7.0 METHOD 3 - HUMAN HEALTH

This section provides guidance on conducting a Method 3 Human Health Risk Characterization. The human health evaluation is just one of *four* distinct assessments which comprise a complete Method 3 Risk Characterization: the risk to safety, public welfare and the environment must also be addressed. The most site-specific of the three risk characterization options available under the MCP, a Method 3 assessment is an option at all c.21E sites.

The specific regulations concerning the Method 3 risk characterization process begin at 310 CMR 40.0990 of the Massachusetts Contingency Plan. Readers are reminded that general requirements applicable or potentially applicable to all risk characterizations are found in 310 CMR 40.0900 through 40.0960, and are described in Section 1.0 through 4.0 of this guidance document.

A Method 3 Risk Characterization Is Complete If...

- Risk to Safety (Section 4.0)
- Risk to Human Health (Section 7.0)
- Risk to Public Welfare (Section 8.0)
- Risk to the Environment (Section 9.0)

...Are Evaluated

The scope and level of effort needed to complete each component of a Method 3 Risk Characterization will vary depending upon site conditions.

The Method 3 human health risk characterization approach involves five steps: hazard identification, dose-response assessment, exposure assessment, risk characterization and uncertainty analysis.

Hazard Identification determines whether a substance causes adverse effects and identifies those effects. This step describes why the substance is of regulatory concern.

The **Dose-Response Assessment** describes the relationship between the level of exposure and the likelihood and/or severity of an adverse effect. Simply speaking, the dose-response information describes the toxicity of the substance.

The **Exposure Assessment** involves identifying potential routes of exposure; characterizing the populations exposed; and determining the frequency, duration and extent of exposure.

The **Risk Characterization** combines information from the previous three steps to describe the type (e.g., carcinogenic or non-carcinogenic) and magnitude of risks to exposed populations. The resulting risks are then compared to the risk management criteria promulgated in the regulations.

The **Uncertainty Analysis** identifies the nature and, when possible, the magnitude of the uncertainty and variability inherent in the characterization of risks. The results of any risk assessment reflect scientific uncertainty resulting from limitations in available data and assumptions that are made in the absence of such data, and the variability in exposure and toxicological response expected given the diversity within the human population. The assumptions and limitations which are a part of all risk characterizations should be explicitly discussed.

Each of these risk assessment steps is described in detail in the following sections of this document.

It is important to remember that risk estimates generated in the risk assessment are not measures of actual or absolute risks. Rather, risk assessments are a tool - a method of providing valuable information regarding potential risks to public health and the environment. Risk assessment is used throughout the regulatory process to provide such information, whether it is to determine "How clean is clean enough?" at a disposal site, to develop drinking water standards for public water supplies, or to evaluate a proposed facility seeking a source permit.

The MCP is explicit in its interpretation of the significance of the risk estimates. The risk management philosophy inherent in the establishment of the risk limits is to ensure that no potential receptor groups would experience an excess lifetime cancer risk greater than the risk limit, regardless of the number of chemicals or exposure routes that exist at a site. The noncancer risk limit reflects a risk management decision that multiple-chemical, multiple-route exposures related to a disposal site will not exceed an estimated "allowable" dose - a dose which would not result in adverse health effects.

Under Method 3, remediation of the disposal site is required if: (1) Exposure Point Concentrations exceed any applicable or suitably analogous public health standards, ***or*** (2) the estimated cancer or non-cancer risks associated with exposure to oil or hazardous material exceed the Cumulative Receptor Risk Limits (310 CMR 40.0993(6)). Remedial alternatives must be evaluated to determine if they eliminate "Significant Risk" as defined in the MCP.

7.1 HAZARD IDENTIFICATION

The Hazard Identification portion of an MCP Method 3 risk characterization describes the hazards associated with each OHM which has been selected as a Contaminant of Concern. More specifically, the Hazard Identification discusses whether exposure to a particular contaminant can cause an increase of a particular adverse health effect and whether the adverse health effect is likely to occur in humans.

The Hazard Identification section of the Risk Assessment should contain: an identification of the OHMs which have been selected as Contaminants of Concern, a summary of the analytical data which have been collected for these OHMs presented by specific environmental medium, and a description of the potential health effects which may be associated with exposure to each OHM.

The description of the potential health effects associated with each contaminant is provided in a ***Toxicity Profile***. A Toxicity Profile should be prepared for each Contaminant of Concern and presented in the documentation of the Risk Characterization.

Toxicity Profiles serve several purposes. They provide a summary of the potential adverse human health effects which may be associated with exposure to a particular contaminant and contain references for the dose-response assessment. Toxicity Profiles also serve as reference material for non-toxicologists who are involved with or interested in activities at the site and who want to understand the potential health impacts associated with contaminants at the site.

The information in Toxicity Profiles may also be used to group chemicals by health endpoint and mechanism of toxicity in order to estimate more detailed Hazard Indices. The reader should refer to Section 7.4.1 for more information on calculating endpoint-specific Hazard Indices.

In general, a Toxicity Profile is a comprehensive, in-depth profile of the toxicokinetics, human and animal mechanisms of toxicity, genotoxicity, carcinogenicity, and developmental/reproductive toxicity for the chemical of interest. A Toxicity Profile should also address Structure Activity relationships and interaction with other chemicals, as appropriate. In preparing the Toxicity Profile, the risk assessor should rely on credible, peer-reviewed sources of information such as controlled, epidemiologic investigations, clinical trials, experimental animal studies, metabolic and pharmacokinetic experiments, *in vitro* studies and structure-activity studies. All references should be provided to document the sources of information used to prepare the Toxicity Profile.

The scope and level of detail of a Toxicity Profile will vary depending upon the nature and quantity of information available for a particular chemical. For many substances (e.g., chemicals for which Method 1 standards have been developed) toxicological information is readily available from many sources, and repetition of that information in great detail in the Toxicity Profile is not necessary. For such cases a short descriptive summary of the known health effects associated with the chemical of interest and the basis for any existing standards or guidelines would be sufficient. The primary purpose of such a descriptive summary is to

provide information to the public in a readily available form.

7.2 DOSE RESPONSE ASSESSMENT

The dose-response assessment describes the observed effects in humans and/or laboratory animals associated with particular exposures (or doses) of the chemical of concern. This information is obtained from published literature describing epidemiologic or toxicologic studies involving the particular chemical. For most chemicals reported at c.21E disposal sites, the dose-response information needed to conduct a risk assessment may be found in secondary sources published by the USEPA or other government agencies, as described below.

The dose-response relationship(s) for each OHM which has been selected as a Chemical of Concern must be identified in the risk assessment report. This information is later coupled with knowledge of the nature and magnitude of potential exposures to characterize risk.

The dose-response information may be divided into three major categories:

- Toxicity information associated with threshold (non-carcinogenic) health effects.
- Toxicity information concerning carcinogenicity, either from human epidemiologic data or from laboratory studies.
- The Relative Absorption Factors (RAFs) used to relate the toxicity information identified from the literature to the exposure pathways of concern at the disposal site under investigation

> ***All*** the chemicals selected as Contaminants Of Concern should be evaluated for potential *non-carcinogenic* health effects. In addition, any substance considered to be a *known*, *probable*, or *possible* human carcinogen (as designated by EPA) should also evaluated for its potential carcinogenic effect. The classification of a chemical as a carcinogen does not preclude an evaluation of that same chemical for potential non-carcinogenic health risks.

7.2.1 Threshold Effects

For non-carcinogenic health effects, it is believed that a dose (or exposure) level exists at and below which no adverse health effects would be expected. Such a level is referred to as a ***threshold dose***. In theory, the threshold dose would be safe for all receptors who might be exposed at that level.

The goal of the dose response assessment is to identify the threshold dose, or a close approximation, given the toxicological information currently available. It may be impossible, however, to specify this theoretical threshold dose for a given chemical due to the inadequacy of the scientific data. Ideally, the threshold dose would be identified from large and well-run human epidemiological and toxicological studies. Unfortunately, such studies are uncommon as they are difficult to conduct, expensive, time-consuming, and often pose ethical concerns. It is possible to approximate this threshold dose in a health-protective manner that accounts for the data limitations by identifying a *sub-threshold* dose: such a value is typically derived from the ***No Observable Adverse Effects Level*** (NOAEL) of an animal study by application of uncertainty factors (UF) and a modifying factor (MF) (Farland and Dourson, 1992). Uncertainty Factors are applied to account for interspecies variation, exposure duration and protection of sensitive populations. Additional Uncertainty Factors may be applied if the toxicological study identified a ***Lowest Observable Adverse Effects Level***, or LOAEL, rather than a NOAEL. Each Uncertainty Factor is typically equal to a factor of ten, and the product of all the Uncertainty Factors may be as high as 10,000 (10 x 10 x 10 x 10). A Modifying Factor may be applied to reflect additional uncertainties in the critical study and the entire data base not addressed by the Uncertainty Factor. The value of the Modifying Factor is greater than zero and less than or equal to ten; the default value for the Modifying Factor is one. Important factors to consider when identifying and using such a sub-threshold dose include, at a minimum:

- the route of administration from the study (inhalation, oral, dermal contact, etc...);
- the duration of exposure to that dose (lifetime, chronic, subchronic, or acute exposure);
- the absorption efficiency (if any) used to calculate that dose; and
- the age of the person receiving the dose.

The subthreshold dose in units of mg/kg/day (with uncertainty spanning perhaps an order of magnitude or greater) to which daily exposure of a human population, including sensitive subgroups, is likely to be free of appreciable risk of deleterious effects during a lifetime is termed a ***Reference Dose*** (RfD) (Barnes and Dourson, 1988). The RfD is derived using the following equation:

$$RfD_{(mg/kg/day)} = \frac{NOAEL\ or\ LOAEL}{U.F.\ and/or\ MF} \qquad \textbf{(7-1)}$$

USEPA (1991) has also proposed a **Reference Dose for developmental toxicity** (RfD_{DT}). The RfD_{DT} is based on a NOAEL derived from short-duration exposures typically used in developmental studies. Uncertainty factors for developmental toxicity generally include a tenfold factor for interspecies variation and a tenfold factor for intraspecies variation; in general an uncertainty factor is not applied to account for duration of exposure. Additional uncertainty factors may be applied due to a variety of uncertainties in the data base (Farland and Dourson, 1992).

A ***Reference Concentration*** (RfC, in units of mg/m^3) is the inhalation exposure concentration (with uncertainty spanning perhaps an order of magnitude or greater) to which daily exposure of a human population, including sensitive populations, is likely to be free of appreciable effects. Interim methods for development of inhalation reference concentrations (USEPA, 1990) describe the conversion of the experimental exposure NOAEL to human equivalent concentrations ($NOAEL_{HEC}$). The conversion is specific both to the type of inhaled agent (particle or gas) and to the observed effect (respiratory or systemic) and adjusts for dosimetric differences between various experimental species and humans. Once the $NOAEL_{HEC}$ is identified, the same equation used to estimate the RfD is used to calculate the inhalation RfC with the application of similar, although not identical, uncertainty factors (Farland and Dourson, 1992). Conversion of an RfC to an inhalation RfD (in units of mg/kg/day) is not recommended.

There are a number of different sources of subthreshold toxicity values. When selecting toxicity information for use in quantitative risk assessment, the risk assessor should ensure that the information is appropriate for the assessment being conducted and that it is up-to-date. Note that sources differ in the frequency at which they are updated and the level of review they receive. The Massachusetts Contingency Plan requires that primary consideration be given to information developed by the U.S. Environmental Protection Agency (310 CMR 40.0993(5)(a)).

The following presents a list of sources of toxicity information in the order of preference:

(1) **Integrated Risk Information System (IRIS)** - IRIS is an USEPA data base that contains only those RfDs/RfCs which represent a consensus judgement of USEPA RfD/RfC Workgroup which is composed of scientists from various EPA offices and the Office of Research and Development. It is the preferred source of toxicity information. The IRIS database is updated monthly and is available on-line. For information on how to access IRIS, call IRIS user support at (513) 569-7254.

(2) **Health Effects Assessment Summary Tables (HEAST)** - HEAST is prepared by USEPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. HEAST contains almost entirely *provisional* toxicity values. These values have undergone review by individual USEPA program

offices, but are not recognized as agency-wide consensus values. HEAST is scheduled to be updated quarterly and can be obtained by contacting the National Technical Information Services (NTIS) Subscriptions Department at (703) 487-4630.

(3) **Other sources.** When information is not available in IRIS or HEAST, the following sources may be reviewed to determine whether comparable values exist and whether those values are appropriate for quantitative risk assessment.

Toxicity Values Developed by MADEP, ORS - The Office of Research and Standards develops chronic and subchronic RfDs and RfCs for some OHMs for which no values are available in IRIS or HEAST. These values are based on available toxicological data and standard USEPA approaches for developing reference doses for threshold effects. The list of chemicals includes a number of carcinogens for which USEPA has not derived non-cancer toxicity values. These values can be accessed through the MA DEP Bulletin Board.

Agency for Toxic Substances Disease Registry (ATSDR) - ATSDR produces Toxicological Profiles for 275 hazardous substances found at NPL sites. The priority list of hazardous substances is published in the Federal Register. An announcement of the release of draft Toxicological Profile documents appears in the Federal Register and the documents are available from ATSDR. Final toxicological profiles which incorporate reviewers comments, are available from the National Technical Information Service (NTIS) at (800) 553-6847 or (703) 487-4650.

In the toxicological profiles, ATSDR develops ***Minimal Risk Levels*** (MRLs) for threshold effects of some chemicals. These values are updated when the profiles are revised, if appropriate. An MRL is defined as an estimate of the daily human exposure to a substance that is likely to be free of appreciate risk of adverse noncancerous effects over a specified duration of exposure. MRLs are derived using the modified risk assessment methodology the U.S. EPA uses to derive reference doses and reference concentrations for lifetime exposure.

Allowable Threshold Concentration (ATC) - The "*Allowable Threshold Concentrations*" are values roughly equivalent to the reference concentration, but they are derived from the Threshold Effects Exposure Limit (TEL) described in CHEM (MA DEP, 1990). (The TEL value represents 20% of an allowable concentration, or ATC. Thus the ATC is equal to five times the TEL. The TEL was derived in a manner considering children to be the most sensitive potential receptors.) The ATC is a concentration of the chemical in air which would not be expected to result in adverse non-carcinogenic health effects. The ATC is derived considering acute and chronic threshold health endpoints, including reproductive effects. These values can be accessed through the MA DEP Bulletin Board.

Allowable Doses Back-Calculated From Drinking Water Standards and Guidelines - Drinking water standards and guidelines, which give the allowable concentration of a contaminant in drinking water supplies include: the Maximum Contaminant Level Goal (MCLG), the Maximum Contaminant Level (MCL), and Health Advisories (HAs). An allowable daily intake (ADI) comparable to an RfD may be obtained by back-calculation, using the same exposure assumptions used to develop the standard or guideline. It is imperative that the assumptions used to develop the standard or guideline be known before an RfD is calculated.

Back-calculating From Standards

When back-calculating from a concentration to a dose, the risk assessor must always use the exposure assumptions on which the concentration is based. For example, if a drinking water standard was derived using a body weight of 70 kg and a water intake rate of 2 liters/day, those factors must be used in back-calculating an allowable daily dose.

Site-specific exposure assumptions (such as a child's body weight and water intake rate) would then be considered in the risk assessment itself to evaluate the potential risk posed by the contamination.

A list of MCLs, MCLGs and HAs is available from USEPA by calling the Safe Drinking Water Hotline (1-800-426-4791). The list is updated twice per year. These values are also available in a chemical's IRIS file.

MCLGs - MCLGs are non-enforceable concentrations of a drinking water contaminant that are protective against adverse human health effects and allow an adequate margin of safety. MCLGs for substances considered to be carcinogenic are set at zero because USEPA assumes that any level of exposure is associated with some level of risk. MCLGs for substances not treated as known or probable human carcinogens are based upon chronic toxicity or other health data and applied uncertainty data. *Back calculation from the MCLG is only appropriate for use in the evaluation of compounds not considered Weight-of-Evidence Group A or B carcinogens.* Documentation for MCLGs is found in the preamble to the final rule for each OHM in the Federal Register.

MCLs - MCLs are the maximum permissible level of a contaminant in water which is delivered to any user of a public water system. MCLs are enforceable standards that are set as close to MCLGs as feasible. MCLs consider factors which are not strictly health based, such as treatment technology and cost. Thus, the basis for an MCL must be carefully examined before an MCL is used to derive an RfD. Generally, an MCL is not used to derive an RfD.

Health Advisories - Health Advisories (HAs) describe concentrations of drinking water contaminants at which adverse non-carcinogenic health effects would not be expected to occur over specific exposure durations. HAs are developed for 1-day, 10-day, longer term (generally up to 2 years), and lifetime exposures based only on data describing non-carcinogenic endpoints of toxicity.

For those substances which are known or probable human carcinogens, HAs for lifetime exposure are not derived. The documentation for each HA should be consulted before proceeding with any calculations. Documentation for HAs is available through the Education Research Information Clearinghouse (ERIC), (614) 292-6717.

Allowable Doses Back-Calculated From Ambient Water Quality Criteria - Ambient Water Quality Criteria (AWQC) are developed by the USEPA Office of Water Regulations and Standards per Section 304(a)(1) of the Clean Water Act of 1977. The AWQC consider both toxicity to aquatic life and human health effects. The AWQC do not consider technical feasibility or cost and may be used to derive a chronic sub-threshold dose for use in a risk assessment. However, it must be noted that the AWQC incorporate factors which account for exposure via both drinking water ingestion and consumption of contaminated fish. The documentation for each AWQC should be consulted before proceeding with any calculations and are available through the National Technical Information Service (NTIS) at (800) 336-4700. Individual AWQC are listed in IRIS.

(4) **Calculation of a dose-response value using toxicity information from the literature.** Dose-response values may be derived by a qualified risk assessor or toxicologist if none of the above sources provides a toxicity value, but adequate toxicity studies are available, *or* if more recent, credible and relevant data becomes available. USEPA approaches to development of RfDs are described in *Risk Assessment Guidance for Superfund* (USEPA, 1989) and in Appendix A to IRIS. Approaches to the development of RfCs are described in *Interim Methods for Development of Inhalation Reference Doses* (USEPA, 1991). The review and approval by the Department of such a proposed value would depend upon the justification and documentation provided to support it. The development of an alternative value when a USEPA or MA DEP derived reference dose or reference concentration is available is rarely justifiable and the risk assessor should contact the MA DEP Office of Research and Standards early on in the site assessment process for prior approval before proceeding.

7.2.2 Carcinogenic Effects

Unlike non-carcinogenic health effects, the dose-response assessment for carcinogens assumes that there is no threshold dose for carcinogenicity; that there is no dose of a carcinogenic substance (other than no exposure) which is associated with zero risk. USEPA evaluates available toxicity data and, based on this evaluation, the chemical is assigned to a weight-of-evidence class. The system for characterizing the overall weight of evidence for a chemical's carcinogenicity developed by USEPA is based on the availability of animal, human, and other supportive data (USEPA, 1986). The weight-of-evidence classification rates the likelihood that an agent is a human carcinogen, and it may qualitatively affect the interpretation of potential health risks. Three major factors are considered in characterizing the overall weight-of-evidence for carcinogenicity: (1) the quality of evidence from human studies, (2) the quality of evidence from animal studies,

and (3) other supportive information, such as mutagenicity data and structure-activity data. The five categories of the USEPA's final classification scheme (adapted from an approach taken by the International Agency for Research on Cancer) are described in Table 7.1.

Table 7.1

USEPA Weight of Evidence Classification

Group A - Human Carcinogen: This category indicates there is sufficient evidence from epidemiological studies to support a causal association between an agent and human cancer.

Group B - Probable Human Carcinogen: This category generally indicates there is at least limited evidence from epidemiologic studies of carcinogenicity to humans (Group B1) or that, in the absence of data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).

Group C - Possible Human Carcinogen: This category indicates that there is limited evidence of carcinogenicity in animals in the absence of data on humans.

Group D - Not Classified: This category indicates that the evidence for carcinogenicity in animals is inadequate, or no data are available.

Group E - No Evidence of Carcinogenicity to Humans: This category indicates that there is evidence of noncarcinogenicity in at least two adequate animal tests in different species or in both epidemiologic and animal studies.

The ability of a chemical to increase the incidence of cancer in a target population is described by one of two measures: the cancer ***slope factor*** or the ***unit risk***. Cancer Slope Factors or Unit Risks are typically calculated for chemicals in Groups A, B1 and B2. Slope factors for chemicals in Group C are calculated on a case-by-case basis.

The cancer Slope Factor (CSF) for a chemical is derived by the USEPA's Cancer Assessment Group (CAG). Using mathematical extrapolation models, commonly the linearized multistage model, the largest possible linear slope (within the 95% Confidence Limit) consistent with the available data is estimated at low extrapolated doses. For some chemicals, human epidemiologic data are the basis of an estimate of the carcinogenic potency, although the most common basis of these values is an animal study. The CSF is expressed as the risk per unit dose, and is typically given in units of $(mg/kg/day)^{-1}$. Use of the slope factor assumes that the calculated dose received is expressed as a lifetime average.

The Unit Risk (UR) is the upper 95% Confidence Limit of the mean incremental lifetime cancer risk estimated to result from lifetime exposure to an agent if it is in the air at a concentration of 1 $\mu g/m^3$ or in the drinking water at a concentration of 1 $\mu g/L$. These values are used in lieu of the chemical's slope factor when an estimate of a lifetime average concentration of the chemical is available.

There are a number of different sources of CSFs and URs. When selecting this information

for use in quantitative risk assessment, the risk assessor should ensure that the information is appropriate for the assessment being conducted and that it is up-to-date. Note that sources differ in the frequency at which they are updated and the level of review they receive. The Massachusetts Contingency Plan requires that primary consideration be given to information developed by the U.S. Environmental Protection Agency (310 CMR 40.0993(5)(a)).

Preferred sources for cancer slope factors or unit risk values are:

(1) **Integrated Risk Information System (IRIS)** - The IRIS data base contains only those CSFs or URs which represent a consensus judgement of the USEPA Carcinogen Risk Assessment Verification Endeavor (CRAVE) which is composed of scientists from various EPA offices and the Office of Research and Development. It is the preferred source of toxicity information. The IRIS database is updated monthly and is available on-line. For information on how to access IRIS, call IRIS user support at (513) 569-7254.

(2) **Health Effects Assessment Summary Tables (HEAST)** - HEAST contains values that have received some form of review by USEPA, but have not been verified and are considered provisional. HEAST is prepared by USEPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. HEAST is scheduled to be updated quarterly and can be obtained by contacting the National Technical Information Services (NTIS) Subscriptions Department at (703) 487-4630.

(3) **Other Sources** - When information is not available in IRIS or HEAST, the following sources may be reviewed to determine whether comparable values exist and whether those values are appropriate for quantitative risk assessment.

California Environmental Protection Agency (Cal/EPA) - Cal/EPA's Office of Environmental Health Hazard Assessment (OEHHA), Department of Pesticide Regulation (DPR) and Department of Toxic Substances Control (DTSC) develop or approve cancer potency factors for use in risk assessments and as the basis for regulatory action. A list of available cancer potency factors is revised semiannually and can be obtained from OEHHA's Hazardous Waste Toxicology Section, at (916) 324-7572.

Toxicity Values Developed by MA DEP/ORS - The Office of Research and Standards may develop CSFs and URs for chemicals for which no values are available in IRIS or HEAST. When available, these values can be accessed through the MA DEP Bulletin Board.

(4) **Calculation of a slope factor or unit risk value using toxicity information from the literature.** CSFs and URs may be derived by a qualified risk assessor or toxicologist if none of the above sources provides a toxicity value, but adequate toxicity

studies are available, *or* if more recent, credible and relevant data becomes available. USEPA approaches to development of cancer slope factors are described in several documents (USEPA, 1989a; USEPA, 1986) and in Appendix B to IRIS. The review and approval by the Department of such a proposed value would depend upon the justification and documentation provided to support it. The development of an alternative value when a USEPA derived CSF or UR is available in IRIS or HEAST is rarely justifiable and the risk assessor should contact the MA DEP Office of Research and Standards early on in the site assessment process for prior approval before proceeding.

7.2.3 Relative Absorption Factors (RAFs)

The Relative Absorption Factor (RAF) is used to account for differences in the absorption of a COC under assumed exposure conditions at the site (exposure route and matrix) relative to the absorption of the COC under the experimental conditions upon which the dose-response value is based. RAFs are used *in lieu of absorption efficiencies* to ensure that the exposures evaluated at the disposal site are comparable to the toxicity information identified in the literature.

The reference doses, reference concentrations, slope factors and unit risks used in quantitative risk assessment are typically based upon controlled laboratory experiments in which animal test species are exposed in some manner to the chemical under study. Many important features vary from study to study: the test animal may vary (e.g., mice, rats, rabbits or even humans may be used); the chemical may be administered orally, dermally, via inhalation or injected; and the material may be administered in different matrices (e.g., neat, dissolved in oil or mixed with food). At disposal sites, the exposures of concern also vary widely and rarely correspond to the exact conditions under which the toxicity information was derived. Typical site-related exposure pathways include the incidental ingestion of contaminated soil by young children and the dermal absorption of a substance from surface water.

The RAF is used to adjust the calculated exposure (e.g., the soil ingestion exposure of a child) in such a way that it is comparable to the toxicity information (e.g., derived from a study in which rats were administered by gavage a chemical dissolved in olive oil).

A unique RAF should be determined or estimated for a chemical for each combination of toxicity value and route of exposure. This means that multiple RAFs may be required in order to conduct the quantitative risk assessment. To estimate an RAF, two factors must be identified:

- the absorption efficiency for the chemical via the route and medium of exposure being evaluated for the disposal site, and

- the absorption efficiency for the route and medium of exposure in the experimental study which is the basis of the dose-response value for the chemical in question.

Thus, the RAF adjusts the dose (or exposure) estimates based on these ***two*** absorption efficiencies. The RAF is calculated as follows:

$$RAF = \frac{Absorption\ Efficiency_{SITE\ route/medium\ of\ exposure}}{Absorption\ Efficiency_{STUDY\ route/medium\ of\ exposure}} \qquad \textbf{(7-2)}$$

It is *very* important to determine whether the toxicity value is based on a *absorbed* or *applied* dose. The above equation is for a dose response value based on an applied dose. If the dose response value has been derived from an absorbed dose, then the RAF is simply equal to the absorption efficiency via the route and medium under consideration.

An example of the calculation of an RAF for dermal exposure to benzo(a)pyrene (carcinogenic effects) in soil is presented in Example 7.1 (taken from MADEP, 1992b).

RAFs developed by MADEP Office of Research and Standards staff are available through the MA DEP's Risk Assessment Bulletin Board. A number of DEP derived RAFs are listed in the Toxicity Information section of the *Risk Assessment ShortForm - Residential Scenario* and accompanying documentation (MADEP, 1992b). USEPA's Risk Assessment Guidance for Superfund (1989a), Appendix A also provides guidance for the "Adjustments For Absorption Efficiency" - a process similar to the development of RAFs.

The risk assessor is reminded that an absorption efficiency (or absorption factor) which does not consider derivation of the toxicity values (Reference Dose, Reference Concentration, Slope Factor or Unit Risk) is <u>not</u> an RAF.

7.2.4 Groups of Chemicals

The discussion in this section has focused on the toxicity information available for specific chemicals. There are several groups of closely related compounds for which alternative approaches to the identification of dose-response values have been proposed and specific guidance has been requested. These groups include:

- Chlorinated dioxins and furans
- Polycyclic Aromatic Hydrocarbons (PAHs)
- Polychlorinated Biphenyls (PCBs)
- Total Petroleum Hydrocarbons (TPH)

Approaches to evaluating the toxicity of each of these groups is described below.

EXAMPLE DERIVATION:
RAF for the Cancer Risk Evaluation of Site Soil Dermal Exposures

The oral slope factor for benzo(a)pyrene (B[a]P) is listed in IRIS as 7.3 $(mg/kg/day)^{-1}$ and is based on a dietary study in mice. The oral absorption of ^{14}C-labeled B[a]P, dissolved in peanut oil and administered by gavage, was studied in rats (Hecht et al., 1979). Absorption was determined by recovery of label in urine and feces. Unchanged B[a]P recovered in feces was estimated at 9% of the total dose, with all other fecal radioactivity (85% of applied dose) recovered as metabolites. This suggests an oral absorption efficiency of 91%.

The percutaneous absorption of ^{14}C-B[a]P was studied in vivo in Swiss Webster mice (Sanders et al., 1986) and in Sprague-Dawley rats (Yang et al., 1986). Absorption was determined by analyzing radioactivity in urine, feces and tissues, and by analysis of residual label at the site of application. Dermal absorption efficiency was measured as 40% (in mice) and 6% (in rats) in 24 hrs. The higher value of 40% is selected as a protective estimate for human dermal exposure to pure compound. In vitro estimates are lower, ranging from 0.1%-15% in humans and animals (Kao et al., 1985; Kao et al., 1988) and are not considered applicable to human exposure. The in vivo percutaneous absorption of soil-adsorbed B[a]P was determined in rats by Yang et al. (1989). The range of absorbed doses was 1.3% - 9.2% depending on the amount of soil applied. More efficient absorption occurred at lower soil application rates. Wester et al. (1990) confirms a low absorption for soil-associated B[a]P in the rhesus monkey with a range of 9% - 18%. The upper limit of 18% is selected as a protective estimate for human exposure to B[a]P contaminated soil.

The dermal penetration of B[a]P, applied as a complex organic mixture, seems to be representative of the dermal penetration of other PAHs examined in this study (Dankovic et al., 1989) including pyrene, benzanthracene, benzofluorene, methylchrysene, chrysene, benzofluoranthene and benzo[e]pyrene. The disappearance half-life of B[a]P was 6.7 hours with the other PAHs ranging from 5.0 - 8.8 hours. The disappearance half-life of B[a]P was decreased to 3 hours when pure B[a]P was applied to skin in acetone. These data suggest a 50% decrease in dermal absorption of B[a]P when applied as an environmental mixture (20%) rather than as neat compound (40%). This compares closely with the upper limit of 18% dermal absorption efficiency selected from the study of Wester et al.(1990) for soil-associated B[a]P.

The RAF specific to the cancer risk evaluation of for soil dermal contact exposures would be the ratio:

$$\text{Absorption Efficiency}_{\text{B[a]P from soil via dermal contact}} \div \text{Absorption Efficiency}_{\text{B[a]P via oral exposure}}$$

$$RAF = 0.18 \div 0.91 = 0.2$$

7.2.4.1 Chlorinated Dioxins and Furans

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzifurans (PCDFs) comprise a family of chemicals containing 210 specific monochlorinated and polychlorinated congeners. In 1987, USEPA formally adopted an interim procedure for estimating risks associated with complex environmental mixtures containing PCDDs and PCDFs (Bellin and Barnes, 1987). The procedure used a set of **toxicity equivalency factors** (TEFs) to convert the concentration of congeners into an equivalent concentration of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), the most toxic of the 210 congeners. The TEFs have been reviewed and updated periodically, the most recent update being USEPA (1989b) and MADEP (Silverman and Hutcheson, 1991).

A list of current TEFs is presented in Table 7.2. Documentation of the derivation of these toxicity equivalency factors is available from the MADEP Office of Research and Standards and may be accessed through the MA DEP Bulletin Board.

7.2.4.2 Polycylic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons are a class of structurally similar chemical compounds characterized by the presence of fused aromatic rings. PAHs

Table 7.2
MADEP Derived Toxicity Equivalency Factors (TEFs) for Polychlorinated Dioxins and Dibenzofurans

Compound	TEF
DIOXINS:	
Mono-, Di- and Trichlorinated dibenzo-p-dioxins	0.001
2,3,7,8-Tetrachlorinated dibenzo-p-dioxin	1
Other tetrachlorinated dibenzo-p-dioxins	0.01
2,3,7,8-Pentachlorinated dibenzo-p-dioxins	0.5
Other Pentachlorinated dibenzo-p-dioxins	0.05
2,3,7,8-Hexachlorinated dibenzo-p-dioxins	0.1
Other Hexachlorinated dibenzo-p-dioxins	0.01
2,3,7,8-Heptachlorinated dibenzo-p-dioxins	0.1
Other Heptachlorinated dibenzo-p-dioxins	0.01
Octochlorinated dibenzo-p-dioxin	0.001
FURANS:	
Mono-, Di- and Trichlorinated dibenzofurans	0.001
2,3,7,8-Tetrachlorinated dibenzofuran	0.1
Other Tetrachlorinated dibenzofurans	0.01
2,3,7,8-Pentachlorinated dibenzofurans	0.5
Other Pentachlorinated dibenzofurans	0.05
2,3,7,8-Hexachlorinated dibenzofurans	0.1
Other Hexachlorinated dibenzofurans	0.01
2,3,7,8-Heptachlorinated dibenzofurans	0.1
Other Heptachlorinated dibenzofurans	0.01
Octochlorinated dibenzofurans	0.001

from MADEP (Silverman and Hutcheson, 1991)

are typically formed during the incomplete burning of organic material including coal, oil, gasoline and garbage. PAHs are also found in crude oil, coal tar, creosote and asphalt. PAHs are associated with human activities (the combustion of fossil fuels) and natural occurrences (such as forest fires), and they are considered to be ubiquitous in the environment at some level.

PAHs are often discussed as a group because they are commonly found as mixtures of two or more compounds in the environment. In addition, they are often treated similarly in risk assessments due to their similar structures and toxicities. It should be noted that, while PAHs are often discussed as a group, the individual chemicals are evaluated as separate chemicals in the risk characterization. There are over 100 chemicals in this family of compounds, although a smaller number are routinely reported at disposal sites (Table 7.3). ***The PAH's which are often present at sites but are unreported may result in the <u>underestimation</u> of potential risks.***

Table 7.3

PAH's Commonly Reported at c.21E Disposal Sites and Carcinogenicity Weight-of-Evidence Classifications

	USEPA[1]	IARC[2]
Acenaphthene	N/A	N/A
Acenaphthylene	D	N/A
Anthracene	D	3
Benz(a)anthracene	B2	2A
Benz(a)pyrene	B2	2A
Benzo(e)pyrene	N/A	3
Benzo(b)fluoranthene	B2	2B
Benzo(g,h,i)perylene	N/A	3
Benzo(j)fluoranthene	N/A	2B
Benzo(k)fluoranthene	B2	2B
Chrysene	B2	3
Dibenz(a,h,)anthracene	B2	N/A
Fluoranthene	D	3
Fluorene	N/A	3
Indeno(1,2,3-cd)pyrene	B2	2B
2-Methylnaphthalene	N/A	N/A
Naphthalene	D	3
Phenanthrene	D	3
Pyrene	D	3

1 - U.S. Environmental Protection Agency. **B2**: Probable Human Carcinogen; **D**: Not Classifiable
2 - International Agency for Research on Cancer. **2A**: Probable Human Carcinogen; **2B**: Possible Human Carcinogen; **3**: Not Classifiable
N/A - Not Available

Among the polycyclic aromatic hydrocarbons, the USEPA (IRIS, 1993) has classified seven chemicals as *probable human carcinogens* (identified in Table 7.3 as USEPA Class B2). The classification of PAHs by the International Agency for Research on Cancer (IARC) is fairly consistent with that of the EPA. PAH's which are considered <u>unclassified</u> (either N/A, D or 3 in Table 7.3) may also contribute to carcinogenic risk (Nisbet and LaGoy, 1992) and should not necessarily be assumed to be "*noncarcinogens*" which would be USEPA Class <u>*E*</u>.

All PAHs identified as contaminants of concern should be evaluated in terms of potential noncancer risk. ***Remember that the carcinogenic PAHs may also be associated with noncancer health effects and must be included in this evaluation.***

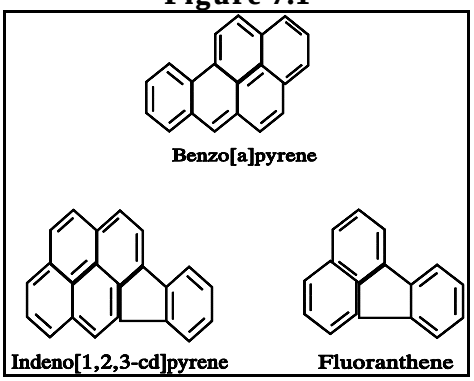
Historically, risk assessments involving PAHs become problematic due to the general lack of toxicity information available for many of the compounds reported at disposal sites. The following paragraphs discuss the MA DEP recommended approaches for the evaluation of cancer and noncancer risk of harm posed by exposure to polycyclic aromatic hydrocarbons.

PAH Cancer Risk:

Until recently the only cancer slope factor the USEPA published for PAH's was for the chemical benzo[a]pyrene (B[a]P). In the absence of further chemical-specific information, the EPA and MADEP guidance instructed risk assessors to assign the B[a]P slope factor to all PAHs considered to be carcinogenic. This approach was considered to be protective of public health as benzo[a]pyrene is thought to be one of the most potent carcinogens among the PAH's. In 1993, USEPA formally adopted provisional guidance for estimating cancer risks associated with polycyclic aromatics hydrocarbons (USEPA, 1993). The procedure uses information from the scientific literature to estimate the carcinogenic potency of several PAHs relative to benz[a]pyrene. These **relative potencies** may be used to modify the CSF developed for benzo[a]pyrene for each PAH, or to calculate B[a]P-equivalent concentrations for each of the PAH's (which would then be used with the B[a]P slope factor). The latter approach is similar to that used for the evaluation of dioxins.

The relative potency values published by the USEPA and others (Chu and Chen, 1984; Clement, 1988; Nisbet and LaGoy, 1992) are being reviewed and may be adopted (perhaps in a modified form) by MA DEP Office of Research and Standards. A list of the USEPA relative potency values is presented in Table 7.4 for use in c.21E risk characterizations pending publication of MADEP recommended values (which will be available through the MA DEP Bulletin Board System).

Figure 7.1



PAH Noncancer Risk:

While the USEPA has published (in *IRIS* and *HEAST*) threshold effects toxicity information for a number of polycyclic aromatic hydrocarbons, for many other members of this chemical family such information has not yet been developed. In order to adequately characterize the noncancer risks associated with these PAHs, MADEP recommends that the published reference dose, reference concentration, or analogous value for a structurally similar PAH be adopted for each compound for which sufficient chemical-specific toxicological information is unavailable.

Examples of how the potential toxicity of individual PAHs may be evaluated are described in Example 7.2.

7.2.4.3 Polychlorinated Biphenyls (PCBs).

Polychlorinated biphenyls (PCBs) is the name given to the general class of compounds in which one or more chlorine atoms are bonded to a biphenyl structure (Figure 7.2). The PCB family is comprised of 209 different variants, or **congeners**, depending upon the number of chlorine atoms present and their position on the biphenyl structure. PCBs may also be described according to *isomeric groups*, which are families of PCBs having the same number of chlorine atoms and thus the same molecular weight. For example, 2,2'-Dichlorobiphenyl is one of 209 chlorinated biphenyl congeners and one of 12 possible dichlorobiphenyls; these 12 dichlorobiphenyls are considered **isomers** of each other.

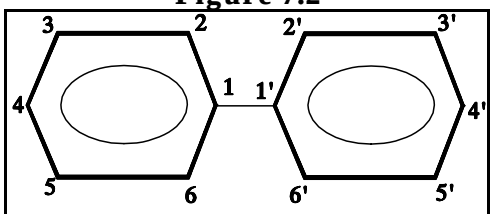
Table 7.4
Relative Potency Values for Individual PAH's: (USEPA, 1993)

Compound	Relative Potency Factor
Acenaphthene	NA
Acenaphthylene	NA
Anthracene	NA
Benz(a)anthracene	0.1
Benz(a)pyrene	1
Benzo(b)fluoranthene	0.1
Benzo(g,h,i)perylene	NA
Benzo(k)fluoranthene	0.01
Chrysene	0.01
Dibenz(a,h,)anthracene	1
Fluoranthene	NA
Fluorene	NA
Indeno(1,2,3-cd)pyrene	0.1
2-Methylnaphthalene	NA
Naphthalene	NA
Phenanthrene	NA
Pyrene	NA

NA - Chemical is not currently considered to be carcinogenic by USEPA so no relative potency value is currently applicable.

PCBs are typically found in the environment as mixtures of different PCB congeners. These mixtures (also known as ***Aroclors***, a trade name of the Monsanto Corporation) are identified by a four digit numbering code in which the first two digits (12) indicate that the parent molecule (the biphenyl) has twelve carbon atoms, and the last two digits indicate the percent chlorine by weight. Thus, Aroclor 1260 is a chlorinated biphenyl mixture with an <u>average</u> chlorine content of 60%. [The only exception to this nomenclature is Aroclor 1016, which retains the name by which it was known during development. Aroclor 1016 is a mixture which has an average chlorine percentage of 41.5%, making it very similar to Aroclor 1242.] It is important to note that an Aroclor mixture may contain dozens of individual PCB congeners representing several isomeric groups.

Figure 7.2



The general chemical structure of
Polychlorinated Biphenyls

Example 7.2

EVALUATION OF POLYCYCLIC AROMATIC HYDROCARBONS (PAH's)

Cancer Risk

A polycyclic aromatic hydrocarbon for which a cancer slope factor has not been developed by USEPA may be evaluated using the relative potency values recommended by USEPA (Table 7.4). These values can be used in one of two ways which are mathematically equivalent. To illustrate, let's assume that **Indeno[1,2,3-cd]pyrene** was reported at a disposal site at a concentration of **2 mg/kg**.

- In the first approach, the relative potency factor for indeno[1,2,3-cd]pyrene (**0.1**, from Table 7.4) is used to estimate a cancer slope factor for this compound by adjusting the slope factor for benzo[a]pyrene (**7.3 mg/kg/day**, from USEPA *IRIS*, 1993):

$$CSF_{i[1,2,3\text{-}cd]p} = 0.1 \times 7.3\ (mg/kg/day)^{-1} = 0.73\ (mg/kg/day)^{-1}$$

- The second approach would be to adjust the concentration of indeno[1,2,3-cd]pyrene (**2 mg/kg**, in this example) by the relative potency value (**0.1**, from Table 7.4) to estimate a benzo[a]pyrene equivalent concentration, to which the B[a]P slope factor would be applied:

$$B[a]P_{equiv.\ conc.} = 0.1 \times 2\ mg/kg = 0.2\ mg/kg$$

Noncancer Risk

A polycyclic aromatic hydrocarbon for which a reference dose (RfD) has not been developed by USEPA may be evaluated using a reference dose from a structurally similar PAH. Using the example above, indeno[1,2,3-cd]pyrene (for which there is currently no RfD) is structurally similar to fluoranthene: both chemicals have a 5-carbon ring structure bound to three aromatic rings, although indeno[1,2,3-cd]pyrene has two additional aromatic rings (see Figure 7.1). The reference dose for fluoranthene is 0.04 mg/kg/day (USEPA *IRIS*, 1993). This value would be adopted to evaluate potential noncancer risks associated with indeno[1,2,3-cd]pyrene.

As described earlier in this section, MADEP relies heavily upon the work of the USEPA and its published collection of agency-reviewed toxicity information published primarily in the *Integrated Risk Information System* (IRIS) and the *Health Effects Assessment Summary Tables* (HEAST). While it is generally unnecessary to duplicate the USEPA's efforts in developing toxicity information, the DEP Office of Research and Standards has staff toxicologists to fill data gaps or review supplemental information. The following is a summary of MADEP's general approach to the selection of toxicity information:

- When it exists , MADEP recommends the use of USEPA toxicity information from *IRIS* or *HEAST* for a given chemical.

- For *mixtures* of chemicals, the USEPA may publish toxicity information for the mixture as a whole or for *some* constituents of the mixture. When information is only available for certain formulations of a mixture, or for a limited number of constituents of a mixture, MADEP must, *as a matter of science policy*, determine how the limited information should be extrapolated to (a) other formulations of the mixture, or (b) the mixture as a whole.

For the evaluation of polychlorinated biphenyls, MADEP has specific policies based upon the information available at the time that this document was prepared. The reader is urged to consult the MADEP Office of Research and Standards or the MADEP Risk Assessment Bulletin Board for the current status of this information. The MADEP/ORS recommends the following:

- the use of the USEPA derived CSF of 7.7 $(mg/kg/day)^{-1}$ for all PCB mixtures. "*Although it is known that PCB congeners vary greatly as to their potency in producing biological effects, for purposes of this carcinogenicity assessment, Aroclor 1260 is intended to be representative of all PCB mixtures.*" (USEPA *IRIS* file for PCBs, 1993)

- the use of the Aroclor-specific USEPA derived chronic, oral reference dose of 7×10^{-5} mg/kg/day for Aroclor 1016 (USEPA *IRIS* file for Aroclor 1016, 1993). This value may also be applicable to PCB mixtures containing similarly chlorinated congeners, such as Aroclor 1242.

- the use of the Aroclor-specific USEPA derived chronic, oral reference dose of 2×10^{-5} mg/kg/day for Aroclor 1254 (USEPA *IRIS* file for Aroclor 1254, 1994). This value may also be applicable to PCB mixtures containing similarly chlorinated congeners, such as Aroclor 1260.

- the use of other Aroclor-specific USEPA derived values, as they become available.

7.2.4.4 Total Petroleum Hydrocarbons.

The Total Petroleum Hydrocarbon (TPH) measure often reported for c.21E disposal sites is generally considered inadequate for the purposes of site specific risk assessment. The commonly used infra-red (IR) analysis technique does not identify individual compounds or related groups of constituents. The mixture of petroleum hydrocarbons reported as the TPH parameter includes a wide range of compounds of different toxicities. Thus, the health effects (or the risk of such effects) associated with exposure to particular concentrations of "TPH" cannot be determined.

The MADEP Bureau of Waste Site Cleanup is developing a "*Policy for the Investigation,*

Assessment and Remediation of Petroleum Releases" (or the Petroleum Policy) which will include a section entitled "*Interim Final Petroleum Report: Development of a Health Based Alternative to the TPH Parameter*." That document identifies an alternative to the TPH parameter which can be used to conduct site-specific risk assessments and the document will propose dose-response values to be used with the specified analytical parameters. The key element of the policy is that the proposed analytical technique would allow the quantification of several ranges of compounds (rather than a single TPH result) and each range would be assigned a "reference compound" whose toxicity would be representative for all chemicals in that range.

The interim final report, *Development of a Risk Based Alternative to the TPH Parameter* (MADEP, 1994a) is currently available through the MA DEP Bulletin Board and the State Bookstore.

7.2.5 Recommended Format

Tables 7.5 and 7.6 present recommended formats for presentation of dose-response information for threshold and nonthreshold effects, respectively.

For threshold effects, separate tables should be presented for chronic and subchronic effects. Information that should be presented in the table includes:

- Name
- Toxicity value
- Source of toxicity value (i.e IRIS, HEAST)
- Date that the toxicity value was last verified
- Study Type - how the OHM was administered
- Confidence Level - identified by USEPA
- Critical Effect - target organ and toxic effect on which the dose-response value is based
- Test Animal - animal species on which the study is based
- Uncertainty of modifying factors - factors listed by agency generating the toxicity value

For nonthreshold effects, the information that should be presented in the table includes:

- Name
- Potency Value or Unit Risk
- Source of toxicity value (i.e IRIS, HEAST)
- Date that the toxicity value was last verified
- Study Type - how the OHM was administered
- Weight of Evidence - USEPA weight of evidence classification
- Test Animal - animal species on which the study is based
- Cancer type - tumor type listed by the agency establishing the toxicity value

7.3 EXPOSURE ASSESSMENT - CONCEPTS

The exposure assessment is a critical component of the site assessment process as it describes, both qualitatively and quantitatively, the contact between the contamination and the people who are potentially affected by the contamination. The exposure assessment must be consistent with the primary questions asked in the risk characterization process:

Given the current and identified foreseeable uses of the site, would the oil or hazardous material present pose significant risk of harm to health, safety, public welfare or environment if no further remedial action were to occur?

or

If a proposed remedial alternative is implemented and meets its identified remediation goals, will a condition of no significant risk of harm to health, safety, public welfare and the environment be achieved given the current and identified foreseeable uses of the site?

Whether the risk characterization is a **baseline** assessment (which answers the first question) or an evaluation of a proposed remedial alternative, the exposure assessment must incorporate site conditions associated with both current use and identified foreseeable uses of the site and surrounding environment. In this context site *use* or site *activity* are short-hand references for the exposures to site contaminants which could occur at or near the disposal site.

There are two important results of the exposure assessment: exposure profiles and quantitative estimates of exposure. An exposure profile is a narrative description of the exposures which may occur at the disposal site, and the information is often summarized in one or more tables for easy reference. The quantification of exposure translates the narrative exposure profile into a series of exposure equations resulting in numerical estimates of exposure. These numerical estimates are subsequently used in the risk calculations.

EXAMPLES: Dose-Response Summary Tables

Table 7.2.5

Chronic Oral Reference Doses

Chemical Name	CAS Number	Chronic Oral RfD	Source	Date Last Verified	Study Type	Confidence Level	Target Organ/ Critical Effect	Test Animal	Uncertainty/ Modifying Factors
Carbon Tetrachloride	56-23-5	7E-04	IRIS	1/94	Gavage, 12 weeks	Medium	Liver/Lesions	Rat	1,000
c-1,2-Dichloroethylene	156-59-2	1E-02	HEAST	1/94	Gavage, 90 day	N/A	Blood/Decreased Hematocrit	Rat	3,000
Dichloromethane	75-09-2	6E-02	IRIS	1/94	Drinking Water, 2-year	Medium	Liver/Liver Toxicity	Rat	100

Table 7.2.6

Oral Cancer Slope Factors

Chemical Name	CAS Number	Oral CSF	Source	Date Last Verified	Study Type	Weight of Evidence	Tumor Type	Test Animal(s)
Carbon Tetrachloride	56-23-5	1.3E-01	IRIS	1/94	Gavage	B2	Hepatocellular carcinomas/hepatomas	Hamster Mouse Mouse Rat
p-Cloronitrobenzene	100-00-5	1.8E-02	HEAST	1/94	Diet	B2	Cardiovascular System Tumors	Mouse
Dichloromethane	75-09-2	7.5E-03	IRIS	1/94	Inhalation Drinking Water	B2	Hepatocellular adenomas or carcinomas Hepatocellular cancer and neoplastic nodules	Mouse Mouse

Baseline Risk Characterizations

Baseline risk characterizations evaluate the "***no action***" alternative: What risks would be posed by the contamination if no remedial action were taken? If risk reduction measures have already been completed, then the baseline risk characterization would evaluate the risks if no *further* remedial action were taken.

Anticipated or proposed remedial actions or land use restrictions should never be incorporated into a baseline risk characterization, as it would no longer be an evaluation of the "no action" alternative. By extension, completed Immediate Response Actions (IRS's), Release Abatement Measures (RAM's) or Utility-related Abatement Measures (URAM's) can be considered in a baseline risk characterization ***only if they are considered to be permanent***.

For example, temporary fencing of an area as an Immediate Response Action to eliminate direct contact with contaminated soils should not be incorporated into a baseline risk characterization. Rather, the conditions which would exist in the absence of the IRA should be evaluated to determine the need for a permanent solution: the exposure assessment would assume that no fence is in place. If, however, a completed IRA, RAM or URAM permanently changes the exposure potential at a disposal site (e.g., the complete removal and disposal of contaminated soil), that impact of that *permanent* response action would be considered in the baseline assessment.

7.3.1 Development of Exposure Profiles

Exposure profiles provide the narrative description of how exposure takes place at the disposal site. The exposure profiles assist the risk assessor in identifying appropriate values for the exposure variables (such as intake rate, frequency of exposure, etc...) by providing a context within which the variables have meaning. Exposure profiles are sometimes referred to as "*exposure scenarios*".

An exposure profile should be developed for each of the receptors identified for all current and foreseeable uses of the site. The number and content of the exposure profiles will vary from site-to-site, reflecting the nature and complexity of the exposures which may occur.

There are also several ways to streamline this process and minimize the number of exposure profiles needed. If the current use of the site is assumed to remain unchanged into the future, then separate exposure profiles need not be developed for both the current and future receptors. For example, if a residential area is being evaluated and the land is likely to remain residential, it is unnecessary to construct exposure profiles to represent other uses. For a property where the frequency and intensity of exposure is low, it is also possible to assume that the use and activities will remain the same, but this assumption requires an activity and use limitation, as detailed in Section 2.1 of this Guidance Document.

Another situation conducive to streamlining exposure profiles is when two (or more) hypothetical receptors share the same exposures but the magnitude of exposure is demonstrably greater for one. In this case, a detailed exposure profile may be developed for the highly exposed receptor, accompanied by the conclusion that lesser exposed receptors will also be protected.

The USEPA *Guidelines for Exposure Assessment* (1992) describes exposure scenarios (exposure profiles) as containing the "*facts, data, assumptions, inferences, and sometimes professional judgement*" about how the exposures take place. Since these factors determine the magnitude of exposure (and thus the magnitude of the risk posed by the disposal site) it is important that there be a clear description and summary of this information. The exposure profiles allow anyone concerned about the disposal site to read and understand what was considered in the risk characterization and what was the basis for the decision on the need for remedial action.

Risk Characterizations for Remedial Alternatives

A risk characterization for a remedial alternative is performed to determine whether that action will achieve (if the alternative is *proposed*) or has achieved (if the alternative has been *implemented*) a condition of No Significant Risk.

The conclusions of the risk characterization report must be explicit about the conditions and assumptions upon which the risk characterization is based. Sections 40.0923(4) and (5) of the MCP require that such conditions and assumptions (such as *Activity and Use Limitations*, or the implementation of a remedial measure) be clearly and concisely stated and it must be noted that the results of the risk characterization are only valid upon if and when the remedial measures (including AULs) are carried out.

Note that the information which goes into an exposure profile (the receptors, exposure points, exposure point concentrations, etc...) comes from the site investigation. Thus the investigation must be designed in such a way to provide the risk assessor with information suitable for the risk characterization. These exposure attributes are interrelated (e.g., the location of the exposure points depends on the migration of the OHM and the activities of the receptors) so the information should be collected and processed in an iterative manner. The following subsections discuss the specific information which must be gathered for the risk characterization, presented in the site assessment report or the documentation of the risk characterization and summarized in the exposure profiles.

7.3.1.1 Site Information Required to Quantify Exposures

The exposure assessment begins with a description of the physical characteristics of the disposal site. This information is typically collected as part of a Phase I (310 CMR 40.0480) or Phase II (310 CMR 40.830) site investigation, although the type of information needed and the appropriate level of detail should reflect the nature and complexity of the site as well as point in time at which the risk characterization is being performed. Relevant site information would include:

- the address and location of the disposal site;
- a detailed map of the site and surrounding area;
- a description of the land uses at and surrounding the disposal site;
- a listing and description of natural resources and vegetation at or near the disposal site (e.g., surface waters, wetlands, forests, grassy areas, etc...);
- a summary of the use of oil or hazardous material and a description of any known and relevant releases which may have occurred;
- a summary of site hydrogeological characteristics, including depth to groundwater, direction and rate of flow, soil types, etc...
- a summary of background concentrations of oil or hazardous materials

WHO ?...WHAT ?...WHEN ?...WHERE ?...HOW ?

The Exposure Profile should contain information to completely describe each receptor's exposures to oil or hazardous material at the disposal site.

- ***Who*** is exposed? The exposure profile should be developed for each receptor likely to be present at the disposal site or in the surrounding environment, and who, as a result, would likely be exposed to OHM.

- ***Where*** does the exposure occur? Is the contamination limited to the area near the original source, or has/will migration of contaminants result in potential exposures at a more distant point?

- ***What*** are the receptors exposed to? What oil or hazardous materials are present at the disposal site? What concentrations of the material have been reported?

- ***When*** does the exposure occur? Are the exposures likely under current site conditions, or will the exposure be of concern if the site use changes in the future?

- ***How*** does exposure occur, and how often? What receptor actions or activities result in contact with the oil or hazardous material? Do these events happen every day or are they rare incidents?

Some of this information may be available through the Massachusetts Geographic Information System (MASS-GIS) which provides color plots or digital data of wetland areas, sole source aquifers, endangered species habitats and other natural resource areas. Several data packages have been developed specific to c.21E site investigations. For a full listing of available data, contact MassGIS, EOEA Data Center, 20 Somerset Street, Boston, MA 02108, (617) 727-3888.

7.3.1.2 Identification of Potential Human Receptors

Section 40.0921 of the Massachusetts Contingency Plan contains regulations specific to the identification of receptors at c.21E sites.

The documentation of the risk characterization should contain a description of the potentially exposed persons who live, work, play, visit, or otherwise come to the disposal site or the surrounding environment. In identifying these receptors, the risk assessor must consider not only those people currently associated with the disposal site, but also those who may frequent the site in the future if the use of the site were to change (See the discussion on Current and Foreseeable Use, Section 2.1).

The human receptors are described as subpopulations (subsets of the more diverse overall population of Massachusetts) rather than specific individuals so that the results of the risk characterization can be generalized. For example "*children*", a specific, identifiable group within the larger general population of humans, are often identified as receptors of concern at c.21E disposal sites. (Hypothetically a risk assessor could identify a specific (real) child who lives at the site and conduct a risk assessment based upon that child's physical characteristics and behavioral patterns, but the result of such an assessment would be valid only for that child and could not be generalized to other children who may visit the site or live there in the future.) Note, though, that while the receptors are described in terms of "subpopulations" or "subgroups", the product of the risk assessment is still an estimate of the risk that applies to the protection of an *individual* within that group. The MCP focuses on individual risk, *not* population risk.

The receptor groups are described in terms that highlight their relationship to the site and the unique characteristics of the subpopulation. For example, the term ***site residents*** describes a diverse group which lives (or may in the future live) at the disposal site. For the purposes of the risk characterization the site residents should be further divided into subpopulations based upon gender and age if those factors are indicative of a higher exposure potential or greater susceptibility to environmental contamination. Young children and women of child-bearing age are often chosen as receptors of concern in residential locations because of these factors. At industrial locations, adults may be the most susceptible receptors. Identification of the most sensitive subpopulation should be done on a site by site basis.

Example 7.3

EXAMPLE RECEPTOR: Site Resident		
Exposure of Concern/ Health Endpoint	**Typical Subpopulation(s) Evaluated**	**Discussion**
Acute Exposure, Noncancer Effects	2 year old child 22 year old woman	The young child is of concern for acute exposures (typically 1 event or several exposures over a short period of time) due to the higher exposure potential while potential developmental effects could be of concern for the woman of child-bearing age.
Subchronic Exposure, Noncancer Effects	2 year old child 22 year old woman	The young child is of concern for subchronic exposures (typically 2 weeks to a year) due to the higher exposure potential while potential developmental effects could be of concern for the woman of child-bearing age.
Chronic Exposures, Noncancer Effects	1-8 year old child	A young child would typically experience the highest exposure in a residential setting. Chronic exposures to adults would not have to be specifically evaluated for noncancer health effects unless the adult is assumed to take part in activities which would result in unusually high exposures.
Chronic Exposures, Cancer Risk	Resident 1-31 years old	Since the magnitude of the cancer risk is dependent upon the total amount of material contacted, a 30 year exposure which incorporates the age groups which experience the highest rates of exposure should be evaluated.

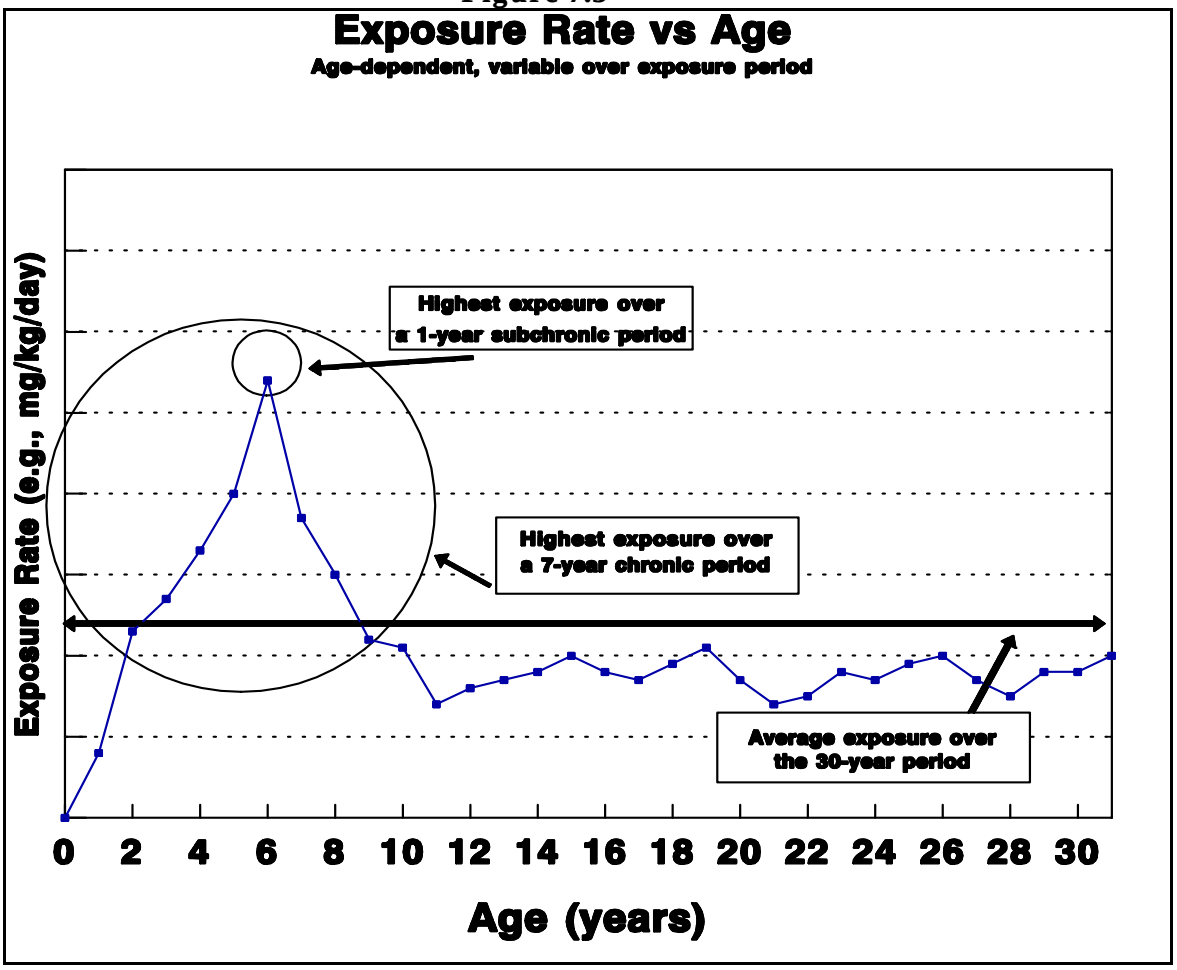
Thus to adequately evaluate the "site residents" the risk assessor may need to look at several specific receptors to insure that all sensitive subpopulations are being protected. Example 7.3 describes typical receptors who might be chosen to evaluate a residential exposure scenario.

By focusing on the subpopulations experiencing the highest rates of exposure the risk assessor may conclude that all other subpopulations at the location would be subject to lower exposures and risks than those calculated. Figure 7.3 illustrates how exposure may vary by age and highlights periods of high exposure which may need to be evaluated by the risk assessor.

7.3.1.3 Identification of Exposure Points

For receptors to be exposed to a contaminant at or from a disposal site, a realistic pathway must be established leading from the source of the oil or hazardous material to the receptor. The point at which the contact occurs is referred to as the *exposure point* (or "exposure setting"). Potential exposure points must be identified per 310 CMR 40.0924. The route by which the material travels from the source to the exposure point is called the *migration pathway*.

Figure 7.3



The migration pathway describes the movement of the material, and it is comprised of three parts: a release *source*, a release *mechanism*, and a release (or transport) *medium*. The documentation of the risk characterization must describe the source of the OHM, how the material was released to the environment and its movement through the environment. This information is routinely gathered during site investigations (see 310 CMR 40.0904), but it is restated here in terms used by risk assessors. A simple example of a migration pathway would be the volatilization of a chemical from a drum to indoor air, where the source of the OHM is the drum, the release mechanism is volatilization, and the transport medium is the air. A migration pathway may include several transport media.

Source $\xrightarrow{\text{Migration Pathway}}$ Exposure Point

Drain Pipe $\xrightarrow{\text{Discharge}}$ *Soil* $\xrightarrow{\text{Leaching}}$ *Groundwater* $\xrightarrow{\text{Migration}}$ *Water Supply Well*

Potential points of exposure may be distant from the original source material, so the risk assessor must consider the current and future migration pathways to identify all potential exposure points.

The regulations also require that *hot spots* (Section 2.2) be identified as separate and distinct exposure points for purposes of risk characterization (310 CMR 40.0924(2)). This requirement ensures that areas with high relative contamination will not simply be averaged into a wider area of lesser contamination, thus minimizing (or diluting) their potential impacts. (The MCP describes a number of risk reduction tools (IRAs, RAMs, URAMs) which can and should be used to address hot spots in a timely fashion, thus reducing overall site risks in an efficient, cost-effective manner.)

While the regulations and guidance use the term *exposure point*, the term may actually describe an area of a disposal site or surrounding environment and not necessarily a single, discrete point. The exposure point should be an area within which the receptor has an equal likelihood of exposure, such as "a backyard" or "a schoolyard". If there are areas within the site which receptors frequent at a higher rate (such as the area surrounding playground equipment within a larger schoolyard) then those areas should be evaluated as separate and distinct exposure points. Figure 7.4 depicts a site within which there are two areas that should be evaluated separately (in addition to the area of generalized contamination) as exposure points: a hot spot and a playground. Additional examples of exposure points include:

- an area where people come into contact with contaminated soil,
- a drinking water well or a potential drinking water well location
- a building into which air contaminants are migrating and accumulating in the indoor air
- an area in which ambient air contains elevated levels of site-related contaminants

In general, an exposure point for soil, sediment or surface water should be delineated by the distribution of oil or hazardous material in the environmental medium. For example, for soil, an exposure point should be a contaminated area within which the exposure of concern is likely. The area outside the boundaries of the contamination should not be included in the exposure point, and data from those areas should not be included in the concentration estimate. There are two reasons for this recommendation:

1) There is rarely enough information on current small-scale exposure patterns in the

vicinity of a contaminated area, for example a residential yard, to justify assumptions about the relative amount of time spent in the area known to be contaminated.

2) The full areal extent of contamination is not always known, unfortunately, at the time of the risk characterization. Sample collection is often focused on the areas where contamination is expected and/or obvious, and other areas are not fully characterized (although those areas may be contaminated as well). The practice of treating the contaminated area as the entire exposure provides a conservative estimate of exposure.

There may be some situations where the default approach described above is not appropriate. In cases where the extent of soil contamination is well defined and clearly constitutes only a fraction of the area over which the receptor group of concern is equally likely to be exposed, the exposure point may be an area that is somewhat larger than the contaminated area. The best example of a situation where this exception might be applied is a residential back yard. If a resident is equally likely to contact the soil at any locations within the yard, and if the contaminated area has been clearly delineated and found to comprise only a fraction of the yard, the risk assessor may opt to define the entire backyard as the exposure point.

When considering whether the exposure point should cover an area larger than that which is contaminated, the scale of the contaminated area relative to the anticipated exposure pattern is an important consideration. For example, consider a vacant lot where children are likely to play. If ¼ of a 2000 ft^2 lot were contaminated, it may be reasonable to assume that activity levels and exposures in the 500 ft^2 contaminated area are not likely to be any higher than those in the rest of the lot. However, if the ¼ of a one acre lot is contaminated, it would be more difficult to justify the assumption that activity levels in the ¼ acre that is contaminated will never be higher than in the surrounding area.

Another important consideration is whether the foreseeable activities are likely to result in more intense or more frequent exposures in some areas than in others. For example, in play parks, exposure intensity at any location depends upon the landscaping, the pattern of open space and the layout of equipment. If a small area of surface soil located within a large park were contaminated, the risk assessor may not be able to rule out the possibility that exposures to individual children will not be higher in that area than in other areas of the park. Therefore it would be more appropriate to designate the contaminated area alone as the exposure point, and not the entire park.

The burden to demonstrate that the designation of an exposure point is appropriate and conforms with this guidance rests with the risk assessor. The documentation of the risk characterization should present summary tables describing the migration pathways identified and the exposure points to be evaluated.

7.3.1.4 Identification of Exposure Routes

The mechanism by which a receptor comes into contact with the oil or hazardous material is called the *Exposure Route*. Typical exposure routes described at c.21E disposal sites include:

- **INGESTION** of contaminated soil, water or food
- **INHALATION** of contaminated air or fugitive dust
- **DERMAL ABSORPTION** from contaminated water, soil or sediments

Remember that a receptor may be exposed to oil or hazardous material at one ***or more*** exposure points, and that at each exposure point the receptor may be exposed via one **or more** routes. The exposure profile for the receptor should describe such multiple exposure scenarios in a way which makes clear to the reader that the combination of exposures to the receptor is being addressed in the risk assessment.

7.3.2.5 Identification of Exposure Pathways

The ***Exposure Pathway*** is the term used to describe the course that the oil or hazardous material takes from the source of the material to the receptor of concern. The term encompasses the source, the migration pathway, the exposure point, the receptor and the exposure route.

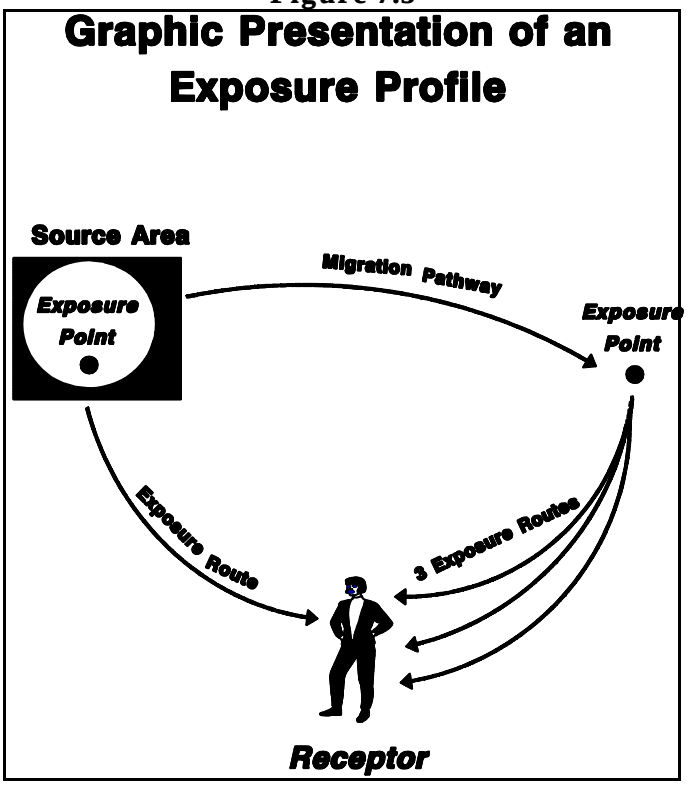
Exposure Pathway:

$$\text{Source} \xrightarrow[\text{Migration Pathway}]{} \text{Exposure Point} \xrightarrow[\text{Exposure Route}]{} \text{Receptor}$$

Thus the Exposure Profile (or exposure scenario) developed for each receptor would describe, in narrative and tabular form, the circumstances under which the receptor is exposed. The exposure profile may be relatively simple if a single receptor group is exposed at one location via one route of exposure. Exposures at c.21E sites are generally a bit more complex, however. A receptor group may be exposed to the oil or hazardous material through a number of exposure routes at several locations. Figure 7.5 illustrates a situation in which there is one receptor, one source of OHM, a migration pathway and four exposure routes. (There are also four exposure pathways, as there are four routes by which chemicals may move from the source to the receptor.) Example 7.4 demonstrates how a more complex example may be clearly presented in a tabular format.

7.3.2 Basic Approach/Assumptions

Figure 7.5



The basic approach which should be taken in an exposure assessment under the MCP is to produce an assessment which is realistic and health protective. The regulations (310 CMR 40.0992(2)) stipulate that the objective of a Method 3 risk characterization is to provide a conservative estimate of the impact that the oil and/or hazardous material may have on the receptors at the site and in the surrounding environment. The assessment should not be a "*worst case*" exposure assessment unless there are site-specific justifications for performing such an evaluation. (Worst case assessments are useful screening tools which may demonstrate that risks are clearly insignificant, but they are not useful in determining whether realistic risks are actually significant.) Conversely, the assessment should not represent an "*average case*" which may underestimate potential risks experienced by a large portion of the exposed subpopulation, and thus would not be considered to be health protective. This section presents guidance on identifying receptor groups that are likely to be most susceptible to contamination at the site, and on selecting exposure parameters that will result in an appropriately conservative estimate of risk to that receptor group.

Numerous attempts have been made to define a combination of exposure assumptions which would result in a reasonable yet health-protective exposure assessment. USEPA (1989) defined a **Reasonable Maximum Exposure** (RME) as "*the maximum exposure that is reasonably expected to occur at a site*" and recommended specific exposure factors (USEPA, 1991) to be used to evaluate the RME. More recently (USEPA, 1992) the concept of "*high-end*" exposure, dose and risk estimates has been introduced:

The high-end risk is taken to be a plausible estimate of the risk for persons at the upper end of the risk distribution. The intent of the high-end descriptor is to convey an estimate of risk in the upper range of the distribution, but to avoid estimates that are beyond the true distribution. Conceptually, high-end risk means risks beyond the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest risk. The discriptor is intended to estimate the risks that are expected to occur in small but definable high-end segments of the subject population. The use of "above the 90th percentile" in the definition is not meant to precisely define the range of this descriptor, but rather to clarify what is meant conceptually by high-end.

Figure 7.5 graphically depicts the "high-end" exposure range (from USEPA, 1992) from a

Example 7.4

Exposure Profile Summary Table

Receptor	Age	Exposure Point	Exposure Route
Resident	Young Child, age 1-6	Residential Backyard	Soil Dermal Contact Soil Ingestion Inhalation of Volatilized Material Ingestion of Groundwater
		School Playground	Soil Dermal Contact Soil Ingestion Inhalation of Fugitive Dust
	Older Child and Adult, age 7 - 30	Residential Backyard	Soil Dermal Contact Soil Ingestion Inhalation of Volatilized Material Ingestion of Groundwater

hypothetical distribution of site exposure for a specified subpopulation.

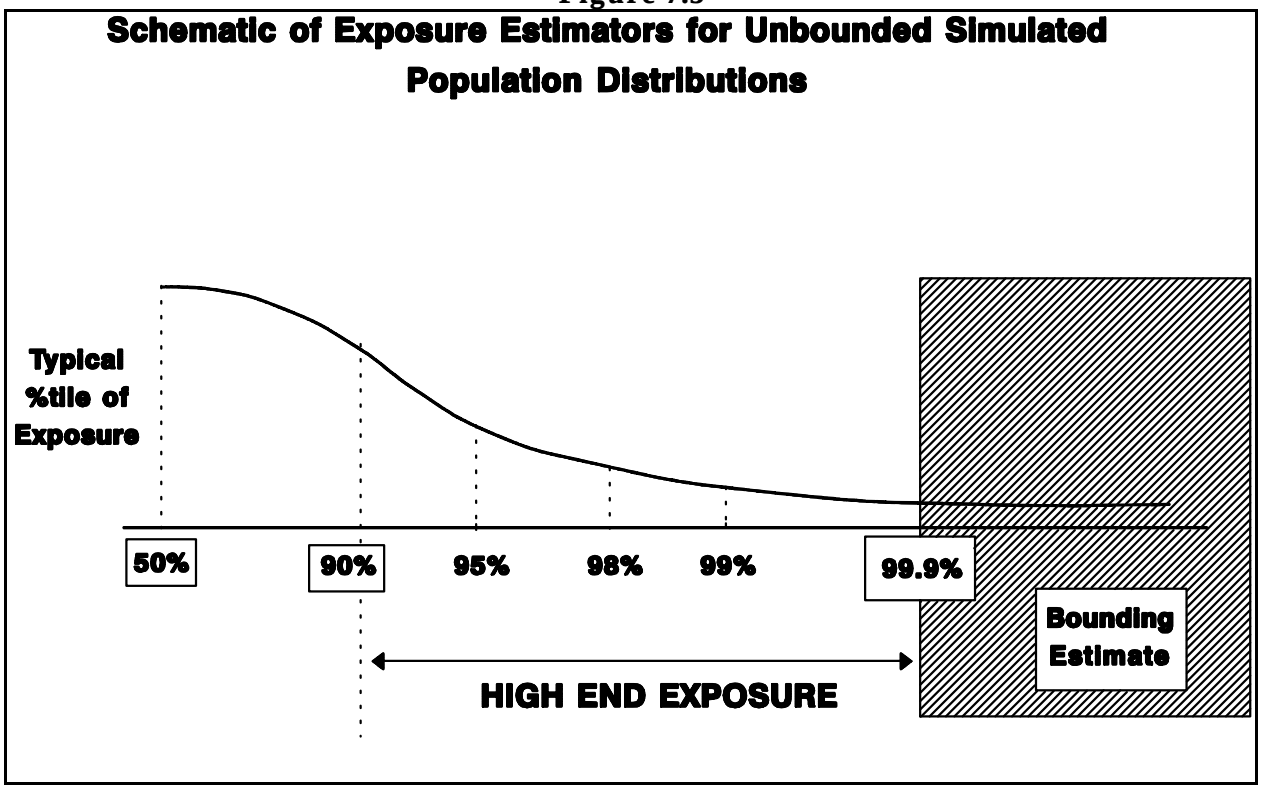
MADEP has in the past recommended (MADEP, 1992b) that the exposure assessments identify the average exposure for the Maximally Exposed Individual (MEI) of a specified receptor group. The term "*Maximally Exposed Individual*" is, therefore, a misnomer for that receptor of concern since the evaluation would focus on the *average* individual within this subpopulation.

For the purposes of Method 3 Risk Characterizations performed under the MCP, the receptor subpopulation of concern would be characterized by those individuals whose activities (described by the frequency and duration of the actions) represent a full and unrestricted use of the site (considering the current and foreseeable uses identified) and who

are most susceptible to the contamination at the site. The quantitative exposure

assessment should describe a conservative estimate of a representative individual within that subpopulation. (Note that the "*fullest use*" does not necessarily mean that the highest possible values for exposure frequency and duration should be used.)

Figure 7.5



The subpopulations or receptor groups evaluated in the quantitative risk assessment should represent the most susceptible individuals and groups of all of those who are exposed to contamination at the site in question. Higher susceptibility is used here to mean a higher probability of experiencing adverse impacts as a result of exposure. Susceptibility is determined by the combination of the intensity of exposure and the sensitivity to toxic effects combined. Examples of receptor groups that are often identified as the most susceptible subpopulations include those described below:

- In typical residential areas, children are usually considered among the most susceptible receptors because (1) their activities are likely to result in more intense exposures than those of adults, (2) they are believed to intake higher amounts of soil by incidental ingestion, and (3) all other things being equal, their lower body weights result in higher normalized doses. Note that the first two factors relate to higher exposure intensity, while the third translates to higher sensitivity, all of which combine to make children generally more susceptible than adults to the contamination.

- In typical industrial areas, adults who work at the site are often considered as one of the most susceptible subgroups because their exposure frequency is higher than for others who may be exposed on occasion.

- Occasionally, pregnant and/or nursing women may be identified as a highly susceptible subgroup. The effects of concern in these cases may be developmental effects on fetuses and babies, not necessarily effects on the mother herself. Fetuses are considered more sensitive than adults to some contaminants because a one-day exposure may be sufficient to cause adverse developmental effects. Babies are more susceptible because they may be exposed to significant levels of fat-soluble contaminants which may become concentrated in mother's milk. Because of their low body weight, a baby's exposure can lead to a relatively high normalized dose. Babies and young children are also more sensitive than adults to the toxic effects of some substances, metals in particular.

It is worth noting that, although we have often spoken in terms of the "most sensitive receptors", most of the factors that lead to a higher susceptibility are in fact related to exposure intensity, and not necessarily a greater sensitivity to the toxin. While higher sensitivity to a toxin may be an important consideration, it is seldom addressed quantitatively in health risk assessments, because the same toxicity values are generally (perhaps unfortunately) applied to all subgroups.

Exposure assessments should use mid-range estimates of exposure parameters, such as such as intake rates, contact rates and bodyweights, which are known to vary among individuals within the specified receptor group. The arithmetic mean of concentrations at exposure points are recommended (See Section 7.3.3.5) for use in the exposure calculations. Again, note that the values used for frequency and duration of exposure should reflect realistic values for receptors making the fullest use of the site or resource (given the current and future uses determined for the location) while considering climatic conditions in Massachusetts.

This mix of mid-range and conservative values is intended to produce realistic upper-end exposure estimates which will be protective of public health and produce risk estimates which will be valid for comparison to the MCP Cumulative Risk Limits. For exposure assessments performed using probabilistic techniques (such as Monte Carlo analysis) the MCP stipulates that the 95th percentile value of the resulting exposure distribution *for the specified receptor subpopulation* be used to calculate risk estimates.

For risk assessors attempting to meet the regulatory requirements of both the MADEP and the USEPA, the risk estimates calculated using the USEPA "high end" exposures would likely be equal to or higher than those estimates using the MADEP approach. Thus, cleanup decisions based upon such "high end" estimates (used with the MCP risk management criteria) are likely to meet the requirements of the MCP, even though the specific mix of exposure parameters used in the calculations will be different in the different programs.

Exposure estimates calculated as described herein are considered to be protective of public health in that they are not likely to be underestimates of the "true risk" for individuals in the specified receptor subpopulation.

7.3.3 Quantitative Estimations of Exposure

Once exposure profiles have been developed describing the contaminants of concern, exposure points, exposure point concentrations and the receptors of concern, the potential exposures experienced by the receptors are quantified. This information will then be used to estimate risk, as described in Section 7.4.

This section of the guidance describes (a) the differences between exposure and dose, (b) the different types of doses which may be employed in the risk assessment, (c) the common factors used to estimate exposure, and (d) the pathway-specific equations employed to quantify exposure.

7.3.3.1 Concepts and Terminology

The concept of exposure is complex, and the numerical value calculated by the risk assessor will depend upon the nature of the exposure pathway under investigation, the duration of the exposure, and the health effects associated with the chemicals of concern.

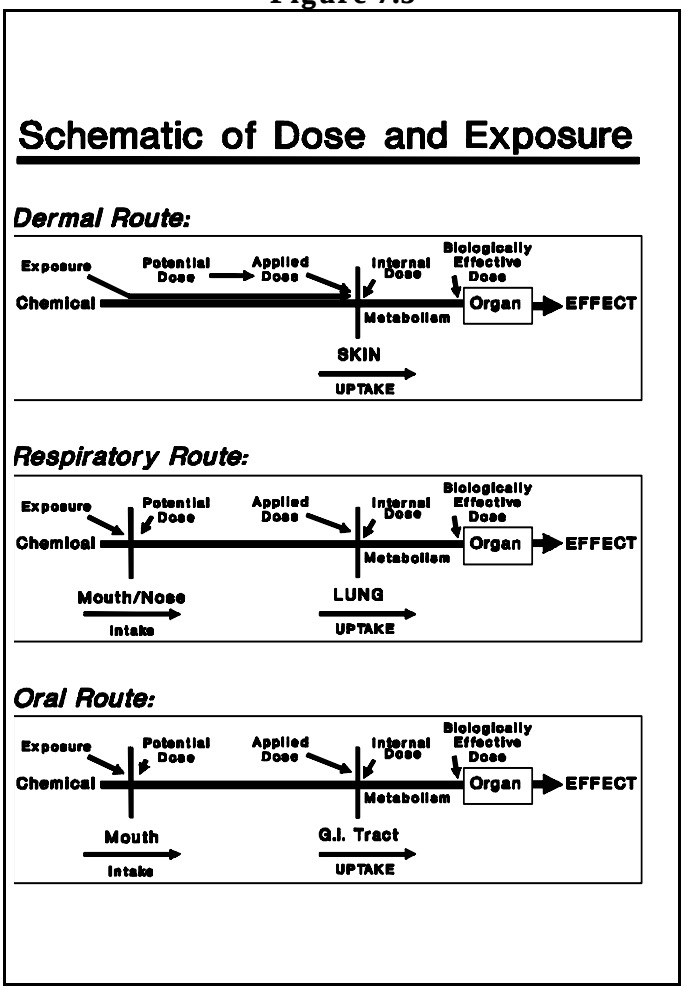
The US EPA Exposure Assessment Group defines *exposure* as the amount of material in contact with an organism and available for absorption. The material which reaches the organism's absorption barrier (such as the skin, lung or gastrointestinal tract) is referred to as the *applied dose*, while the *absorbed* (or *internal*) *dose* is defined as the amount of material which actually crosses the organism's exchange boundary. [Note that exposure is often thought of as the "*potential dose*" and taken as an approximation of the applied dose, as it represents the amount which could be absorbed if it were 100% bioavailable. Figure 7.6 (adopted from USEPA, 1992) illustrates the differences in these terms for the dermal, respiratory, and oral routes of exposure.

The type of exposure or dose used to characterize risk will depend upon the exposure pathway under evaluation and the nature of the toxicity information available for each chemical:

- Typically *respiratory* exposures are evaluated using the exposure point concentration in combination with a published Reference Concentration or Unit Risk value.

- Oral and dermal exposures are typically evaluated by modifying the applied dose with a *Relative Absorption Factor* (RAF) to insure that the calculated exposure is comparable to the Reference Dose or Cancer Slope Factor employed. (See Section 7.2 for a discussion of RAFs.)

Figure 7.5



Where appropriate, the equations given in the following pages include a Relative Absorption Factor. Under certain conditions the quantitative estimate of exposure will in fact be an estimate of the applied dose (or exposure) rather than an absorbed dose. For simplicity, the term "average daily dose" is used to describe the product of an "average daily exposure" and a Relative Absorption Factor.

7.3.3.2 Types of Average Daily Doses

The equations presented below outline the procedure for the calculation of an Average Daily Dose of an oil or hazardous material. Depending upon the duration of the exposure under evaluation and the type of health effect (cancer or noncancer) of concern, the calculations may yield one of several results:

- ***Lifetime Average Daily Dose (LADD):*** A LADD in units of milligrams per kilogram body weight per day (mg/kg/day) should be calculated to estimate carcinogenic risk. The total intake during that exposure is normalized to a lifetime, taken to be 75 years. [Note that exposure may occur for all or some fraction of the receptor's lifetime.]

- ***Chronic Average Daily Dose ($ADD_{chronic}$:*** Chronic human exposures are defined by MADEP to be those lasting seven years or more. The $ADD_{chronic}$ (in units of mg/kg/day) is calculated for the characterization of potential noncancer risk resulting from long-term exposures, and the value must be an estimate of exposure experienced by the receptor *during the period of exposure.*

- ***Subchronic Average Daily Dose ($ADD_{subchronic}$):*** Subchronic human exposures are defined by MADEP to be those lasting from several days up to seven years. The $ADD_{subchronic}$ (in units of mg/kg/day) is calculated for the characterization of potential noncancer risk associated with such mid-range exposures, and the value must be an estimate of exposure experienced by the receptor *during the period of exposure.*

- ***Acute Average Daily Dose (ADD_{acute}):*** The Acute exposure may range from the instantaneous to those lasting up to several days, and the ADD_{acute} (in units of mg/kg/day) is calculated for the evaluation of potential noncancer risks resulting from such short-term exposures.

Inhalation risks are characterized by calculating the exposure concentration rather than the dose. Therefore, the terminology used for inhalation exposures differs from that used for ingestion and dermal exposures. To estimate carcinogenic risk from an inhalation exposure, the ***Lifetime Average Daily Exposure (LADE)*** (milligrams per cubic meter air per day) is calculated rather than the LADD. To estimate risks of non-cancer effects from inhalation exposures, the ***Average Daily Exposure (ADE)*** is calculated for chronic, subchronic and acute exposures rather than the ADD.

Note that it is often necessary to calculate several different daily doses of a chemical to a receptor in order to evaluate all relevant exposure scenarios. For chemicals which are considered carcinogenic, a lifetime average daily dose must be calculated as well as all appropriate average daily doses (*chronic, subchronic and/or acute*) for the evaluation of noncancer health risks. For noncarcinogens, all appropriate average daily doses (*chronic, subchronic and/or acute*) must be calculated.

7.3.3.3 General Form of Dose Equations

The general form of the equations to estimate average daily exposure (ADE) and average daily dose (ADD) is presented as:

$$ADE = \frac{(Total\ \ Amount\ \ of\ \ OHM\ \ Contacted)}{(Averaging\ \ Period)} \qquad \textbf{(7-3)}$$

and

$$ADD = \frac{(Total\ \ Amount\ \ of\ \ OHM\ \ Contacted) * (Relative\ \ Absorption\ \ Factor)}{(Body\ \ Weight) * (Averaging\ \ Period)} \qquad \textbf{(7-4)}$$

Note that "*dose*" is taken to be "*exposure*" normalized to the receptor's body weight and

adjusted for absorption/bioavailability (as described in section 7.2.3).

At c.21E disposal sites it is common to have situations where a receptor may be exposed to a chemical through multiple exposure pathways, such as ingesting contaminated soil and absorbing the material following dermal contact with contaminated soil. In such cases, the doses of an oil or hazardous material received via different routes of exposure are assumed to be additive unless there is strong evidence otherwise.

$$\begin{matrix} Cumulative \\ Risk \end{matrix} = \sum \sum (Chemical_i , Exposure\ \ Pathway_j) \qquad \textbf{(7-5)}$$

General equations for the calculation of Average Daily Dose are presented in this section for some frequently encountered exposure pathways. These equations are not intended to represent the universe of potential models and they must be tailored to site-specific conditions. It is expected that additional exposure pathways may be identified, and an average daily dose may be calculated, using appropriate models, for each receptor of concern.

There are a number of common exposure factors that are employed in virtually all of the exposure equations, and the discussion which follows describes some of the issues which may arise when using these elements. Exposure factors which are specific to a particular pathway are discussed in the subsection which presents the equations for that pathway.

The daily dose(s) of each OHM calculated for each potential receptor should be summarized in the risk characterization report in a manner which is clear and concise. Summary tables presenting the equations and the exposure assumptions used to calculate the daily dose should also be presented and well referenced.

7.3.3.4 Descriptions of General Exposure Factors

There are eight exposure factors which recur throughout the equations used to estimate the dose of oil or hazardous material experienced by a potential receptor:

- Chemical Concentration
- Body Weight
- Frequency of Exposure
- Duration of the Exposure Event
- Duration of the Exposure Period
- Relative Absorption Factor
- Averaging Period
- Units Conversion Factors

These factors are generally used in the same manner regardless of the exposure pathway under investigation, so it is useful to discuss them separately.

Chemical Concentration

The concentration of the oil or hazardous material used to quantify exposure is the Exposure Point Concentration, or EPC, described in section 7.3.4.5. The exposure point concentration is expressed in terms of mass of the material per unit mass (or volume) of the exposure medium: mg_{OHM}/kg_{soil}, $\mu_{OHM}/liter_{water}$, and $\mu g_{OHM}/m^3_{air}$. When concentrations are expressed in terms of parts-per-million (ppm) or parts-per-billion (ppb), care must be taken to convert the concentrations to the appropriate units.

Soil, sediment, food: 1 mg/kg = 1 µg/g = 1 ppm
1 µg/kg = 1 ppb

Water: 1 mg/liter = 1 ppm
1 µg/liter = 1 ppb

Air:

$$1\ \frac{mg}{m^3} = \frac{1\ ppm\ *\ M.W.}{22.4\ *\ \frac{T}{273°K}\ *\ \frac{P}{760\ Torr}} \qquad \textbf{(7-6)}$$

Where T is the air temperature (often assumed to be 25˜ C or 298˜ K) and P is the atmospheric pressure (often assumed to be 1 atmosphere or 760 Torr), and M.W. is the molecular weight of the chemical under evaluation.

The exposure point concentration is represented in these exposure equations by the term: $[OHM]_{exposure\ medium}$. The exposure point concentration should *not* be adjusted for receptor exposure frequency, duration, etc... as those factors are generally addressed in the exposure calculations.

Body Weight

A receptor's body weight is relevant throughout the dose equations as dose is expressed in terms of mass of contaminant per unit body weight per day (mg/kg/day). When each receptor of concern is identified, the receptor is often described in terms of occupation (resident, construction worker), age (a child age 1 to 6 years) and sometimes gender. The receptor's body weight is dependent upon its age and gender. Since body weight is easily measured, there are numerous summaries of age and gender-specific body weights. A table of such values used by ORS is included in Appendix B.

The receptor body weight (**BW**, typically expressed in kilograms, kg) must be matched to the age and gender identified in the exposure profile. Since exposure is often assumed to occur over a period of several years, the changes in body weight which might occur during the period of exposure must also be considered. (See section 7.3.3.6 for the mathematical treatment of age groups.)

Even within a given age/sex combination, there is some variability of body weight for

that subpopulation: some 8 year old boys weigh more/less than other 8 year old boys. This variation is well defined, and the distribution of body weights for this subpopulation of concern may be used as part of a probabilistic assessment of exposure. For evaluations requiring a point estimate of body weight, ORS recommends using the 50th percentile body weigh for that subpopulation, unless there is strong evidence that the potentially exposed subpopulation is biased in some manner. Note that for a normal distribution, the 50th percentile approximates the arithmetic mean.

Frequency of Exposure and Duration of the Exposure Event

A receptor may be exposed to oil or hazardous material continuously, at regular intervals, or in a sporadic manner. The Frequency of Exposure (**EF**) and the Duration of the Exposure Event (**ED**) in combination describe the pattern of exposure being modelled.

The frequency of exposure term describes how often the exposure event occurs over a given period of time. The term answers the questions: *How many times a day does exposure occur?, How many times per week?, per month?, per year?* Exposure Frequency may, in fact, be a string of terms which ultimately reduce to one expression:

$$\frac{1\,event}{day} * \frac{3\,days}{week} * \frac{4\,weeks}{month} * \frac{12\,months}{year} = \frac{144\,events}{year} \qquad \textbf{(7-7)}$$

The Duration of the Exposure Event, as the name implies, describes how long each individual exposure event might last. The term is somewhat more complex than it sounds, however, because it must be consistent with the scale of the contact rate for the exposure being modelled. For some exposure pathways, the information available describing the contact rate is broken down to a small scale (such as hours). The respiratory pathway is perhaps the best example of this case as ventilation (breathing) rates are often measured and expressed in terms of cubic meters per hour, and breathing occurs throughout the day. For such exposures ED may be described as some number of hours/event. More common, however, are contact rates which are on the scale of days rather than hours. The ingestion pathway is typical of this case. While estimates have been published on the amount of water ingested during a *day*, there can be no reliable estimate of average *hourly* ingestion rates as drinking water is a sporadic event depending upon thirst and habit. For such exposures (including drinking water ingestion, soil ingestion and dermal contact) **ED is by definition 1 day/event**. During that "1 event" the receptor is assumed to receive the daily intake of the contaminant.

Duration of the Exposure Period

The exposure period (EP) describes the length of time over which the receptor comes into contact with the oil or hazardous material. The exposure period depends upon the type of activities which lead a receptor to be exposed. Remember that the receptor may be exposed continuously, at regular intervals, or sporadically, depending upon the activity being modelled, so the exposure period would be the length of time between the first exposure experienced and the last. The EP term is typically expressed as some unit of time: days, months, years.

Averaging Period

The equations which follow calculate *average* daily doses or *average* daily exposures, and the averaging period (AP) is the time (in days, months or years) over which the total intake is normalized.

Remember that a Lifetime Average Daily Dose (LADD) is calculated for the evaluation of *cancer risk*. While the duration of the exposure period (EP) might range from one day to an entire lifetime, the total intake during that exposure is normalized to 75 years (a lifetime). The averaging period is thus assigned a value of 75 years, and, for exposures lasting less than a lifetime, the values for EP and AP will be different.

For the evaluation of *noncancer risk*, however, the Average Daily Dose calculated should be representative of the exposure received while exposure is on-going (i.e., during the exposure period). Thus the duration of the exposure period (EP) and the averaging period (AP) for an chronic, subchronic or acute Average Daily Dose are variable factors depending upon the exposure being modelled, *but the AP is set equal to EP by definition*.

Relative Absorption Factor

As described in the Dose-Response section of this guidance, the Relative Absorption Factor (RAF) relates the exposure and absorption estimated for the exposure pathway under evaluation to the exposure and absorption in the toxicological study on which the dose-response information is based. The RAF is dimensionless and is chemical and pathway specific.

EXPOSURE DURATION (EP) and AVERAGING PERIOD (AP)

The Averaging Period (AP) used in the equations to calculate dose will be equal to the Exposure Period (EP) for the evaluation of ***noncancer*** risks. When estimating ***cancer*** risk, AP is always equal to a lifetime (75 years) while EP may vary depending upon the exposure under investigation:

Example: The risk assessor is asked to evaluate the carcinogenic risk associated with a ten year exposure to chemical A. Estimation of carcinogenic risk requires the calculation of a Lifetime Average Daily Dose. Thus, the Averaging Period used for calculating the LADD would be 75 years while the Exposure Period would be equal to 10 years.

The risk assessor is also asked to evaluate the likelihood of non-carcinogenic health effects associated with that same ten year exposure. The assessor would calculate an Average Daily Dose Chronic ($ADD_{chronic}$) where EP = 10 years and AP = 10 yrs.

Units Conversion Factors

One of the most valuable habits a risk assessor can develop is to routinely conduct dimensional analyses on the equations used to quantify exposure. The exposure factors and analytical data used for a given calculation may come in several forms. For example, ventilation rates may be expressed as cubic meters per day or liters per hour; exposure point concentrations in drinking water may be in milligrams per liter or micrograms per liter. Dimensional analysis will reveal whether units conversion factors are necessary to insure that the result of the calculation (the dose) is expressed in the correct units (mg/kg/day).

Use of a units conversion factors (C) is equivalent to multiplication by one. The numerator and denominator of the factor must be an equivalent quantity expressed in different terms. It is not uncommon to need several conversion factors in the same equation to reconcile the dimensions of mass, volume and time.

EXAMPLES OF UNITS CONVERSION FACTORS (C)

Relationship	The numerator and denominator may be reversed depending upon the form of the equation.	
1,000,000 mg = 1 kg	C = 10^6 mg/kg	C = 10^{-6} kg/mg
1 year = 365 days	C = 365 d/yr	C = 0.00274 yr/d
1,000 liters = 1 meter3	C = 10^3 l/m^3	C = 10^{-3} m^3/l

7.3.3.5 Estimating Exposure Point Concentrations - General Considerations

Sampling and Analysis

To assure that site sampling efforts provide adequate data for the risk assessment, the sampling and analysis plan should be developed in consultation with the risk assessor. Analytical data is collected during the site investigation to fully characterize the nature, extent, severity and horizontal and vertical distribution of the oil and hazardous materials at the disposal site. Some or all of the data obtained may be used for the risk assessment. The data obtained or selected for the risk assessment must be representative of actual and foreseeable exposures, and it must be compatible with the dose response value that will be used in the assessment.

Averaging

The exposure point concentration should represent the arithmetic mean of the concentrations to which an individual may be exposed over the exposure period at the exposure point.

As previously stated, the exposure point concentration should be compatible with the toxicity values that will be used to characterize health risks. Chronic and subchronic reference doses are generally based on time-weighted averages of exposure concentrations used in toxicological experiments, and are expressed in terms of an allowable average daily dose. Therefore, the exposure point concentrations used with those reference doses should approximate the time weighted average concentration to which the receptor may be exposed at the exposure point during the exposure period being evaluated. Cancer slope factors are also based on an average daily dose, and exposure point concentrations for evaluating cancer risks should represent the average daily dose for a 30 year exposure.

Four types of exposures are routinely evaluated in disposal site risk assessments: (1) acute (typically 24 hour exposures), (2) subchronic (several months to seven years) exposures to substances with non-carcinogenic effects, (3) chronic exposures (greater than seven years) to substances with non-carcinogenic effects and (4) lifetime exposures (typically 30 years and averaged over a lifetime of 75 years) to carcinogens. For each type of exposure, the risk assessment should focus on the time-segment during which the highest dose is likely to be received. The exposure point concentration should be a conservative estimate of the average exposure concentration over that period of time. For example, to evaluate three month subchronic drinking water exposure when the concentration in the water supply is known to fluctuate seasonally, the exposure point concentration should represent the highest average to which a person could be exposed within a three month time frame.

Acute Exposures

For acute exposure assessments, the exposure point concentration should represent a conservative estimate of the concentration to which a receptor might be exposed over the period of one day. Generally, the highest detected concentration should be employed when

one-time exposure could result in adverse health effects.

Using Qualified Data

Non-Detects

In estimating exposure point concentrations, it is not uncommon for the risk assessor to be presented with analytic data for a chemical at the site which includes a number of samples reported to be below the Method Detection Limit (MDL). Such results are referred to as "Non-Detects".

Non-Detect results may be classified into two general situations. First, if a chemical is truly not present at the disposal site (virtually all the samples are reported as Non-Detect), and there is no history of a release of that chemical, then the risk assessor may conclude that the chemical should be dropped from the quantitative risk assessment. Second, if the chemical is reported at the site at concentrations ranging from Non-Detect to some site maximum, the risk assessor may conclude that the reported Non-Detects actually represent a distribution of concentrations between zero and the MDL. These Non-Detect results contribute to the information known about the disposal site and should be incorporated into the quantitative risk assessment in a meaningful way. (There is a third possible situation, where the spatial pattern of positive and Non-Detect results indicate that contamination is localized to specific areas. This would represent a combination of the previous two examples.)

There are several options for the treatment of "Non-Detects" described in the literature (Travis, 1990; Helsel, 1990; Klassen, 1986 and Slymen et al., 1994). The methodologies described include the use of log-probit analysis, maximum likelihood estimation and probability plotting procedures. The level of effort and number of data points required to effectively employ these methods vary, and the risk assessor is encouraged to exercise professional judgement in the selection of a method to treat Non-Detect results.

For estimating exposure point concentrations at most c.21E sites, the Department believes that a more straightforward approach is often appropriate. When a contaminant is detected or likely to be present in the area under investigation and the laboratory reports the concentration of an OHM in a sample taken from the area as "Non-Detect", the concentration of the OHM in that sample should be assumed to be one-half of the Sample Quantitation Limit (SQL). The SQL is the actual quantitation limit for each analysis, and it accounts for sample dilution that may occur. If only the Method Detection Limit is reported, and if the sample is heavily contaminated with any constituent, the risk assessor should attempt to determine whether the sample was diluted. For samples that have been diluted (a factor of 10 is not unusual), the risk assessor could substantially underestimate the concentration by using the Method Detection Limit or the Practical Quantitation Limit as a basis for the estimate.

This methodology is simple and easy to use. These benefits must be weighed against the bias which is introduced in the resulting EPC estimate. The Non-Detect method

selection should also consider, the often high level of uncertainty which is often inherent in environmental sampling and analysis procedures. This uncertainty may result from failure to take an adequate number of samples, mistakes on the part of the sampler, the heterogeneity of the matrix being sampled, and intentional bias in the sample collection. For relatively small disposal sites, these inherent uncertainties may overwhelm the bias introduced by using 1/2 the MDL. A more statistically oriented ND method may not, in such cases, significantly reduce the uncertainty inherent in the resulting EPC. It is up to the risk assessor to judge the level of sophistication appropriate to the data set.

As always, there may be exceptions to this guidance, particularly when the site history and the NDs may indicate the absence of an OHM at a site (or areas within a site). In the latter case, the chemical may be dropped from the quantitative risk assessment or the NDs may be factored into the Exposure Point Concentration as a zero value with appropriate justification.

Tentatively Identified Compounds

Tentatively identified compounds (TICs) are compounds which are detected during sample analysis, but are not target compounds. TICs are often reported when gas-chromatography-mass spectrometry (GC-MS) is used to analyze organic compounds. Target compounds are those for which the instrument was calibrated, using a chemical standard, prior to analysis. The ability of the MS system to store mass spectra electronically in a "library" enables the analyst to compare the library spectra with the spectra produced by a non-target contaminant when one shows up in an environmental sample. Identification based on a "library" comparison is much more uncertain, however, than one based on calibration with a standard for the target compound.

There is no rule of thumb for whether TICs should be included in the risk assessment. Confidence in a TIC identification depends on a number of factors, including site history and the presence of similar compounds at the site. The EPA's *Guidance for Data Useability in Risk Assessment* provides the following guidance:

Confidence in the identification of a TIC can be increased in several ways. ...An analytical chemist trained in the interpretation of mass spectra and chromatograms can review TIC data and eliminate many false positive identifications. The use of retention indices or relative retention times can confirm TICs identified by the GC-MS computer (Eckel, et al. 1989). Examination of historical data, industry-specific compound lists, compound identifications from iterative sampling episodes, and analyses performed by different laboratories may also increase confidence in the identification of a TIC. The final identification step is to re-analyze the sample after calibrating the GC-MS instrument with an authentic standard of the compound that the TIC is believed to be.

Many compounds that appear as TICs during broad spectrum analyses belong to compound classes. Examples of compound classes are saturated aliphatic hydrocarbons and polycyclic aromatic hydrocarbons (PAHs). The risk assessor may be

able to make a preliminary judgement of toxicity at the compound class level without a definitive identification of each compound present.

The identification of a TIC can be confirmed definitively only by further analysis. However, depending on the analytical and historical information available, and the potential impact of the TIC on the results of the risk assessment, confirmatory analysis may not be warranted. The risk assessor should work with the project manager and an analytical chemist to make a prudent decision about the need for follow-up analysis.

Measured vs. Modeled Concentrations

Direct measurement of environmental concentrations is generally preferred, but estimation by an analytical or numerical model may be acceptable when direct measurement is impossible or extremely impractical. If a model is used, modeling methods, input parameters and assumptions, and model validation should be fully referenced and described. Modeling considerations are discussed further in subsequent sections on exposures to specific environmental media.

7.3.3.6 Soil Exposure Point Concentrations

Direct Contact

Direct contact with soil can result from such diverse activities as work, play and gardening on residential properties; recreational activities on public and private land; landscaping of commercial properties; grading or excavation of soil for construction or utility repair; agricultural work; outdoor work on industrial properties; and exploration of any area sufficiently unattractive to appeal to young people's curiosity. Exposure occurs primarily by dermal absorption of contaminants from soil and incidental ingestion of contaminated soil. To calculate an exposure point concentration for a particular exposure scenario, the selected samples should be representative of the area and depth within which the particular exposure is likely to occur.

Generally, for surface soil exposures, **the arithmetic mean soil concentration in an exposure area may be used as the exposure point concentration estimate**. The accuracy of this method depends on three underlying assumptions:

- Over time, soil concentrations remain constant;
- The detected concentrations represent a uniform or random distribution of soil samples over the exposure area; and
- Over time, exposure is equally likely at any location within the exposure area.

If these assumptions hold true, the arithmetic mean concentration in the exposure area will represent the arithmetic mean concentrations with which a person comes into contact over time. In other words, the spatial average may be used as a surrogate for the temporal average.

The first assumption stated above is consistent with current DEP practices. Laboratory derived degradation rates often are not observed in the field, and the conservative assumption that concentrations will not decrease over the time of the exposure period is encouraged.

There are cases, however, when the second and/or third assumptions do not hold true. Sampling locations are not always distributed evenly over the site, and exposure frequencies are often higher in some areas than others. In these cases, a weighted average of the detected concentrations should be used.

Figure 7.7 illustrates a situation where the sampling points are not evenly distributed over the site. In this example, an area weighted average exposure point concentration is considered to be a representative estimate of the exposures at the site over time. In this method, analytical data should be weighted in a manner which reflects the sampling frequency as follows:

> *If 6 equidistant samples were taken in a portion of a site approximately 10 meters by 60 meters each sample can be said to represent 100 m^2 (600 m^2/6 samples). If two additional equidistant samples were obtained from another portion of the site approximately 10 meters by 40 meters, each sample could be said to represent 200 m^2. The sample values should be weighted according to the relative area each represents. The area-weighted average obtained from this exercise represents the arithmetic mean concentration over the exposure area. If exposures are equally likely throughout the entire area over time, this area-weighted average also represents the time-weighted average, or the average exposure concentration over time.*

Figure 7.8 illustrates a scenario where the sampling locations are distributed evenly, but exposure occurs more frequently in one portion of the site than the other. In this example, a person is not equally likely to be exposed at all locations, and the time-weighted average could account for different exposure frequencies in different areas as follows:

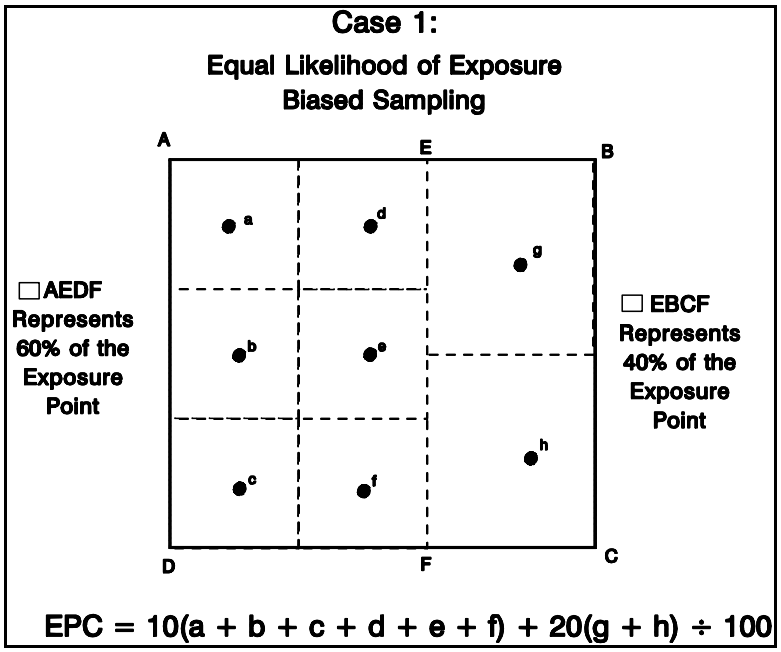


Figure 7.7

> *If 90% of the exposure time takes place on half of the site, and 10% of the exposure time takes place on the other half of the site. The average concentration for each half could be calculated separately, and then weighted to obtain a frequency-weighted average. Again the result represents the arithmetic mean of the concentrations to which the person is exposed over time.*

Note that there may be situations in which weighting for both exposure time and area are appropriate.

These examples represent simple approaches to obtaining a weighted average. More refined techniques for weighting soil or sediment data to estimate an areal average are available. Those that appear to be best suited for exposure assessment are polygon techniques. In general, these procedures involve construction of a polygon around each data point so that each polygon contains the locations that are closer to the data point at its center than any other data point. Such methods are useful for deriving area weighted average soil concentrations which may be used as surrogates for time-weighted exposure point concentrations.

Other approaches often suggested in risk assessment literature and guidance are oriented toward estimating the most likely concentrations at locations between data points. Kriging and triangulation are examples of such methods. The problem of determining concentrations between data points is related to but different from the problem of estimating the average concentration over an exposure area. To date, DEP has found no compelling argument for the applicability and utility of these techniques for calculating exposure point concentrations, and therefore recommends against employing them at this time.

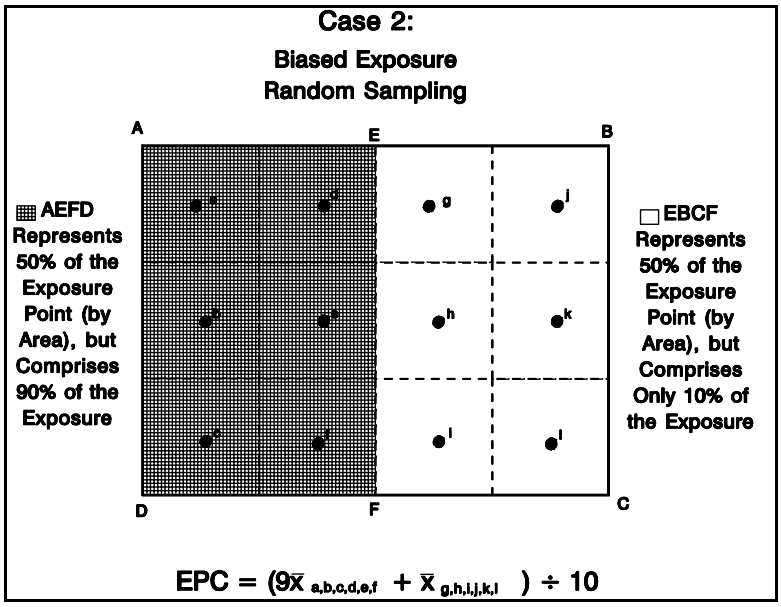


Figure 7.8

Composite Soil Samples

The concentration of a composite soil sample may be used to approximate the arithmetic average of the subsample concentrations. The use of composites can provide an arithmetic mean concentration of several locations at the same cost as analyzing an individual sample. However, the concentration detected in a composite is representative of the average concentration of subsamples only if: (1) the subsamples are representative of the exposure area (2) the composite sample is well mixed and (3) the process of compositing does not result in analyte loss. These conditions can be verified by comparing the average concentration of a set of single location samples with the concentration of a composite of sample collected from the same area. If a composite sample from one area is checked in this manner and demonstrated to be accurate for each sampling event, it is not necessary to check all composites from all areas.

Consumption of Homegrown Fruits and Vegetables

Consumption of fruits and vegetables grown in contaminated soil will result in exposure if the plant takes up a portion of contaminant from the soil. Ideally, produce concentrations should be measured directly. However, sometimes produce concentration data cannot be obtained quickly enough to be used in site management decisions, and must therefore be estimated from soil concentration data. The contaminant concentration in the produce itself is related to the soil concentration and the plant uptake factor, as follows:

$$[OHM]_{plant} = [OHM]_{soil} \ x \ Ksp_{plant/soil} \quad \textbf{(7-8)}$$

Where:

$[OHM]_{plant}$ = plant contaminant concentration (mg_{OHM}/kg_{plant})
$[OHM]_{soil}$ = soil contaminant concentration (mg_{OHM}/kg_{soil})
$Ksp_{plant/soil}$ = plant/soil uptake factor (kg_{soil}/kg_{plant})

Default plant/soil uptake factors are listed in Appendix B.

When estimating contaminant concentrations in produce, it is necessary to assure that the uptake factors and produce consumption estimates are compatible. Plant uptake factors are generally reported on a dry weight basis. Dry weight produce concentrations must be used with intake estimates that are expressed in terms of dry weight, not wet weight.

Inhalation of Particulate Matter from Contaminated Soil

Inhalation of contaminated particulate matter is of concern in cases where contaminated soil is unvegetated or is likely to be graded or excavated for site work or for development.

The exposure point concentration (mass of contaminant/volume air) should be calculated as follows:

$$EPC_{air} = [OHM]_{soil} \ x \ PM_{10} \ x \ CF \quad \textbf{(7-9)}$$

Where:

EPC_{air} = Exposure Point Concentration ($\mu g_{contaminant}/m^3_{air}$)
$[OHM]_{soil}$ = Soil concentration ($mg_{contaminant}/kg_{soil}$)
PM_{10} = Respirable particulate concentration in air ($\mu g/m^3_{air}$)
CF = Conversion factor (10^{-9} kg/μg)

When evaluating exposure to airborne particulate matter at a sparsely vegetated or unvegetated site, or at a construction site, it should be assumed that all of the PM10 is contributed by the contaminated area. This may overestimate the contribution of site soil to airborne particulate concentrations, but the data necessary to obtain a more accurate estimate for these conditions is not available. On a site-specific basis, with appropriate justification (e.g. dense vegetation), the percentage of PM10 that is soil-derived may be reduced to as low as 40% (Thurston and Spengler, 1983).

It may generally be assumed that the concentration of the contaminant in PM10 is equal to the concentration of the contaminant in soil. This assumption may underestimate the concentration of contaminant in the PM10 fraction, since smaller particulate fractions sometimes contain contaminant concentrations that are enriched relative to larger fractions. However, the data needed to derive more accurate concentration estimates is not

available.

Ideally, to assess current conditions, both the concentration of PM10 in the air and the concentrations of contaminants in the PM10 fraction should be measured directly. However, to assess future conditions, it is necessary to estimate the contaminant concentrations in air from the contaminant concentrations in soil. Default values for air concentrations of PM10 from one of two situations are usually required. The first situation is an open field condition, in which contaminated soil is sparsely vegetated or bare, and soil particulate matter readily becomes airborne. The second is a grading or excavation scenario, in which earth working activities may raise elevated levels of dust.

For open field situations, 32 $\mu g/m^3$ should be used as an estimate of the ambient PM10 concentration. This value represents the highest (from 17 sampling stations) annual arithmetic mean concentration measured in Massachusetts in 1994 by DEP's Air Quality Surveillance Branch (1994 Air Quality Report, Commonwealth of Massachusetts). A contribution factor of 100% should be used to estimate the contribution of soil to airborne particulate matter/TSP concentrations under sparsely vegetated open field conditions. If particulate exposures are being evaluated for heavily vegetated open field conditions, the contribution factor may range from 100% to 40%.

For grading and excavation scenarios, a PM10 value of 61 should be used to estimate ambient concentrations. This value is the arithmetic mean of the 24 hour maximum PM10 values from 20 samplers (at 17 locations) in the Commonwealth during 1994 (1994 Air Quality Report, Commonwealth of Massachusetts). A contribution factor of 100% should be used to estimate the portion of ambient particulate level contributed by the construction activities.

There are a number of uncertainties associated with use of the default PM10 values, including:

- The published 24 hour averages may underestimate PM10 concentrations attained during the work day.

- The sampling locations are not necessarily located near construction activities or large areas of sparsely vegetated soil.

Therefore, these PM10 values are recommended for use only in the absence of more representative data.

7.3.3.7 Groundwater Exposure Point Concentrations

Private Wells

Exposure Points

Within a GW-1 area, the risk assessment should address both the risks associated with any well in use and the foreseeable risks from the installation of a private supply well anywhere within the contaminated area. Thus, the exposure points of concern should include both existing wells and the groundwater at any location where a well could potentially be installed. In other words, the groundwater at each monitoring well should be considered a foreseeable exposure point.

Thus, regardless of the risk assessment method employed, **exposure point concentrations and risks should be evaluated separately for each well in use and for each location (monitoring well) where a well could be installed within the contaminated area.** The risk assessor should assume that any one individual would be exposed only to water from one supply well. A single exposure point concentration should include data from locations within an area likely to be influenced by one supply well.

In general, BWSC recommends against averaging concentrations detected in different monitoring wells because monitoring wells are seldom clustered closely enough to lie within an area that would affect a single well. However, in exceptional cases where the locations of monitoring wells are clustered closely enough so that several would sample from an area of groundwater from which a single private supply well could draw, concentrations may be averaged.

The monitoring wells with the highest levels of contamination should be selected to represent potential supply well locations for the risk assessment. At some sites, one monitoring well may clearly represent the highest contaminant levels. At other sites where the groundwater is contaminated by a mixture of substances of varying relative concentrations, several monitoring wells may have to be evaluated as potential supply well locations.

Averaging Periods

The exposure point concentration for a private well should represent an estimate of the average concentration to which a user is likely to be exposed over the period of concern. Lifetime exposure assessments are based on a 30 year time period, chronic exposure evaluations typically focus on a seven year period, and subchronic exposure evaluations focus on period of three months (sometimes longer, but always less than seven years). Thus, the exposure point concentration should represent an estimate of a one year, seven year or lifetime average.

A three month average for a subchronic evaluation should be based on samples collected at a time when the concentrations can reasonably be expected to represent a maximum for the year. One sampling round is generally insufficient to obtain a reliable concentration estimate, and, confirmatory samples should always be collected.

Of course, site management decisions have to be made within time periods that are much shorter than seven years or a lifetime. Unless, as is discussed in the following paragraph, there is evidence that contaminant levels are increasing, it is reasonable to use the current annual average as an estimate of the seven year or lifetime average.

If the data suggest or show an increasing trend, the exposure point concentration estimate should reflect the predicted increase, and the assessment report should fully describe uncertainty about that estimate. However, such an estimate should only be used for preliminary site management decisions. Given the uncertainty associated with exposure estimates for wells where contamination is increasing, such estimates should not be used to support a conclusion that "no further action" is required.

If the data show a decreasing trend, it may be appropriate to use current values as an estimate of the long-term average. Including historical data in the calculation may lead to exposure estimates that are not consistent with respect to current or future conditions, and could lead to risk management decisions that are problematic. For example, it would be inappropriate to conclude that groundwater remediation is necessary in a situation where the concentrations are already below levels of concern for human health and are continuing to decrease.

Use of Mathematical Models

The use of mathematical models to estimate current exposure point concentrations for private wells is inappropriate. Existing wells should be sampled on a continuing basis to determine representative exposure point concentrations. Samples from the most highly contaminated monitoring wells (in *or* upgradient from the GW-1 area) should be used to represent potential exposures under foreseeable use and future conditions.

To Filter or Not to Filter

The nature of the samples analyzed to obtain exposure point concentrations at private water supplies should represent, as closely as possible, the nature of the water drawn from the wells in question. Often the water drawn from a private supply well is unfiltered, so, in theory, unfiltered groundwater samples from monitoring wells should be used to estimate potential exposure point concentrations. However, monitoring wells, especially newly developed monitoring wells, often produce samples that are quite turbid, and obviously are not representative of water that would be drawn from a supply well. For example, if the water from a monitoring well exceeds the turbidity standard for drinking water, it is reasonable to assume that the particulate levels are not representative of the water being drawn from the supply well. In such cases, BWSC recommends using **filtered samples** to estimate exposure point concentrations.

A promising alternative to filtering is using a peristaltic pump to purge monitoring wells and collect groundwater. In comparison to samples collected with a bailer, peristaltic pumps operated at a low flow rate (0.2 liters per minute) have reportedly produced samples that are less turbid and more representative with respect to groundwater metals concentrations (Acquisition of Representative Ground Water Quality Samples for Metals. Robert W. Puls and Robert M. Powell, *Ground Water Monitoring Review*. Summer 1992.) Although this technique has not been universally accepted or widely applied in field investigations to date, it appears to offer a reasonable alternative to the choice between filtering and not filtering, both of which have serious drawbacks. ORS would consider samples collected at low flow from monitoring wells to be reasonably representative of water drawn from a private supply well at the same location.

EPCs For Comparison to Drinking Water Standards

Massachusetts Drinking Water Quality Standards (310 CMR 22) are compared to exposure point concentrations as applicable suitably analogous standards. Each exposure point concentration, including those measured at monitoring wells, is compared with drinking water standards as a component of the Method 3 risk characterization. The drinking water quality regulations should be consulted for details concerning sampling and analysis required as part of these regulations. In general, the MMCLs are compared with average exposure point concentrations. For public water supply wells, the average of four quarterly samples is used.

Public Wells

Exposure point concentrations representing current conditions at public water supply wells are measured directly at the wellhead. Samples collected for baseline risk assessment purposes should represent pre-mixing, pre-treatment conditions. Neither mixing nor well head treatment is considered permanent, and these risk reduction activities should not be considered when estimating a baseline exposure point concentration.

Estimating exposure point concentrations under future conditions for public water supply wells is slightly more complicated than for private supplies. At supply wells located some distance away from the contamination source area, future concentrations depend on contaminant fate and transport processes such as dilution and dispersion. Even in future public supply wells that could potentially be installed in the most highly contaminated area, the exposure point concentrations are likely to be lower than current monitoring well concentrations because of dilution during pumping. As a consequence, a predictive model is needed to estimate exposure point concentrations at a public supply well under future conditions. Either a simple analytical model or a complex numerical model may be used.

BWSC generally recommends the use of a simple, conservative analytical approach to predict concentrations under future conditions. The results of a complex numerical model will not affect the conclusion of the risk assessment because of the requirement to characterize foreseeable risks by comparing standards to concentrations at each foreseeable exposure point. **The MCP requires the comparison of all current and foreseeable exposure point concentrations in GW-1 areas to applicable or suitably analogous standards** (310 CMR 40.0993(3)). Thus, current groundwater concentrations at each monitoring well in a GW-1 area must be compared with drinking water standards. If the monitoring well concentration exceeds the standard, the risk assessment will conclude that Significant Risk of harm to public health exists. This direct comparison of groundwater concentrations to standards is more likely to indicate the need for remediation than are risk estimates based on a model that incorporates dilution. Since modeled concentrations are not likely to affect the conclusions of the risk assessment, extensive mathematical modeling efforts are seldom warranted.

7.3.3.8 Indoor Air Exposure Point Concentrations

At disposal sites where soil or groundwater beneath a building is contaminated with volatile organic compounds, the potential for exposure to those substances must be considered in the human health risk assessment. Organic compounds can accumulate in indoor air by migrating from soil or groundwater, through the soil gas in the overlying unsaturated soil and into buildings through pores, cracks or openings in the foundation.

Exposure point concentrations in the air in any particular building are dependent upon a combination of conditions:

- The Henry's Law coefficients of the contaminant of concern, which provides an indication of their tendency to partition from the groundwater to the air spaces in

the overlying soil

- the concentrations of contaminant in the groundwater
- the depth of the water table below the surface of the soil
- the depth of the groundwater table below the building structure
- the physical characteristics of the soil at the location of concern
- the structure of the building
- the heating and ventilation features of the building which affect the rate at which soil gas will enter the building.

Measurement vs Modeling

The two basic approaches to estimating indoor air concentrations are direct measurement (air sampling followed by laboratory analysis) and estimation using a contaminant transport model. While each approach has advantages and disadvantages, direct measurement is preferable overall and is generally recommended for evaluating conditions in existing buildings associated with current groundwater concentrations.

It is often difficult to model indoor air concentrations with confidence from concentrations detected in groundwater, or even soil gas, for three reasons. First, the information needed to determine the validity of a model for a particular location and building is often not available. Second, the site-specific soil and building parameters needed to accurately model transport at a specific site may not be available. Third, models generally focus on water-soil gas partitioning and soil gas-indoor air diffusion, and don't account for other transport pathways, such as utility lines, that may provide the dominant migration route into a particular building.

Direct measurement also has some drawbacks. It is more resource intensive than modeling, and it is often logistically challenging. One of the most serious technical concerns is the fact that a single measurement event cannot provide an integrated estimate of the exposure point concentration over time. Indoor air concentrations in a building are heavily influenced by weather and by variations in use and activities. Thus, indoor air concentrations can vary substantially over time, and it may not be possible to predict whether concentrations at a given point in time represent a high, low or average estimate. (It should be noted that modeling does not necessarily provide an integrated estimate either, but the problem of temporal variation can be addressed to some extent by the selection of conservative modeling parameters; after that the question is generally set aside.)

The following sections discuss measurement and modeling considerations in more detail.

Indoor Air Sampling

To obtain a representative estimate of the concentration to which a person is likely to be exposed over time in a building, sampling locations, times, and methodology must be

planned carefully. Each of these considerations is discussed briefly in the following paragraphs.

Sampling Locations

Sampling locations should include areas where concentrations are likely to be highest and areas where the frequency and duration of exposure is high. Concentrations are normally expected to be highest in the basement, if there is one. However, people who live or work in the building are likely to spend more time in other areas. Results from all areas of a building should be incorporated in the exposure point concentration estimates, but data from different areas should be weighted to reflect exposure frequency. Samples from various rooms in a living area or a commercial building can vary substantially, so a number of areas should be sampled during each sampling round.

Sampling Over Time

In planning a sampling program, both **sampling time and sampling duration** are important to consider in obtaining a representative estimate.

In most buildings where volatile organic compounds migrate from groundwater into indoor air, the indoor air concentrations are likely to vary substantially over time. Seasonal changes in the depth to groundwater, temperature, and in building use can affect indoor air concentrations. Even daily changes in ambient air pressure may have a significant effect. For a long-term exposure evaluation (as opposed to an imminent hazard evaluation) sampling should be conducted several times a year. However, air sampling is time consuming and expensive, and it is not always possible to obtain samples that fully reflect temporal variations in concentration.

If sampling is only to be done once or twice because of resource constraints, the site assessment report must demonstrate that the concentrations would be highest at those times, considering depth to groundwater, heating system operating conditions, and building tightness (closed doors and windows).

The sampling duration should correspond as closely as possible to the duration of the exposure being evaluated. Since the duration of most indoor air sampling events ranges from a couple of hours to a day, and the results are often used to evaluate subchronic exposures (longer than a few months) and chronic exposures (longer than seven years), sampling durations should be as long as possible. Other factors that affect sampling duration are discussed in the following section on Sampling and Analysis Methodology.

Sampling and Analysis Methodology

Although an extensive discussion of sampling and analysis methodology is beyond the scope of this guidance, a few words of caution may be appropriate. Air sampling should

be planned and conducted by specialists in the field. Designing and executing an air sampling program requires a thorough understanding of the complexities and subtleties of air sampling theory and technology.

Method validation is crucial in enabling risk managers to make reasonable decisions based on sampling results. When available and appropriate, standard EPA methods should be employed. However, the utility of a standard method to the specific situation of concern should always be carefully evaluated.

Method sensitivity is one factor that often limits the applicability of standard methods at specific exposure situations. Because air intake rates are high relative to drinking water intake or soil intake rates, the concentration of a substance in air that is associated with a significant risk is relatively low. Therefore, it is particularly important to verify that a proposed air sampling methodology can achieve the necessary detection limits before conducting a sampling program.

Whatever the duration of an indoor air sampling event (from several hours to one day), the results are usually used to represent exposures that occur over much longer periods of time (from several months to a lifetime). In planning the duration of a sampling event, a balance must be struck between the need to collect samples that are reasonably representative of long term exposures and the technical constraints of available technologies. In many cases, the sampling duration is limited by the potential for breakthrough (desorption of contamination from the sample collection medium), which can be a serious problem if the volume of air drawn through the sampling tube is higher than that specified in the protocols. In some cases, a lower flow rate can be used to achieve a longer sampling duration. Again, it is recommended that sampling plans be developed by specialists with extensive experience in order plan flow rates and sampling durations that balance risk assessment and technical considerations.

Modeling Indoor Air Concentrations

Before a model is used, the validity of the model for conditions similar to those at the location of concern must be determined. **Precedent is not an indication of validation.** Validation must include obtaining or identifying data showing that the model can predict indoor air concentrations with a degree of accuracy that is sufficient for the risk assessment and the risk management decisions at hand.

Both groundwater and soil gas concentrations have been used as source terms for models. In principle, soil gas concentrations offer a preferable starting point, since they eliminate the need to model partitioning from groundwater into soil gas, and thus eliminate a significant source of uncertainty about the final estimate. However, soil gas measurements have a somewhat uneven track record, and in many cases, potential error associated with measuring soil gas concentrations may be a larger source of uncertainty than the partition model.

(MADEP/ORS is in the process of determining whether there are any existing models

that are generally valid and conservative and could be considered default models).

7.3.3.9 Exposure Point Concentrations Related To Surface Water and Sediment Contamination

Fish Consumption

Exposure point concentrations for fish consumption should be consistent with the type of exposure being evaluated. For chronic and subchronic exposure point concentration estimates, an average of the concentration detected in tissue of individual fish fillets may be used to represent the average concentration in fish that a person might consume over time. Ideally, sufficient data would be available to calculate exposure point concentrations for each fish species present so that the risk assessment could consider exposures to populations partial to eating certain species. For substances that could have acute toxic effects, the highest concentration detected should be used as the exposure point concentration estimate when evaluating the risks from acute exposures.

In many cases, it is not possible to obtain a large enough number of fish to calculate an average concentration with a reasonable degree of certainty. The risk assessor and project manager must then decide how to deal with the uncertainty. One option would be to use an upper Confidence Limit on the mean as a conservative estimate of the average concentration. An alternative would be to describe the uncertainty in the assessment report, and compensate for it by making a very conservative risk management decision. However, a sample number smaller than three would be insufficient basis for a public health-protective decision.

Appendix D contains a detailed discussion of fish tissue sampling and analysis considerations.

Swimming

Sediment and surface water exposure point concentrations used to evaluate swimming and wading exposures should represent conservative estimates of the arithmetic mean concentration in the shoreline area used for swimming or wading. If contamination is reaching a surface water body by groundwater discharge or by surface runoff, near shore areas may be more heavily contaminated. Concentrations of samples collected over large areas of a water body will not necessarily be representative, and should not be averaged. Likewise, if a model is used to predict concentrations likely to be attained in the future, the model should focus on the near shore area, and not the entire water body.

7.3.4 Exposure Equations

The following equations, organized by exposure medium, are provided to assist the risk assessor in quantifying a receptor's potential exposure to oil or hazardous material at a c.21E disposal site. The variables specific to each equation are discussed in this section while variables common to most of the equations were presented in the previous section. Default assumptions for these variables are provided in Appendix B.

7.3.4.1 Air

The toxicity information generally used to evaluate the risk of harm to health associated with inhalation exposures, Reference Concentrations and Units Risk values, are air *concentrations*. These values are intended to be used in combination with Average Daily Exposures expressed as applied concentrations, *not* dose. In the absence of RfCs or Unit Risk values, an oral Reference Dose or Slope Factor may be used to estimate risk either by: (a) calculating an Average Daily Dose from the inhalation pathway; or (b) converting the Reference Dose to a Reference Concentration and the Slope Factor to a Unit Risk. Thus, the equation chosen to evaluate the site inhalation exposures will depend upon the availability and nature of the toxicity information.

Calculation of Average Daily Exposure$_{air}$

Gaseous oil or hazardous material (for example, OHM volatilized from contaminated soil or groundwater) may be inhaled by the receptor of concern whenever the receptor is near the disposal site. The Average Daily Exposure to the contaminated air (ADE_{air}) is dependent upon the frequency and duration of the assumed exposures. The result of this calculation should be an estimate of applied concentration, *not* dose. Note that the equation is a simple adjustment of the exposure point concentration to account for the amount of time the receptor spends in the area with contaminated air.

$$ADE_{air} = \frac{[OHM]_{air} * EF * ED * EP * C}{AP} \qquad \textbf{(7-10)}$$

Where:

[OHM]$_{air}$ = Exposure point concentration of gaseous oil or hazardous material in the air at the Exposure Point during the period of exposure (dimensions: mass/volume; typical units: $\mu g/m^3$).

EF = Number of exposure events (frequency) during the exposure period divided by the number of days in the exposure period (dimensions: events/time; typical units: events/day)

ED = Duration of each exposure event (dimensions: time/event; typical units: hours/event)

EP = Duration of the exposure period (dimensions: time; typical units: years)

AP = Averaging Period (dimension: time; typical units: years)

C = Appropriate units conversion factor(s) (e.g., 10^{-6} kg/mg, 1 week/7 days)

For receptors assumed to be exposed constantly (such as for many residential

exposures), the Average Daily Exposure would be equal to the Exposure Point Concentration:

$$ADE_{air} = \frac{[OHM]_{air} * 1\frac{event}{day} * 24\frac{hours}{event} * 6\ years * \frac{1\ day}{24\ hours}}{6\ years} \qquad \textbf{(7-11)}$$

$$ADE_{air} = [OHM]_{air} \qquad \textbf{(7-12)}$$

Calculation of Average Daily Dose$_{air}$

As noted above, there are circumstances under which the *dose* or hazardous material experienced by a receptor breathing contaminated air may be calculated. The equation for estimating such an Average Daily Dose (ADD_{air}) is given as:

$$ADD_{gaseousOHM} = \frac{[OHM]_{air} * VR * RAF * EF * ED * EP * C}{BW * AP} \qquad \textbf{(7-13)}$$

Where:

[OHM]$_{air}$ = Exposure point concentration of gaseous oil or hazardous material in the air at the Exposure Point during the period of exposure (dimensions: mass/volume; typical units: $\mu g/m^3$)

VR = Ventilation (inhalation) rate for the receptor of concern during the period of exposure. (dimensions: volume/time; typical units: m^3/hour)

RAF = Relative Absorption Factor (unitless)

EF = Number of exposure events during the exposure period divided by the number of days in the exposure period (dimensions: events/time; typical units: events/day)

ED = Duration of each exposure event (dimensions: time/event; typical units: hours/event)

EP = Duration of the exposure period (dimensions: time; typical units: years)

BW = Body weight of the receptor of concern during the averaging period (dimension: mass; typical units: kg)

AP = Averaging Period (dimension: time; typical units: years)

C = Appropriate units conversion factor(s) (e.g., 10^{-6} kg/mg, 1 week/7 days)

7.3.4.2 Soil

The Average Daily Dose received by a receptor via direct contact with soil containing OHM (ADD_{soil}) is the sum of the average daily doses resulting from absorption via dermal contact with the contaminated soil and the incidental ingestion of that soil.

$$ADD_{Soil} = ADD_{dermal\ absorption} + ADD_{ingestion}\ [\ + ADD_{particulate\ inhalation}\ ] \qquad \textbf{(7-14)}$$

Additional soil-related exposures may result from the inhalation of fugitive dust originating from the contaminated soil.

Note: The general procedures for assessing soil exposure described in this section have been adapted from an on-going project within the Office of Research and Standards to develop methodology for deriving soil advisory levels (MADEP, 1995b).

Dermal Contact with Contaminated Soil

Dermal absorption of oil or hazardous material is a potentially significant route of exposure whenever direct contact with soil may occur. In fact, dermal absorption from soils may be more significant than incidental ingestion for chemicals which have a percent absorption exceeding about 10% (USEPA, 1992). (Even chemicals exhibiting percentage absorption less than 10% may contribute significantly to cumulative risk estimates and thus, these chemicals must also be evaluated.) The absorption of OHM from soil depends upon chemical-specific factors as well as the characteristics of the soil (such as particle size and organic carbon content).

The Average Daily Dose due to dermal contact with OHM contaminated soil ($ADD_{dermal\ absorption}$) may be calculated:

$$ADD_{dermal\ absorption} = \frac{[OHM]_{soil} * SA * AF * RAF * EF * ED * EP * C}{BW * AP} \qquad \textbf{(7-15)}$$

Where:

[OHM]$_{soil}$ = Representative concentration of OHM in the soil at the exposure point during the period of exposure (dimensions: mass/mass)
SA = Skin surface area in contact with the soil on days exposed (dimensions: area/time)
AF = Mass of soil adhered to the unit surface area of skin exposed (dimensions: mass/area)
RAF = Relative Absorption Factor (unitless)
EF = Exposure Frequency: the number of exposure events during the exposure period divided by the number of days in the exposure period (dimensions: events/time)
ED = Exposure Duration: the typical duration of each exposure event (dimensions: time/event)

EP = Exposure Period: the period of time over which exposure may occur (dimension: time)
BW = Body Weight of the receptor of concern during the averaging period (dimension: mass)
AP = Averaging Period (dimension: time)
C = Appropriate units conversion factor(s)

Incidental Ingestion of Contaminated Soil

The Average Daily Dose due to the incidental ingestion of OHM contaminated soil (ADD_{soi}) may be calculated:

$$ADD_{ingestion} = \frac{[OHM]_{soil} * IR * RAF * EF * ED * EP * C}{BW * AP} \quad \textbf{(7-16)}$$

Where:

ADD_{ing} = Average daily dose of oil or hazardous material received through the ingestion of soil, during the period of exposure (dimensions: mass/mass ▸ time, typical units: mg/kg ▸ day).
$[OHM]_{soil}$ = Exposure point concentration of the oil or hazardous material in soil (dimensions: mass/mass, typical units: mg/kg).
IR = Daily soil ingestion rate on days exposed during the exposure period (dimensions: mass/time, typical units: mg/day)
RAF = Relative Absorption Factor (dimensionless).
EF = Number of exposure events during the exposure period divided by the number of days in the exposure period (dimensions: events/time, typical units: events/day).
ED = Average duration of each exposure event (dimensions: time/event, typical units: day/event).
EP = Duration of the exposure period (dimensions: time, typical units: years).
C = Appropriate units conversion factor(s)
BW = Body weight of the receptor of concern during the averaging period (dimensions: mass, typical units: kg).
AP = Averaging Period (dimension: time, typical units: years).

DERMAL EXPOSURES:
COMPARISON WITH EPA-RECOMMENDED METHOD

Equation 7-15 incorporates the USEPA recommended approach of estimating dermally absorbed doses from ***any chemical*** present in soil. The USEPA equation (USEPA, 1992; equation 6.18) is based upon an experimentally determined (or theoretically derived) absorption fraction (ABS) to determine the absorbed dose per event:

$$DA_{event} = C_{soil} * AF * ABS \quad \textbf{(7-17)}$$

Where:

DA_{event} = Absorbed dose per event (mg/cm^2-event)
C_{soil} = Contaminant concentration in soil (mg/kg)(10^{-6} kg/mg)
AF = Adherence factor of soil to skin (mg/cm^2-event)
ABS = Absorption Fraction

Note that C_{soil} and AF of the USEPA equation correspond to $[OHM]_{soil}$ and AF in Equation 7-15. The Absorption Fraction (ABS) of the USEPA equation is incorporated into the Relative Absorption Factor (RAF) shown in Equation 7-15 (See Section 7.2.3 for a discussion of the derivation of RAFs).

This comparison of USEPA and MADEP approaches is included here to address a common misperception that EPA guidance recommends evaluating dermal absorption for only cadmium and PCBs.

Inhalation of OHM Contaminated Particulates

Airborne particulates (fugitive dust) may carry oil or hazardous material to receptors, resulting in soil-related inhalation exposures. An Average Daily Dose due to the inhalation of OHM contaminated particulates (ADD_{inhp}) may be calculated:

$$ADD_{particulate\ inhalation} = \frac{[RP]_{air} * [OHM]_{particulate} * VR * RAF * EF * ED * EP * C}{BW * AP} \quad \textbf{(7-18)}$$

Where:

$[RP]_{air}$ = Exposure point concentration of respirable particulates (i.e., PM_{10}) in the air at the Exposure Point during the exposure event. (dimensions: mass/volume; typical units: $\mu g/m^3$)

$[OHM]_{part}$ = Representative concentration of OHM in the respirable particulates at the Exposure Point during the period of exposure. (dimensions: mass/mass; typical units: mg/kg)

VR = Ventilation (inhalation) rate for the receptor of concern during the period of exposure. (dimensions: volume/time; typical units: m^3/hour)

RAF = Relative Absorption Factor (dimensionless)

EF = Number of exposure events during the exposure period divided by the number of days in the exposure period. (dimensions: events/time; typical units: events/day)

ED = Duration of each exposure event. (dimensions: time/event; typical units: hours/event)

EP = Duration of the exposure period (dimensions: time; typical units: years)

BW = Body weight of the receptor of concern during the averaging period (dimension: mass; typical units: kg)

AP = Averaging Period (dimension: time; typical units: years)

C = Appropriate units conversion factor(s)

For airborne chemicals which act at the point of contact (e.g. the lungs) when inhaled, the Average Daily Exposure of these chemicals calculated in the manner described in Section 7.3.4.1 would be used in combination with a *Reference Concentration* or *Unit Risk* to estimate potential risks. Under such conditions, the $ADD_{particulate\ inhalation}$ would not be calculated.

In situations with high particulate concentrations, the larger (greater than 10 μm) inhaled particulates may result in significant <u>*oral*</u> exposures which should also be quantified.

7.3.4.3 Sediment

The Average Daily Dose received by a receptor via direct contact (dermal absorption and incidental ingestion) with OHM contaminated sediment will be estimated in a manner similar to the calculation of the ADD for soil exposure, including both dermal contact with the sediment and incidental ingestion of that sediment. The inhalation of fugitive dust originating from contaminated sediments would not generally be evaluated unless climatic conditions resulted in such sediments becoming dry, thus increasing the potential for dust generation.

7.3.4.4 Drinking Water

The exposure experienced by a receptor using contaminated water is not limited to exposure received when actually drinking the water. Several studies indicate that significant exposure may also result from the inhalation of material volatilized from the water and through the absorption of contaminants from water in contact with the receptor's skin (Jo et al., 1990a and 1990b). Each of these exposure pathways should be evaluated separately, as described herein. The calculated oral and dermal doses are assumed to be equitoxic and may be mathematically combined:

$$ADD_{oral,\ dermal} = ADD_{oral} + ADD_{dermal} \qquad \textbf{(7-19)}$$

The assumption of equitoxicity is *not* assumed to apply to the dose received via the inhalation of volatilized material from the water, and the risk associated with this exposure must be evaluated separately using appropriate toxicity information.

Ingestion of Contaminated Drinking Water

The Average Daily Dose due to the ingestion of OHM contaminated drinking water (ADD_{dwi}) may be calculated:

$$ADD_{ingestion} = \frac{[OHM]_{water} * VI * RAF * EF * ED * EP * C}{BW * AP} \qquad \textbf{(7-20)}$$

Where:

[OHM]$_{water}$ = Exposure point concentration of oil or hazardous material in the drinking water at the exposure point during the exposure period (dimensions: mass/volume; typical units: µg/liter)

VI = Volume of drinking water ingested by the receptor of concern at (or from) the exposure point during the exposure period (dimensions: volume/time; typical units: liters/day)

RAF = Relative Absorption Factor (unitless)

EF = The exposure frequency, or the number of exposure events during the exposure period divided by the number of days in the exposure period (dimensions: events/time; typical units: events/day)

ED = Duration of each exposure event (dimensions: time/event; typical units = days/event)

EP = Duration of the exposure period (dimension: time; typical units: years)

BW = Body weight of the receptor of concern during the averaging period (dimensions: mass; typical units: kg)

AP = Averaging Period (dimension: time)

C = Appropriate units conversion factor(s)

Dermal Absorption of OHM Via Drinking Water

Dermal absorption of oil or hazardous material may occur while the receptor is in contact with the contaminated drinking water. Everyday activities such as showering, bathing, washing floors and cooking lead to direct contact with water and may result in dermal absorption of the chemicals.

DEP/ORS has assessed the magnitude of the dermal exposure received during showering (Brown et al., 1984) and has evaluated this exposure relative to that which a receptor would be expected to receive from drinking the same water. For most organic compounds, the shower/dermal absorption exposures are estimated to be approximately

20% (or less) than the estimated drinking water ingestion exposures (MADEP, 1992a). For chemicals which penetrate the skin the fastest (i.e., those with high permeability constants of approximately 1 $cm^3/cm^2 \cdot hr$ or greater), the dermal doses received are roughly equivalent to the ingestion doses (Hutcheson, et al., in press). Based upon these observations, BWSC recommends that the following streamlined approach be adopted[1]:

- For the majority of organic compounds, the absorbed dermal dose may be approximated as 20% of the calculated dose received from drinking water ingestion:

$$ADD_{dermal} = 0.2 * ADD_{ingestion}$$

- For organic compounds which have a permeability constant greater the 0.5 $cm^3/cm^2 \cdot hr$ (including ethylbenzene and toluene), the absorbed dermal dose may be approximated as the calculated dose received from drinking water ingestion:

$$ADD_{dermal} = ADD_{ingestion}$$

- For metals and inorganic compounds, the dermal exposures experienced during showering may be assumed to be negligible when compared with the exposures received while ingesting the contaminated water.

These approximations are considered protective for most chemicals, and when applied within the stated limitations, would be generally be acceptable to the BWSC. However, the approach is generic, and will yield less accurate dose estimates for some compounds than others. Therefore, as an alternative, the risk assessor may choose to explicitly calculate the dose received when the receptor comes into dermal contact with contaminated water. The equation presented under *Surface Water Exposures* may be used with assumptions appropriate to the specific exposure being modelled.

Inhalation of OHM Volatilized from Drinking Water

As with the dermal exposures associated with the use of drinking water, numerous studies (Andelman, 1985; Foster and Chrostowski, 1987; McKone, 11987; McKone, 1991) have looked at the magnitude of the inhalation exposures associated with household water use. Based on a review of those studies, ORS has concluded that for *volatile* organic compounds (i.e. compounds with a Henry's Law Constant equal to or

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1 These approaches assume 100% absorption via ingestion. The equation should be modified (dividing the ADD ingestion by the oral absorption efficiency) if less oral absorption is assumed to occur.
Example: **ADDdermal = 0.2 * ADDingestion ÷ Oral Absorption Efficiency**
Note that assuming lower oral absorption increases the fraction of the total dose attributable to dermal contact.

greater than 5 x 10^{-4} atm*m^3/mol*K), the shower/inhalation exposures are likely to be approximately equal to and no greater than the estimated drinking water ingestion exposures. However, exposures to compounds with lower Henry's Law Constants are likely to be lower.

Based upon these observations, BWSC recommends that the following streamlined approach be adopted for the evaluation of shower/inhalation exposures:

- For chemicals with a Henry's Law constant equal to or greater than 5 x 10^{-4} atm ▸ m^3/mol ▸ ˜K (at 20→25˜C), the applied dose (in mg/kg/day) received via inhalation may be approximated as the calculated *applied* dose received from drinking water ingestion (This value would correspond to the result of Equation 20 *if the RAF factor were removed*.)

- For chemicals with a Henry's Law constant less than 5 x 10^{-4} but greater than or equal to 1 x 10^{-5} atm ▸ m^3/mol ▸ ˜K (at 20→25˜C), the applied dose (in mg/kg/day) received via inhalation may be approximated as one half the calculated *applied* dose received from drinking water ingestion (or ½ The value which would result from Equation 20 *if the RAF factor were removed*.)

- For chemicals with a Henry's Law constant less than 1 x 10^{-5} atm ▸ m^3/mol ▸ ˜K (at 20→25˜C), the inhalation exposures experienced during showering are assumed to be negligible relative to the ingestion exposures and would not need to be evaluated unless the chemical under investigation is significantly more toxic when inhaled than when ingested.

Unlike the dermal exposures, however, it cannot be assumed that the chemicals have equal toxicity by inhalation and oral exposures. In order to estimate risk using the Reference Concentration or Unit Risk toxicity values, the doses approximated as above must be converted to an applied inhalation exposure (in concentration units such as μg/m^3) using the following equation:

$$ADE_{inhalation} = \frac{ADD_{inhalation} * BW * C}{VR} \qquad \textbf{(7-21)}$$

Where:

ADE_{inh} = The average daily exposure to the contaminant in air resulting from one shower exposure per day (dimensions: mass/volume; typical units: μg/m^3).
ADD_{inh} = Average daily dose of OHM (ia inhalation) approximated from the water ingestion pathway (dimensions: mass/mass ▸ time; typical units: mg/kg ▸ day).
BW = Body weight of the receptor of concern during the averaging period (dimension: mass; typical units: kg).
C = Appropriate units conversion factor(s).
VR = Ventilation (inhalation) rate for the receptor of concern during the exposure event (dimensions: volume/time; typical units: m^3/hr.)

NOTE: Equation 21 provides the calculation of an Average <u>Daily</u> Exposure. If the goal is to calculate the exposure point concentration during the shower event, Exposure Frequency and Exposure Duration terms should be inserted in the <u>denominator</u> of Equation 21:

EF = Exposure frequency. The number of shower events during the exposure period divided by the number of days in the exposure period. (Dimensions: events/time; typical units: event/day).

ED = Duration of shower exposure event (dimensions: time/event; typical units: minutes/event).

Alternatively, shower models available in the literature (Foster and Chrostowski, 1987) may be used to estimate chemical-specific air exposures.

7.3.4.6 Surface Water

Contamination in surface water can result in receptor exposures from the incidental ingestion of the water, through dermal contact with the water, and through the inhalation of material volatilized from the water. As with the drinking water evaluation, the ingestion and dermal doses are assumed to be equitoxic and the estimated values may be mathematically combined:

$$ADD_{oral,\ dermal} = ADD_{oral} + ADD_{dermal} \qquad \textbf{(7-22)}$$

The assumption of equitoxicity is *<u>not</u>* assumed to apply to the dose received via the inhalation of volatilized material from the water, and the risk associated with this exposure must be evaluated separately using appropriate toxicity information.

<u>Surface Water Ingestion</u>

The equation used to estimate the Average Daily Dose received by a receptor via the ingestion of contaminated surface water ($ADD_{surface\ water\ ingestion}$) is identical to that used to evaluate drinking water ingestion exposures, which is described earlier in this section. The assumptions chosen to describe the exposure (the volume of water ingested, the duration of the exposure event, etc...) should be representative of the exposure scenario being modelled.

<u>Surface Water, Dermal Contact</u>

The Average Daily Dose of a chemical received via dermal absorption from surface water ($ADD_{dermal,\ water}$) may be calculated using the following equation. This approach is recommended by BWSC for all chemicals when the dermal exposure is explicitly calculated.

$$ADD_{dermal,water} = \frac{[OHM]_{water} * SA * K_p * RAF * EF * ED * EP * C}{BW * AP} \quad \textbf{(7-23)}$$

Where:

ADD_{dermal} = Average daily dose of oil or hazardous material associated with dermal contact exposure to contaminated water. In units: mg/kg/day.

$[OHM]_{water}$ = The concentration of contaminant in water which is contacting the skin during the exposure event. (Dimensions: mass/volume; typical units: µg/liter).

SA = Body surface area exposed to contaminated water during the exposure event. (dimensions: area; typical units: cm^2).

K_p = Permeability Constant. (dimensions: volume/(time * area); typical units: cm^3/(hr * cm^2), which is often simplified to cm/hr).

RAF = Relative Absorption Factor for dermal contact with water. **Note:** when the permeability constant (K_p) is used to determine the flux of contaminant through the skin, it results in an *absorbed* dose of OHM. The RAF is used here to adjust this absorbed dose to make it comparable to the toxicity value employed to estimate risk. The numerator of the RAF must be assigned a value of 1, and the denominator depends upon the absorption in the study which is the basis of the toxicity value (See Section 7.2.3). If the toxicity value itself is based on an *absorbed* dose, then the RAF_{dermal} is 1 by definition. Dimensionless.

EF = The exposure frequency, or the number of exposure events during the exposure period divided by the number of days in the exposure period (dimensions: events/time; typical units: **events/day**).

ED = The duration of each exposure event (dimensions: time/event; typical units: hours/event).

EP = Duration of exposure period (dimension: time; typical units: years).

C = Appropriate units conversion factor(s).

BW = Body weight of the receptor of concern during the averaging period (dimensions: mass; typical units: kg).

AP = Averaging Period (dimension: time; typical units: years).

Alternatively, another model, specific to organic compounds and assuming some exposure period before a steady-state condition is established, is described in a USEPA Interim Report (USEPA, 1992). The USEPA cautions in that document that this procedure is still under review by the scientific community and that further refinement of the approach is expected.

Inhalation Exposures Associated With Contaminated Surface Water

Under some circumstances the volatilization of oil or hazardous material from surface water may contribute to exposure experienced by the receptor of concern. Such exposures are more likely to be of concern if the material is volatilizing into a confined space or if the concentrations in the surface water are relatively high. The exposures associated with this scenario may be evaluated following the equation presented in Section 7.3.4.1, with the $[OHM]_{air}$ term being either measured or modelled air concentrations of the contaminant.

7.3.4.7 Food

The average daily dose (ADD_{food}) experienced by the receptor as a result of consuming food (e.g. garden produce) containing oil or hazardous material may be estimated using the following equation. The general form of this equation may be applied to the ingestion of contaminated fish, meat, or vegetables. The evaluation of exposure to infants from ingesting mother's milk or other fluids may be estimated using the general equation for drinking water exposures in combination with the appropriate exposure factors.

$$ADD_{food} = \frac{[OHM]_{food} * FI * RAF * EF * ED * EP * C}{BW * AP} \qquad \textbf{(7-24)}$$

Where:

[OHM]food = Representative concentration of OHM in the food of concern during the period of exposure (dimensions: mass/mass, typical units: mg/kg)
FI = Daily intake of the food of concern on days exposed during the exposure period (dimensions: mass/event; typical units: mg/meal)
RAF = Bioavailability Adjustment Factor
EF = Number of exposure events during the exposure period divided by the number of days in the exposure period (dimensions: events/time. typical units: meals/day)
ED = Duration of the exposure period (dimension: time, typical units: years)
BW = Body weight of the receptor of concern during the averaging period (dimension: mass, typical units: kg)
AP = Averaging Period (dimensions: time, typical units: years)
C = Appropriate units conversion factor(s)

7.3.4.8 Calculation of Lifetime Average Daily Dose (For All Media)

The lifetime average daily dose should be calculated to reflect age-related differences in exposure rates that are experienced by a receptor throughout his or her lifetime of exposure. Because of their low body weight and behavioral characteristics, young children receive greater exposure per unit body weight than older children and adults. Furthermore, young children typically have more dermal contact with soil and more hand-to mouth activity. Therefore, the LADD should be calculated in a way that does not "dilute" the higher exposure rates experienced by young children with lower exposure rates experienced by older children and adults.

For example, a LADD (based on a 30-year exposure period) which uses an average body weight and skin surface area value for all ages of receptor (1<31) will not be protective of the high exposure rates in young children and is <u>not</u> a recommended procedure.

There are a number of averaging methods that can be used to calculate a LADD that reflects the higher exposure rates experienced by young children. One method is to calculate average annual dose rates, normalized to body weight, for each year of exposure. The sum of the dose rates is then averaged over a lifetime (75 years). The equation below

shows this averaging approach. However, this type of calculation can be tedious, even when performed by computer.

$$LADD = \frac{\sum_{i=0}^{30\ years} \frac{IR_i \times EP_i}{BW_i}}{AP} \qquad \textbf{(7-25)}$$

Where:

IR_i = Average Intake rate for the exposure period (mg/day)
EP_i = Exposure period, one year
BW_i= Age-dependent body weight, ages 0 to 30
AP = Averaging Period, lifetime (75 years)

As an alternative, there is a simpler averaging approach which can be used to calculate the lifetime average daily dose. This simpler approach gives essentially the same results as the year-by-year averaging method. The simpler averaging approach uses a weighted average for younger children aged 1 to 6. Children aged 1 to 6 is a logical choice for the weighted group because the default soil ingestion rate for children aged 1 to 6 is 100 mg per day (double the rate used for older children and adults). Thus, children aged 1 to 6 have a much higher rate of exposure because of the higher rate of soil ingestion assumed.

As the equation below shows, only two Average Daily Doses need to be calculated instead of 30. This greatly simplifies the calculations. The Average Daily Dose for children aged 1 to 6 is calculated using average exposure parameters for children in this age group. Similarly, the Average Daily Dose for the receptors aged 6 to 31 is calculated using average values for receptors in this group. The LADD is then calculated as the sum of the two doses averaged over a lifetime. The equation below shows this weighted calculation.

$$LADD = \frac{\frac{IR_{1<6}}{BW_{1<6}} \times EP_1 + \frac{IR_{6<31}}{BW_{6<31}} \times EP_2}{AP} \qquad \textbf{(7-26)}$$

Where:

$IR_{1<6}$ = Average Intake rate for receptors aged 1<6 (mg/day)
EP_1 = Exposure period, 5 years
$BW_{1<6}$ = Average body weight for children ages 1 to 6
$IR_{6<31}$ = Average Intake rate for receptors aged 6 to 31 (mg/day)
EP_2 = Exposure period, 25 years
$BW_{6<31}$ = Average body weight for receptors aged 6 to 31
AP = Averaging Period, lifetime (75 years)

As stated above, this weighted average approach can be used to calculate the LADD and

will result in essentially the same results as the more complicated year-by-year averaging approach.

7.4 RISK CHARACTERIZATION

Risk Characterization is the final step in the risk assessment process. In this step, the results of the Hazard Identification, Dose-Response Assessment and Exposure Assessment are integrated to yield quantitative measures of cancer and noncancer risk. The Risk Characterization can be thought of as providing a link between risk assessment and risk management because it presents the numerical estimates of risk posed by the site in a context that can be used easily by risk managers to make decisions about remediation.
In accordance with the MCP (310 CMR 40.0993(6)), the Risk Characterization step must also must include a comparison of Exposure Point Concentrations (EPCs) with applicable or suitably analogous public health standards.

A critical component in the presentation of risk estimates is the discussion of major assumptions, scientific judgements and uncertainties inherent in the numerical risk estimates. The importance of this component cannot be overstated. The discussion of uncertainties should place the numerical estimates of risk and hazard in the overall context of what is known about the site and what is uncertain. The numerical risk estimates should never be interpreted as a characterization of absolute risk but should always be interpreted in the context of the uncertainties.

The regulations provide clear direction regarding the way numerical estimates of risk are to be presented in the Risk Characterization (310 CMR 40.0933). The MCP requires that chemical-specific and medium-specific estimates of risk be combined to yield Cumulative Cancer and Noncancer Risks for each Receptor. These Cumulative Risks are then compared with specific risk management criteria which include public health standards and Cumulative Receptor Risk Limits (310 CMR 40.0933(6)). The result of this comparison determines whether a condition of No Significant Risk of harm to human health exists or has been achieved at the site.

This Section of the *Guidance* describes methods for characterizing cancer and noncancer risks and discusses the interpretation of Risk Characterization results within the context of the MCP. This Section also addresses the identification of Applicable or Suitably Analogous Public Health Standards and the comparison of such standards with EPCs. Lastly, this Section addresses how uncertainties in the Risk Assessment should be addressed.

7.4.1 Noncancer Risk

The measure used to describe the potential for noncarcinogenic health effects is the Hazard Index (HI). For a given chemical, the HI is the ratio of a receptor's exposure level (or dose) to the *"acceptable"* (or allowable) exposure level. A Hazard Index of 1.0 or less indicates that the receptor's exposure is equal to or less than the allowable exposure level, and it is considered unlikely that adverse health effects will occur. When the HI is less than or equal to 1.0, a conclusion of "No Significant Risk of harm to human health" based on non-cancer effects, is appropriate.

A HI of greater than 1.0 indicates that noncancer health effects could occur, and cannot be ruled out. It does not mean that noncancer effects *will* occur. Uncertainty inherent in most Reference Doses precludes identifying a specific dose above which adverse effects are likely *and* below which effects are unlikely. Accordingly, the probability of an effect cannot be quantified from a HI. *For any one chemical*, it is always true that the likelihood of an effect increases as the exposure level (and therefore the HI) increases.

The uncertainty inherent in RfDs for different chemicals differs both qualitatively and quantitatively. Therefore, for different substances, the probability of an effect increases at different rates. For example, a HI of 20 for one substance may indicate a very high probability of an effect, but may represent only a moderate probability of an effect for another chemical.

In interpreting the HI, one must consider the appropriateness of the exposure assumptions and the basis of the toxicity information used to develop the RfD. As a general rule, the greater the HI is above 1.0, the greater the level of concern.

In its most general form, the Hazard Index associated with a chemical via a given route of exposure is calculated as:

$$HI = \frac{ADD}{RfD} \qquad \textbf{(7-27)}$$

or, for inhalation exposures,

$$HI = \frac{[OHM]_{air}}{RfC} \qquad \textbf{(7-28)}$$

Where:

HI =	The Hazard Index associated with exposure to the chemical via the specified route of exposure.
ADD =	The estimated Average Daily Dose of the chemical via the specified exposure route. In **mg/kg/day**.
RfD =	The oral Reference Dose or appropriate substitute toxicity value identified for the chemical of concern. In **mg/kg/day**.
$[OHM]_{air}$ =	The Exposure Point Concentration of the Oil or Hazardous Material in air. In **µg/m³**.
RfC =	The Reference Concentration or substitute toxicity value identified for the chemical of concern. In **µg/m³**.

The Average Daily Dose (ADD) in equation 7-27 is calculated from the Exposure Point Concentration using exposure assumptions consistent with the Exposure Profiles developed for each receptor being evaluated. Section 7.3 of this Guidance describes the process for calculating a receptor's ADD.

The allowable dose or exposure (denominators in equations 7-27 and 7-28) will typically be the EPA Reference Dose (RfD) for most exposure routes or the EPA Reference Concentration (RfC) for air exposures. Selection of an appropriate "acceptable" dose is discussed in Section 7.2.

It is important to calculate separate HIs for acute, subchronic or chronic exposures if these have been identified as exposure periods of concern in the development of exposure profiles.

As mentioned previously, the MCP requires that *cumulative* noncancer risks be calculated. A cumulative HI represents the cumulative noncarcinogenic impact that the site has on a particular receptor group. The cumulative HI accounts for exposures that a receptor may receive from multiple chemicals and multiple exposure routes.

Again, remember that separate cumulative HIs are calculated for acute, subchronic or chronic exposures that have been identified as exposure period of concern for the site.

As shown by the following two equations, the cumulative HI can be calculated by summing the exposure route-specific HI. Route specific HI are calculated as the sum of all chemical-specific HIs.

$$Total\ HI_{route\text{-}specific} = \sum HI_{chemical\text{-}specific} \quad \textbf{(7-29)}$$

$$Cumulative\ HI = \sum HI_{route\text{-}specific} \quad \textbf{(7-30)}$$

If the risk calculations are being performed using a probabilistic analysis, the risk assessor must identify the dose or concentration associated with the 95th percentile estimate of exposure (310 CMR 0993(5)). This dose or concentration should be compared with the toxicity value identified following the dose/response section of this Guidance. This HI is then compared with the HI Limit of 1.0 in order to determine whether the site poses a significant risk of harm to human health based on the risk of noncancer health effects.

The documentation of the Risk Characterization must clearly present all mathematical equations used to calculate Cumulative Noncancer Risks (310 CMR 40.0993(9).

7.4.1.1 Screening Hazard Index

Initially, the risk assessor should use equation 7-30 above to calculate a *Screening Hazard Index* for a given receptor group based on all chemicals of concern at the site in all exposure routes at all exposure points. A HI calculated in this way will provide a conservative estimate of the true HI because it treats as additive, different toxic effects from multiple chemicals acting on different organ systems by different mechanisms of action. In fact, in a true HI, the only endpoints which should be treated as additive are those which produce adverse effects on the same organ system by the same mechanism. Thus, the Screening HI will provide a conservative estimate of the actual HI because it reflects the sum of toxicities for multiple chemicals, regardless of the chemical's health endpoint, target organ or mechanism of action.

Recall that there may be multiple adverse health effects associated with exposure to a given chemical and it is the most sensitive adverse health effect observed in the scientific data which drives estimation of the Reference Dose. Thus, for a given group of chemicals, Reference Doses may be based on a different toxic effects on different organ systems by different mechanisms of action.

The screening HI should be compared with the MCP Cumulative Receptor Noncancer Risk Limit which is a HI equal to 1.0 (310 CMR 40.0933(6)). If the screening HI is less than 1.0, then no additional effort is needed to characterize noncancer risks. However, if the screening HI exceeds 1.0, the risk assessor should then calculate separate HIs for chemicals with similar toxic effects and mechanisms of action.

Remember that separate screening HIs should be calculated for different exposure periods (i.e., chronic, subchronic, acute).

7.4.1.2 Health Endpoint-Specific Hazard Index

The procedure for segregating HIs by effect and mechanism of action is not simple and should be performed by a toxicologist. If the segregation is done improperly, an underestimate of the true hazard could result. Segregation of HIs requires identification of the major health endpoints of each chemical, including effects observed at higher doses than the critical effect on which the toxicity value is based. This is because the critical effect for one chemical may not be relevant for other chemicals and doses of other chemicals may not be additive for that effect. On the other hand, additive impacts could be important for other health endpoints that are only expected at higher doses.

Major effect categories that should be considered in segregating chemicals include neurotoxicity, developmental toxicity, reproductive toxicity and immunotoxicity. Adverse effects also should be categorized by target organ (i.e., hepatic, renal, respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal and dermal/ocular). The effects and mechanism of action should be discussed in the toxicological profile.

Once chemicals have been categorized, the Cumulative Hazard Index for chemicals with similar health endpoints and mechanisms of toxicity should be calculated. Each HI should be compared with the MCP Cumulative Receptor Noncancer Risk Limit which is a HI equal to 1.0. If any of the HIs exceeds one, then the Risk Characterization must conclude that the site poses Significant Risk of harm to human health based on the risk of noncancer health effects.

7.4.2 Cancer Risk

The potential for carcinogenic (i.e., nonthreshold) health effects is characterized as the Excess Lifetime Cancer Risk (ELCR). The ELCR represents the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen. For a given chemical, the estimated ELCR is the product of the receptor's quantified exposure and a measure of carcinogenic potency. The typical measures of carcinogenic potency are the EPA Cancer Slope Factor (SF) for most exposure routes and the Unit Risk (UR) for inhalation.

In its basic form, the ELCR associated with exposure to a given chemical via a particular exposure pathway is estimated as follows:

$$ELCR = LADD \times SF \quad \textbf{(7-31)}$$

or, for inhalation exposures,

$$ELCR = [OHM]_{air} \times UR \quad \textbf{(7-32)}$$

Where:

ELCR = The Excess Lifetime Cancer Risk associated with exposure to the chemical via the specified route of exposure.

LADD = The estimated Lifetime Average Daily Dose of the chemical via the specified exposure route. In **mg/kg/day**.

SF = The Cancer Slope Factor identified for the chemical, appropriate to the specific exposure pathway. In **(mg/kg/day)$^{-1}$**. The selection of this toxicity value is discussed in Section 7.2.2 of this Guidance.

$[OHM]_{air}$ = The Exposure Point Concentration of the Oil or Hazardous Material in air. In **µg/m^3**.

UR = The Unit Risk for the particular chemical of concern. In **µg/m^3**. The identification and selection of UR values is described in Section 7.2.2.

The Lifetime Average Daily Dose (LADD) in equation 7-31 is calculated from the Exposure Point Concentration using exposure assumptions consistent with the Exposure Profiles developed for each receptor being evaluated. Section 7.3 of this Guidance describes the process for calculating a receptor's LADD. The selection of Cancer Slope Factors and Unit Risk values is discussed in greater detail in Section 7.2.2.

As mentioned previously, the MCP requires that *cumulative* cancer risks be calculated. The cumulative cancer risk must be estimated for all Class A and B carcinogens (i.e., chemicals classified by EPA as being known human carcinogens and probable human carcinogens). For most Class C Carcinogens (i.e., those classified by EPA as being possible human carcinogens), the available toxicity data is insufficient to quantify cancer risks. In general, potential carcinogenic effects of these substances should be discussed qualitatively in the Uncertainty Section of the Risk Assessment. However, the Department may in the future identify some Class C carcinogens for which there is sufficient data to include these substances in the quantitative assessment of carcinogenic risk.

The cumulative ELCR represents the cumulative carcinogenic impact that the site has on a particular receptor group. The cumulative ELCR accounts for exposures that a receptor may receive from multiple chemicals and multiple exposure routes.

As shown by the following two equations, the cumulative ELCR can be calculated by summing all of the exposure route-specific ELCRs. Route-specific ELCRs are calculated as the sum all the chemical-specific ELCRs.

This is represented by the following equations:

$$Total\ ELCR_{route\text{-}specific} = \sum ELCR_{chemical\text{-}specific} \quad \textbf{(7-33)}$$

$$Cumulative\ ELCR = \sum ELCR_{route\text{-}specific} \quad \textbf{(7-34)}$$

The Cumulative ELCR should be compared with the MCP Cumulative Receptor Cancer Risk Limit which is an ELCR equal to one-in-one hundred thousand (1×10^{-5}). If the Cumulative Cancer Risk exceeds the ELCR Limit then the Risk Characterization must conclude that the site poses significant risk of harm to human health based on the risk of cancer health effects.

If the risk calculations are being performed using a probabilistic analysis, the risk assessor must identify the dose or concentration associated with the 95th percentile estimate of exposure (310 CMR 0993(5)). This dose or concentration should be compared with the toxicity value identified following the dose/response section of this Guidance. This ELCR is then compared with the Cancer Risk Limit of 1×10^{-5} in order to determine whether the site poses a significant risk of harm to human health based on the risk of cancer health effects.

The documentation of the Risk Characterization must clearly present all mathematical equations used to calculate Cumulative Cancer Risks (310 CMR 40.0993(9).

7.4.3 Comparison to Applicable or Suitably Analogous Public Health Standards

The MCP requires that the characterization of risk of harm to human health include a comparison of EPCs to applicable or suitably analogous public health standards. The list of such standards, as provided in the MCP includes, but is not limited to:

- Massachusetts Drinking Water Quality Standards, promulgated in 310 CMR 22.00 *(these standards are considered applicable only to category GW-1 groundwater).*
- Massachusetts Air Quality Standards promulgated in 310 CMR 6.00; and
- Massachusetts Surface Water Quality Standards promulgated in 314 CMR 4.00.

It should be noted that the MCP Method 1 Soil and Groundwater Standards listed in 310 CMR 40.0970 are <u>not</u> considered applicable or suitably analogous, as those standards represent an alternative risk characterization approach to Method 3. MADEP staff have noted a tendency to include a list of the Method 1 standards in Method 3 risk characterizations, but including those standards only confuses the reader and brings into question how the risks were actually characterized.

As provided in the MCP, if any EPC exceeds an applicable or suitably analogous standard, the Risk Characterization must conclude that a condition of Significant Risk exists at the site.

7.4.4 Risk Characterization Conclusions

The documentation of the Method 3 Human Health Risk Characterization must contain a clear statement of whether or not a condition of No Significant Risk of harm to human health exists or has been achieved, based upon the criteria contained at 310 CMR 40.0993(7).

As provided in the MCP, a condition of No Significant Risk of harm to human health exists or has been achieved at the site if:

- no Exposure Point Concentration of oil or hazardous material is greater than an applicable or suitably analogous public health standard; *AND*

- no Cumulative Receptor Cancer Risk calculated is greater than the Cumulative Cancer Risk Limit; *AND*

- no Cumulative Receptor Noncancer risk is greater than the Cumulative Receptor Noncancer Risk Limit.

Note that all three criteria must be met in order for a conclusion to be reached that the site poses No Significant Risk of harm to human health.

7.5 UNCERTAINTY ANALYSIS

The Uncertainty Analysis is a critical component of the Risk Characterization. The Uncertainty Analysis should contain a narrative section which places the numerical risk estimates in the overall context of what is known and what is not known about the site and in the context of decisions that the site manager will make about remediation. The Uncertainty Analysis does not modify the risk characterization conclusions themselves. However, a Risk Characterization is not considered complete unless the numerical risk estimates are accompanied by an explanation which interprets and qualifies the risk results.

Inherent in all risk assessments are many assumptions, scientific judgements and a wide variety of uncertainties, which can be introduced at each step in the risk assessment process. In addition, dose response and exposure assessment guidance presented in this document are intended to produce conservative, consistent estimates of the potential for adverse impacts. For all of these reasons, the numerical risk estimates calculated in the Risk Characterization should never be interpreted as absolute, purely scientific estimates of the risk of harm to health.

General sources of uncertainty in the risk assessment which should be discussed in the Uncertainty Analysis include, but are not limited to:

- Identification of all site-related contaminants in sampling of the environmental media at the site.

- Modeling used to develop Exposure Point Concentrations.
- Quantitative toxicological data used to develop cancer and noncancer toxicity values.
- Development of Exposure Profiles and selection of exposure assumptions used in dose calculations.

Although the Uncertainty Analysis may be a qualitative evaluation of uncertainties affecting the risk estimates, the risk assessor should attempt to describe the magnitude and direction of effect that a particular area of uncertainty is likely to have on the numerical risk estimates.

Monte Carlo Analysis can be a powerful tool for expressing the uncertainties in risk assessments. The reader should refer to Appendix C for a discussion about the use of Monte Carlo Analysis.

7.6 SHORTCUTS

Under certain circumstances, it may be possible to substantially reduce the level of effort necessary to conduct a Method 3 risk assessment. Two possible shortcuts, the "Screening" Risk Characterization and the *DEP Risk Assessment ShortForm - Residential Scenario* are specifically discussed.

Other shortcuts, if they are logical, clearly identified and defensible (usually with a quantitative demonstration) may be used as well and are encouraged. Using a shortcut *without* adequate justification is inappropriate.

7.6.1 Screening Risk Characterization

One shortcut option that may be considered is to conduct a *"Screening"* Human Health Risk Characterization using worst-case exposure assumptions (310 CMR 40.0902(5)). The objective of a screening evaluation is to quickly demonstrate that a condition of No Significant Risk exists or has been achieved at a disposal site. To do this, the risk assessor should use worst-case exposure assumptions and conservative toxicity values. For example, the risk assessor might assign the toxicity value for the most toxic substance at a site to all substances at the site and use the maximum reported concentration for each chemical as the EPC. Assuming residential exposures at an industrial site is another possible overly-conservative assumption that may be used in a screening risk characterization.

The objective of the screening risk characterization is to save time and money by using readily available data and information that will result in risk estimates that will not underestimate the risks posed by the disposal site. Thus, if the resulting risks are below the MCP Risk Limits, clearly, remediation would not be required based on risk of harm to human health. It is important to note that remediation may still be required based on risk of harm to the environment, public welfare or safety.

A screening risk characterization may also be used to demonstrate that certain exposure pathways result in risks which are trivial, compared with the MCP Cumulative Risk Limits. Such a demonstration would justify the elimination of that exposure pathway from consideration in the risk characterization. In general, "trivial" is considered as being a level of risk that is at least one order of magnitude smaller than the MCP Risk Limit, based on a conservative risk characterization as described in the preceding paragraphs.

A screening risk characterization is intended as an option to reduce the cost and level of effort involved in conducting a risk characterization, *not site characterization*. The results of a "Screening" risk characterization should never be used to justify inadequate site characterization.

7.6.2 DEP Risk Assessment ShortForm - Residential Scenario

The *Residential ShortForm* is an optional tool which has been developed by the Department to provide a streamlined method of evaluating potential human health risks at 21e sites. The *ShortForm* streamlines the process by providing a rapid, low cost procedure for assessing health risks. The *ShortForm* is a *LOTUS 1-2-3* (or *Quattro Pro*) spreadsheet incorporating standard assumptions for assessing residential exposures and equations which are used to estimate human health cancer and noncancer risks. The *ShortForm* is intended for use at "residential" sites which are to be evaluated via a Method 3 risk assessment. The output of the *Residential ShortForm* is a series of summary tables which describe the EPCs, toxicity information and potential chemical-specific, medium-specific and cumulative health risks. These output tables can be submitted as the Risk Assessment portion of a Phase II Report.

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