or adult]
ED	=	Exposure duration (yr) [child or adult]
BW	=	Body weight (kg) [child or adult]
AT_n	=	Averaging time, noncarcinogens (d)
AT_c	=	Averaging time, carcinogens (d)
DA_{soil}	=	Absorbed dose per soil contact event (mg/cm^2·event)
C_s	=	Contaminant concentration in soil (mg/kg)
AF	=	Soil-to-skin adherence factor (mg/cm^2·event) [child or adult]
CF_{km}	=	Conversion factor (10^{-6} kg/mg)
DAF	=	Dermal absorption factor (unitless)
BW	=	Body weight (kg) [child or adult]

In general, EPA (2004) should be consulted for guidance on the evaluation and use of the dermal absorption factor (DAF). 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. Table A-4 provides default DAF values.

A.3 Water (Ground or Surface)

A.3.1 Ingestion

Carcinogens, residential

$$ADD_{wi} = \frac{C_w \cdot IRW_{adj} \cdot (EF_{hd} / CF_{hd}) \cdot EF_{dy}}{AT_c}$$

Carcinogens, occupational

$$ADD_{wi} = \frac{C_w \cdot IRW \cdot (EF_{hd} / CF_{hd}) \cdot EF_{dy} \cdot ED}{BW \cdot AT_c}$$

Noncarcinogens

$$ADD_{wi} = \frac{C_w \cdot IRW \cdot (EF_{hd} / CF_{hd}) \cdot EF_{dy} \cdot ED}{BW \cdot AT_n}$$

where:

ADD_{wi}	=	Average daily dose from tap water ingestion (mg/[kg·d])
C_w	=	Contaminant concentration in water (mg/L)
IRW_{adj}	=	Age-adjusted water ingestion factor ([L·yr]/[kg·d])
IRW	=	Drinking (tap) water ingestion rate (L/d) or incidental water ingestion rate (L/d) [child or adult]
EF_{hd}	=	Exposure frequency (hr/d) [child or adult]
CF_{hd}	=	Conversion factor (24 hr/d)
EF_{dy}	=	Exposure frequency (d/yr) [child or adult]
ED	=	Exposure duration (yr) [child or adult]
BW	=	Body weight (kg) [child or adult]
AT_n	=	Averaging time, noncarcinogens (d)
AT_c	=	Averaging time, carcinogens (d)

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.

A.3.2 Dermal

Carcinogens, residential

$$ADD_{wd} = \frac{DA_{water} \cdot SAS_{adj} \cdot EF_{evd} \cdot EF_{dy}}{AT_c}$$

Carcinogens, occupational

$$ADD_{wd} = \frac{DA_{water} \cdot SA \cdot EF_{evd} \cdot EF_{dy} \cdot ED}{BW \cdot AT_c}$$

Noncarcinogens

$$ADD_{wd} = \frac{DA_{water} \cdot SA \cdot EF_{evd} \cdot EF_{dy} \cdot ED}{BW \cdot AT_n}$$

Inorganics in water

$$DA_{water} = K_P \cdot (C_w \cdot CF_{cl}) \cdot t_{event}$$

Organics in water

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

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

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

where:

ADD_{wd}	=	Absorbed daily dose from contact with water (mg/[kg·d])
SA	=	Exposed skin surface area (cm^2)
SAS_{adj}	=	Age-adjusted water dermal contact factor ([cm^2·yr]/kg)
EF_{evd}	=	Event frequency (events/d)
EF_{dy}	=	Exposure frequency (d/yr)
ED	=	Exposure duration (yr)
BW	=	Body weight (kg)
AT_n	=	Averaging time, noncarcinogens (d)
AT_c	=	Averaging time, carcinogens (d)
DA_{water}	=	Dose absorbed per unit area per water contact event (mg/cm^2·event)
C_w	=	Contaminant concentration in water (mg/L)
CF_{cl}	=	Conversion factor (10^{-3} L/cm^3)
t_{event}	=	Duration of exposure event (hr/event)
K_p	=	Dermal permeability coefficient (cm/hr) [10^{-3} for inorganics]
τ	=	Lag time (hr/event)
t*	=	Time to reach steady-state (hr)
B	=	Relative contribution of permeability coefficients (unitless)
K_{ow}	=	*n*-Octanol-water partition coefficient
MW	=	Contaminant-specific molecular weight (g/mol)

For most chemicals, values for τ, t*, K_p, and B may be obtained from Appendix B of EPA (2004).

For chemicals not listed in Appendix B, these parameters may be calculated using equations given by EPA (2004). EPA's regional screening table (EPA 2010c) is also a source of dermal factor values.

Many semi-volatile organic compounds of interest at sites, such as PAHs, PCBs, and dioxins, have properties that fall outside of the effective prediction domain used by EPA to develop the table of parameter values for dermal contact with water. These chemicals are identified in Appendix B of EPA 2004. Our recommendation is to not include these chemicals in the quantitative risk characterization because the results can be unrealistically health protective. Instead, water dermal contact with these chemicals should be discussed in the uncertainty section, or otherwise addressed in a qualitative manner.

A.4 Air

A.4.1 Vapors

Volatile chemicals are defined as those with a Henry's Law constant [atm·m^3/mol] greater than 10^{-5} (DEQ 2010). We no longer use a molecular weight limit of 200 g/mol from EPA 1996 in the definition of a volatile chemical. Quantitatively evaluate this pathway if the properties of the chemicals released at the site indicate they meet the definition of volatile chemicals. Follow the screening steps in DEQ's vapor intrusion guidance (DEQ 2010) to determine if inhalation of vapors is a pathway of concern at the site.

Carcinogens, residential

$$ADC_{av} = \frac{C_a \cdot (ET / 24hr / day) \cdot EF \cdot ED}{AT_c}$$

Carcinogens, occupational

$$ADC_{av} = \frac{C_a \cdot (ET / 24hr / day) \cdot EF \cdot ED}{AT_c}$$

Noncarcinogens

$$ADC_{av} = \frac{C_a \cdot (ET / 24hr / day) \cdot EF \cdot ED}{AT_n}$$

where:

ADC_{av}	=	Average daily concentration from inhalation of vaporized contaminant (mg/m^3)
C_a	=	Contaminant concentration in air (mg/m^3)
C_s	=	Contaminant concentration in soil (mg/kg)
ET_{hd}	=	Exposure time (hr/d)
EF	=	Exposure frequency (d/yr)
ED	=	Exposure duration (yr)
AT_n	=	Averaging time, noncarcinogens (d)
AT_c	=	Averaging time, carcinogens (d)

The value of C_a is determined by direct measurement of soil gas or air concentrations in the area of interest (indoors or outdoors) as described in (DEQ 2010).

A.4.2 Soil Particles

Chemicals adhering to soil can result in an inhalation hazard if the soil particles become airborne. The following equations are used to evaluate this pathway.

Carcinogens, residential

$$ADC_{ap} = \frac{C_{air} \cdot (ET / 24hr / day) \cdot EF \cdot ED}{AT_c}$$

Carcinogens, occupational

$$ADC_{ap} = \frac{C_{air} \cdot (ET / 24hr / day) \cdot EF_{dy} \cdot ED}{AT_c}$$

Noncarcinogens

$$ADC_{ap} = \frac{C_{air} \cdot (ET / 24hr / day) \cdot EF_{dy} \cdot ED}{AT_n}$$

$$C_{air} = \left(\frac{C_s}{PEF} \right) \cdot F_s$$

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

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

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

where:

ADC_{ap}	=	Average daily concentration from inhalation of particulates (mg/m^3)
C_{air}	=	Concentration of chemical in air (mg/m^3)
INF_{adj}	=	Age-adjusted inhalation factor ($[m^3 \cdot yr]/[kg \cdot d]$)
ET	=	Exposure time (hr/d)
EF_{dy}	=	Exposure frequency (d/yr)
ED	=	Exposure duration (yr)
BW	=	Body weight of *k*th age (kg)
AT_n	=	Averaging time, noncarcinogens (d)
AT_c	=	Averaging time, carcinogens (d)
C_s	=	Chemical concentration in soil/dust (mg/kg)
PEF	=	Particulate emission factor for 10-micron particles (m^3/kg)
F_s	=	Fraction of soil contaminated (unitless)
Q/C	=	Inverse of mean concentration at center of source area ($g/m^2 \cdot s$ / kg/m^3); from Exhibit 11 in EPA (1996)
CF_{sh}	=	Conversion factor (3600 s/hr)

R_f = Respirable fraction (g/[m^2·h])
G = Fraction of vegetative cover (unitless)
U_m = Mean annual wind speed (m/s)
U_t = Erosion threshold wind speed (m/s)
U_f = Friction velocity (m/s)
H_e = Erosion threshold height (cm)
Z_o = Roughness height (cm)
F(x) = Function dependent of U_t/U_m (unitless)

The value of C_{air} 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 the equation for C_{air}, where the PEF term is an estimate of the emission rate of particulates from soils. The equation may make more sense by considering that 1/PEF is the 10-micron dust concentration in air. The concentration of a chemical in air (C_{air}) is calculated by multiplying the airborne dust concentration (1/PEF) by the chemical concentration in the dust (C_s), taking into account the fraction of soil contamination (F_s). 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 1.32×10^9 m^3/kg (EPA 1996).

A.5 Ingestion of Food

A.5.1 Fish/Shellfish

Carcinogens

$$ADD_f = \frac{C_f \cdot IRF_{adj} \cdot F_f \cdot CF_{gg} \cdot EF_{dy}}{AT_c}$$

Noncarcinogens

$$ADD_f = \frac{C_f \cdot IRF_f \cdot F_f \cdot CF_{gg} \cdot EF_{dy} \cdot ED}{BW \cdot AT_n}$$

$$C_f = C_{sed} \cdot BSAF \cdot f_{lipid} / f_{oc}$$

where:
ADD_f = Average daily dose from ingestion of local fish (mg/[kg·d])
C_f = Concentration of contaminant in finfish (mg/kg)
C_{sed} = Concentration of contaminant in sediment (mg/kg)
C_w = Concentration of contaminant in water (mg/L)
BSAF = Biota-Sediment Accumulation Factor, lipid/carbon normalized (kg_{oc}/kg_{lipid})
f_{lipid} = Lipid content in fish (fraction)
f_{oc} = Organic carbon content in sediment (fraction)
IRF_{adj} = Age-adjusted fish ingestion factor ([g·yr]/[kg·d])
IRF_f = Daily finfish ingestion rate (g/d) [child or adult]
F_f = Fraction of finfish obtained from site (unitless)
CF_{gg} = Conversion factor ($kg/10^3$ g)
EF_{dy} = Exposure frequency (d/yr) [child or adult]

ED = Exposure duration (yr) [child or adult]
BW = Body weight (kg) [child or adult]
AT_n = Averaging time, noncarcinogens (d)
AT_c = Averaging time, carcinogens (d)

The concentration in fish (C_f) is best obtained by sampling fish or shellfish tissue. Most consumption will be of fish fillet tissues, but there may be groups of fishers that use or consume all portions of the fish in broth or stews for example. This should be considered in a fish sampling and analysis plan.

It is preferable to use actual tissue measurements in most cases, however, in some cases where this is impracticable, you may need to model the concentration of chemical in fish or shellfish. The modeling can be from chemical concentrations in sediment or in water. Given the nature of bioaccumulating chemicals, the chemical will more likely be present in sediment than in water. In this case, biota-sediment accumulation factor (BSAF) values can be used to model fish tissue concentrations. BSAF values are carbon normalized, so the calculation of fish tissue concentrations must take into account the lipid content of fish and the fraction of organic carbon in sediment. Site-specific BSAF values are preferred. If site-specific data are not available, use BSAFs from Oregon DEQ guidance (DEQ 2007) or if not available there, use alternative sources such as Washington Department of Health study (WDOH 1995) or the U.S. Army Corps of Engineers database (USACE 2008). DEQ may also consider other scientifically defensible approaches to modeling bioaccumulation of chemicals from sediment to tissue.

The fraction of fish from site parameter ($0 \leq F_f \leq 1$) is an estimate of the fraction of total fish consumed which are caught within the contaminated site (exposure unit). As a default, you should assume that all fish consumed originate from contaminated waters onsite, so that $F_f = 1$. This is particularly relevant for subsistence (high-consumption) fishing and tribal fishing. If site-specific information is available on the sources of the fish, an appropriate distribution may be used to model a range of F_f values. The default fish consumption rate for all consumers is 17.5 g/day (EPA 2000a). At sites with subsistence fishing activity and fish populations capable of sustaining such a fishery, the default fish consumption rate from DEQ sediment bioaccumulation guidance is 142.4 g/day (DEQ 2007). If site-specific fish consumption data are available, they can be used to estimate IRF_f.

A.5.2 Vegetables and Fruits

Carcinogens

$$ADD_{vf} = \frac{C_v \cdot \left(IRQ_{adj-v} \cdot F_v + IRQ_{adj-fr} \cdot F_{fr}\right) \cdot V_{cf} \cdot CF_{gg} \cdot EF_{dy}}{AT_c}$$

Noncarcinogens

$$ADD_{vf} = \frac{C_v \cdot \left(IRV_v \cdot F_v + IRV_f \cdot F_{fr}\right) \cdot V_{cf} \cdot CF_{gg} \cdot EF_{dy} \cdot ED}{AT_n}$$

$$C_v = \left(\frac{C_s}{PEF}\cdot K_{ap}^{pt}\right) + \left(\frac{C_s}{VF_s}\cdot K_{ap}^{gs}\right) + \left(C_s \cdot K_{ps}\right) + \left(C_s \cdot K_{ps(roots)}\right)$$

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

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

$$K_{ps} = 0.784 \cdot \left\{\exp-\left[\frac{\left(\log_{10} K_{ow} - 1.78\right)^2}{2.44}\right]\right\}\cdot \frac{1}{K_{oc}\cdot f_{oc}}$$

where:

ADD_{vf}	=	Average daily dose from ingestion of vegetables/fruit (mg/[kg·d])
C_v	=	Contaminant concentration in vegetables and fruit (mg/kg)
$IRQ_{adj\text{-}v}$	=	Age-adjusted food ingestion factor - vegetables ([g·yr]/[kg·d])
$IRQ_{adj\text{-}fr}$	=	Age-adjusted food ingestion factor - fruit ([g·yr]/[kg·d])
IRV_v	=	Total vegetable ingestion rate (g/[kg·d])
IRV_f	=	Total fruit ingestion rate (g/[kg·d])
CF_{gg}	=	Conversion factor (kg/10^3 g)
F_v	=	Fraction of vegetables obtained from site (unitless)
F_{fr}	=	Fraction of fruit obtained from site (unitless)
V_{cf}	=	Vegetable/fruit correction factor (unitless)
EF_{dy}	=	Exposure frequency (d/yr)
ED	=	Exposure duration (yr)
AT_n	=	Averaging time, noncarcinogens (d)
AT_c	=	Averaging time, carcinogens (d)
C_s	=	Concentration of contaminant in soil (mg/kg)
PEF	=	Particulate emission factor (m^3/kg)
K_{ap}^{pt}	=	Plant-air partition coefficient for particle-bound contaminant (m^3 air/kg plant fresh mass)
K_{ap}^{gs}	=	Plant-air partition coefficient for gas-phase contaminant (m^3 air/kg plant fresh mass)
VF_s	=	Volatilization factor for soil (m^3/kg)
f_{pa}	=	Volume fraction of plant tissue in air (unitless)
f_{pw}	=	Volume fraction of plant tissue in water (unitless)
f_{pl}	=	Volume fraction of plant tissue lipid (unitless)
R	=	Universal gas constant (8.31 Pa·m^3/mol·K)
T	=	Temperature (K)
H	=	Henry's law constant (Pa·m^3/mol)
K_{ps}	=	Plant-soil partition coefficient from root-zone soil to above-ground plant parts (kg soil/kg plant fresh mass)
$K_{ps(roots)}$	=	Plant-soil partition coefficient from root-zone soil to roots (kg soil/kg plant fresh mass)
K_{ow}	=	*n*-Octanol-water partition coefficient (unitless)
K_{oc}	=	Organic carbon-water partition coefficient (L water/kg carbon)
f_{oc}	=	Fraction of organic carbon in soil (g/g)

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 kilogram 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). This modeling approach to estimate concentrations in fruits and vegetables introduces additional uncertainty into the assessment. If this is an important pathway, DEQ recommends measuring chemical concentrations in the food crops directly.

Briggs et al. (1982) 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, that remove this contaminated surface layer, are anticipated to 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 ($0 \le F_v \le 1$, $0 \le F_{fr} \le 1$) is an estimate of that fraction of total vegetables consumed which are grown within the contaminated site (exposure unit). To start, you should assume that all vegetables consumed are grown onsite in contaminated soils, so that F_v or F_{fr} = 1. If you have site-specific information on sources of vegetables, an appropriate distribution may be used to model a range of F_v or F_{fr} values.

A.5.3 Animal Products

Carcinogens

$$ADD_m = \frac{\left(IRQ_{adj-m} \cdot C_m \cdot F_m + IRQ_{adj-dp} \cdot C_{dp} \cdot F_{dp} + IRQ_{adj-e} \cdot C_e \cdot F_e\right) \cdot CF_{gg} \cdot EF_{dy}}{AT_c}$$

Noncarcinogens

$$ADD_m = \frac{\left(C_m \cdot IRM_m \cdot F_m + C_{dp} \cdot IRM_{dp} \cdot F_{dp} + C_e \cdot IRM_e \cdot F_e\right) \cdot CF_{gg} \cdot EF_{dy} \cdot ED}{AT_n}$$

$$C_{dp} = C_s \cdot 7.9 \times 10^{-9} \cdot K_{ow}$$

$$C_m = C_s \cdot 2.5 \times 10^{-8} \cdot K_{ow}$$

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

where:

ADD_m = Average daily dose from ingestion of homegrown meat, milk, and eggs (mg/[kg·d])
C_m = Contaminant concentration in meat (mg/kg)
C_{dp} = Contaminant concentration in dairy products (mg/kg)
C_e = Contaminant concentration in eggs (mg/kg)
$IRQ_{adj\text{-}m}$ = Age-adjusted food ingestion factor - meat ([g·yr]/[kg·d])
$IRQ_{adj\text{-}dp}$ = Age-adjusted food ingestion factor - dairy products ([g·yr]/[kg·d])
$IRQ_{adj\text{-}e}$ = Age-adjusted food ingestion factor - eggs ([g·yr]/[kg·d])
IRM_m = Meat ingestion rate (g/[kg·d]) [child or adult]
IRM_{dp} = Dairy product ingestion rate (g/[kg·d]) [child or adult]
IRM_e = Egg ingestion rate (g/[kg·d]) [child or adult]
F_m = Fraction of meat from site (unitless)
F_{dp} = Fraction of dairy products from site (unitless)
F_e = Fraction of eggs from site (unitless)
CF_{gg} = Conversion factor (kg/10^3 g)
EF_{dy} = Exposure frequency (d/yr) [child or adult]
ED = Exposure duration (yr) [child or adult]
AT_n = Averaging time, noncarcinogens (d)
AT_c = Averaging time, carcinogens (d)
C_s = Concentration of contaminant in soil (mg/kg)
K_{ow} = *n*-Octanol-water partition coefficient (unitless)

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 $2.5 \times 10^{-8} \times K_{ow}$ (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 $7.9 \times 10^{-9} \times K_{ow}$ (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 $1.6 \times 10^{-6} \times K_{ow}$ (see also Travis & Arms 1988). For chemicals with high K_{ow} (i.e., log $K_{ow} > 6$), this relationship does not hold, and will result in inaccurate estimates of uptake to animal flesh. In these cases, this relationship should not be used (see Birak et. al. 2001).

The fraction of meat, milk, or eggs from site parameter ($0 \leq F_{m,dp,e} \leq 1$) is an estimate of that fraction of total meat, milk, or eggs consumed which are raised within the contaminated site (exposure unit). To start, you should assume that all meat, milk, or eggs consumed are produced onsite in contaminated soils, so that $F_{m,dp,e} = 1$.

Default values for various meat, egg, and dairy food item consumption rates are not provided as these rates are anticipated to be highly site specific. Information with which to begin constructing site-specific rates can be found in the Exposure Factors Handbook (EPA, 1997).

A.5.4 Human Milk Consumption

People who are exposed to bioaccumulative compounds such as PCBs, polybrominated diphenylethers (PBDEs), dioxins/furans, and DDTs, accumulate chemical concentrations in lipid tissue. These bioaccumulating chemicals will be present in breast milk and may pose a threat to breastfeeding infants. EPA guidance is available to evaluate this exposure pathway (EPA 1998b and EPA 2005a). Because occupational and residential exposure includes exposure to women who may became mothers in the future, human milk ingestion is a relevant pathway for all sites with bioaccumulating compounds. For PCBs, exposure to the mother prior to breastfeeding will be the most important exposure pathway. Appendix D provides detailed information on the breastfeeding pathway and how to address it in a risk assessment. Use the infant risk adjustment factors in Table D-3 to calculate risks to infants based on risks to occupational and residential receptors.

Note that an evaluation of breastfeeding risks is complicated by the known substantial health benefits of breastfeeding, as discussed in Appendix D. Including the breastfeeding exposure pathway in risk assessments is important to ensure that our environment is protective of infants. However, it is critical to understand that calculated risks are not intended to advise women about whether or not to breastfeed their infants. Rather, the purpose is to inform site clean-up managers so that they can make decisions that will lead to decreased exposure to women, and ultimately lower concentrations of contaminants in the milk women produce for their infants. Calculated risks to infants support public health actions that encourage women to limit their own exposure to environmental contaminants so that their infants can receive the optimal health benefits from breastfeeding.

A.6 Exposure Frequency Considerations

Dose is a function of concentration and 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 occur and for how long. In some cases, such as a person staying in a home and not going to a separate workplace, it may be possible for an individual to experience one exposure scenario for 24 hours per day (ET = 24 hours/day) and 365 days per year (EF = 365 days/year). This is therefore an appropriate screening scenario. For other individuals, this type of exposure may be unlikely. Under special circumstances, DEQ may accept modified factors for current exposure conditions. For example, if an area of an industrial site is only visited once per week, this can be taken into account by modifying the EF. However, with no guarantee that the exposure frequency will always apply, DEQ will use the default EF for potential future exposure.

Note that 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 day (IRS), EF days per year. It is not appropriate to modify this rate for exposure occurring less than 24 hr/day because the values were derived based on typical contact throughout a day.

THIS PAGE LEFT BLANK

Table A-1
Default Reasonable Maximum Exposure (RME) Values

Table A-1. Default Reasonable Maximum Exposure Values

Exposure Factor (Symbol)	Units	Residential		Urban Residential		Non-Residential		
		Child	Adult	Child	Adult	Occupational	Construction	Excavation
Averaging time, carcinogens (AT_c)	yr d	70 a 25550	70 a 25550	70 a 25550	70 a 25550	70 a 25550	70 a 25550	70 a 25550
Averaging time, noncarcinogens (AT_n)	yr	= ED a	= ED a	= ED a	=ED a	=ED a	=ED c	=ED c
Body weight (BW)	kg	15 c	70 a	15 c	70 a	70 a	70 a	70 a
Exposure duration (ED)	yr	6 c	24 c	6 c	5 b	25 c	1 c	1 c
Exposure frequency (EF_{dy})								
general (includes soil contact and bathing)	d/yr	350 c	350 c	175/350 f	175/350 f	250 c	250 c	9 g
swimming	d/yr	150 b1	150 b1	150 b1	150 b1	NA d	NA	NA
Event frequency (EF_{evd})								
soil contact	ev/d	1 c	1 c	1 c	1 c	NA	2 e	2 e
bathing and swimming	ev/d	1 b1	1 b1	1 b1	1 b1	NA	NA	NA
groundwater contact	ev/d	NA	NA	NA	NA	NA	2 e	2 e
Event time (t_{event})								
bathing	hr/ev	0.25 b1	0.25 b1	0.25 b1	0.25 b1	NA	NA	NA
swimming	hr/ev	1 b1	1 b1	1 b1	1 b1	NA	NA	NA
groundwater contact	hr/ev	NA	NA	NA	NA	NA	2 e	2 e
Skin surface area (SA)								
soil contact	cm^2	2800 c	5700 c	2800 c	5700 c	3300 c	3300 c	3300 c
water contact (bath/swim)	cm^2	7300 b2	23000 b3	7300 b2	23000 b3	NA	NA	NA

Table A-1. Default Reasonable Maximum Exposure Values

Exposure Factor (Symbol)	Units	Residential		Urban Residential		Non-Residential		
		Child	Adult	Child	Adult	Occupational	Construction	Excavation
groundwater	cm^2	NA	NA	NA	NA	NA	5700 b	5700 b
Soil ingestion rate (IRS)	mg/d	200 a	100 a	200 a	100 a	100 c	330 c	330 c
Soil to skin adherence factor (AF)	mg/ cm^2-ev	0.2 i4	0.07 i1	0.2 i4	0.07 i1	0.1 i2	0.3 i3	0.3 i3
Water ingestion rate (IRW)	L/d	1.5 b4	2.0 a	1.5 b4	2.0 a	0.7 h	NA	NA
Incidental Water ingestion Rate (IRWi)	L/hr	0.05 a	0.05 a	0.05 a	0.05 a	NA	NA	NA

Notes:

(a) EPA 1989 and EPA 2008
(b) EPA 1997
  (1) Table 15-18, upper values
  (2) Table 6-6, 90th percentile male child, 3-4 years old
  (3) Table 6-14
  (4) Table 3-30, 90th percentile, 3-5 year olds
  (5) Table 5-23, mean for 3-5 year olds
(c) EPA 2002b
(d) NA = not applicable, or applicable on a site-specific basis.
(e) Assumes that direct soil contact or groundwater contact activities occur twice a day (morning, afternoon) for a total of four hours per day.
(f) Assumed equal to ½ residential value for soil contact, equal to residential value for other pathways (vapor inhalation, water ingestion).
(g) The value of 9 days per year was based on standard dimensions for a residential excavation site from DEQ (1997a) and construction worker excavation statistics from EPA and *Means Heavy Construction Cost Data, 8th Annual Edition, R.S. Means Company, Inc., Kingston, MA*.
(h) EPA 1997, adjusted for time spent at work (8 hours / 24 hours).
(i) EPA 2004
  (1) Exhibit 3-3, mean for residential adult gardener
  (2) Exhibit 3-3, mean for commercial gardener used to represent upper end commercial exposure
  (3) Exhibit 3-3, 95th percentile construction worker
  (4) Exhibit 3-3, mean for children playing

Table A-2
Default Central Tendency Exposure (CTE) Values

Table A-2. Default Central Tendency Exposure Values

Exposure Factor (Symbol)	Units	Residential		Urban Residential		Non-Residential		
		Child	Adult	Child	Adult	Occupational	Construction	Excavation
Averaging time, carcinogens (AT_c)	yr d	70 a 25500	70 a 25500	70 a 25500	70 a 25500	70 a 25500	70 a 25500	70 a 25500
Averaging time, noncarcinogens (AT_n)	yr	= ED a	= ED a	= ED a	= ED a	= ED a	= ED c	= ED c
Body weight (BW)	kg	15 c	70 a	15 c	70 a	70 a	70 a	70 a
Exposure duration (ED)	yr	6 c	3 c	4	4	6 c	0.5 c	1 c
Exposure frequency (EF_{dy})								
general (includes soil contact and bathing)	d/yr	350 c	350 c	175 f	175 f	250 c	250 c	9 g
swimming	d/yr	5 b1	5 b1	5 b1	5 b1	NA	NA	NA
Event frequency (EF_{evd})								
soil contact	ev/d	1 c	1 c	1 c	1 c	NA	2 e	2 e
bathing and swimming	ev/d	1 b1	1 b1	1 b1	1 b1	NA	NA	NA
groundwater contact	ev/d	NA	NA	NA	NA	NA	2 e	2 e
Event time (t_{event})								
bathing	hr/ev	0.16 b1	0.16 b1	0.16 b1	0.16 b1	NA	NA	NA
swimming	hr/ev	0.5 b1	0.5 b1	0.5 b1	0.5 b1	NA	NA	NA
groundwater contact	hr/ev	NA	NA	NA	NA	NA	2 e	2 e
Skin surface area (SA)								
soil contact	cm^2	2800 c	5700 c	2800 c	5700 c	3300 c	3300 c	3200 c
water contact (bath/swim)	cm^2	6600 b2	20000 b3	6600 b2	20000 b3	NA	NA	NA
groundwater	cm^2	NA	NA	NA	NA	NA	5700 b	5700 b

Table A-2. Default Central Tendency Exposure Values

Exposure Factor (Symbol)	Units	Residential		Urban Residential		Non-Residential		
		Child	Adult	Child	Adult	Occupational	Construction	Excavation
Soil ingestion rate (IRS)	mg/d	100 a	50 a	100 a	50 a	50 c	100 c	100 c
Soil to skin adherence factor (AF)	mg/c m^2-ev	0.04 i4	0.01 i1	0.04 i4	0.01 i1	0.02 i2	0.1 i3	0.1 i3
Water ingestion rate (IRW)	L/d	0.87 b4	1.4 b4	0.87 b4	1.4 b4	0.5 h	NA	NA
Incidental Water Ingestion Rate	L/hr	0.05 a	0.05 a	0.05 a	0.05 a	NA	NA	NA

Notes:

(a) EPA 1989 and EPA 2008
(b) EPA 1997
  (1) Table 15-18, central values
  (2) Table 6-6, 50th percentile male child, 3-4 years old
  (3) Table 6-14, central tendency
  (4) Table 3-30, mean, 3-5 year olds and adults
  (5) Table 5-23, mean for 3-5 year olds
(c) EPA 2002b
(d) NA = not applicable, or applicable on a site-specific basis.
(e) Assumes that direct soil contact or groundwater contact activities occur twice a day (morning, afternoon) for a total of four hours per day.
(f) Assumed equal to ½ residential value for soil contact, equal to residential value for other pathways (vapor inhalation, water ingestion).
(g) The value of 9 days per year was based on standard dimensions for a residential excavation site from DEQ (1997a) and construction worker excavation statistics from EPA and *Means Heavy Construction Cost Data, 8th Annual Edition, R.S. Means Company, Inc., Kingston, MA*.
(h) EPA 1997, adjusted for time spent at work (8 hours / 24 hours).
(i) EPA 2004
  (1) Exhibit 3-3, mean for residential groundskeeper
  (2) Exhibit 3-3, mean for commercial groundskeeper
  (3) Exhibit 3-3, mean for construction worker
  (4) Exhibit 3-3, mean for children playing in dry soil

Table A-3
Default Values for Exposure Model Variables

Exposure Model Variable	Symbol	Units	Value
Contaminant concentration in soil	C_s	mg/kg	---
Conversion factor	CF_{km}	kg/mg	0.000001
Fraction of soil contaminated	F_s	unitless	1
Contaminant concentration in water	C_w	mg/L	---
Fraction of water contaminated	F_w	unitless	1
Contaminant concentration in vegetables and fruit	C_v	mg/kg	---
Fraction of vegetables from site	F_v	unitless	1
Fraction of fruit from site	F_{fr}	unitless	1
Vegetable correction factor	V_{cf}	unitless	0.01
Conversion factor	C_{gg}	kg/g	0.001
Particulate emission factor	PEF	m^3/kg	1.32×10^9
Plant-air partition coefficient for particle-bound contaminant	K_{ap}^{pt}	m^3/kg	3300
Plant-air partition coefficient for gas-phase contaminant	K_{ap}^{gs}	m^3/kg	---
Volatilization factor for soil	VF_s	m^3/kg	---
Volume fraction of plant tissue in air	f_{pa}	unitless	0.5
Volume fraction of plant tissue in water	f_{pw}	unitless	0.4
Volume fraction of plant tissue lipid	f_{pl}	unitless	0.01
Universal gas constant	R	$Pa \cdot m^3/mol \cdot K$	8.31
Temperature	T	K	---
Henry's law constant	H	$Pa \cdot m^3/mol$	---
Plant-soil partition coefficient from root-zone soil to above-ground plant parts	K_{ps}	unitless	---
Plant-soil partition coefficient from root-zone soil to roots	$K_{ps(roots)}$	unitless	---
n-Octanol-water partition coefficient	K_{ow}	unitless	---
Organic carbon-water partition coefficient	K_{oc}	L/kg	---
Fraction of organic carbon in soil	f_{oc}	unitless	0.006
Contaminant concentration in meat	C_m	mg/kg	---
Contaminant concentration in dairy products	C_{dp}	mg/kg	---
Contaminant concentration in eggs	C_e	mg/kg	---
Fraction of meat from site	F_m	unitless	1
Fraction of dairy products from site	F_{dp}	unitless	1
Fraction of eggs from site	F_e	unitless	1
Concentration of contaminant in finfish	C_f	mg/kg	---
Fraction of finfish from site	F_f	unitless	1
Contaminant-specific bioconcentration factor for finfish	BCF_f	L/kg	---

Exposure Model Variable	Symbol	Units	Value
Dermal absorption factor	DAF	unitless	---
Absorbed dose per soil contact event	DA_{soil}	mg/cm^2-event	---
Absorbed dose per water contact event	DA_{water}	mg/cm^2-event	---
Conversion factor	CF_{cl}	L/cm^3	0.001
Lag time	τ	hr	---
Time to reach steady-state	t*	hr	---
Relative contribution of permeability coefficients	B	unitless	---
Dermal permeability coefficient	K_p	cm/hr	---
Contaminant-specific molecular weight	MW	g/mol	---
Inverse of mean concentration at center of source area; from Exhibit 11 in EPA (1996)	Q/C	$g/m^2 \cdot s$ / kg/m^3	68.81
Apparent diffusivity	D_a	cm^2/s	---
Exposure interval	I_e	s	9.5×10^8
Dry soil bulk density	ρ_b	g/cm^3	1.5
Air-filled soil porosity	θ_a	unitless	---
Diffusivity in air	D_i	cm^2/s	---
Water-filled soil porosity	θ_w	unitless	0.15
Diffusivity in water	D_w	cm^2/s	---
Total soil porosity	*n*	unitless	---
Soil-water partition coefficient	K_d	cm^3/g	---
Soil particle density	ρ_s	g/cm^3	2.65
Respirable particulate concentration	C_{air}	mg/m^3	---
Respirable fraction	R_f	$g/m^2 \cdot hr$	0.036
Fraction of vegetative cover	G	unitless	0.5
Mean annual wind speed	U_m	m/s	4.69
Erosion threshold wind speed	U_t	m/s	11.32
Friction velocity	U_f	m/s	0.5
Function dependent of U_t/U_m	F(x)	unitless	0.194

Table A-4
Default Dermal Absorption Factors

Chemical	Default DAF
Arsenic	0.03
Cadmium	0.001
Chlordane	0.04
2,4-Dichlorophenoxyacetic acid (2,4-D)	0.05
DDT	0.03
TCDD (TOC ≤ 10%)	0.03
TCDD (TOC > 10%)	0.001
Lindane	0.04
Benzo[a]pyrene and other PAHs	0.13
Aroclor 1242, Aroclor 1254 and other PCBs	0.14
Pentachlorophenol	0.25
Semi-volatile organic compounds	0.1

Notes:

DAF = dermal absorption factor

Default DAF values are not provided for general volatile organic compounds or classes of inorganic chemicals. VOCs will tend to volatilize from soil on skin, and should be accounted for by inhalation routes. For inorganics, the speciation of the chemical is critical to dermal absorption, and there are too little data to determine a reasonable default value.

Source: EPA 2004

APPENDIX B

Incorporating Early-Life Exposure

B.1 Introduction

This appendix covers the evaluation of early-life exposure for certain compounds. At Cleanup Program sites, the chemicals for which incorporation of early-life exposure will be necessary are vinyl chloride and the carcinogenic polycyclic aromatic hydrocarbons. These chemicals are discussed separately below. In the future as more information becomes available, early-life exposure may need to be considered for other chemicals. DEQ's RBC table (DEQ 2003) incorporates the early-life exposure evaluation in the residential scenario.

B.2 Background

In March 2005, EPA issued new *Guidelines for Carcinogenic Risk Assessment* (EPA 2005b), updating the 1986 guidelines and 1999 interim final guidelines. Also included was *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (EPA 2005c). In a 29 March 2005 memorandum on Application of New Cancer Guidelines, then Acting Administrator Stephen Johnson stated that:

1. For all[1] newly initiated carcinogenicity risk assessments, the Cancer Guidelines and Supplemental Guidance will be used from this point forward;
2. For risk assessments currently being performed, the guidance will be used on a case-by-case basis; and
3. For completed risk assessments, reassessments may be performed on a case-by-case basis when a new decision is required that needs to be supported by an updated risk assessment. Until that time, the current completed risk assessment will continue to be considered scientifically sound based on the guidance used when the assessment was completed.

DEQ typically follows EPA risk assessment guidance. In the *Supplemental Guidance,* EPA concluded that some chemicals (carcinogens acting by a mutagenic mode of action) have a greater cancer impact if exposure occurs during childhood. You should evaluate early-life exposure for the relevant chemicals. Where applicable, evaluate cancer risk using different adjusted potency factors for three life stages (0 – 2 years, 2 – 16 years, and adult). If early-life exposure is not of concern, you can use one potency factor for two life stages (child 0 – 6 years, and adult).

EPA created workgroups to provide additional information on how to implement the Supplemental Guidance, and provide consistency. One outcome of the workgroups is an EPA memorandum clarifying which chemicals should be evaluated for early-life exposure (EPA 2006b). The list of chemicals is provided in Table B-1. EPA determined that vinyl chloride should continue to be evaluated using a specific procedure for evaluating early-life exposure. This procedure was included in DEQ's 2003 revisions to the risk-based decision making (RBDM) guidance, and is presented in Section B.3. The only other early-life chemicals of interest to the Cleanup Program are the carcinogenic

[1] Emphasis in original.

polycyclic aromatic hydrocarbons (cPAHs). The procedure for evaluating cPAHs is presented in Section B.4.

EPA uses an evaluation of early-life exposure in calculating their regional screening values (EPA 2010c). DEQ uses the same early-life exposure values recommended by EPA.

DEQ requires the consideration of early-life exposure on all human health risk assessments for sites where the relevant exposure scenarios include residential site use or other uses where childhood exposure is likely. Therefore, we developed risk-based concentrations (RBCs) for vinyl chloride and cPAHs to include early-life exposure. In the future, if EPA includes additional chemicals for early-life consideration, the appropriate RBC screening values can be calculated using the RBC spreadsheet. To activate the early-life option, download the RBC workbook, go to the Toxicological Data sheet, and change the Early Life designation from "n" to "y". Then click the Recalculate button to automatically calculate RBCs based on early-life exposure.

B.3 Calculation of RBCs for Vinyl Chloride

EPA's Integrated Risk Information System (IRIS) report for vinyl chloride includes two derivations of cancer slope factors, one based on the linearized multistage (LMS) procedure, and one based on the LED_{10} approach. The LED_{10} is the lower 95% limit on a dose that is estimated to cause a 10% response. The results are similar, but the LMS approach is used here because that is what is currently used by the EPA regions. For vinyl chloride, LMS values are slightly less conservative than slope factors based on the LED_{10} approach.

Slope factors are provided separately for lifetime exposure as an adult, and lifetime exposure beginning from birth. The values differ by a factor of 2. Following the precise method recommended by EPA (2000b), only the adult slope factor is needed. The oral slope factor is 0.72 $(mg/kg/day)^{-1}$.

Unit risk factors are provided in IRIS for inhalation exposure. These are 4.4×10^{-6} risk per $\mu g/m^3$ for adult exposure, and 8.8×10^{-6} risk per $\mu g/m^3$ for adult/child exposure. Rather than use the child/adult unit risk factors, DEQ uses EPA's more precise approach to incorporate early-life exposure. An example is presented below for the calculation of the inhalation RBC for vinyl chloride. You can use similar concepts in a forward risk assessment.

Incorporation of Early-Life Exposure in Derivation of RBCs for Vinyl Chloride

The standard residential exposure scenario considers exposure to both children and adults. Ingestion rates, inhalation rates, and other factors are different for children and adults, and these differences are taken into account when calculating residential RBCs. Because the exposure calculations for carcinogens and noncarcinogens have different underlying assumptions, the method used to account for these adult/child differences depends on whether the contaminant is classified as a carcinogen or a noncarcinogen.

For carcinogens that are not evaluated for early-life exposure, residential exposure is calculated for the first 30 years of life, and then averaged over a 70-year lifetime. To

account for differences in exposures to children and adults over the 30-year period, weighted averages are calculated for the exposure factors assuming 6 years of exposure at a childhood exposure rate, and 24 years of exposure at an adult exposure rate. This general approach cannot be used for vinyl chloride.

For vinyl chloride, EPA concludes that because the effects of early-life exposure are qualitatively and quantitatively different from those of later exposures, it is not appropriate to prorate early-life exposures as if they were received at a proportionately lesser rate over a full lifetime. This feature of vinyl chloride toxicity must be considered in the derivation of RBCs for residential exposure.

Following EPA's example, early-life exposure is estimated assuming a lifetime of exposure using the lower (adult) slope factor. For an exposure scenario involving both early-life and adult exposure, the early-life exposure (which is a single value and is not pro-rated for reduced exposure time) is added to exposure as an adult (which can be pro-rated). "Adult" exposure in this case can also include child exposure beyond the initial early-life exposure.

To show explicitly how early-life and adult exposure are incorporated, the following shows how the site-specific water ingestion RBC was calculated:

$$RBC_{air} = \frac{ARL_c \cdot AT_c \cdot 365\ days/yr}{EF_r \cdot ED \cdot IUR} \cdot 10^3 \mu g/mg$$

Where:
RBC_{air} = Risk based concentration for inhalation of air ($\mu g/m^3$)
ARL = Acceptable risk level (10^{-6})
ATc = Averaging time, carcinogens (70 years)
EF_r = Exposure frequency, residential (350 days/year)
ED = Exposure duration (yr)
IUR = Inhalation unit risk (risk per $\mu g/m^3$)

Early-life exposure was assumed to be equivalent to a lifetime of adult exposure (70 years).

$$RBC_{early-life} = \frac{10^{-6} \cdot 70\ yr \cdot 365\ days/yr}{350\ days/yr \cdot 70yr \cdot 4.4x10^{-6}\ (\mu g/m^3)^{-1}}$$

$$= 0.24\ \mu g/m^3$$

For a residential adult, the RBC is:

$$RBC_{adult} = \frac{10^{-6} \cdot 70\ yr \cdot 365\ days/yr}{350\ days/yr \cdot 30yr \cdot 4.4x10^{-6}\ (\mu g/m^3)^{-1}}$$

$$= 0.55\ \mu g/m^3$$

The RBC for combined exposure as a child and adult is calculated using the following relationship:

$$\frac{1}{RBC_{early-life/adult}} = \frac{1}{RBC_{early-life}} + \frac{1}{RBC_{adult}}$$

$$RBC_{early-life/adult} = \frac{1}{\frac{1}{RBC_{early-life}} + \frac{1}{RBC_{adult}}}$$

$$RBC_{early-life/adult} = \frac{1}{\frac{1}{0.24} + \frac{1}{0.55}}$$

$$= 0.17\ \mu g/m^3$$

A similar approach was used to calculate vinyl chloride RBCs for the standard exposure scenarios. The same approach should be used in performing a risk assessment for vinyl chloride. Alternatively, risk values can be calculated from RBCs as discussed in Section 3.4.1.

B.4 Early-Life Risk Assessment Calculations for cPAHs

Risk assessments for cPAHs (and other carcinogens acting by a mutagenic mode of action, excluding vinyl chloride discussed above) include a term called an age dependent adjustment factor (ADAF) to account for increased carcinogenic potency during early life stages. For ages up to 2 years, the ADAF is 10, indicating a ten-fold increase in carcinogenic potency during this period. For ages from 2 years to 16 years, the ADAF is 3. For ages 16 years and older, the ADAF is 1. Using ADAFs, the differences in potency are incorporated by a factor separate from the slope factor, so only one cancer slope factor is needed. Risk assessments for carcinogens that do not act by a mutagenic mode of action should be conducted using the slope factor without adjustments for age.

In developing exposure parameters for children, EPA decided that it would be more accurate to divide the 2- to 16-year-old stage into two stages (2 to 6 years, and 6 to 16 years). Both stages have the same ADAF value, but body weights, skin surface area, and intakes differ.

The incorporation of ADAFs is best included in the calculation of age-adjusted intake factors. These factors are used both in forward risk assessments and calculations of RBCs. Equations for age-adjusted intake factors are presented in Appendix A and in DEQ's RBDM guidance (DEQ 2003). For carcinogens acting by a mutagenic mode of action, these equations should be modified as follows:

Soil ingestion

$$IRS_{adj} = \frac{ED_2 \cdot IRS_2 \cdot ADAF_2}{BW_2} + \frac{ED_6 \cdot IRS_6 \cdot ADAF_6}{BW_6} + \frac{ED_{16} \cdot IRS_{16} \cdot ADAF_{16}}{BW_{16}} + \frac{ED_{adult} \cdot IRS_{adult} \cdot ADAF_{adult}}{BW_{adult}}$$

Water ingestion

$$IRW_{adj} = \frac{ED_2 \cdot IRW_2 \cdot ADAF_2}{BW_2} + \frac{ED_6 \cdot IRW_6 \cdot ADAF_6}{BW_6} + \frac{ED_{16} \cdot IRW_{16} \cdot ADAF_{16}}{BW_{16}} + \frac{ED_{adult} \cdot IRW_{adult} \cdot ADAF_{adult}}{BW_{adult}}$$

Soil dermal contact

$$SFS_{adj} = \frac{ED_2 \cdot AF_2 \cdot SA_2 \cdot ADAF_2}{BW_2} + \frac{ED_6 \cdot AF_6 \cdot SA_6 \cdot ADAF_6}{BW_6} + \frac{ED_{16} \cdot AF_{16} \cdot SA_{16} \cdot ADAF_{16}}{BW_{16}} + \frac{ED_{adult} \cdot AF_{adult} \cdot SA_{adult} \cdot ADAF_{adult}}{BW_{adult}}$$

Water dermal contact

$$SAS_{adj} = \frac{ED_2 \cdot SA_2 \cdot ADAF_2}{BW_2} + \frac{ED_6 \cdot SA_6 \cdot ADAF_6}{BW_6} + \frac{ED_{16} \cdot SA_{16} \cdot ADAF_{16}}{BW_{16}} + \frac{ED_{adult} \cdot SA_{adult} \cdot ADAF_{adult}}{BW_{adult}}$$

Inhalation

$$ED_{adj} = ED_2\ ADAF_2 + ED_6\ ADAF_6 + ED_{16}\ ADAF_{16} + ED_{adult}\ ADAF_{adult}$$

where:

IRS_{adj} = Age-adjusted incidental soil ingestion factor ([mg·yr]/[kg·d])
IRW_{adj} = Age-adjusted water ingestion factor ([L·yr]/[kg·d])
SFS_{adj} = Age-adjusted soil dermal contact factor ([mg·yr]/[kg·event])
SAS_{adj} = Age-adjusted water dermal contact factor ([cm^2·yr]/kg)
ED_{adj} = Age-adjusted exposure duration (yr)
$ADAF_2$ = Age-dependent Adjustment Factor, child 0 to <2 years old (unitless)
$ADAF_6$ = Age-dependent Adjustment Factor, child 2 to <6 years old (unitless)
$ADAF_{16}$ = Age-dependent Adjustment Factor, child 6 to <16 years old (unitless)
$ADAF_{adult}$ = Age-dependent Adjustment Factor, adult (unitless)
ED_2 = Exposure duration, child 0 to <2 years old (yr)
ED_6 = Exposure duration, child 2 to <6 years old (yr)
ED_{16} = Exposure duration, child 6 to <16 years old (yr)
ED_{adult} = Exposure duration, adult (yr)
BW_2 = Body weight, child 0 to <2 years old (kg)
BW_6 = Body weight, child 2 to <6 years old (kg)
BW_{16} = Body weight, child 6 to <16 years old (kg)
BW_{adult} = Body weight, adult (kg)
IRS_2 = Soil ingestion rate, child 0 to <2 years old (mg/d)
IRS_6 = Soil ingestion rate, child 2 to <6 years old (mg/d)

IRS_{16} = Soil ingestion rate, child 6 to <16 years old (mg/d)
IRS_{adult} = Soil ingestion rate, adult (mg/d)
IRW_2 = Water ingestion rate, child 0 to <2 years old (L/d)
IRW_6 = Water ingestion rate, child 2 to <6 years old (L/d)
IRW_{16} = Water ingestion rate, child 6 to <16 years old (L/d)
IRW_{adult} = Water ingestion rate, adult (L/d)
AF_2 = Adherence factor, child 0 to <2 years old ($mg/cm^2 \cdot event$)
AF_6 = Adherence factor, child 2 to <6 years old ($mg/cm^2 \cdot event$)
AF_{16} = Adherence factor, child 6 to <16 years old ($mg/cm^2 \cdot event$)
AF_{adult} = Adherence factor, adult ($mg/cm^2 \cdot event$)
SA_2 = Exposed skin surface area, 0 to <2 years old child (cm^2)
SA_6 = Exposed skin surface area, 2 to <6 years old child (cm^2)
SA_{16} = Exposed skin surface area, child 6 to <16 years old (cm^2)
SA_{adult} = Exposed skin surface area, adult (cm^2)

The default parameter values are shown in Table B-2.

Table B-1
Chemicals Determined by EPA to be Carcinogens Having a Mutagenic Mode of Action

Chemical[a]	Chemical Abstract Service Registration Number
Chemicals typically found at Cleanup Program sites	
Benz[a]anthracene[b]	56-55-3
Benzo[b]fluoranthene[b]	205-99-2
Benzo[k]fluoranthene[b]	207-08-9
Benzo[a]pyrene	50-32-8
Chrysene[b]	218-01-9
Dibenz[a,h]anthracene	53-70-3
Dimethylbenz[a]anthracene	57-97-6
Indeno[1,2,3-cd]pyrene[b]	193-39-5
Vinyl chloride[c]	75-01-4
Chemicals not typically found at Cleanup Program sites	
Benzidine	92-87-5
N-Nitrosodiethylamine (diethylnitrosamine)	55-18-5
N-Methyl-N-nitrosomethanamine (dimethylnitrosamine)	62-75-9
N-Nitrosoethylurea (ethylnitrosourea)	759-73-9
3-Methylcholanthrene	56-49-5
N-Nitroso-N-methylurea (methylnitrosourea)	684-93-5
Safrole	94-59-7
Urethane (ethyl carbamate)	51-79-6

Notes:

a) Source: EPA 2006.
b) Although not explicitly included in EPA's list, EPA states that carcinogenic PAHs with a relative potency factor relating the toxicity to the slope factor for benzo[a]pyrene should also be evaluated for early-life exposure.
c) Early-life exposure to vinyl chloride should be evaluated using the chemical-specific analysis presented in EPA's Integrated Risk Information System (www.epa.gov/iris), and not by using the general ADAF approach. The appropriate IRIS method is used to calculate RBC values for vinyl chloride.

Table B-2
Default Parameter Values for Early-Life Exposure

Parameter	<2 Years Old	2 to <6 Years Old	6 to <16 Years Old	Adult
ADAF (unitless)[a]	10	3	3	1
ED (yr)[b] residential[c]	2	4	10	14
urban residential[d]	2	4	0	5
BW (kg)[b]	15	15	70	70
IRS (mg/d)[b]	200	200	100	100
IRW (L/d)[b]	1	1	2	2
AF ($mg/cm^2 \cdot event$)[e]	0.2	0.2	0.07	0.07
SA (cm^2)[b]	2,800	2,800	5,700	5,700
IRA (m^3/d)[b]	10	10	20	20

Notes:

a) Age-dependent adjustment factor (ADAF) values taken from EPA 2005.
b) Exposure values taken from *Exposure Factors Handbook* (EPA 1997) and *Risk Assessment Guidance for Superfund* (EPA 1989).
   ED = exposure duration BW = body weight
   IRS = ingestion rate, soil IRW = ingestion rate, water
   AF = adherence factor SA = skin surface area
   IRA = inhalation rate, air
c) The standard residential default exposure duration is 30 years. For adult ED, DEQ uses 30 years minus the time exposed as a child (6 years), for a total of 14 years as an adult.
d) The standard urban residential default exposure duration is 11 years. DEQ's assumption is 6 years as a child, 5 years as an adult.
e) Adherence factors taken from EPA 2004.

APPENDIX C

Incorporating Inhalation Exposure for RBC calculations

C.1 Introduction

To make DEQ's RBDM guidance consistent with EPA's revised inhalation guidance (EPA 2009), we needed to revise the RBCs equations to incorporate toxicity based on concentrations instead of dose. This appendix presents the modifications that have been incorporated into the generic RBC equations. For consistency with the RBDM guidance, we will use the same numbering system for the equations.

As described on Page B-23 of DEQ's RBDM guidance document (DEQ 2003), the basic equations for dose-based calculations to estimate risk-based concentrations are:

$$RBC_{air} = \left[\frac{AT \cdot BW}{ED \cdot EF \cdot IRA}\right] \cdot \frac{Hazard\,Quotient}{(1/RfD)} \qquad \text{[old RBDM B-66]}$$

$$RBC_{air} = \left[\frac{AT \cdot BW}{ED \cdot EF \cdot IRA}\right] \cdot \frac{Risk}{(Slope\,Factor)} \qquad \text{[old B-67]}$$

The modified RAGs part F approach (EPA 2009) is concentration-based rather than dose-based, and eliminates both inhalation rates and body weights. Therefore, the above two equations were modified to remove inhalation rates and body weights. In addition, slope factors are replaced with inhalation unit risks (IUR) in units of $(\mu g/m^3)^{-1}$, and reference doses are replaced with reference concentrations (RfCs) in units of (mg/m^3). The corresponding general equations are now:

$$RBC_{air} = \left[\frac{AT}{ED \cdot EF}\right] \cdot \frac{Hazard\,Quotient}{(1/RfC)} \qquad \text{[B-66]}$$

$$RBC_{air} = \left[\frac{AT}{ED \cdot EF}\right] \cdot \frac{Risk}{IUR} \qquad \text{[B-67]}$$

Where:

AT	=	Averaging time (days)
ED	=	Exposure duration (years)
EF	=	Exposure frequency (days/year)
RfC	=	Reference concentration ($\mu g/m^3$)
IUR	=	Inhalation unit risk ($\mu g/m^3$)
Hazard Quotient	=	1 (acceptable level for noncarcinogens)
Risk	=	1×10^{-6} (acceptable level for individual carcinogens)

The equations that require modification are presented here with their equation numbers

corresponding to their presentation in the RBDM guidance document (DEQ 2003).

C.2 Age-Adjusted Inhalation Factor (Air)

Revisions to equations B-72 and B-73, shown in Block 1 below, replace age-adjusted inhalation factors with inhalation unit risk and reference concentrations. Because ingestion and dermal exposure are not affected, equations B-74 through B-79 are still needed and remain unchanged.

Block 1

Residential

$$IFAadj_r = \frac{ED_{cr} \cdot IRA_c}{BW_c} + \frac{(ED_r - ED_{cr}) \cdot IRA_r}{BW_a} \quad [B\text{-}72]$$

Urban Residential

$$IFAadj_u = \frac{ED_{cu} \cdot IRA_c}{BW_c} + \frac{(ED_u - ED_{cu}) \cdot IRA_u}{BW_a} \quad [B\text{-}73]$$

where:

*IFAadj	=	Age-adjusted inhalation factor for air ([m^3-yr]/[kg-d])
*IFSadj	=	Age-adjusted ingestion factor for soils ([mg-yr]/[kg-d])
*IFWadj	=	Age-adjusted ingestion factor for water ([L-yr]/[kg-d])
*SFSadj	=	Age-adjusted skin contact factor for soils ([mg-yr]/[kg-d])
*AF	=	Adherence factor (mg/cm^2-d)
*BW	=	Body weight (kg)
*ED	=	Exposure duration (yr)
*IRA	=	Inhalation rate (m^3/d)
*IRS	=	Soil ingestion rate (mg/d)
*IRW	=	Water ingestion rate (L/d)
*SA	=	Skin surface contact area (cm^2)

* The subscripts on these parameters in the equations refer to the following: a = adult; c = child; cr = residential child; cu = urban residential child; r = residential; and u = urban residential.

C.3 Air RBCs – Three Phase Calculations

Blocks 2 and 3 below present the modified equations for three-phase calculations for air RBCs.

Block 2

Residential - Carcinogens

$$RBC_{air}\ (\mu g/m^3) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr \cdot (24hr/d)}{ET_r \cdot ED_r \cdot EF_r \cdot IUR} \quad [B\text{-}80]$$

Residential – Noncarcinogens

$$RBC_{air}\ (\mu g/m^3) = \frac{ARL_n \cdot AT_{nc} \cdot (24hr/day) \cdot 365\ d/yr \cdot 10^3\ \mu g/mg}{ET_r \cdot ED_r \cdot EF_r \cdot (1/RfC)} \quad [B\text{-}81]$$

Urban Residential – Carcinogens

$$RBC_{air}\ (\mu g/m^3) = \frac{ARL_c \cdot (24hr/day) \cdot AT_c \cdot 365\ d/yr}{ET_u \cdot ED_u \cdot EF_r \cdot IUR} \quad [B\text{-}82]$$

Urban Residential – Noncarcinogens

$$RBC_{air}\ (\mu g/m^3) = \frac{ARL_n \cdot AT_{nc} \cdot 365\ d/yr \cdot (24hr/day) \cdot 10^3\ \mu g/mg}{ET_u \cdot ED_u \cdot EF_r \cdot (1/RfC)} \quad [B\text{-}83]$$

Occupational - Carcinogens

$$RBC_{air}\ (\mu g/m^3) = \frac{ARL_c \cdot (24hr/1day) \cdot AT_c \cdot 365\ d/yr}{ET_o \cdot ED_o \cdot EF_o \cdot\ IUR} \quad [B\text{-}84]$$

Occupational - Noncarcinogens

$$RBC_{air}\ (\mu g/m^3) = \frac{ARL_n \cdot (24hr/day) AT_{no} \cdot 365\ d/yr \cdot 10^3\ \mu g/mg}{ET_o \cdot ED_o \cdot EF_o \cdot (1/RfC)} \quad [B\text{-}85]$$

where:

RBC_{air}	=	Risk-based air concentration ($\mu g/m^3$)
ARL_c	=	Acceptable Risk Level for Carcinogens (unitless)
ARL_n	=	Acceptable Risk Level for Noncarcinogens (unitless)
AT_c	=	Averaging time – carcinogens (yr)
*AT_n	=	Averaging time – noncarcinogens (yr)
*ED_r	=	Exposure duration (yr)-residential
ED_u	=	Exposure duration (yr)-urban residential
*EF	=	Exposure frequency (d/yr)
*ET_r	=	Exposure Time (hours/day) - residential
*ET_u	=	Exposure Time (hours/day) - urban residential
*ET_o	=	Exposure Time (hours/day) - occupational
RfC	=	Noncancer reference concentration (mg/m^3)
IUR	=	inhalation Unit Risk ($\mu g/m^3)^{-1}$

* The subscripts on these parameters in the equations refer to the following: o = occupational; r = residential and; u=urban residential.

Block 3

Residential - Carcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr}{EF_r[(\frac{IFSadj_r \cdot SF_o}{10^6 mg/kg}) + (\frac{SFSadj_r \cdot RAF_d \cdot SF_o}{10^6 mg/kg}) + (\frac{ED_r \cdot ET_r \cdot (1d/24hr) \cdot IUR \cdot TF_{ss}}{10^{-3} mg/\mu g})]} \quad [B\text{-}100]$$

Residential - Noncarcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_n \cdot AT_{nc} \cdot 365\ d/yr \cdot BW_c}{ED_{cr} \cdot EF_r \cdot [(\frac{IRS_{cr}}{RfD_o \cdot 10^6 mg/kg}) + (\frac{SA_c \cdot AF_c \cdot RAF_d}{RfD_o \cdot 10^6 mg/kg}) + (\frac{ET_r \cdot (1d/24hr) \cdot BW_c \cdot TF_{ss}}{RfC})]} \quad [B\text{-}101]$$

Urban Residential – Carcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr}{EF_u \cdot [(\frac{IFSadj_u \cdot SF_o}{10^6 mg/kg}) + (\frac{SFSadj_u \cdot RAF_d \cdot SF_o}{10^6 mg/kg}) + (\frac{ED_u \cdot ET_u \cdot (1d/24hr) \cdot IUR \cdot TF_{ss}}{10^{-3} mg/\mu g})]} \quad [B\text{-}102]$$

Urban Residential – Noncarcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_n \cdot AT_{nc} \cdot 365\ d/yr \cdot BW_c}{ED_{cu} \cdot EF_u \cdot [(\frac{IRS_{cu}}{RfD_o \cdot 10^6 mg/kg}) + (\frac{SA_c \cdot AF_c \cdot RAF_d}{RfD_o \cdot 10^6 mg/kg}) + (\frac{ET_u \cdot (1d/24hr) \cdot BW_c \cdot TF_{ss}}{RfC})]} \quad [B\text{-}103]$$

Occupational – Carcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr \cdot BW_a}{ED_o \cdot EF_o \cdot [(\frac{IRS_o \cdot SF_o}{10^6 mg/kg}) + (\frac{SA_o \cdot AF_o \cdot RAF_d \cdot SF_o}{10^6 mg/kg}) + (\frac{ET_o \cdot (1d/24hr) \cdot IUR \cdot TF_{ss} \cdot BW_a)}{10^{-3} mg/\mu g})]} \quad [B\text{-}104]$$

Occupational – Noncarcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_n \cdot AT_{no} \cdot 365\ d/yr \cdot BW_a}{ED_o \cdot EF_o \cdot [(\frac{IRS_o}{RfD_o \cdot 10^6 mg/kg}) + (\frac{SA_o \cdot AF_o \cdot RAF_d}{RfD_o \cdot 10^6 mg/kg}) + (\frac{ET_o \cdot (1d/24hr) \cdot BW_a \cdot TF_{ss}}{RfC})]} \quad [B\text{-}105]$$

Construction Worker – Carcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr \cdot BW_a}{ED_k \cdot EF_k \cdot [(\frac{IRS_k \cdot SF_o}{10^6 mg/kg}) + (\frac{SA_k \cdot AF_k \cdot RAF_d \cdot SF_o}{10^6 mg/kg}) + (\frac{ET_k \cdot (1d/24hr) \cdot IUR \cdot TF_{ss} \cdot BW_a)}{10^{-3} mg/\mu g})]} \quad [B\text{-}106]$$

Construction Worker – Noncarcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_n \cdot AT_{nk} \cdot 365\ d/yr \cdot BW_a}{ED_k \cdot EF_k \cdot [(\frac{IRS_k}{RfD_o \cdot 10^6 mg/kg}) + (\frac{SA_k \cdot AF_{nk} \cdot RAF_d}{RfD_o \cdot 10^6 mg/kg}) + (\frac{ET_k \cdot (1d/24hr) \cdot BW_a \cdot TF_{ss}}{RfC})]} \quad [B\text{-}107]$$

Excavation Worker – Carcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr \cdot BW_a}{ED_e \cdot EF_e \cdot [(\frac{IRS_e \cdot SF_o}{10^6 mg/kg}) + (\frac{SA_e \cdot AF_e \cdot RAF_d \cdot SF_o}{10^6 mg/kg}) + (\frac{ET_e \cdot (1d/24hr) \cdot IUR \cdot TF_{ss} \cdot BW_a)}{10^{-3} mg/\mu g})]} \quad [B\text{-}108]$$

Excavation Worker – Noncarcinogens

$$RBC_{ss}\ (mg/kg) = \frac{ARL_n \cdot AT_{nc} \cdot 365\ d/yr \cdot BW_a}{ED_e \cdot EF_e \cdot [(\frac{IRS_e}{RfD_o \cdot 10^6 mg/kg}) + (\frac{SA_e \cdot AF_e \cdot RAF_d}{RfD_o \cdot 10^6 mg/kg}) + (\frac{ET_e \cdot (1d/24hr) \cdot BW_a \cdot TF_{ss}}{RfC})]} \quad [B\text{-}109]$$

where:

RBC_{ss}	=	Risk-based concentration for soils (mg/kg)
*AF	=	Adherence factor (mg/cm^2-d)
ARL_c	=	Acceptable Risk Level for Carcinogens (unitless)
ARL_n	=	Acceptable Risk Level for Noncarcinogens (unitless)
*AT_n	=	Averaging time – noncarcinogens (yr)
AT_c	=	Averaging time – carcinogens (yr)
*BW	=	Body weight (kg)
*ED	=	Exposure duration (yr)
*EF	=	Exposure frequency (d/yr)
*ET	=	Exposure time (hours/d)
*IFSadj	=	Age-adjusted ingestion factor for soils ([mg-yr]/[kg-d])
*IRS	=	Ingestion rate for soils (mg/d)
*SFSadj	=	Age-adjusted dermal contact factor for soils ([mg-yr]/[kg])
RAF_d	=	Relative dermal absorption factor (unitless)
RfC	=	Reference concentration – inhaled (mg/m^3)
IUR	=	Inhalation Unit Risk $(\mu g/m^3)^{-1}$
*SA	=	Skin surface contact area (cm^2)
SF_o	=	Cancer slope factor – oral $(mg/kg\text{-}d)^{-1}$
TF_{ss}	=	Transport factor for surface soil, which is either a volatilization factor (VFss, kg/m^3), or a particulate emission factor (PEF, kg/m^3)

* The subscripts on these parameters in the equations refer to the following: a = adult; c = child; cr = residential child; cu = urban residential child; e = excavation worker; k = construction worker; o = occupational; r = residential; and u = urban residential.

C.3 Air RBCs – Four-Phase Calculations

Block 4 presents the modified four-phase equations used to calculated air RBCs.

Block 4

Residential - Noncarcinogens

$$HQ^i_{ss} = \frac{C^i_{soil} \cdot ED_{cr} \cdot EF_r}{AT_{nc} \cdot 365\ d/yr \cdot BW_c} \cdot [(\frac{IRS_{cr}}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{SA_c \cdot AF_c \cdot RAF_d}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{ET_r \cdot (1d/24hr) \cdot BW_c \cdot TF^i_{ss}}{RfC})] \quad [B\text{-}110]$$

Urban Residential – Noncarcinogens

$$HQ^i_{ss} = \frac{C^i_{soil} \cdot ED_{cu} \cdot EF_u}{AT_{nc} \cdot 365\ d/yr \cdot BW_c} \cdot [(\frac{IRS_{cu}}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{SA_c \cdot AF_c \cdot RAF_d}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{ET_u \cdot (1d/24hr) \cdot BW_c \cdot TF^i_{ss}}{RfC})] \quad [B\text{-}111]$$

Occupational – Noncarcinogens

$$HQ^i_{ss} = \frac{C^i_{soil} \cdot ED_o \cdot EF_o}{AT_{nc} \cdot 365\ d/yr \cdot BW_a} \cdot [(\frac{IRS_o}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{SA_o \cdot AF_o \cdot RAF_d}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{ET_o \cdot (1d/24hr) \cdot BW_a \cdot TF^i_{ss}}{RfC})] \quad [B\text{-}112]$$

Construction Worker – Noncarcinogens

$$HQ^i_{ss} = \frac{C^i_{soil} \cdot ED_k \cdot EF_k}{AT_{nc} \cdot 365\ d/yr \cdot BW_a} \cdot [(\frac{IRS_k}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{SA_k \cdot AF_k \cdot RAF_d}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{ET_k \cdot (1d/24hr) \cdot BW_a \cdot TF^i_{ss}}{RfC})] \quad [B\text{-}113]$$

Excavation Worker – Noncarcinogens

$$HQ^i_{ss} = \frac{C^i_{soil} \cdot ED_e \cdot EF_e}{AT_{nc} \cdot 365\ d/yr \cdot BW_a} \cdot [(\frac{IRS_e}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{SA_e \cdot AF_e \cdot RAF_d}{RfD^i_o \cdot 10^6 mg/kg}) + (\frac{ET_e \cdot (1d/24hr) \cdot BW_a \cdot TF^i_{ss}}{RfC})] \quad [B\text{-}114]$$

where:

HQ^i_{ss}	=	Hazard quotient for TPH fraction "i" – surface soils (unitless)
C^i_{soil}	=	The concentration of TPH fraction "i" in surface soil (mg/kg)
ET	=	Exposure Time (hours/day)

All other terms are as previously defined.

C.4 Groundwater RBCs – Three-Phase Calculations

Blocks 5 and 6 present the modified equations for three-phase groundwater RBCs.

Block 5

Residential – Carcinogens

$$RBC_{tw}(\mu g/L) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr}{EF_r \cdot [(IFWadj_r \cdot SF_o \cdot 10^{-3} mg/\mu g) + (ET_r \cdot (1d/24hr) \cdot ED_r \cdot VF_w \cdot IUR)]} \quad [B\text{-}143]$$

Residential – Noncarcinogens

$$RBC_{tw}(\mu g/L) = \frac{ARL_n \cdot AT_{nr} \cdot 365\ d/yr \cdot BW_a}{ED_r \cdot EF_r \cdot [(IRW_a / RfD_o) + (ET_r \cdot (1d/24hr) \cdot VF_w \cdot (1/RfC) \cdot BW_a)]} \cdot 10^3 \mu g/mg \quad [B\text{-}144]$$

Urban Residential – Carcinogens

$$RBC_{tw}(\mu g/L) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr}{EF_u \cdot [(IFWadj_u \cdot SF_o \cdot 10^{-3} mg/\mu g) + (ET_u \cdot (1d/24hr) \cdot ED_u \cdot VF_w \cdot IUR)]} \quad [B\text{-}145]$$

Urban Residential – Noncarcinogens

$$RBC_{tw}(\mu g/L) = \frac{ARL_n \cdot AT_{nu} \cdot 365\ d/yr \cdot BW_a}{ED_u \cdot EF_u \cdot [(IRW_a / RfD_o) + (ET_u \cdot (1d/24hr) \cdot VF_w \cdot (1/RfC) \cdot BW_a)]} \cdot 10^3 \mu g/mg \quad [B\text{-}146]$$

Occupational – Carcinogens

$$RBC_{tw}(\mu g/L) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr \cdot BW_a}{ED_o \cdot EF_o \cdot [(IRW_a \cdot SF_o \cdot 10^{-3} mg/\mu g) + (ET_o \cdot (1d/24hr) \cdot VF_w \cdot IUR \cdot BW_a)]} \quad [B\text{-}147]$$

Occupational – Noncarcinogens

$$RBC_{tw}(\mu g/L) = \frac{ARL_n \cdot AT_{no} \cdot 365\ d/yr \cdot BW_a}{ED_o \cdot EF_o \cdot [(IRW_a / RfD_o) + (ET_o \cdot (1d/24hr) \cdot VF_w \cdot (1/RfC) \cdot BW_a)]} \cdot 10^3 \mu g/mg \quad [B\text{-}148]$$

where:

RBC_{tw}	=	Risk-Based Concentration for Ingestion & Volatiles in Tap Water (µg/L)
ARL_c	=	Acceptable risk level – carcinogens (unitless)
ARL_n	=	Acceptable risk level – noncarcinogens (unitless)
AT_c	=	Averaging time – carcinogens (yr)
*AT_n	=	Averaging time – noncarcinogens (yr)
*BW	=	Body weight (kg)
*ED	=	Exposure duration (yr)
*EF	=	Exposure frequency (d/yr)
ET	=	Exposure Time (hrs/day)
*IFWadj	=	Age-adjusted ingestion factor for water ([L-yr]/[kg-d])
IRW_a	=	Water ingestion rate – adult (L/d)
RfC	=	Reference concentration – inhaled (mg/m^3)
RfD_o	=	Reference dose – oral (mg/kg-d)
IUR	=	Inhalation Unit Risk ($\mu g/m^3$) $^{-1}$
SF_o	=	Cancer slope factor – oral $(mg/kg\text{-}d)^{-1}$
VF_w	=	Volatilization factor from tap water (L/m^3)

* The subscripts on these parameters in the equations refer to the following: a = adult; r = residential; and u = urban residential, o = occupational.

Block 6

Construction and Excavation Worker – Carcinogens

$$RBC_{we}\ (\mu g/L) = \frac{ARL_c \cdot AT_c \cdot 365\ d/yr \cdot BW_a}{ED_e \cdot EF_e \cdot (ET_e \cdot (1d/24hr) \cdot VF_{we} \cdot IUR \cdot BW_a) + (DA_w \cdot EvF_w \cdot SA_w \cdot SF_o \cdot 10^{-3} mg/\mu g)]} \quad [B\text{-}161]$$

Construction and Excavation Worker – Noncarcinogens

$$RBC_{we}\ (\mu g/L) = \frac{ARL_n \cdot AT_{ne} \cdot 365\ d/yr \cdot BW_a}{ED_e \cdot EF_e \cdot [(\frac{ET_e \cdot (1d/24hr) \cdot VF_{we} \cdot BW_a}{RfC}) + (\frac{DA_w \cdot EvF_w \cdot SA_w}{RfD_o})]} \cdot 10^3 \mu g/mg \quad [B\text{-}162]$$

where:

RBC_{we}	=	Risk-Based Concentration for Excavation or Construction Worker Exposure to Groundwater (µg/L)
ARL_c	=	Acceptable risk level – carcinogens (unitless)
ARL_n	=	Acceptable risk level – noncarcinogens (unitless)
AT_c	=	Averaging time – carcinogens (yr)
$*AT_{ne}$	=	Averaging time – noncarcinogens (yr)
BW_a	=	Body weight (kg)
DA_w	=	Dermal absorption factor for groundwater (L/cm^2-event)
$*ED_e$	=	Exposure duration (yr)
$*EF_e$	=	Exposure frequency (d/yr)
$*ET_e$	=	Exposure Time (hrs/d)
EvF_w	=	Event frequency for groundwater contact (event/d)
RfC	=	Reference concentration – inhaled (mg/m^3)
RfD_o	=	Reference dose – oral (mg/kg-d)
SA_w	=	Skin surface contact area to groundwater (cm^2)
IUR	=	Inhalation Unit Risk – inhaled $(\mu g/m^3)^{-1}$
SF_o	=	Cancer slope factor – oral $(mg/kg\text{-}d)^{-1}$
VF_{we}	=	Volatilization factor for water in an excavation (L/m^3)

* In this scenario, the subscript “e” can represent either the excavation OR construction worker depending on the situation being modeled.

APPENDIX D

Evaluating Potential Risks to Infants from Consuming Human Milk

INTRODUCTION

This appendix presents a standard approach for evaluating potential risks to infants from consumption of human milk. The approach was developed in conjunction with EPA Region 10 risk assessors. The following is consistent with the approach recommended by EPA, and with Oregon Administrative Rules.

Including the breastfeeding exposure pathway in risk assessments is important to ensure that our environment is protective of infants. However, it is critical to understand that risks calculated using the model presented here are not intended to advise women about whether or not to breastfeed their infants. Rather, the purpose is to inform site clean-up managers so that they can make decisions that will lead to decreased exposure to women, and ultimately lower concentrations of contaminants in the milk women produce for their infants. Calculated risks to infants support public health actions that encourage women to limit their own exposure to environmental contaminants so that their infants can receive the optimal health benefits from breastfeeding.

DEQ evaluated the feasibility of conducting a risk assessment based on exposure to human milk using EPA's *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (MPE Guidance, EPA 1998), *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities* (Combustion Guidance, EPA 2005), *Exposure Factors Handbook* (EPA 1997), *Child-Specific Exposure Factors Handbook* (EPA 2008), and examples from other hazardous waste sites. We determined that it is feasible to include exposure to human milk in human health risk assessments, and that this is an important exposure pathway for bioaccumulating chemicals. Risk assessments for sites contaminated with polychlorinated biphenyls (PCBs), chlorinated dibenzenzo-*p*-dioxins (CDDs) and chlorinated dibenzofurans (CDFs), and/or DDT compounds (including DDE and DDD) should include potential risks from the breastfeeding pathway. Although DEQ considers these chemicals to be the most important contributors to risk from this pathway, we may require that you include other bioaccumulating chemicals released at the facility in your risk assessment.

To assist risk assessors in incorporating the human milk consumption pathway into the human health risk assessment, we prepared this appendix to present relevant exposure and risk equations, and exposure and toxicity parameters (summarized in Tables D-1 and D-2). We include example calculations using total PCB Aroclors to show how the various equations in EPA's MPE guidance can be modified to focus on fish consumption, one of the most important exposure pathways for bioaccumulating chemicals. However, the general calculations for infants apply to every exposure pathway that the mother may experience prior to and during breastfeeding. Actual risk assessments should include the exposure pathways relevant for the site. Risk assessments should also include all relevant chemicals, such as total PCBs (from Aroclors or congeners), 2,3,7,8-TCDD equivalents (from chlorinated dibenzo-*p*-dioxins, chlorinated dibenzofurans, and dioxin-like PCB congeners, evaluating each chemical class separately and collectively as the

sum of all dioxin-like chemicals), and DDT and its degradation products.

The methods discussed in this appendix are appropriate for lipophilic compounds that are present mostly in milkfat rather than the aqueous phase. Equations are also available for compounds that will partition more to the aqueous portion of human milk. Specific guidance for hydrophilic compounds is not presented in this document.

We include infant risk adjustment factors in this appendix (Table D-3) so that all the risk calculations for human milk ingestion do not need to be included in risk assessments. Instead, the potential risk to infants can be calculated based on the exposure to the mother, which should already be evaluated for relevant exposure pathways. This will simplify incorporation of the breastfeeding pathway into the risk assessment.

Generally, risk assessments are limited to an evaluation of risk, and do not consider comparative risks or benefits. For example, eating fish is health beneficial compared with eating other animal protein. Public health agencies commonly address the health tradeoffs of eating contaminated fish, but the issue is not typically discussed in a Superfund risk assessment. For breastfeeding, however, the benefits to infants are so substantial that we consider it appropriate to discuss the issue in the risk assessment report. Risk assessments that include this pathway should state clearly that information in the risk assessment should not be used or interpreted to advise women about whether or not they should breastfeed their infants. The Oregon Office of Environmental Public Health (OEPH) has prepared a statement that presents the risks and benefits of consuming contaminated human milk. DEQ recommends that information presented in this statement (Appendix D, Attachment 2) be included with risk assessments that include the breastfeeding pathway.

PROPOSED RISK ASSESSMENT APPROACH

Exposure Assessment

We mainly relied on the equations presented in EPA's MPE document (EPA 1998), using a fish ingestion scenario for the mother to illustrate the approach for calculating risk to the breastfeeding infant. The key concept is that the concentration of a chemical in milk can be calculated from the long-term body burden in the mother. This is consistent with the information presented in the Agency for Toxic Substances Disease Registry (ATSDR) *Toxicological Profile for Polychlorinated Biphenyls.(ARSDR 2000)*

Average Daily Dose to Mother

We start with the average daily intake of chemicals to the mother. The general Equation D-1, modified from Table C-1-4 of the Combustion Guidance (EPA 2005), is then further modified to consider absorbed dose (Equation D-2) so that body burden in the mother can be estimated.

Equation D-1
General Equation for Average Daily Dose to Mother

$$ADD_{mat} = \frac{C \times CR \times EF \times ED}{AT \times BW_{mat}}$$

Where:
ADD_{mat} = Average daily dose of chemical to mother (mg/kg/day)
C = Chemical concentration in medium of interest (e.g., mg/kg in soil)
CR = Contact rate (e.g., mg/day)
EF = Exposure frequency (e.g., days/year)
ED = Exposure duration (e.g., years)
AT = Averaging time (70 years = 25550 days for carcinogens, ED for noncarcinogens)
BW_{mat} = Body weight of mother (66 kg for average adult female aged 15 to 44)

Equation D-2
Daily Absorbed Intake to Mother

$$DAI_{mat} = ADD_{mat} \times AE_{mat}$$

Where:
DAI_{mat} = Daily maternal absorbed intake of chemical (mg/kg/day)
ADD_{mat} = Average daily dose of chemical to mother (mg/kg/day); Equation D-1
AE_{mat} = Absorption efficiency of chemical (fraction)

For the example exposure evaluated in this appendix, we consider exposure to the mother by consumption of fish contaminated with PCBs:

Equation D-3
Average Daily Dose to Mother from Fish Ingestion

$$ADD_{mat} = \frac{C_{fish} \times IR_{fish} \times CF \times F_{fish}}{BW_{mat}}$$

Where:
ADD_{mat} = Average daily dose of chemical to mother (mg/kg/day)
C_{fish} = Chemical concentration in fish (mg/kg)
IR_{fish} = Ingestion rate of fish for mother (standard default rate of 17.5 g/day)
CF = Conversion factor (0.001 kg/g)
F_{fish} = Fraction of fish contaminated (1)
BW_{mat} = Body weight of mother (66 kg)

The ingestion rate used in the example is the default rate used by EPA in developing ambient water quality criteria. The fish ingestion rate is an annualized rate (*i.e.*, it includes the assumption that fish are eaten throughout the year), so exposure frequency, exposure duration, and averaging time are not included in the equation. Loss of chemicals during cooking, which has been considered at other sites, is not included in EPA's Combustion Guidance. However, cooking loss can be addressed in the uncertainty section of a risk assessment. For body weight, we follow recent EPA guidance and consider it appropriate to use the average female weight aged 15 years to 44 years of 66 kg (EPA 2009). Prior default values were 70 kg (average adult weight) used in EPA's Combustion Guidance (EPA 2005), and 60 kg (average female weight) used in EPA's MPE Guidance (EPA 1998).

For this example, the calculations are performed assuming a total PCB concentration of 1 mg/kg in whole-body tissue. This value is for illustration only, and to develop a relative risk ratio. An actual risk assessment should use chemical concentrations appropriate for the various species of fish sampled.

$$ADD_{mat} = 1 \text{ mg/kg} \times 17.5 \text{ g/day} \times 0.001 \text{ kg/g} \times 1 \ / \ 66 \text{ kg} = 0.000265 \text{ mg/kg/day}$$

Assuming an absorption efficiency of 1 (AE_{mat} in Equation D-2), DAI_{mat} is equal to ADD_{mat}.

Equation D-3 is appropriate for evaluating non-carcinogenic effects to the mother. For an excess lifetime cancer risk calculation for the mother, the equation would be modified to incorporate exposure duration (typically 30 years) and averaging time (lifetime of 70 years). The resulting average daily dose would be reduced by a factor of 30/70, or 0.43 times the ADD calculated above, resulting in a lifetime dose of 0.00011 mg/kg/day.

Chemical Concentration in Milkfat

EPA found that dietary intake of PCBs by the mother during pregnancy and lactation is only weakly correlated with PCB concentrations in human milk. The more important determinant is long-term consumption of PCBs. The following simplified equation is an initial approximation to calculate the PCB concentration in milkfat.

Equation D-4
Steady State Chemical Concentration in Milkfat

$$C_{milkfat,ss} = \frac{DAI_{mat} \times h \times f_f}{\ln(2) \times f_{fm}}$$

Where:

$C_{milkfat,ss}$	=	Chemical concentration in milkfat (mg/kg-lipid)
DAI_{mat}	=	Daily absorbed chemical intake to mother (mg/kg/day)
h	=	Half-life of chemical (days)
f_f	=	Fraction of absorbed PCB stored in fat (0.9)
f_{fm}	=	Fraction of mother's weight that is fat (0.3 kg-lipidBW/kg-totalBW)

Equation D-4 was modified from Table C-3-1 of the Combustion Guidance,(EPA 2005) and is consistent with equations 1 through 3(b) in Section 3.4.4.2 of the ATSDR *Toxicological Profile* (ATSDR 2000). The equation is for steady-state conditions, assuming that maternal intake occurs over a time-period greater than the chemical half-life. Another important assumption is that chemical concentrations in human milk reflect the maternal body burden.

For the PCB example, the assumed half-life is 7 years (2555 days) following the Combustion Guidance (EPA 2005). The calculated PCB concentration in milkfat is:

$$C_{milkfat,ss} = \frac{0.000265 \text{ mg/kg-totalBW/day} \times 2555 \text{ days} \times 0.9}{0.693 \times 0.3 \text{ (kg-lipidBW/kg-totalBW)}}$$

= 2.9 mg/kg-lipid

In EPA's MPE guidance, a more complex equation is used to explicitly consider two factors relevant to average milkfat concentrations over the time that an infant is breastfeeding. The modified approach avoids overestimating human milk concentrations by: 1) estimating maternal body burden at the start of breastfeeding taking into account the exposure period of the mother relative to the metabolic halflife of the chemical, rather than assuming steady-state conditions, and 2) accounting for the reduction in chemical concentrations over time during the breastfeeding period as chemical mass is transferred from mother to infant. Both of these factors are incorporated into the following equation (taken from Equation 9-4 in MPE guidance, EPA 1998).

Equation D-5
Average Chemical Concentration in Milkfat During Breastfeeding

$$C_{milkfat,avg} = \frac{DAI_{mat} \times f_f}{k_{elim} \times f_{fm}} \times \left[\frac{k_{elim}}{k_{elac}} + \frac{1}{k_{elac} \times t_{bf}} \left(1 - e^{-k_{elim} t_{pn}} - \frac{k_{elim}}{k_{elac}} \right) (1 - e^{-k_{elac} t_{bf}}) \right]$$

Where:

$C_{milkfat,avg}$ = Average chemical concentration in milkfat (mg/kg-lipid)
DAI_{mat} = Daily absorbed chemical intake to mother (mg/kg/day); Equation D-2
f_f = Fraction of ingested chemical stored in fat (0.9)
f_{fm} = Fraction of mother's weight that is fat (0.3 kg-lipidBW/kg-totalBW)
k_{elim} = Elimination rate constant for non-lactating women ($days^{-1}$); Equation D-6
k_{elac} = Elimination rate constant for chemical in milkfat during breast feeding ($days^{-1}$); Equation D-7
t_{pn} = Duration of mother's exposure prior to breastfeeding (25 years = 9125 days)
t_{bf} = Duration of breastfeeding (365 days)

The elimination rate constants k_{elim} and k_{elac} are calculated as shown below.

Equation D-6
Biological Elimination Rate Constant for Women Prior to Breastfeeding

$$k_{elim} = \frac{\ln(2)}{h}$$

Where:

k_{elim} = Elimination rate constant for non-lactating women
h = Half-life of chemical (days)

Equation D-7
Biological Elimination Rate Constant for Women who are Breastfeeding

$$k_{elac} = k_{elim} + \frac{IR_{milk} \times f_f \times f_{mbm}}{f_{fm} \times BW_{mat}}$$

Where:

k_{elac} = Elimination rate constant for chemical in milkfat during breast feeding ($days^{-1}$)
K_{elim} = Elimination rate constant for non-lactating women ($days^{-1}$)
IR_{milk} = Ingestion rate of milk over duration of breast feeding (0.98 kg/day)
BW_{mat} = Body weight (66 kg)
f_f = Fraction of ingested chemical stored in fat (0.9)
f_{mbm} = Fraction of fat in mother's milk (0.04)
f_{fm} = Fraction of mother's weight that is fat (0.3 kg-lipidBW/kg-totalBW)

The first term in Equation D-5 ($DAI_{mat}\ f_f / k_{elim}\ f_m$) is equivalent to Equation D-4, given the definition of k_{elim} in Equation D-6. The other terms in Equation D-5 account for chemical concentrations in the mother prior to steady-state concentrations (if the duration of exposure to the mother is less than about three times the elimination halflife), and also chemical losses during breastfeeding.

In the PCB example, with an assumed half-life of 7 years (2555 days), the calculated rate constants are:

$$k_{elim} = \ln(2) / 2555 \text{ days} = 0.693 / 2555 \text{ days} = 0.00027 \text{ (days)}^{-1}$$

$$k_{elac} = 0.00027 + (0.98 \times 0.9 \times 0.04)/(0.3 \times 66) = 0.0021 \text{ (days)}^{-1}$$

Using these values, the average concentration of PCB in milkfat over a year of breastfeeding calculated from Equation D-5 is:

$$C_{milkfat} = 2.0 \text{ mg/kg-lipid}$$

This more realistic value is similar to the value of 2.9 mg/kg-lipid calculated using simplified Equation D-4.

In order to explicitly evaluate the magnitude of effect of accounting for 1) steady state concentrations not being reached, and 2) chemical loses during breastfeeding, we present a simplified version of Equation D-5 in Attachment 1. For the PCB example, we show that the steady-state maternal concentration (Equation D-4) is reduced by a factor of 0.86 to account for non-steady state conditions, and another factor of 0.70 to account for mass loses during breastfeeding, to calculate a mean PCB concentration in human milk during breastfeeding.

Average Daily Dose to Infant from Milkfat

For lipophilic chemicals such as PCB, the majority of the chemical will be partitioned in milkfat, so once we have calculated $C_{milkfat}$ we can calculate the average daily dose to a breastfeeding infant using the following equation (modified from Equation 9-1 of the MPE Guidance):

Equation D-8
Average Daily Dose to Breastfeeding Infant

$$ADD_{inf} = \frac{C_{milkfat} \times f_{mbm} \times CR_{milk} \times ED_{inf}}{BW_{inf} \times AT}$$

Where:

ADD_{inf} = Average daily dose to breast-feeding infant (mg/kg/day)
$C_{milkfat}$ = Concentration of chemical in milk fat (mg/kg-lipid)
f_{mbm} = Fraction of fat in mother's milk (0.04)
CR_{milk} = Consumption rate of human milk (0.98 kg/day)
ED_{inf} = Exposure duration of breastfeeding infant (365 days)
BW_{inf} = Average body weight of infant over ED (7.8 kg)
AT = Averaging time: carcinogen (70 years x 365 days/year)
non-carcinogen (AT = ED)

Equation 9-1 in EPA's MPE guidance (EPA 1998) includes two additional terms: f_{am} for fraction absorbed by the mother (called AE_{mat} in this document), and f_{ai} for fraction of ingested chemical absorbed by the infant. Because AE_{mat} is already included in the calculation of $C_{milkfat}$, it is inappropriate to also include the term f_{am} in Equation D-8. The term f_{ai} was included in the original calculations because infant body burden was used to evaluate risk (Smith 1987). However, for typical evaluations of risk using either reference doses or slope factors, we use administered dose, not absorbed dose. Therefore, the term f_{ai} is not included in the calculation of dose to infant in Equation D-8. Note, however, that Equation D-2 does use the fraction of chemical absorbed by the mother (AE_{mat}). This is because $C_{milkfat}$ is based on body burden in the mother, which is dependent on absorbed dose, not administered dose.

For the PCB example, the ADD values for carcinogenic and noncarcinogenic effects are calculated as follows:

$ADD_{ca\text{-}infant}$ = $\frac{\text{2.0 mg/kg-lipid x 0.04 kg-lipid/kg-milk x 0.98 kg/day x 365 day/yr}}{\text{7.8 kg x 1 x 70 yr x 365 day/yr}}$

= 0.00014 mg/kg/day

$ADD_{nc\text{-}infant}$ = $\frac{\text{2.0 mg/kg-lipid x 0.04 kg-lipid/kg-milk x 0.98 kg/day x 365 day/yr}}{\text{7.8 kg x 1 x 365 day/yr}}$

= 0.010 mg/kg/day

Aqueous Component

EPA's MPE Guidance includes an approach for evaluating infant exposure to chemicals in the aqueous phase of human milk. The approach presented here in DEQ guidance is limited to lipophilic compounds that are preferentially present in milkfat. We do not anticipate expanding the evaluation to include hydrophilic compounds at this time. If we determine that this pathway should be included in a risk assessment, the equations in Chapter 9 of EPA's MPE Guidance can be used (EPA 1998).

Mercury Exposure

EPA has extended their MPE Guidance with additional information on how to evaluate exposure to mercury in human milk (EPA 2009). They refer to a study that showed that methyl mercury concentrations in blood of nursing infants were similar to methyl mercury concentrations in the mothers' blood. EPA concludes that, for the purpose of a risk assessment, the dose of mercury to the infant can be assumed to be approximately equal to the dose to the mother during breastfeeding. EPA's guidance on risk assessment methodologies can be referred to if mercury exposure is a relevant pathway (EPA 2009).

Toxicity Assessment

EPA's hierarchy for selecting toxicity factors is to first obtain factors from EPA's Integrated Risk Information System (IRIS). For example, the cancer slope factor for PCBs presented in IRIS is 2 $(mg/kg/day)^{-1}$. This value is applied to total PCBs.

The reference dose (RfD) for PCBs in IRIS is 2×10^{-5} mg/kg/day for chronic exposure (7 years to lifetime). There is no RfD for subchronic exposure in IRIS. Following EPA's hierarchy for toxicity values, the next source for a subchronic RfD is ATSDR. The ATSDR minimal risk level (MRL, comparable to an RfD) is 3×10^{-5} mg/kg/day for intermediate-duration (subchronic) oral exposure to PCBs. ATSDR defines intermediate-duration exposure as two weeks to one year. The intermediate-duration MRL was derived from a study on monkeys that approximated exposure during breastfeeding using a mixture of PCB congeners typically found in human milk. For this reason, it is a better indicator of toxicity than the chronic RfD (which is equal to the chronic MRL).

Table D-1 provides the slope factors, chronic reference doses, and where available, subchronic reference doses for bioaccumulating chemicals.

Risk Characterization

Calculated Cancer Risk to Infants

Using the standard risk characterization equations, excess lifetime cancer risk and non-cancer hazards are calculated separately. Excess lifetime cancer risk is approximated by:

Equation D-9
Calculation of Excess Lifetime Cancer Risk to Breastfeeding Infant

$$ELCR_{infant} = ADD_{ca\text{-}infant} \times SF_o$$

Where:
$ELCR_{infant}$ = Excess lifetime cancer risk to infant from breastfeeding
$ADD_{ca\text{-}infant}$ = Average daily dose (cancer) for breastfeeding infant (mg/kg/day)
SF_o = Cancer slope factor – oral [$(mg/kg/day)^{-1}$]

Using the slope factor of 2 $(mg/kg/day)^{-1}$ for total PCBs, the ELCR for the example is:

$ELCR_{infant}$ = 0.00014 mg/kg/day x 2 $(mg/kg/day)^{-1}$ = 3×10^{-4}

Calculated Non-Cancer Risk to Infants

The non-cancer hazard quotient is:

Equation D-10
Calculation of Non-Cancer Hazard Quotient to Breastfeeding Infant

$$HQ_{infant} = \frac{ADD_{nc\text{-}infant}}{RfD}$$

Where:
HQ_{infant} = Hazard quotient for breast-feeding infant
$ADD_{nc\text{-}infant}$ = Average daily dose (non-cancer) for breastfeeding infant (mg/kg/day)
RfD = Non-cancer reference dose (mg/kg/day)

Using the intermediate-duration MRL of 3×10^{-5} mg/kg/day for total PCBs, the calculated hazard quotient is:

$$HQ_{infant} = 0.010 \text{ mg/kg/day} / 3 \times 10^{-5} \text{ mg/kg/day} = 330$$

Developing Infant Risk Adjustment Factors (IRAFs)

For comparison, the calculated risks to the mother given the exposure assumptions are the following. For carcinogenic effects, using the long-term ADD and the oral slope factor:

$$ELCR_{mother} = 0.00011 \text{ mg/kg/day} \times 2 (mg/kg/day)^{-1} = 2 \times 10^{-4}$$

For noncarcinogenic effects, using ADD without factoring in exposure duration, exposure frequency, and averaging time, and using the chronic reference dose:

$$HQ_{mother} = 0.000265 \text{ mg/kg/day} / 2 \times 10^{-5} \text{ mg/kg/day} = 13$$

The relative ratios of risk to the infant compared with risk to the mother can be used to develop infant risk adjustment factors (IRAFs):

$$IRAF_{ca} = ELCR_{infant} / ELCR_{mother}$$

$$= 2.87 \times 10^{-4} / 2.28 \times 10^{-4} = 1.3$$

$$IRAF_{nc} = HQ_{infant} / HQ_{mother}$$

$$= 330 / 13 = 25$$

Where:

$ELCR_{infant}$	=	Excess lifetime cancer risk for breastfeeding infant
$ELCR_{mother}$	=	Excess lifetime cancer risk for mother
HQ_{infant}	=	Hazard quotient for breast-feeding infant
HQ_{mother}	=	Hazard quotient for mother
$IRAF_{ca}$	=	Infant risk adjustment factor for cancer effects
$IRAF_{nc}$	=	Infant risk adjustment factor for noncancer effects

This evaluation shows that the breastfeeding infant's excess lifetime cancer risk is slightly less than the calculated risk to the mother, and the non-cancer risk to the infant is 25 times greater than the non-cancer risk to the mother for PCB exposure. Although the example was performed using the fish ingestion pathway, this result is independent of the exposure pathway or dose to the mother. For PCBs, regardless of the exposure pathway to the mother or the dose, the excess lifetime cancer risk to the infant will always be approximately equal to the cancer risk to the mother, and the hazard quotient to the infant will always be 25 times the hazard quotient to the mother. This assumes that the conditions used to derive dose to the mother are appropriate for calculating maternal body burdens. We know this condition is not met for residential soil exposure, as discussed below. IRAF values depend on the metabolic half-life of the compound, and the difference between the subchronic and chronic reference doses. At a half-life greater than 60 days, the exposure to the infant will be greater than the exposure to the mother.

An IRAF of 25 for PCBs is not unexpected. It is based on a dose to the infant 38 times the dose to the mother. Other estimates are that the PCB dose to the infant is 50 times the dose to the mother, so our calculated result is consistent with empirical studies (ATSDR 2000).

IRAF values developed above apply to risk calculated for the mother prior to having her first child using a constant dose and body weight to determine chemical body burden in the mother. This is appropriate for occupational soil and groundwater exposure to an adult, and residential groundwater exposure to an adult. However, it is not appropriate for noncancer risk from residential soil exposure, which is typically calculated using child exposure parameters and body weight. To calculate body burden to the mother from residential exposure, a combination of childhood exposure and adult exposure needs to be considered, using the body weight of the mother. We performed these calculations using the simple default assumption of 6 years of exposure as a child, and 24 years of exposure as an adult. IRAF values differ not only by chemical and whether it applies to

cancer or noncancer effects, but also by general type of exposure pathway (using adult exposure factors or child exposure factors).

Table D-3 provides a summary of IRAFs for the major bioaccumulating chemicals. The IRAFs in Table D-3 provide a convenient method of including a breastfeeding pathway evaluation in risk assessments without having to perform the intermediate calculations. For example:

$$HQ_{infant} = HQ_{mother} \times IRAF_{nc}$$

$$ELCR_{infant} = ECLR_{mother} \times IRAF_{ca}$$

Where:

HQ_{infant}	=	Hazard quotient for breast-feeding infant
HQ_{mother}	=	Hazard quotient for mother
$ELCR_{infant}$	=	Excess lifetime cancer risk for breast-feeding infant
$ELCR_{mother}$	=	Excess lifetime cancer risk for mother
$IRAF_{nc}$	=	Infant risk adjustment factor for noncancer effects (Table D-3)
$IRAF_{ca}$	=	Infant risk adjustment factor for cancer effects (Table D-3)

For any exposure pathway in a risk assessment where women (including girls) are exposed to bioaccumulating chemicals, the risk to their infants from future breastfeeding can be calculated by multiplying the calculated excess lifetime cancer risk or hazard quotient for the mother by the factors shown in Table D-3. Example risk assessment calculations are shown in Table D-4.

Because they are developed for risk calculations, IRAFs are a combination of adjustment factors for exposure and toxicity. For CDDs/CDFs, PCB TEQ, and DDT, chronic RfDs are used for subchronic infant exposure, so there is no adjustment for differences in toxicity factors. For these chemicals, the IRAF is equivalent to an infant exposure adjustment factor (IEAF). For PCB non-cancer effects, there is a difference in the chronic RfD for child/adult exposure, and the subchronic RfD used for infant exposure. Toxicity differences are factored into the IRAF along with exposure differences. In most cases, we expect the IRAF approach to be used for the infant pathway because it is a simple and comprehensive approach. However, if you want to conduct a more explicit risk assessment for PCBs instead of using the comprehensive IRAF values, Table D-3 provides IEAF values. This may be helpful if, for example, the PCB RfD values are revised.

Comparison of Calculated Risks with Acceptable Levels

Using the approach presented in this appendix, the example excess lifetime cancer risk is approximately 3×10^{-4} for an infant consuming total PCBs in human milk for one year. This is substantially above the acceptable excess lifetime cancer risk of 1×10^{-6}.

For non-cancer effects of PCB exposure, the calculated hazard quotient is 330. For hazard quotients above 1, unacceptable exposures may be occurring and there may be concern for potential non-cancer effects. Generally, the greater the magnitude of the hazard quotient above 1, the greater the level of concern for non-cancer health effects.

The calculated cancer risks and non-cancer hazards are based on a total PCB concentration in whole-body resident fish composites of 1 mg/kg. Although this concentration was used as a convenient value to demonstrate the calculations, it is within the range of total PCB concentrations measured in resident fish tissue at contaminated sites in Oregon. Because the calculated excess lifetime cancer risk and hazard quotient are considerably above acceptable levels, we conclude that infant exposure to chemicals in human milk will be an important pathway for sites contaminated with bioaccumulating chemicals.

UNCERTAINTY EVALUATION

Following standard guidance, the risk assessment for this pathway should include an evaluation of the associated uncertainties. During our evaluation of this pathway, we considered the following issues.

Exposure Assessment

The only exposure to infants evaluated was consumption of human milk. We did not consider other potential exposure routes, such as transplacental transfer of PCBs from mother to fetus during pregnancy.

Unlike Equation D-4, Equation D-5 does not rely on the assumption that intake to the mother has occurred for a period of time long enough relative to the half-life of the chemical that steady-state conditions are reached. For chemicals such as PCBs or CDDs/CDFs with half-lives on the order of 7 years, approximately 90 percent of steady-state concentration is reached after 21 years of exposure to the mother (3 half-lives). If the mother is exposed for only 7 years prior to breast feeding, the concentration of chemical in milkfat will be only one-half the concentration calculated for steady-state conditions, and risks calculated using the steady-state equation (D-4) will be overestimated by a factor of 2.

We assumed that chemical concentrations in milkfat are equal to chemical concentrations in the mother's body fat. This may overestimate milkfat concentrations. However, EPA considers this to be a reasonable assumption for lipophilic compounds, based on human data for CDDs/CDFs (EPA 1998).

Comparison of Model Results with PBPK Models

Before we selected the EPA model for use in standard risk assessment guidance, we compared it to two more complex and validated models that predict concentrations of environmental contaminants in breast milk. Haddad, *et al*. have created an 8-compartment physiologically-based pharmacokinetic (PBPK) model and validated this model using observed data from 75 Canadian Inuit women and their infants.(Verner 2009) The simulated breast milk concentrations for PCB-153 from the Haddad model correlated tightly (r = 0.96) with observed concentrations. The Yang model is a 3-compartment PBPK model developed for ATSDR by Yang, *et al*. at Colorado State University (Redding 2008).

We compared the EPA model proposed in this guidance with the Haddad and Yang

models; this was done by running simulations in each model using PCB-153 as a case study. ADDm was back-calculated using the Haddad model based on PCB-153 blood concentrations measured in 8 of the 75 human subjects in the Haddad study. These 8 subjects were chosen for several reasons: they had all breastfed their infants for at least 11 months; observed data were available for every validated parameter in the Haddad study; they covered a representative distribution of the PCB-153 values observed among subjects in the study; and there were an equal number of male and female infants. Using the derived ADDm values, we ran the three models to simulate the milk concentration ($C_{milkfat}$) of PCB-153 and average daily dose to infant (ADD_{inf}) for each subject pair. Calculated milk concentrations (Figure D-1) and ADD_{inf} (Figure D-2) were then compared for each subject pair across the three models. The results from the three models were similar within a factor of 2 (Figures D-1 and D-2). These results suggest that the EPA model presented in this guidance (Equation C-5) yields results that are comparable to those from more complex and data-validated PBPK models, and is adequately protective of health. The model has the additional benefit of being simple enough to run on standard spreadsheet software.

Toxicity Assessment

The ATSDR intermediate-duration MRL was derived from a study on monkeys that approximated exposure during breastfeeding using a mixture of PCB congeners typically found in human milk. The uncertainty factors were LOAEL to NOAEL conversion (10), sensitive members (10), and interspecies extrapolation (3), for a total of 300.

The chronic PCB RfD is also based on LOAELs developed from studies on monkeys. The health effects included inflammation of glands in the eye, distorted growth of finger and toe nails, and decreased antibody responses. The uncertainty factors used in the derivation of the human health RfD total 300, applied to an animal LOAEL of 0.005 mg/kg/day.

If a chronic RfD is used instead of a subchronic RfD, another uncertainty is the application of the RfD to one year of exposure, rather than long-term (lifetime) exposure. EPA's Superfund guidance defines chronic exposure as that between seven years and a lifetime. However, in its Combustion Guidance, EPA considered it appropriate to apply the chronic RfD to one year of exposure to human milk, at least for screening purposes (EPA 2005). Application of the chronic RfD to one year of exposure may also be appropriate considering the potential sensitivity of infants to adverse health effects.

Risk Characterization

Using the chronic RfD for PCBs instead of the intermediate duration MRL, the calculated hazard quotient is:

$$HQ_{infant} = 0.010 \text{ mg/kg/day} / 2 \times 10^{-5} \text{ mg/kg/day} = 500$$

The chronic HQ is 1.5 times the subchronic HQ of 330.

Body Burden Reductions

Incorporation of body burden reduction during a year of breastfeeding was included in Equation D-4 for the first infant that is breastfed. For additional infants that are breastfed by the same woman, the mother's body burden will be reduced to about half of the body burden prior to the previous breastfed child. However, evaluation of infant risk should be based on the most exposed infant, which will almost always be the first-born child.

Relative Exposure

EPA considered presenting the potential risks from human milk consumption as a ratio to background risk rather than as an excess lifetime cancer risk or hazard quotient. Background total PCB concentrations reported in the literature include 0.27 mg/kg-lipid in milk (Greizerstein et al. 1999), 0.32 mg/kg-lipid (Korrick and Altschul 1998), and 0.38 mg/kg-lipid (Noren and Meironyte 2000). Using the assumed total PCB concentration of 1 mg/kg in fish tissue and the assumed fish consumption rate, the calculated total initial PCB concentration in human milk is 1.9 mg/kg-lipid. As an alternative presentation of risk in the uncertainty section, this result can be discussed as corresponding to a risk approximately 5 to 7 times that of background concentrations.

Fish Advisories

DEQ is aware that in some major rivers, consumption of resident fish by mothers who are breastfeeding is already discouraged by fish advisories. For example, the Oregon Office of Environmental Public Health (OEPH) advisory for PCBs in the Willamette River states that:

> Women of childbearing age, particularly pregnant or breastfeeding women, children and people with weak immune systems, thyroid or liver problems, should avoid eating resident fish from Portland Harbor, especially carp, bass and catfish.

For this reason, there may currently be limited infant exposure to human milk contaminated as a result of consumption of resident fish in the lower Willamette River. In addition, OEPH advice on preparing fish for consumption, including removing fat from fillets (rather than consuming whole-body fish), could substantially lower risks to fish consumers, and also subsequently to breastfeeding infants. However, the results presented here appear to quantitatively support the advisory, and indicate that there are potentially significant unacceptable risks by the breastfeeding pathway.

HEALTH CONSULTATION ON BREASTFEEDING PATHWAY

DEQ asked the Oregon Office of Environmental Public Health (OEPH) to develop recommendations on how to address the potential health risks for infants exposed to PCBs in human milk in the context of the many health benefits of breastfeeding. OEPH's evaluation and recommendations are included in Attachment 2 of this appendix.

REFERENCES

Allan H. Smith. 1987. **Infant Exposure Assessment for Breast Milk Dioxins and Furans Derived from Waste Incineration Emissions**. Risk Analysis, Vol. 7, No. 3. 1987.

ATSDR 2000. **Toxicological Profile for Polychlorinated Biphenyls.** Agency for Toxic Substances and Disease Registry (Update, November 2000).

EPA 1997. **Exposure Factors Handbook.** National Center for Environmental Assessment, Office of Research and Development. August 1997.

EPA 1998. **Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions.** EPA 600/R-98/137, December 1998.

EPA 2005. **Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.** EPA 530-R-05-006, September 2005.

EPA 2008. **Child-Specific Exposure Factors Handbook**. National Center for Environmental Assessment, Office of Research and Development. EPA/600/R-06/096F. September 2008.

EPA 2009. **Risk and Technology Review (RTR). Risk Assessment Methodologies: For Review by the EPA's Science Advisory Board.** EPA-452/R-09-006. June 2009.

Greizerstein, H.B., C. Stinson, P. Mendola, G.M. Buck, P.J. Kostyniak, and J.E. Vena. 1999. **Comparison of PCB congeners and pesticide levels between serum and milk from lactating women.** Environ. Res. 80(3):280-6.

Korrick, S. and L. Altschul. 1998. **High breast milk levels of polychlorinated biphenyls (PCBs) among four women living adjacent to a PCB-contaminated waste site.** Environ. Health Perspect. 34 106:513.

Noren, K. and D. Meironyte. 2000. **Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years.** Chemosphere 40:1111-23.

Redding, L.E., et al., 2008. **Population physiologically based pharmacokinetic modeling for the human lactational transfer of PCB-153 with consideration of worldwide human biomonitoring results.** Environ Health Perspect. 116(12): p. 1629-35

Verner, M.A., et al., 2009. **A physiologically based pharmacokinetic model for the assessment of infant exposure to persistent organic pollutants in epidemiologic studies.** Environ Health Perspect, 2009. 117(3): p. 481-7.

Table D-1
Half-lives and Toxicity Values for Bioaccumulating Chemicals

Chemical	Half-life (days)	Oral RfDsubchronic (mg/kg/day)	Oral RfDchronic (mg/kg/day)	Oral Slope Factor $(mg/kg/day)^{-1}$
CDDs/CDFs TEQ	2550	-	1×10^{-9}	1.3×10^{5}
DDD	120	-	-	0.24
DDE	120	-	-	0.34
DDT	120	-	5.0×10^{-4}	0.34
Total PCB	2550	3×10^{-5}	2×10^{-5}	2
PCB TEQ	2550	-	1×10^{-9}	1.3×10^{5}

Notes:

Source of half-lives:
ATSDR 1977. Toxicological Profile for Chlorinated Dibenzo-p-dioxins. Draft for Public Comment. Agency for Toxic Substances and Disease Registry. September 1997.
ATSDR 2002. Toxicological Profile for DDT/DDD/DDE (Update). Agency for Toxic Substances and Disease Registry. September 2002.
ATSDR 2000. Toxicological Profile for Polychlorinated Biphenyls (Update). Agency for Toxic Substances and Disease Registry. November 2000.

Source of reference doses (RfDs) and oral slope factors (SFo):
EPA 2010. Regional Screening Levels.
www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm.

Source of subchronic RfD (taken from ATSDR Minimal Risk Level [MRL]):
ATSDR 2000. Toxicological Profile for Polychlorinated Biphenyls (Update). Agency for Toxic Substances and Disease Registry. November 2000.

Table D-2
Parameters for Evaluation of Risk to Infant Consuming Human Milk

Parameter	Units	Description	Value[a]	Source
ADD_{mat}	mg/kg/day	Average daily dose to mother	Calculated	-
DAI_{mat}	mg/kg/day	Average daily absorbed dose to mother	Calculated	-
AE	unitless	Absorption efficiency of chemicals	1	EPA 1998
ADD_{infant}	mg/kg/day	Average daily dose to infant	Calculated	-
C_{fish}	mg/kg	Chemical concentration in fish	Site-specific	-
IR_{fish}	g/day	Ingestion rate of fish	17.5 (recreational fishers)	EPA 2000
CF	kg/g	Conversion factor	0.001	
F_{fish}	unitless	Fraction of fish contaminated	1	
BW_{mat}	kg	Body weight of mother (mean for ages 15 to 44)	66	EPA 2009
BW_{inf}	kg	Average body weight of infant over 1 year	7.8	EPA 1998
$C_{milkfat,ss}$	mg/kg-lipid	Steady state chemical concentration in milkfat prior to breastfeeding	Calculated	-
$C_{milkfat,avg}$	mg/kg-lipid	Average chemical concentration in milkfat during breastfeeding	Calculated	-
h	days	Half-life of chemical	Chemical-specific	-
f_f	unitless	Fraction of ingested chemicals stored in fat	0.9	EPA 1998
f_{fm}	unitless	Fraction of mother's weight that is fat	0.3	EPA 1998
f_{mbm}	unitless	Fraction of human milk that is fat	0.04	EPA 1998
k_{elim}	$days^{-1}$	Elimination constant for chemical in non-lactating mother	Calculated	-
k_{elac}	$days^{-1}$	Elimination constant for chemical in lactating mother	Calculated	-

Table D-2
Parameters for Evaluation of Risk from Consuming Human Milk

Parameter	Units	Description	Value[a]	Source
t_{pn}	days	Duration of mother's exposure prior to breastfeeding	25 years = 9125 days	Assumption
t_{bf}	days	Duration of breastfeeding	1 year = 365 days	EPA 1998
ED_{inf}	days	Exposure duration of breast-feeding child	1 year = 365 days	EPA 1998
CR_{milk}	kg/day	Average consumption rate of human milk	0.98 kg/day	EPA 2009
AT	days	Averaging time – carcinogen	70 years[c] = 25550 days	-
		Averaging time – non-carcinogen	= ED	-
ELCR	risk	Excess lifetime cancer risk	Calculated	-
HQ	hazard	Hazard quotient	Calculated	-
SF_o	$(mg/kg/day)^{-1}$	Cancer slope factor – oral	Table D-1	-
RfD	(mg/kg/day)	Reference dose (chronic)	Table D-1	-
MRL	(mg/kg/day)	Minimal risk level (intermediate duration)	Table D-1	-

Notes:

a) Exposure assumptions taken from:
EPA 1997. *Exposure Factors Handbook*. Office of Research and Development, U.S. Environmental Protection Agency. (Update to EPA/600/P-96/002Babc, August 1997).

EPA 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000)*. U.S. Environmental Protection Agency, Office of Water. EPA-822-B-00-004, October 2000.

EPA 2005. *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.* EPA 530-R-05-006. September 2005.

EPA 2008. *Child-Specific Exposure Factors Handbook.* National Center for Environmental Assessment, Office of Research and Development. EPA/600/R-06/096F. September 2008.

EPA 2009. Risk and Technology Review (RTR). Risk Assessment Methodologies: For Review by the EPA's Science Advisory Board. EPA-452/R-09-006. June 2009.

b) EPA combustion facilities guidance (EPA 2005) uses 1 year. We consider this too conservative, and use the lifetime AT_c value typically used at Superfund sites.

Table D-3
Default Infant Risk Adjustment Factors (IRAFs)

For Calculating Human Milk Consumption Risks
Based on Risks Calculated for Exposure to the Mother

Chemical	IRAF to Convert Chronic HQ for Mother to Subchronic HQ for Infant[a,e]		IRAF to Convert ELCR for Mother to ELCR for Infant[b]	
	Adult Exposure Pathways[c]	Residential Soil Exposure Pathway[d]	Adult Exposure Pathways[c]	Residential Soil Exposure Pathway[d]
CDDs/CDFs	2	0.3	1	0.7
DDT/DDE/DDD	2	0.3	0.007	0.004
Total PCB	25	4	1	0.6
PCB TEQ	2	0.3	1	0.7

Notes:

a) HQinfant = HQmother x IRAF
b) ELCRinfant = ELCRmother x IRAF

$IRAF_{nc}$ = Infant risk adjustment factor for noncancer effects
$IRAF_{ca}$ = Infant risk adjustment factor for cancer effects
HQ = hazard quotient
ELCR = excess lifetime cancer risk
CDD = chlorinated dibenzo-*p*-dioxin
CDF = chlorinated dibenzofuran
DDT = dichlorodiphenyltrichloroethane
DDE = dichlorodiphenyldichloroethene
DDD = dichlorodiphenyldichloroethane
PCB = polychlorinated biphenyl
TEQ = 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxicity equivalent

c) Adult pathways include occupational exposure pathways and exposure pathways such as residential groundwater ingestion and food ingestion that are often evaluated using adult exposure parameter values for noncancer risk. Residential exposure pathways for cancer effects use time-integrated exposure, which generally reflects adult exposure, with the exception of residential soil exposure.
d) Residential soil exposure for noncancer effects is typically evaluated using child exposure parameter values. Residential soil exposure for cancer effects uses time-integrated exposure.
e) IRAFs reflect differences in child/adult exposure and infant expoure, except for total PCBs, where the IRAFs include both differences in exposure and differences in chronic and subchronic RfDs. Considering exposure alone, the infant exposure adjustment factors (IEAFs) for total PCBs are:
Noncancer adult exposure pathways, IEAF = 38
Noncancer residential soil exposure pathways, IEAF = 5

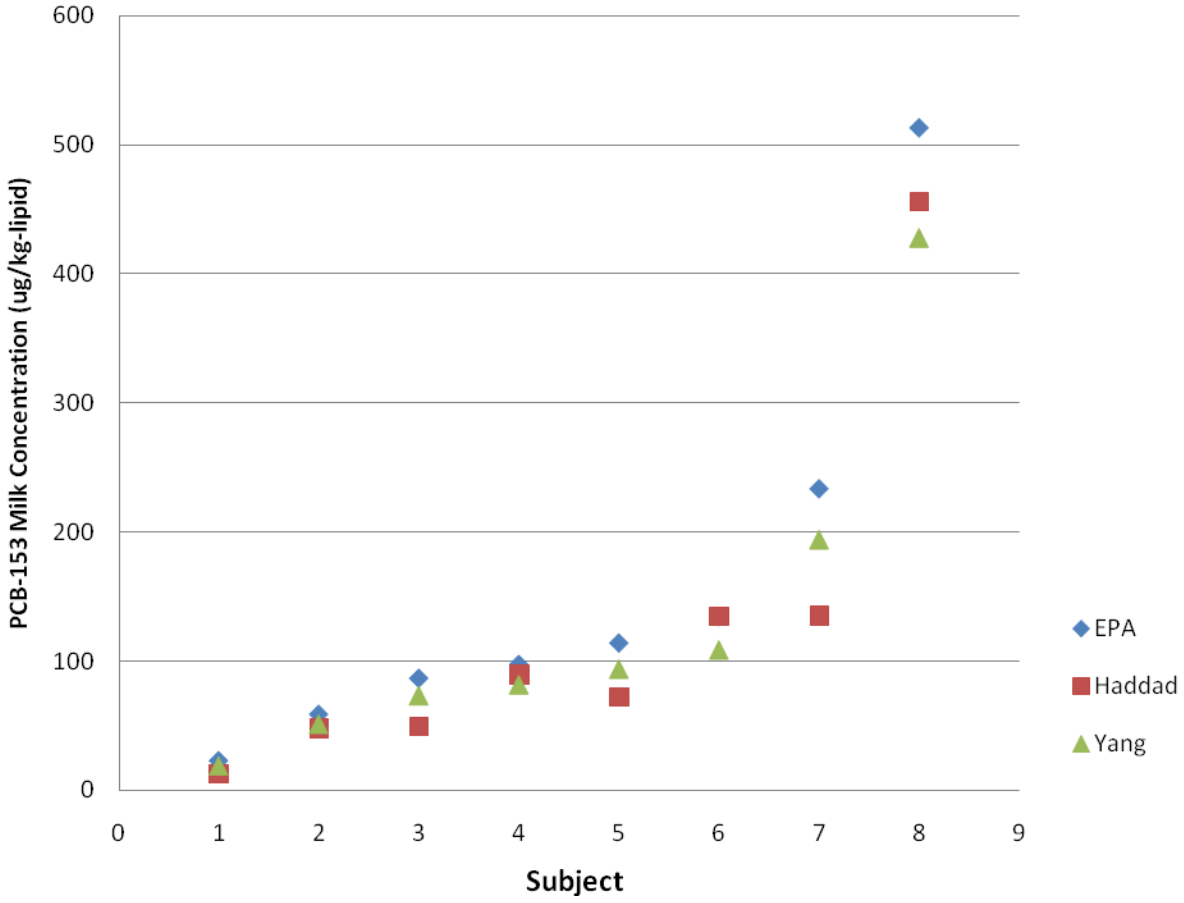
Table D-4
Example Calculation of Human Milk Consumption Risks Based on Risks Calculated for Exposure to the Mother

	Hazard Quotient		Excess Lifetime Cancer Risk	
Chemical	**Adult**	**Infant[a]**	**Adult**	**Infant[a]**
Occupational soil ingestion exposure pathway				
Arsenic	0.13	-	2×10^{-6}	-
Total PCB	0.38	9.5	7×10^{-6}	7×10^{-6}
PCB TEQ	0.08	0.16	*5×10^{-6}*	*5×10^{-6}*
CDD/CDF TEQ	0.016	0.032	*1×10^{-6}*	*1×10^{-6}*
Total TEQ	-	-	6×10^{-6}	6×10^{-6}
Total	**0.61**	**9.7**	**1×10^{-5}**	**1×10^{-5}**
Adult fish ingestion exposure pathway				
Total PCB	13	325	2×10^{-4}	2×10^{-4}
PCB TEQ	3.2	6.4	*2×10^{-4}*	*2×10^{-4}*
CDD/CDF TEQ	1.3	2.6	*8×10^{-5}*	*8×10^{-5}*
Total TEQ	-	-	3×10^{-4}	3×10^{-4}
Total	**18**	**334**	**5×10^{-4}**	**5×10^{-4}**
Total for all exposure pathways				
Total	**18**	**344**	**5×10^{-4}**	**5×10^{-4}**

Notes:

a) Calculated using infant risk adjustment factors (IRAFs) shown in Table D-3.

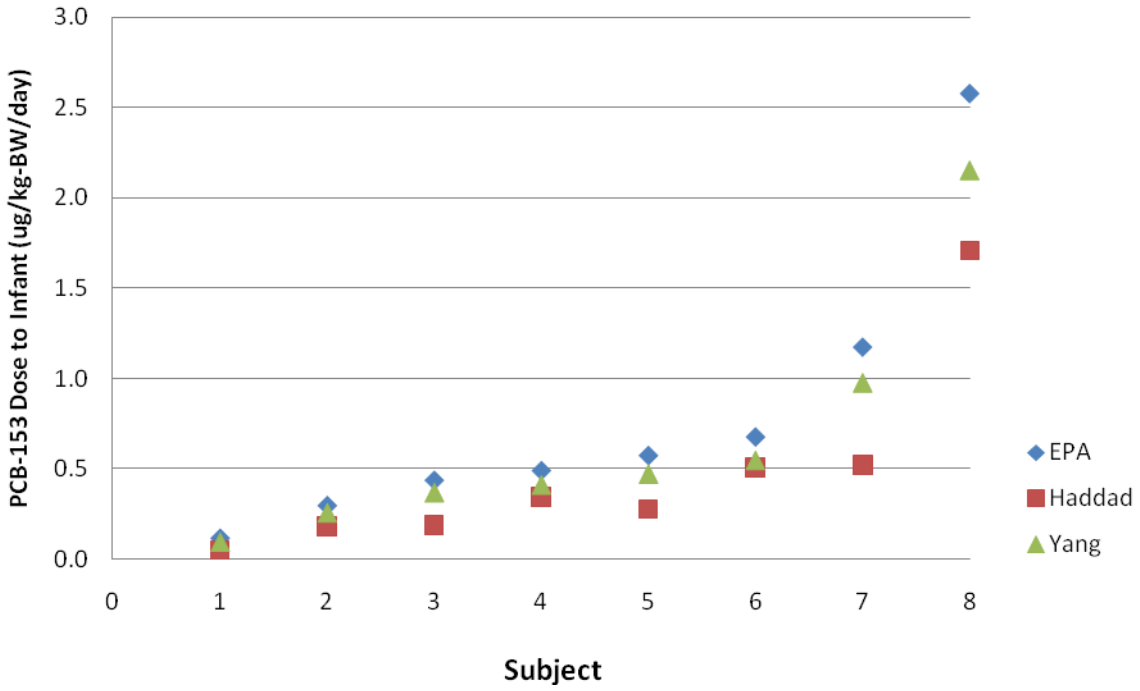
Figure D-1
Model Comparison
1-Year Average PCB-153 Milk Concentrations



Notes:

EPA model as described in this document, modified from EPA 1998.
Haddad model from Verner *et al*., 2009.
Yang model from Redding *et al*., 2008.

Figure D-2
Model Comparison
1-Year Average PCB-153 Dose to Infant



Notes:

EPA model as described in this document, modified from EPA 1998.
Haddad model from Verner *et al*., 2009.
Yang model from Redding *et al*., 2008.

Appendix D - Attachment 1

Consideration of Non-Steady State Chemical Concentrations in Milkfat and Losses During Breastfeeding

The EPA combustion facility guidance document (EPA 2005) and ATSDR's Toxicological Profile (ATSDR 2000) do not elaborate on the derivation of the equation for calculation of chemicals present in milkfat. The main EPA reference for the equation is Allan Smith's evaluation of infant exposure to chlorinated dibenzodioxins and chlorinated dibenzofurans in human milk (Smith 1987). In this attachment, we first explicitly derive the non-steady state equation used to approximate chemical concentrations in maternal body fat, which is assumed to be equivalent to the concentration in human milk. Then we consider chemical mass losses in the mother as a result of breastfeeding. Additional information on the derivation of Equation D-5 is presented in Appendix C of EPA's MPE Guidance. (EPA 1998)

Non-Steady State Chemical Concentration in Milkfat

The chemical body burden in the mother is calculated assuming first-order kinetics:

Equation D-11
General Body Burden Calculation

$$B_t = B_0 e^{-kt}$$

Where:
t = Time period (years)
B_t = Body burden at time t (mg)
B_0 = Body burden at time t = 0 (mg)
k = Rate constant ($days^{-1}$)

Using this standard approach, the maternal daily chemical intake, m (mg/kg/day), is used to calculate the concentration of chemical in the mother's tissue. The maternal chemical concentration (C_{mother} in mg/kg-body-weight) at time T is:

$$C_{mother} = \int_0^T me^{-kt}\, dt$$

where the mother is exposed to the chemical from time t = 0 to time t = T (in days). The general solution to this equation is:

$$\int_0^T me^{-kt} dt = \frac{me^{-kT}}{-k} - \frac{me^{0}}{-k} = \frac{me^{-kT}}{-k} + \frac{m}{k} = \frac{m}{k}(1 - e^{-kT})$$

This results in the following equation, noting that the elimination rate constant $k = k_{elim}$:

Equation D-12
Chemical Concentration in Mother

$$C_{mother} = \frac{m}{k_{elim}}(1 - e^{-k_{elim}T})$$

Where:
C_{mother} = Chemical concentration in mother at time T (days)
m = Absorbed chemical dose to mother (mg/kg/day)
k_{elim} = Elimination rate constant for non-lactating women ($days^{-1}$)
T = Time of exposure to mother before breastfeeding (days)

Equation D-12 is applicable to conditions that have not reached steady state. To derive the steady state equation, we will use the definition of k_{elim} in the main text to convert from elimination rate constant to chemical halflife.

Equation D-6
Biological Elimination Rate Constant for Women Prior to Breastfeeding

$$k_{elim} = \frac{\ln(2)}{h}$$

Where:
k_{elim} = Elimination rate constant for non-lactating women ($days^{-1}$)
h = Half-life of chemical (days)

Using this relationship, Equation D-12 can be presented as:

Equation D-12A
Chemical Concentration in Mother (Version 2)

$$C_{mother} = \frac{mh}{\ln(2)}\left(1 - e^{\frac{-\ln(2)}{h}T}\right)$$

For exposure periods to the mother (T) that are long relative to the halflife of the chemical (h), ln(2)T/h becomes large, $e^{-ln(2)T/h}$ becomes much less than 1, and $(1 - e^{-ln(2)T/h})$ approaches a value of 1. At steady state:

Equation D-13
Steady State Chemical Concentration in the Mother

$$C_{mother} = \frac{mh}{\ln(2)}$$

Using the PCB example and Equation D-12A, if the exposure period of the mother to contaminated fish (*T*) is equal to the chemical half-life (*h*) of 7 years for PCBs, then the chemical concentration in the mother's tissue is:

$$C_{mother} = 0.5\frac{mh}{\ln(2)}$$

If the mother is exposed to PCBs for 7 years prior to breast-feeding, the PCB concentration in lipid tissue is one-half the value obtained assuming steady-state conditions.

If the exposure period of the mother to contaminated fish is equal to four half-lives (T = 4h = 28 years), then the chemical concentration in the mother's tissue is:

$$C_{mother} = 0.94\frac{mh}{\ln(2)}$$

Equation D-12 can be adjusted to make it a lipid-based concentration by considering the fraction of the chemical stored in fat tissue (f_f) and the fraction of the mother's weight that is fat (f_{fm}).

$$C_{mother} = \frac{mh}{\ln(2)}\frac{f_f}{f_{fm}}$$

Substituting the symbol DAI_{mat} for m, and assuming that the chemical concentration in milkfat is equivalent to the chemical concentration in the mother's fat tissue, yields the steady state equation for $C_{milkfat}$ shown in the main text as Equation D-4.

$$C_{milkfat} = \frac{DAI_{mat}\ h}{\ln(2)}\frac{f_f}{f_{fm}}$$

Or, alternatively, using the elimination rate constant instead of the chemical halflife:

Equation D-4A
Steady State Chemical Concentration in Milkfat (Version 2)

$$C_{milkfat} = \frac{DAI_{mat}}{k_{elim}}\frac{f_f}{f_{fm}}$$

To calculate non-steady state concentrations, a modified version of Equation D-12 can be used:

Equation D-14
Chemical Concentration in Milkfat Prior to Breastfeeding

$$C_{milkfat,pn} = \frac{DAI_{mat}}{k_{elim}}\frac{f_f}{f_{fm}}(1 - e^{-k_{elim}t_{pn}})$$

Where:

$C_{milkfat,pn}$ = Chemical concentration in milkfat prior to breastfeeding (mg/kg-lipid)
DAI_{mat} = Daily absorbed chemical intake to mother (mg/kg/day); Equation D-2
f_f = Fraction of ingested chemical stored in fat (0.9)
f_{fm} = Fraction of mother's weight that is fat (0.3 kg-lipidBW/kg-totalBW)
k_{elim} = Elimination rate constant for non-lactating women; Equation D-6
t_{pn} = Duration of mother's exposure prior to breastfeeding (days)

Using Equation D-4 for the steady state concentration in milkfat, $C_{milkfat,ss}$, Equation D-14 can be modified to express $C_{milkfat,pn}$ in terms of $C_{milkfat,ss}$.

Equation D-14A
Chemical Concentration in Milkfat Prior to Breastfeeding (Version 2)

$$C_{milkfat,pn} = C_{milkfat,ss}(1 - e^{-k_{elim}t_{pn}})$$

Reduction in Chemical Dose to Infant Over Time

The loss of chemical mass through breastfeeding will reduce the chemical body burden in the mother, thereby reducing breast milk concentrations and dose to the infant over time. Equation D-5 accounts for reduction in chemical concentrations over time by including the rate constant k_{elac} for elimination of chemicals in milkfat. Because Equation D-5 is complex, it is difficult to determine the impact of reducing dose to the infant during the breastfeeding period. In this section, we look specifically at the reduction in chemical concentration in human milk over time to determine the magnitude of this effect on the dose to the infant.

One of the reasons Equation D-5 is complex is because it assumes that the mother continues to be exposed to chemicals during lactation. We make the simplifying assumption that the mother ceases to be exposed to chemicals during breastfeeding. Because the default breastfeeding period of 1 year is short relative to the default exposure duration of the mother before breastfeeding (25 years), this assumption should have little impact on the average chemical concentration in milk.

With this simplifying assumption, the reduction in chemical concentration in milkfat can be approximated by the following equation based on Equation D-11.

Equation D-15
Chemical Concentration in Milkfat

$$C_{milkfat,t} = C_{milkfat,pn}e^{-kt})$$

Where:

$C_{milkfat,t}$ = Chemical conc. in milkfat during breastfeeding at time t (mg/kg-lipid)
$C_{milkfat,pn}$ = Chemical concentration in milkfat prior to breastfeeding (mg/kg-lipid)
k = Elimination rate constant for lactating women ($days^{-1}$)
t = Duration of breastfeeding (days)

The average chemical concentration in milkfat during breastfeeding is:

Equation D-16
Average Chemical Concentration in Milkfat

$$C_{milkfat,avg} = \frac{1}{T}\int_0^T C_{milkfat,pn} e^{-kt} dt$$

Where:

$C_{milkfat,avg}$ = Average chemical conc. in milkfat during breastfeeding (mg/kg-lipid)
$C_{milkfat,pn}$ = Chemical concentration in milkfat prior to breastfeeding (mg/kg-lipid)
k = Elimination rate constant for lactating women ($days^{-1}$)
T = Duration of breastfeeding (days)

Using modified nomenclature, considering that all concentrations are for milkfat, the general solution to this equation is:

$$C_{avg} = \frac{1}{T}\int_0^T C_{pn} e^{-kt} dt = \frac{C_{pn}}{T}\int_0^T e^{-kt} dt = \frac{C_{pn}}{T}\left(\frac{e^{-kT}}{-k} - \frac{e^{-k(0)}}{-k}\right) = \frac{C_{pn}}{T}\left(\frac{e^{-kT}}{-k} - \frac{1}{-k}\right)$$
$$= \frac{C_{pn}}{kT}(1 - e^{-kT}) = C_{pn}\left(\frac{1 - e^{-kT}}{kT}\right)$$

This simplified equation shows that the average chemical concentration in milkfat can be approximated by the chemical concentration in milkfat prior to lactation (C_{pn}) times the factor $(1 - e^{-kT})/kT$ to account for loss of chemical mass during breastfeeding.

Using Equation D-14A for C_{pn}, the above equation can be modified so that the average chemical concentration in milkfat over the duration of breastfeeding can be expressed in terms of the steady state concentration in the mother:

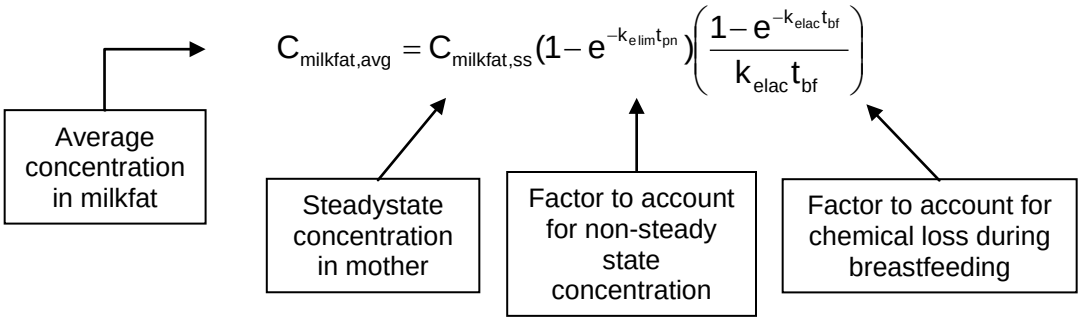
Equation D-17
Average Chemical Concentration in Milkfat as a Function of the Steady State Concentration in the Mother

$$C_{milkfat,avg} = C_{milkfat,ss}(1 - e^{-k_{e\,lim} t_{pn}})\left(\frac{1 - e^{-k_{elac} t_{bf}}}{k_{elac} t_{bf}}\right)$$

Where:

$C_{milkfat,avg}$ = Average chemical conc. in milkfat during breastfeeding (mg/kg-lipid)
$C_{milkfat,ss}$ = Steady state chemical concentration in milkfat prior to breastfeeding (mg/kg-lipid)
k_{elim} = Elimination rate constant prior to lactation ($days^{-1}$)
k_{elac} = Elimination rate constant during lactation ($days^{-1}$)
t_{pn} = Duration of exposure to mother prior to breastfeeding ($days^{-1}$)
t_{bf} = Duration of exposure to infant during breastfeeding ($days^{-1}$)

The PCB example can be used to show the relative impact resulting from considering non-steady state chemical concentrations in the mother, and chemical mass loss during breastfeeding.



In the PCB example, the factor for non-steady state concentration is 0.86, and the factor for chemical loss during breastfeeding is 0.70, so

$$C_{avg} = C_{ss} \times 0.86 \times 0.70 = C_{ss} \times 0.61$$

Collectively, consideration of these two factors reduces the estimate of average PCB concentration in milkfat by about 40 percent.

The lower the half-life of the chemical relative to the exposure period of the mother, the less accurate this approximation of average chemical concentration in milkfat will be.

REFERENCES

ATSDR 2000. **Toxicological Profile for Polychlorinated Biphenyls.** November 2000.

EPA 1998. **Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions.** EPA 600/R-98/137, December 1998.

EPA 2005. **Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.** EPA 530-R-05-006, September 2005.

Allan H. Smith. 1987**. Infant Exposure Assessment for Breast Milk Dioxins and Furans Derived from Waste Incineration Emissions.** Risk Analysis, Vol. 7, No. 3. 1987.

Appendix D - Attachment 2

Statement from
Oregon Office of Environmental Public Health

Statement from the Oregon Office of Environmental Public Health (OEPH) March 2010

Oregon DEQ has asked the Office of Environmental Public Health (OEPH) to develop recommendations on how to address the health risks for infants exposed to lipophilic, bioaccumulative environmental contaminants in human milk in the context of the many health benefits of breastfeeding. This statement is designed to answer their request.

Inclusion of the breastfeeding exposure pathway in risk assessments is important to ensure that our environment is safe for our most vulnerable citizens – namely human infants. However, it is critical to understand that risk estimates calculated using the model presented here are ***not*** intended to advise women about whether or not to breastfeed their infants. Rather, the purpose is to inform site clean-up managers so that they can make decisions that will lead to decreased exposure to women and ultimately lower concentrations of contaminants in the milk women produce for their infants. Calculated risks to infants support public health actions that encourage women to limit their own exposure to environmental contaminants so that their infants can receive the optimal health benefits from breastfeeding. Therefore, OEPH supports DEQ in adding the breastfeeding exposure pathway to their human health risk assessment guidance.

The breastfeeding exposure pathway for environmental contaminants presents unique challenges to public health officials in their efforts to manage health risks by modifying people's behaviors. In most health/risk assessments, the exposure medium is considered only a delivery vehicle for the contaminant of concern. In the case of human milk, however, the exposure medium contains a multitude of healthful compounds that have been well documented to produce measurable health benefits in infants such as improved immunity to infectious diseases. In fact, not breastfeeding is considered a risk factor in many acute and chronic health conditions. Therefore, treatment of this exposure pathway requires not a simple assessment of risk, but rather, a balancing of the risks associated with contaminant exposure against the well documented health benefits of breastfeeding.

Hence, OEPH strongly supports the inclusion of the breastfeeding exposure pathway in risk assessments to be protective of infants. OEPH also encourages risk assessors to review thoroughly the information provided below regarding the health risks and benefits associated with breastfeeding. We ask that risk assessors follow the advice provided in the recommendations section to include appropriate risk messages in their documents that will not discourage women from breastfeeding.

Background Information

Health benefits of breastfeeding

Breastfeeding has been shown to be the healthiest option for infants under most conditions. Human milk is an inexpensive, ideally balanced source of nutrition[1]. The infant immune system is matured and bolstered by human milk components. Immunoglobulin A (IgA) in human milk reduces the uptake of dietary antigens, protecting against development of food allergies[2]. IgA in human milk also protects the infant against microbes from the maternal gut and prevents

microbes from binding to the intestinal mucosal surface[3]. Human milk also has anti-inflammatory properties[2], stimulates maturation of the intestinal epithelium and enhances the protective character of the intestinal mucosa[4]. This overall enhancement of immune function means reduced risk of multiple types of infectious disease for the infant.

Breastfeeding is also associated with improved IQ scores and neurological development and reduced risk of SIDS (Sudden Infant Death Syndrome), type I and type II diabetes, leukemia, obesity, asthma, and high cholesterol[1]. Recent research suggests that exclusive breastfeeding may reduce the risk of celiac disease[5]. There are also psychological benefits to the improved mother-infant bonding that accompanies consistent breastfeeding. Overall, non-breastfed babies have a 21 percent higher mortality rate than breastfed babies[1].

Mothers who breastfeed also enjoy health benefits including reduced postpartum bleeding, reduced risk of breast and ovarian cancer, easier loss of excess adipose accumulated during pregnancy, and enhanced psychological well-being with increased bonding between mother and child. Breastfeeding also benefits society by reducing health care costs (healthier babies) and increasing worker productivity (children sick less often)[1].

Environmental contaminants in human milk and children's health
The model and guidance detailed in Appendix C of this Human Health Risk Assessment (HHRA) guidance document can be applied to any environmentally stable, lipophilic chemical with potential to bioaccumulate in the adipose tissue and milk of women who are exposed. However, preliminary simulations run so far suggest that polychlorinated biphenyls (PCBs) are often the contaminant class leading to the greatest increase in infant/mother risk ratio when the breastfeeding pathway is incorporated into standard HHRA scenarios. Therefore, this discussion on the health/risk balance of environmental contaminants in human milk will focus on PCBs.

Background concentrations of PCBs in human milk vary by region and culture, but overall, these concentrations appear to be decreasing over time now that PCBs are not widely in use. The Agency of Toxic Substances and Disease Registry (ATSDR) suggests that 0.247 µg PCB/g-lipid might be the best current estimate.[6] PCB concentrations as high as 10-15 µg/g-lipid have been reported in instances where mothers were occupationally exposed[7]. People who consume large amounts of PCB-contaminated fish have also been shown to have higher milk PCB concentrations.[6] Studies found a negative correlation between PCB concentrations in the milk of nursing mothers and the health of their children. The adverse health effects in children associated with increasing concentrations of PCBs in their mothers' milk included deficits in neurobehavioral function, alterations within the immune system, and altered thyroid function (see table 1).[6]

In most cases, toxicity was attributed to prenatal exposure to PCBs. One study, known as the "Dutch PCB/Dioxin Study," compared the neurological performance of children exposed to PCBs only prenatally with that of children exposed prenatally and postnatally via human milk. While children consuming milk containing higher PCBs fared worse than children consuming milk with lower levels, all groups of breastfed children fared better than bottle-fed children. The lowest performing children had been exposed to high levels of PCBs prenatally but had been

formula fed after birth. These data suggest that breastfeeding, even with PCB-contaminated milk, served to counter the negative effects of prenatal PCB exposure[8,9]. The studies cited in this report conclude that, even at the highest human milk PCB levels measured, the health benefits of breast feeding still outweigh the risks associated with contaminant exposure.

Generalized risk assessment information for breastfed infants
The doses of PCBs that a breastfeeding infant may be expected to receive, given breast milk PCB concentrations measured in the literature, are presented in table 1. These doses range from 0.0019 to 0.0081 mg/kg/day and are 63-270 times higher than ATSDR's minimal risk level (0.00003 mg/kg/day) for PCB exposures that last between 15 and 364 days. These doses are slightly higher than that shown to cause health effects in monkeys (0.005 mg/kg/day). Health effects that occurred in monkeys at 0.005 mg/kg/day include altered finger and toe nails and nail beds, inflammation of eye-lid glands, and decreased immunity.[6]

Although PCB doses delivered to the infant over the course of breastfeeding may be significant in some cases, PCB exposure via breast milk necessarily follows additional prenatal exposures during critical developmental windows. Studies cited here suggest that breast milk, even with significant PCB contamination, may serve to reverse or stabilize developmental lesions initiated by prenatal exposure[8,9].

The primary goal for environmental and health agencies should be to reduce PCB exposure to women of childbearing age. As PCB-contaminated fish can be a major source of maternal PCB body burdens, these findings reinforce the importance of fish advisories for Portland Harbor and other waterways in Oregon issued by OEPH (http://www.oregon.gov/DHS/ph/envtox/fishconsumption.shtml). However, the recommended course for infants who have already had prenatal exposure to PCBs is clear. Breastfeeding is best for infants regardless of PCB levels in the milk.

Main Conclusions

- For lipophilic environmental contaminants such as PCBs, the nursing infant receives the highest dose of contaminant, and infants are more sensitive to the effects of PCBs than adults.

- Human milk containing high background PCB concentrations could result in doses to infants as much as 270 times higher than the minimal risk level for PCBs. However, due to the significant benefits of human milk, breastfeeding should still be recommended.

- Elevated levels of PCBs in breast milk indicate significant prenatal exposure to PCBs.

Recommendations

- Risk assessors using the new DEQ guidance are asked to include language in their reports and presentations encouraging women to continue breast feeding regardless of contaminant exposure levels. This language should include information on the well-documented health benefits of breast feeding, and be placed in proximity to conclusions and summaries related

to risk from the breastfeeding pathway. The following text is offered as an example:

- "Breastfeeding is still the healthiest way to feed a baby, even if the milk contains (Contaminant X). Even though infants may receive a (significant, moderate, insignificant, low, etc.) dose of (Contaminant X) from their mothers' milk, human milk also contains hundreds of healthy nutrients, vitamins, minerals, and immune system boosters. These natural, healthy substances more than compensate for any health risks from (Contaminant X) and may even help repair damage caused by (Contaminant X) before the baby was born. Breastfeeding has been shown to boost immunity and IQ and prevent many diseases. Calculated risk to infants from breastfeeding presented in this report should ***not*** discourage any mother from breastfeeding her infant."

- Risk assessors using the guidance in this appendix should contact the Environmental Health Assessment Program in OEPH at 971-673-0977 for consultation about how to craft messages about breastfeeding risk.

- Young girls and women of childbearing age should check for and abide by local fish advisories at OEPH's website (http://www.oregon.gov/DHS/ph/envtox/fishconsumption.shtml) before consuming locally-caught fish. The same website also has information for women about general consumption guidelines for store-bought and restaurant fish.

- OEPH recommends that all women continue to breastfeed their infants regardless of exposure situation unless directed otherwise by their physician.

Table 1[a]
Health Effects in Human Infants Associated with PCBs in Breast Milk

Study	Mean Breast Milk PCB Conc. (µg/g-lipid)	$ADD_{nc\text{-}infant}$[b] (mg/kg/day)	Observed Health Effects[c]	Comparison with Formula-fed Controls
Michigan Cohort	0.87 (fisheaters) 0.62 (nonfisheaters) Total PCBs	0.0039 (fisheaters) 0.0028 (nonfisheaters)	Reduced birth weight, head circumference, and gestational age in newborns. Neurobehavioral alterations in newborn and older children.	Deficits correlated with prenatal exposure but not postnatal exposure via breast milk.
Dutch Cohort	0.62 Total PCBs	0.0028	Reduced birth weight. Reduced growth during first 3 months in formula-fed, but not breast-fed children. Neurobehavioral alterations and changes in T-lymphocyte subpopulations and thyroid hormone levels in infants.	Slight increased incidence of mild hypotonia and neurological function in children breastfed with high PCBs relative to formula fed, but mental performance was enhanced with breastfeeding regardless of PCB contamination. Minor effects associated with postnatal exposure via breast milk resolved by 18 months of age.
German Cohort	0.43 Sum of PCB congeners[d]	0.0019	Neurodevelopmental and thyroid hormone alterations in infants	Breast-fed children did better than formula-fed in all parameters tested.
Inuit Infant Study	0.62 Sum of PCB congeners[d]	0.0028	Immunologic alterations.	No difference in immunological parameters between breast fed and formula fed infant
North Carolina Cohort	1.8 Sum of PCB congeners[d]	0.0081	Neurobehavioral alterations in infants	No comparison.
Intermediate-duration MRL[e] for Aroclor 1254:		**0.00003 mg/kg/day**		

Notes:

a) Adapted from Table A-1 in ATSDR's Toxicological Profile for PCBs[6].

b) $ADD_{nc\text{-}infant}$ = Non-cancer Average Daily Dose to infant via breast milk. Parameter not reported in studies, but doses were calculated for infants nursing from mothers with mean breast milk PCB concentrations reported. This exposure pathway is not applicable to formula-fed infants. It is important to note that any exposure via breast milk follows unquantified prenatal exposure.

$$ADD_{nc\text{-}infant} = \frac{C_{milkfat} \times CR_{milk} \times f_{mbm} \times f_f}{BW_i}$$

Where:

$ADD_{nc\text{-}infant}$ = Average daily dose for breast-feeding infant (mg/kg/day)
$C_{milkfat}$ = Concentration of PCBs measured in milk fat (µg/g-lipid)
CR_{milk} = Ingestion rate of breast milk (0.98 kg-milk/day)
f_{mbm} = Fraction of breast milk that is fat (0.04 kg-lipid/kg-milk)
f_f = Fraction of ingested PCB that is absorbed (0.9)
BW_i = Body weight of breast-feeding infant (7.8 kg)

Assumptions and calculations are modified from Table C-3-2 of EPA combustion guidance[10].

c) No distinction between effects due to prenatal exposure and effects due to postnatal exposure via breast milk (unless otherwise noted in table).

d) PCB value is the sum of three non-dioxin-like congeners (PCB 138, PCB 153, and PCB 180).

e) MRL = minimal risk level for intermediate-duration exposure (two weeks to one year).

References

[1] Department of Health and Human Services, National Women's Health Information Center (2008) website http://www.4women.gov/breastfeeding/index.cfm?page=227

[2] Kelly D. and Coutts A.G.P. (2000). Early nutrition and the development of immune function in the neonate. *Proceedings of the Nutritional Society,* **59,** 177-185.

[3] Hanson L.A., Korotkova M., Lundin S., Haversen L., Silfverdal S.A., Mattsby-Baltzer I. (2003). The transfer of immunity from mother to child. *Annals of the New York Academy of Sciences,* **987,** 199-206.

[4] Newburg D.S. (2005). Innate immunity and human milk. *Journal of Nutrition,* **135,** 1308-1312.

[5] Chertok I.R. (2007). The Importance of Exclusive Breastfeeding in Infants at Risk of Celiac Disease. *MCN. The American Journal of Child and Maternal Nursing,* **32,** 50-54.

[6] Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polychlorinated Biphenyls (2000). U.S. Department of Health and Human Services, Atlanta, GA.

[7] Hara I. (1985). Health Status and PCBs in Blood of Workers Exposed to PCBs and of Their Children. *Environmental Health Perspectives,* **59,** 85-90.

[8] Huisman M., Koopman-Esseboom C., Fidler V., Hadders-Algra M., et al. (1995). Perinatal Exposure to Polychlorinated Biphenyls and Dioxins and its Effect on Neonatal Neurological Development. *Early Human Development,* **41,** 111-127.

[9] Koopman-Esseboom C., Weisglas-Kuperus N., de Ridder M.A.J., Van der Paauw C.G., et al. (1996). Effects of Polychlorinated Biphenyl/Dioxin Exposure and Feeding Type on Infants' Mental and Psychomotor Development. *Pediatrics,* **97,** 700-706.

[10] U. S. EPA. *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.* EPA 530-R-05-006, September 2005.