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U.S. DEPARTMENT OF ENERGY PUBLIC AND ONSITE POPULATION HEALTH RISK EVALUATION METHODOLOGY FOR ASSESSING RISKS ASSOCIATED WITH ENVIRONMENTAL RESTORATION AND WASTE MANAGEMENT

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ACRONYMS

ANL	Argonne National Laboratory
ARAR	Applicable or Relevant and Appropriate Requirement (cleanup standard)
ATSDR	Agency for Toxic Substances and Disease Registry
CEDE	Committed Effective Dose Equivalent
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
CM	Conceptual Model
DE	Dose Equivalent
DCF	Dose Conversion Factor
DDT	dichloro-diphenyl-trichloro-ethane
DITTY	Dose Integrated Over Ten Thousand Years
DOE	Department of Energy
DQO	Data Quality Objective
DUST	Disposal Unit Source Term
EDE	Effective Dose Equivalent
EPA	Environmental Protection Agency
ER	Environmental Restoration
GENII	Generation II
HEAST	Health Effects Assessment Summary Tables
HQ	Hazard Quotient
ICRP	International Commission on Radiological Protection
IR	Intake Risk
IRIS	Integrated Risk Information System
ISC2	Industrial Source Complex
ISC2-LT	Industrial Source Complex-Long Term
ISC2-ST	Industrial Source Complex-Short Term
LOAEL	Lowest Observed Adverse Effect Levels
MEPAS	Multimedia Environmental Pollutant Assessment System

MEI	Most Exposed Individual
MRL	Minimum Risk Level
MULTIMED	Multimedia Exposure Assessment Model
NCRP	National Council for Radiological Protection
NEPA	National Environmental Policy Act
NOAA	National Oceanographic and Atmospheric Association
NOAEL	No Observed Adverse Effect Level
NRC	Nuclear Regulatory Commission
PNL	Pacific Northwest Laboratory
PEIS	Programmatic Environmental Impact Statement
PRESTO	Prediction of Radiation Effects from Shallow Trench Operations
RAGS	Risk Assessment Guidance for Superfund
RASCAL	Radiological Assessment System for Consequence Analysis
RCRA	Resource Conservation and Recovery Act
RESRAD	Site-specific RESidual RADioactive Material Guidelines
RfC	Reference Concentration
RfD	Reference Dose
SMCM	Spatial Multimedia Compartmental Model
TSD	Treatment, Storage, and Disposal
USGS	United States Geological Survey
VOC	Volatile Organic Compound
WM	Waste Management

EXECUTIVE SUMMARY

This document describes a public health risk evaluation methodology for assessing risks associated with both Environmental Restoration (ER) and Waste Management (WM). The methodology can be used to estimate risks at Department of Energy (DOE) sites on a site-specific, installation-wide, or programmatic basis. This document forms the basis for the offsite and onsite health risk methodology used to perform the human health risk assessment portion of the DOE Programmatic Environmental Impact Statement (PEIS), although it has applications beyond the PEIS, including installation-wide assessments, screening-level assessments, and more site-specific assessments.

The human health risk evaluation presented here, in accordance with Environmental Protection Agency (EPA) guidance, involves four steps:

- Source term estimation,
- Exposure assessment,
- Toxicity assessment, and
- Risk characterization.

The source term is estimated to determine contaminant releases and release rates. Once representative contaminants of concern are selected, fate and transport modeling can be used to predict present and future contaminant movement through various media from sources to receptors. The fate and transport models discussed in this document are the Multimedia Environmental Pollutant Assessment System (MEPAS) and Generation II (GENII) for ER and WM, respectively. These models can predict risks up to 10,000 yr into the future.

With the use of fate and transport models, the exposure assessment portion of the human health risk evaluation is then conducted to identify possible transport media and exposure routes through which contaminants might reach the public or employees at a site or facility. Intake values or doses are calculated as part of the exposure assessment based on contaminant concentrations, exposure durations, and other necessary parameters. The exposure scenario being evaluated determines the exposure pathways that are assessed. This document provides detailed equations and formulas used to estimate intakes and doses from given exposure routes.

The toxicity assessment evaluates the potential for contaminants of concern to cause adverse health effects in exposed individuals. Appropriate cancer potency factors or unit risk estimates (for chemical carcinogens), risk factors (for radionuclides), or reference doses (RfDs) (for noncarcinogens) are used to assess the toxicity of the contaminants to which people might be exposed. Depending on the exposure

duration, toxicity values can be chronic, subchronic, or acute. When nonroutine events are evaluated, acute toxicity values for chemicals are required.

Finally, the toxicity and exposure assessments are integrated and summarized into qualitative and quantitative expressions of risk in the risk characterization step. Intake or dose estimates are multiplied by toxicity values to estimate chemical or radionuclide cancer risks (for populations and most exposed individuals) or to calculate a noncarcinogenic hazard index for individuals potentially exposed via a given exposure pathway and route in a given 70-yr period. Risks to individuals are expressed in various ways, including average and maximum individual risk. Risks to populations are expressed as the number of cases of cancer incidence, cancer fatalities, and genetic effects in the population, or the number of individuals exposed above a threshold noncarcinogenic effect level.

Once risk estimations are complete, an uncertainty evaluation is performed to document the uncertainties inherent in the risk assessment process, those associated with the chosen models, and those associated with scenarios and parameters used. While it is always preferable to perform a detailed, quantitative uncertainty analysis, in some cases (such as screening-level or programmatic analyses), detailed, quantitative uncertainty analyses are neither feasible nor warranted.

The human health risk evaluation methodology described here is applicable to onsite employees (i.e., general office workers, as opposed to remediation workers directly involved with waste handling), the offsite public, and onsite residents that might live onsite during periods with no institutional controls. This methodology does not include methods to quantitatively evaluate sensitive subpopulations.

The methodology outlined in this document can be applied to evaluate routine operations of ER and WM facilities, baseline risks, and nonroutine occurrences. In addition, this methodology can be used to determine risks posed by sites or facilities *after* remedial actions and waste treatment (i.e., residual risks). Although to calculate residual risks, contaminant concentrations and site-specific conditions may differ, the methodology framework and equations remain the same. The methodology used to evaluate health risks to workers involved in ER and WM activities is documented separately (ORNL 1995a). The specific assumptions and approaches relevant to the PEIS health risk evaluation are also described in a separate document (ORNL 1995b).

1. INTRODUCTION

1.1 BACKGROUND

The goals of Environmental Restoration (ER) and Waste Management (WM) activities are to reduce health risks through site remediation or to contain contamination until new technologies are available to clean up hazardous and/or radiological waste sites. To determine the best way to meet this goal, the U. S. Department of Energy (DOE) must estimate human health risk posed by hazardous and/or radiological waste sites as a result of ongoing and future ER and WM activities. Health risks are estimated through the risk assessment process, which involves the following steps:

- Source term estimation
- Exposure assessment
- Toxicity assessment
- Risk characterization.

The risk assessment methodology described in this document is an integrated ER and WM risk assessment methodology for assessing risks to offsite and onsite populations, combining the Environmental Protection Agency's (EPA) *Risk Assessment Guidance for Superfund (RAGS)* (EPA 1989a) with fate and transport modeling.

1.2 PURPOSE

The purpose of this report is to provide a generic, screening-level methodology for evaluating the health risk to offsite (i.e., members of the public) and onsite populations resulting from DOE hazardous and radiological waste sites and WM activities. In addition, this document describes the risk assessment approach for evaluating risks resulting from the cleanup of waste sites (i.e., risks posed during remediation), and risks remaining after cleanup activities have been completed. The offsite population is defined as the public within a 80-km (50-mi) radius of the location being evaluated. The onsite population is defined as employees who are not directly involved in site remediation activities (e.g., office workers). Onsite populations may include additional receptors (e.g., onsite residents such as a farm family), depending on the exposure scenario being evaluated.

The objectives of this methodology are as follows:

- To establish a general risk assessment methodology that is applicable to ER and WM activities across the DOE complex

- To document the conceptual basis for the approach followed in the EM PEIS for determining offsite and onsite population health risks associated with selected ER and WM alternatives; the specific Programmatic Environmental Impact Statement (PEIS) risk evaluation approach is documented elsewhere (ORNL 1995b).

- To define specific parameter values that are useful for DOE risk assessment applications.

Topics discussed in this document include models and equations used to estimate exposures and numerical parameter values used in those models and equations. Other topics include selecting contaminants of potential concern, determining environmental transport and exposure pathways for quantitative evaluation, quantifying risks corresponding to estimated exposures, and identifying and selecting appropriate human receptors for quantification of exposure scenarios.

The methodology described in this report can be used to assess baseline (i.e., before remediation) public and onsite population health risk as well as residual risks (i.e., risks remaining after the implementation of ER and WM alternatives). In addition, this methodology can be used to assess risks from both normal operations and nonroutine events. The implementation of ER scenarios changes site conditions, requiring site-specific modifications of parameters such as contaminant concentrations and exposure pathways to assess a future (after implementation of alternative), rather than current, condition. Likewise, the occurrence of nonroutine events in WM changes exposure parameters such as exposure duration because of the acute nature of such events.

Conditions other than baseline must be evaluated to decide which ER or WM alternatives will achieve the greatest reduction of baseline risks. Remedial actions at release sites could potentially release contaminants to the environment that would not otherwise be released (e.g., volatilization of contaminants from soils disturbed during excavation). In addition, after remedial actions have been implemented, source and site or facility conditions will have changed, necessitating an evaluation of potential residual risks. Similarly, WM operations such as the treatment, storage, and disposal (TSD) of waste could potentially release contaminants to the environment that would not otherwise be released (e.g., routine vent releases of contaminants during ion-exchange or incineration processes). After treatment technologies have been implemented, the physical form of the waste will have changed, necessitating a risk evaluation of potential remaining risks. For waste operations, risks posed by storage and disposal of waste also need to be evaluated. Once these remediation and WM alternatives have been evaluated, the risk evaluations for each condition can be compared to the baseline risk to determine how much each alternative reduces risk.

The methodology, along with other EPA and DOE guidance documents, is intended to serve as prospective guidance for evaluating health risks for a number of potential applications, including the DOE PEIS. The

methodology is useful for both screening-level and more site-specific applications. For example, a screening-level analysis might include programmatic assessments based on generic assumptions and planning phase assessments for identifying potential concerns before beginning remediation operations. In these types of assessments, exposure data may be limited or unavailable due to the hypothetical nature or "planning phase" nature of the assessments.

For more site-specific analyses, the basic methodology for assessing health risks is also applicable. However, exposure calculations and modeling can be replaced with actual monitoring data, known exposure durations, and known air concentrations.

Two separate documents have been produced (ORNL 1995a; ORNL 1995b) to describe the specific applications of this conceptual methodology for the PEIS; in particular, these documents describes a unit risk approach to assessing human health risks.

Risk estimates resulting from use of this methodology are useful for comparative analyses, but in most cases they should not be construed to represent actual, site-specific risks unless known, site-specific data are used. The methodology described here is often based on conservative assumptions and hypothetical scenarios that may not represent actual site-specific conditions. When actual data are unavailable, this type of approach is feasible. Moreover, this approach can be useful for providing insight into the relative contribution of certain activities to exposures and risks. The use of site-specific parameters, when such values are available, is preferred and is useful in providing less conservative risk estimates.

This methodology is primarily addressed to individuals conducting offsite and onsite population risk evaluations for sites within the DOE complex. This document is written as general risk assessment guidance to apply to various screening-level risk assessments and to be used by individuals with varying levels of risk assessment experience, ranging from experienced risk assessors to novices. Although this document offers risk assessment guidance, no single methodology can address all site circumstances. Therefore, the risk assessors play key roles in the process because their professional judgement must be exercised in some instances.

1.3 ORGANIZATION

This document follows the outline suggested in *RAGS* (EPA 1989a). Major topics are presented in the following order:

- **Chapter 1. Introduction.** Chapter 1 includes the background, objectives, and purpose of the public and onsite population health risk evaluation. This chapter also outlines the important topics presented in the document, including models, equations, parameters, exposure pathways, and exposure scenarios.

- **Chapter 2. Site Characterization.** Chapter 2 discusses the importance of becoming familiar with a site to determine characteristics that could enhance or retard the migration of contaminants through the environment.

- **Chapter 3. Data Collection and Analysis.** Chapter 3 describes the importance of site or facility characterization data, source term data, fate and transport modeling data, exposure assessment data, and toxicity data that adhere to data quality objectives (DQOs). Chapter 3 also discusses DQOs and the process of selecting contaminants of concern and the criteria for selection.

- **Chapter 4. Source Term Estimation.** This chapter describes the steps in source term estimation, including analyzing contaminant releases and release rates. This chapter also discusses identifying potential release mechanisms.

- **Chapter 5. Fate and Transport Modeling.** Chapter 5 describes estimation of exposure point concentrations using fate and transport models. This chapter also discusses identifying and characterizing exposed populations and potential exposure routes and pathways. Chapter 5 also discusses possible fate and transport models and modeling parameters used to evaluate risks associated with ER and WM activities and the criteria used for model selection.

- **Chapter 6. Exposure Assessment.** Chapter 6 provides an overview of the quantification of exposure. This chapter discusses estimating contaminant intakes for various exposure pathways.

- **Chapter 7. Ingestion Exposure Route.** Chapter 7 describes formulations used to estimate exposure via ingestion of contaminants, addressing ingestion of contaminated water and food chain products and inadvertent ingestion of soil.

- **Chapter 8. Inhalation Exposure Route.** Chapter 8 describes formulations used to estimate exposure via inhalation of contaminants, addressing inhalation of contaminants found in the atmosphere and generated by showering.

- **Chapter 9. Dermal Contact Exposure Route.** Chapter 9 describes formulations used to estimate exposure via dermal contact, addressing dermal contact with contaminated soil and water.

- **Chapter 10. Exposure to Radiation.** Chapter 10 describes estimation of external and internal exposure to radionuclides.

- **Chapter 11. Exposure to Tritium.** Chapter 11 describes formulations for estimating exposure to tritium, a special case radionuclide.

- **Chapter 12. Toxicity Assessment.** Chapter 12 describes components of the toxicity assessment such as toxicity values, addressing chemical carcinogens, noncarcinogens, and radionuclides.

- **Chapter 13. Risk Characterization.** Chapter 13 describes components of the risk characterization process, delineating quantification of risk from carcinogens, noncarcinogens, and multiple substances. This chapter describes types of risk that can be estimated, such as population risk and maximum individual risk. Chapter 13 also discusses uncertainties in risk assessment.

- **Appendix A: Exposure Factors**. Appendix A provides EPA exposure factor values for various exposure scenarios.

1.4 Risk Assessment Approach

The goal of the offsite and onsite population health risk evaluation process is to provide a framework for developing the risk information necessary to assist decision-making at ER and WM sites. The risk assessment process provides the following information:

- A consistent process for evaluating and documenting potential off-site and on-site health threats at DOE sites

- An analysis of baseline risks and the need for action at DOE sites and installations

- A basis for comparing potential health impacts of various remedial or TSD alternatives

- A basis for the risk management process of determining levels of contaminants that can remain on-site after remediation activities

- A basis for determining the risks of various waste management activities.

EPA has adopted, in its Risk Characterization Policy, a stance that all statements about health risks must:

- Indicate how the risks vary across population groups, exposure scenarios, and other important categories,

- Indicate the degree of uncertainty in the assessed risks, together with the sources of those uncertainties, and

- Identify to what extent any aspects of the assessed risks derive from policy or doctrine, as separate from scientific and data considerations.

2. SITE CHARACTERIZATION

To analyze the potential threat posed by contamination at a site, the risk assessor must first become familiar with the site, its setting, and its history. Site visits, regulatory reports, and personal contacts provide such necessary site information. Site characterization provides descriptions of the site's physical setting, demographic surroundings, and environmental conditions as well as clues about the nature and extent of contamination at the site.

2.1 BACKGROUND INFORMATION

Background information provides an idea of the magnitude and extent of contamination at a site and possible health concerns related to site operations. Site descriptions provide background information about the geographic location of the site. The location of the site within the community indicates the size of the population potentially affected by site contamination. Visual representations of the site (e.g., site plans, topographic maps, and aerial photographs) provide geographic information as well as indications of site operations, the extent of surface contamination, underground conduits for potential contaminant transport, and land uses near the site.

A history of the site provides an idea of the contaminants that may be present, the extent of contamination, the rate of migration, and the magnitude of potential human exposure. The following general information on the site operations and history is collected to characterize the broad status of the site:

- The types of activities performed at the site indicate potential contaminants of concern at the site (e.g., past WM practices).
- The duration of site activities is likely to influence the extent of contamination and contaminant migration.
- The length of time that contamination has been present at the site indicates the extent of contaminant migration and potential receptors.
- Any changes in site size or development at the site may drastically affect rates and patterns of contaminant migration.

Past community health concerns related to the site are also important to site characterization. Perceived health problems and past or current exposure may be documented in environmental and health complaints

by the public or community health studies. Physical hazards (e.g., stacked drums, unsafe structures) at a site may constitute a public health concern. Populations for which these health concerns exist must be identified through demographic information as part of the site characterization.

2.2 Demographic Information

As part of the site characterization, the populations that are most likely to be exposed to contaminants must be determined. Population characteristics include the distance from the site to the closest residence, the size of the population within a specific radius of the site, size of the onsite population, and the presence of populations at higher risk from exposure (i.e., sensitive subpopulations such as children). For the PEIS, when information on sensitive subpopulations is available, the subpopulations are treated qualitatively. Areas of high risk receptors (e.g., nursing homes, hospitals, schools, day care centers) are identified, but risks are not estimated for these subpopulations because estimation of risks to subpopulations is considered beyond the scope of a screening-level analysis. Demographic information can be provided by census information, local governmental sources, and health agencies.

Demographic and behavioral information should also be characterized to evaluate the potential exposure of humans to contaminants at a release site or TSD facility. Numbers of nearby residents or employees near the release site or treatment facility should be considered in the exposure assessment.

2.3 Natural Resource Information

Use of natural resources such as lakes, land, or forests is also important information used to characterize a site. For example, the existence of a nearby lake that is used for boating, fishing, and swimming will potentially affect the migration of contaminated groundwater to potential exposure points. Land use at or near a site provides valuable information on the activities of potentially exposed populations. Land use determines the intensity of contact with contaminated media.

The risk assessor should consider past, present, and future land use to determine site accessibility. The residential, recreational, and industrial activities of potentially exposed populations must be obtained to characterize a site. For example, residential areas at or near the site should be identified because they may include gardens, livestock, and private wells that increase exposures. Recreational areas such as parks, playgrounds, lakes, and beaches introduce additional potential exposure routes (e.g., ingestion of contaminated fish) that may also increase exposures. Depending on land use, activity types and levels can determine exposure frequency and duration for a population.

2.4 ENVIRONMENTAL INFORMATION

Site characterization also requires environmental contamination and migration pathway information. The minimum environmental information required to characterize a site for risk assessment is the contaminants identified in specific environmental media. Estimation of the contaminant concentrations in each environmental medium are necessary in determining the magnitude of potential exposure. This knowledge is obtained as part of the source term, as discussed in chapter 4.

In addition to environmental contamination information, the setting in which potential exposures occur must be evaluated. The physical characteristics of a site or TSD facility are important because they provide the information necessary for evaluating potential release and migration of contaminants into the environment. Information necessary for release site or facility characterization and subsequent evaluation of exposure pathways may include the following:

- Climatology and meteorology
- Topography
- Regional, local, and release site- or facility-specific geology
- Surface hydrology
- Hydrogeology
- Soil characteristics
- Regional, local, and release site- or facility-specific ecology (EPA 1989a).

This information and sources of such information are discussed in chapter 3. Portions of site characterization overlap with other steps in the risk assessment such as data collection, source term estimation, and exposure assessment. The characterization parameters used to quantify exposure can generally only be approximations, and inherent uncertainty is associated with each.

3. DATA COLLECTION AND ANALYSIS

Data collection and analysis involves gathering and evaluating data relevant to the public and onsite population health risk evaluation. The completion of the data collection process does not necessarily indicate that data collection efforts are complete. Data should be continually updated as significant improvements and new data become available. These data must also adhere to DQOs, statements that specify the quality of data required to support decisions about ER and WM activities. This chapter describes the data requirements and DQOs for conducting a health risk assessment.

3.1 RISK ASSESSMENT DATA

The categories of data required for risk assessment include the following:

- Site characterization data
- Source term data
- Fate and transport modeling data
- Exposure estimation data
- Toxicity data.

These data categories and their uses are described in the following subsections. As these descriptions indicate, the data requirements for each category may overlap.

3.1.1 Site Characterization Data

Site-specific data are always preferable over generic assumptions when conducting a risk assessment. In the absence of site-specific data, however, default exposure values and modeling parameters can be used. When neither site-specific nor standard values are available, reasonable assumptions about these values can be made based on EPA guidance and/or professional judgement.

Site characterization data enable the risk assessor to become more familiar with a site to analyze the potential threat posed by contamination at a site. These data are collected through site visits, site reports, and regulatory reports. Accurate and complete site-specific data enable greater accuracy of the final risk estimates. Site characterization data describe the site, its physical and demographic surroundings, meteorological and environmental conditions that affect the extent of contamination at the site. Environmental and population data are used to characterize sites or facilities and the location of current populations relative to the site or facility.

Environmental data provide information on climate, meteorology, geologic setting, vegetation, soil type, groundwater hydrology, and the location and description of surface water. The following environmental data are examples of data needed to characterize a site:

- **Topography**. Topographic information is required to characterize soil runoff pathways. The steepness of slopes and elevation of the site may affect the direction and rate of water runoff, rate of soil erosion, and the potential for flooding.

- **Annual precipitation rates**. Precipitation rates at a site are useful in determining the amount of surface water runoff, groundwater recharge, and soil moisture content. High precipitation rates coupled with highly water soluble contaminants of concern indicate a high potential for contaminant migration. A site's precipitation rate indicates the speed that particulates and soluble vapors are taken from the atmosphere.

- **Temperature**. The temperature at a site affects the volatilization rates of contaminants. For example, frozen ground can retard contaminant movement. Temperature is also important because it determines the frequency of outdoor activities at a site.

- **Wind speed, direction, and stability class**. The wind's speed and direction influence generation rates of dust. For WM the stability class affects the concentrations in the plume.

- **Seasonal and diurnal conditions**. A site's seasonal and diurnal conditions affect rates of contaminant migration where precipitation rates or temperatures vary by season or time of day.

- **Geomorphological characteristics**. The geomorphological characteristics of a site play a significant role in determining stream flow velocity, volume and speed of runoff, erosion, and soil characteristics.

- **Hydrogeologic characteristics**. The hydrogeological composition and structure of a site affects the direction and extent of contaminant transport in water. The types and locations of aquifers (i.e., water table or confined aquifers) and hydraulic conductivity are important in determining the threat posed by the site to drinking water supplies.

- **Surface water channels**. The location, width, and depth of surface water channels and associated floodplains near the site may affect the extent of contaminant migration. The use of these water bodies affects the migration of contaminants off site and into other environmental media.

- **Soil characteristics**. Soil characteristics such as soil configuration, composition, organic content, bulk density, porosity, and permeability influence rates of percolation, groundwater recharge, contaminant release, and transport.

- **Ground cover.** A site's ground cover and vegetative characteristics influence soil erosion, percolation, and evaporation rates.

- **Presence of plants and animals.** Plants and animals at or near a site that could be used as sources of food.

Sources of physical setting data include site or facility descriptions, preliminary assessments, site inspections, and remedial investigation reports. Other sources include county soil surveys, wetlands maps, aerial photographs, and reports by the National Oceanographic and Atmospheric Association (NOAA) and the U.S. Geological Survey (USGS). In addition, appropriate technical experts (e.g., hydrogeologists, air modelers) should be consulted as needed.

3.1.2 Source Term Data

Source term data characterize the rate of contaminant release from a source at a site or facility. Site reports and documents should provide data on contaminant sources and contaminant concentrations in environmental media. Sufficient information should be provided to assess current and potential contamination of surface water, groundwater, soil, air, plants, and animal to which receptors might be exposed. The following physical and chemical characteristics of a site are considered when determining a source term:

- Disposal operations at the site (e.g., injection, burial of contaminants)
- Time period of disposal operations
- Chemical and radionuclide inventory
- Contaminant concentrations at or near source unit
- Distribution of contaminant in environmental media
- Emission rates for TSD facilities.

A key element of the human health risk evaluation is identification of contaminants that may be hazardous to human health. Selection of contaminants of potential concern requires a close examination of contaminant concentrations at a site or facility, the physical and chemical properties of the contaminants, the quality of environmental sampling data, and the potential human exposure to contaminants. At large sites such as DOE facilities, an important consideration is attribution of contamination to the particular source(s) being evaluated. Other sources may be contributing to the contamination at a site for which the risk assessment is being performed.

Site reports and documents should identify contaminants found in environmental media. Such reports also provide information about sampled media, sampling techniques, detection limits, and detected concentrations. Before using these environmental data to reach conclusions in the risk assessment,

however, the data should be checked for completeness. In addition, sampling and analytical techniques used to obtain the data should be examined for accuracy to ensure data quality.

All contaminants detected at a site or facility should initially be considered potential contaminants of concern. For certain sites and facilities, the list of contaminants may be lengthy. Including a large number of contaminants in the risk assessment may be complex, as well as time- and resource-consuming. In most cases, relatively few contaminants will drive the overall risk posed by a site. Thus, the final contaminant data set should consist of the contaminants that represent the largest portion of the risk to human health.

Several considerations must be made when separating the contaminants that represent the greatest risk from those contaminants that can be reasonably eliminated from the human health risk evaluation (EPA 1989a). To determine which contaminants can be excluded from the evaluation, the following initial considerations should be made:

- **Historical information.** Contaminants that have been reliably associated with site or facility activities based on historical records generally should not be eliminated from the risk evaluation, even if the other criteria included in this section suggest that such an elimination is feasible. Historical information from other, similar sites may also be used.

- **Concentration and toxicity.** Each contaminant's concentration level should be compared with background concentration levels. Contaminants exceeding reported maximum background concentrations should be included in the risk evaluation as contaminants of concern. For remedial alternatives, it is important to consider that a significant risk could be posed by non-elevated background contaminant concentration levels for certain alternatives (e.g., naturally elevated soil levels of arsenic for an alternative involving excavation activity) but not for others (e.g., same area under remedial alternative involving a non-intrusive activity). When evaluating remedial alternatives, therefore, selection of contaminants cannot always be based solely on elevated background levels of contaminants.

Site and facility reports and documents contain concentration data that provide a preliminary indication of the contaminants detected at the highest concentrations and the greatest frequencies. Before eliminating potentially carcinogenic contaminants, however, the EPA weight-of-evidence classification (an indication of the quality and quantity of data underlying a chemical's designation as a potential human carcinogen) should be considered along with the contaminant concentrations detected at a site or facility. For example, it may be practical and conservative to retain a chemical that is present in low concentrations if that chemical is a Group A carcinogen, which is a human carcinogen (see section 12.2).

- **Mobility, persistence, and bioaccumulation.** A contaminant's mobility, persistence, and bioaccumulation potential provide another basis for its selection as a contaminant of concern. For example, a highly volatile (i.e., mobile) contaminant such as benzene, a long-lived (i.e., persistent)

contaminant such as dioxin, or a readily taken-up and concentrated (i.e., bioaccumulated) contaminant such as DDT, should remain in the risk evaluation.

- **Special exposure routes.** Certain exposure routes must be considered carefully. For example, highly volatile contaminants may pose a significant inhalation risk due to the home use of contaminated water, particularly for showering. Conversely vaporization potentials (e.g., metals) that are present in high concentrations could pose a risk due to the volume of contaminant available. Such contaminants should be listed as contaminants of concern.

- **Treatability.** Some contaminants are more difficult to treat than others. Due to their importance during the selection of remedial alternatives, those contaminants that are difficult to treat should be included as contaminants of concern.

- **Applicable or Relevant and Appropriate Requirements (ARARs).** The term ARAR covers two sets of cleanup requirements: (1) applicable requirements (applicable by law) and (2) relevant and appropriate requirements (not legally applicable). ARARs can be contaminant-specific, location-specific, or action-specific. Contaminants with ARARs usually should not be excluded from the risk evaluation. This may, however, depend in part on how the contaminant's concentrations in specific media compare with its ARAR concentrations for those media (EPA 1989a).

After these initial considerations have been addressed, a more quantitative evaluation of the potential contaminants of concern is made, as described in EPA's *RAGS* (EPA 1989a).

At most DOE sites, the prevalent types of contaminants include radionuclides, carcinogenic chemicals, and noncarcinogenic chemicals. These contaminants fall into groups such as inorganic chemicals, volatile organic compounds (VOCs), semi-volatiles, and radionuclides. These types of contaminants react differently in environmental or structural media depending on temperature, climate, soil types, wind speed, precipitation, and presence of moisture. Chemicals have different affinities for binding to water, lipids, different solubilities, and different diffusivities that determine the general behavior of contaminants in environmental media. The chemicals and radionuclides selected as contaminants of concern should be analyzed for their potential to move through the environment and reach potential receptors.

3.1.3 Fate and Transport Modeling Data

Fate and transport modeling data support the development and implementation of models that are used in risk assessment to predict the migration of contaminants from the site or facility through environmental media, as discussed in section 5.1. The types of data required for fate and transport modeling include information on the geology, hydrogeology, surface hydrology, and meteorology of the site and vicinity. For example, hydrogeologic information such as unsaturated and saturated zone materials and groundwater

flow rate is required to support fate and transport modeling for predicting the extent of contamination at each site or facility. Similarly, information on surface water hydrology is used by models to characterize potential impacts to surface water. Specific fate and transport modeling parameters based on these data are discussed in section 5.2.

3.1.4 Exposure Assessment Data

Exposure assessment data are used to estimate intakes of chemicals and radionuclides and direct radiation exposures. Data used to estimate exposures include the results of fate and transport modeling and values for parameters that quantitatively describe exposure scenarios. These parameters include the following:

- Ingestion (or consumption) rate
- Inhalation rate
- Event time
- Exposure frequency
- Exposure duration
- Biotransfer factors
- Averaging time
- Absorption factors
- Body weight.

Exposure duration is the time period over which an exposure event occurs. EPA recommends an exposure duration of 30 yr (a 90% upper bound exposure duration) to represent the time spent by an individual at one residence (EPA 1989a); 9 yr is the national median time at one residence. The measurement (i.e., hr/event) of a single contact event is the event time. Exposure frequency tells how often (i.e., events/yr) the exposure occurs. The averaging time selected depends on the type of toxic effect being assessed (e.g., 30 yr for noncarcinogens and 70 yr for carcinogens). The value for body weight is the average body weight over the exposure period. If exposure occurs only during childhood, the average child body weight during the exposure period should be used to estimate intake. For example, exposure from some pathways (such as soil ingestion) can occur throughout the lifetime, but the majority of exposure occurs during childhood due to higher contact rates. In these cases, exposures should be calculated by age groups based on the body weight of each age group (e.g., 15 kg [33 lbs] for age 1-6, 70 kg [174 lbs] for ages 7-31). Body weight is not always independent of other variables in exposure calculation (e.g., intake); therefore, a constant body weight over the period of exposure is used. Default values for such exposure factors as exposure duration, averaging time, ingestion and inhalation rates, and body weight are listed in appendix A.

3.1.5 Toxicity Data

Toxicity data are used to quantify human health risks from exposure to chemicals and radionuclides. Toxicity data used for the human health risk evaluation can be obtained from EPA sources such as the Integrated Risk Information System (IRIS), EPA's on-line toxicity value database (EPA 1988; 1991a). IRIS is the preferred source for numerical toxicity values and information. This system provides chemical-specific cancer potency factors, RfDs, reference concentrations (RfC), supporting discussion, and references. Health Effects Assessment Summary Tables (HEAST) is a tabular presentation of toxicity information and values including interim RfDs, cancer potency factors, and other toxicity information. HEAST is the secondary source to be used for toxicity values after IRIS has been consulted.

3.2 Data Quality Objectives

DQOs are qualitative and quantitative statements that specify the quality of data required to support decisions about remedial activities. Data of known or acceptable precision, accuracy, completeness, representativeness, and comparability are required for risk assessment. Indeed, the development of DQOs is an important step in assuring quality data for site or facility characterization, fate and transport modeling, and exposure estimation. These statements are established before data collection during the project scoping and sampling and analysis planning phases.

To define DQOs, the problem to be resolved at the site or facility must be defined. Each site and facility has a unique history; therefore, a unique set of DQOs is developed for each site and facility. DQOs also vary according to the intended uses of the collected data. For instance, data may be collected to support risk assessment decisions. Hence, the DQOs serve as the full set of constraints necessary to specify a level of acceptable uncertainty during risk assessment decisions.

EPA guidance provides a three-stage process for developing DQOs (EPA 1989b). These interactive stages include the following:

Stage 1: Identification of decision types. The first stage of the DQO process defines the types of decisions that will be made by identifying data users, evaluating available data, developing a conceptual model, and specifying objectives for the project. During this stage of DQO development, all available site or facility information is compiled and analyzed to develop a conceptual model that describes suspected sources, contaminant pathways, and potential receptors. This stage defines objectives and identifies data gaps.

Stage 2: Identification of data uses/needs. This stage stipulates criteria for determining data adequacy. Risk assessments, for example, use data to evaluate the threat posed by a site or facility

to human health and the environment. Thus, risk assessment data needs include site- or facility-specific contaminants, environmental transport, and exposure data. These data are generated through the sampling and analysis of environmental and biological media, particularly where human exposure is likely. Risk assessment data must represent as closely as possible the conditions at a site or facility, allow accurate estimation of risk, and meet regulatory requirements for risk assessment data.

Stage 3: Design of data collection program. In determining DQOs, the methods by which data of acceptable quality and quantity are obtained to make decisions must be defined. This information is summarized in documents such as work plans or sampling and analysis plans.

The use of DQOs ensures that data collected to conduct a risk assessment are of known and documented quality. For risk assessments conducted on a programmatic basis rather than a site-specific basis, the data collection process involves using the best available data rather than sampling data. First, data extracted from Resource Conservation and Recovery Act/Comprehensive Environmental Response, Compensation, and Liability Act (RCRA/CERCLA) action-confirmed analytical documents that have met rigorous DQOs should be used. In cases where no such data exist, unconfirmed or projected data are used and identified as such in accordance with the National Environmental Policy Act (NEPA) implementing regulations. The quality of unconfirmed data will vary between DOE installations depending on the maturity of that installation's ER and WM programs. However, all data used in DOE ER and WM evaluations should be reviewed for concurrence by appropriate staff at each DOE Operations Office.

4. SOURCE TERM ESTIMATION

The source term describes the amount of material available for release from a source (i.e., contaminant inventory) and the rate of contaminant release. The source term represents situations of existing contamination or potential environmental risk and is, therefore, an important initial step in the risk assessment. To determine possible release sources at a site, all available site descriptions and contaminant-specific data from site investigations and reports should be examined. The contaminant release may be at the site or facility or in the surrounding environment.

Source term data are used to determine the quantity of contaminant released from a source at a site. A description of the source term is determined from actual site data and includes data necessary to conduct remedial activities and to support the risk evaluation. The physical and chemical characteristics of a site are considered when determining a source term for ER. For WM, rate of contaminant release is considered for the waste inventory of a site and the TSD technologies applied to that inventory.

Site reports should also provide data on contaminant inventories, contaminant concentrations detected in the environmental media, and contaminant emission rates for TSD facilities. After the contaminants of concern have been identified, the contaminant concentrations in each environmental medium are needed to determine the magnitude of exposure. Sufficient information should be provided to assess potential release mechanisms and receiving media for past, current, and future releases that could result in the contamination of surface water, groundwater, soil, plants, and animals to which receptors may be exposed.

Potential release mechanisms that lead to contaminant migration through the environment involve the transport of gases, liquids, and particulate solids into a given medium and across the interfaces between air, water, and soil. The assessor should determine whether contaminants are released from a source and transported via surface water runoff, leaching, volatilization, airborne suspension/resuspension or biological uptake. Typical release mechanisms and receiving media for various release sources are listed in table 4.1-1. Release rates may be determined by using computer codes such as MEPAS, which is used in the PEIS and is described in section 5.1.1.1.

Source term information provides a basis for determining the extent of environmental release. After determining that a source of contamination exists, the assessor must determine whether the source can be contained, whether the contamination can be removed or disposed of, and whether the source can be treated. If the source is or has the potential for releasing contamination into the environment, fate and transport modeling may then be used to simulate and predict contaminant movement from the source through the environment to receptors.

For each of the relevant release sources in table 4.1-1, the aim is to develop and present estimates of the characteristic expected release rates of the contaminants of interest. This will include both some long-term average release rates (e.g., averaged over one year or some longer time period) and an indication of the variability of those rates — from year to year, across seasons during a year, and possibly for shorter time periods, as appropriate. If episodic releases are possible and potentially important for estimating acute risks or risks to some particularly vulnerable populations, then some estimate of the magnitude and frequency of such episodes should be presented. This estimate may come from historical data and/or from estimates made using probabilistic risk assessments or by more informal means. In addition to these indications of variability, the release information should be accompanied by some quantitative or qualitative estimate of the uncertainties. This will include both some estimate of the sampling error and measurement error associated with the data collection activities used to generate the data that underlie the release estimates.

Table 4.1-1. Potential Release Sources, Release Mechanisms, and Receiving Media

Receiving Medium	Release Mechanism	Release Source
Air	Volatilization	Surface wastes (lagoons, ponds, pits, spills) Contaminated surface water Contaminated surface soil Contaminated wetlands Leaking drums Contaminated groundwater used for irrigation and washing Emissions for treatment/storage facilities
	Fugitive dust generation	Contaminated surface soil Waste piles
Surface water	Surface runoff	Contaminated surface soil
	Episodic overland flow	Lagoon overflow, spills, leaking containers
Groundwater	Leaching	Surface or buried wastes Contaminated soil
Soil	Leaching	Surface or buried wastes
	Surface runoff	Contaminated surface soil
	Episodic overland flow	Lagoon overflow, spills, leaking containers
	Fugitive dust generation/deposition	Contaminated surface soil Waste piles
Sediment	Surface runoff, episodic overland flow	Surface wastes (lagoons, ponds, pits, spills) Contaminated surface soil
	Groundwater seepage	Contaminated groundwater
	Leaching	Surface or buried wastes Contaminated soil
Biota	Uptake (direct contact, ingestion, inhalation)	Contaminated soil, surface water, sediment, groundwater, or air Other biota

Source: EPA 1989a.

5. FATE AND TRANSPORT MODELING

Health risk assessments can be performed with the aid of computer codes, which are widely used to predict contaminant movement through various media from sources to receptors. Fate and transport modeling has become an integral part of exposure assessment. Fate and transport models are generally used in conjunction with monitoring data and release estimates (i.e., source term) to predict contaminant movement from sources to receptors and estimate exposure point concentrations for quantifying exposure (i.e., intakes) for various exposure pathways. Potential exposure pathways are discussed as part of the exposure assessment in section 6.1.

Models are used to simulate potential exposures under present and future conditions and to simulate potential contaminant transport through environmental media based on assumptions made from present conditions. For example, if contaminants are found at a well, it can be assumed that the contamination may also travel to a nearby well in the future. A groundwater transport model can then be used to estimate exposure for this exposure pathway. Fate and transport models can also estimate contaminant concentrations to predict future exposures for which measured data are not available. In addition, nonroutine events (e.g., fires, tornadoes) can be simulated by fate and transport models.

5.1 MODEL SELECTION AND EVALUATION

Models are selected based on their appropriateness for specific applications in the risk assessment and on the availability of required input information. The modeling process requires input parameters and default values that are consistent with EPA recommendations. Data required for fate and transport modeling include (but are not limited to) information on the geology, hydrogeology, surface hydrology, and meteorology of the site and vicinity. Such data are also required at other stages of the risk assessment such as site characterization and exposure assessment to provide chemical and physical information. Other important information necessary for modeling exposures and information that is necessary for performing exposure assessment in general includes exposure pathways, location of the receptors (and locations of drinking water wells), duration of exposure, and location of exposure points.

Various models are available for use in risk assessment, including groundwater models, short-term and long-term exposure models, atmospheric models, and multimedia models. Typically, models are designed to solve specific problems such as groundwater contamination, onsite exposure, and offsite population exposure due to atmospheric transport and dispersion. However, multimedia models sometimes can perform a combination of these tasks. For specific exposure pathways, models should be investigated to determine the amount of data they require. For example, groundwater models may require groundwater-

specific input such as the volume of the aquifer, mode of irrigation used to withdraw water from a contaminated well, and the volume of water that a receptor population could use.

Human health risks from ER and WM activities can be evaluated with the aid of computer codes designed to simulate environmental transport of contaminants, shielding from radiation, and/or nonroutine atmospheric releases. Computer codes that may be used in the worker health risk evaluation are described elsewhere (ORNL 1995a). Computer codes that may be used for offsite and onsite population risk evaluations are summarized in the following sections.

Before computer codes are selected for use in risk assessment, a model survey should be conducted to determine the most appropriate models available for the evaluation. The models surveyed in this chapter were selected for the evaluation of risks resulting from ER and WM. However, risk assessors are not limited to these models if other models are better suited for their assessments.

5.1.1 Environmental Restoration Models

DOE has a wide variety of waste sites that release contaminants to environmental media; therefore, multimedia models are preferred for ER evaluations since the integration of models that address only individual pathways of contaminant transport is extremely complex and time-consuming. Ideally, multimedia models offer a consistent means for describing risks across many sites. Model selection should be based on the following criteria:

- Ability to consider multiple exposure pathways
- Capability of assessing risk from organic, metal, and radionuclide contaminants and their progeny
- Conformity to EPA and Nuclear Regulatory Commission (NRC) guidelines
- Availability of necessary modeling parameters
- Capability of computing environmental transport over both short and long time periods
- Availability of relatively short computational time requirements
- Degree of conservatism as compared to other models that may be used simultaneously
- Model reliability and performance quality (i.e., quality of output).

The following multimedia models were surveyed for this methodology:

- Multimedia Environmental Pollutant Assessment System (MEPAS)
- Site-specific RESidual RADioactive material guidelines (RESRAD)
- Prediction of Radiation Effects from Shallow Trench Operations (PRESTO)
- Generation II (GENII)
- RiskPro
- Multimedia exposure assessment model (MULTIMED)
- PATHRAE

- GEOTOX
- Spatial Multimedia Compartmental Model (SMCM).

MEPAS (Droppo et al. 1989) simulates releases and transport of contaminants from each source category to potential receptors. MEPAS was originally developed by Pacific Northwest Laboratory (PNL) to provide a management tool for assisting in the prioritization of funding and human resource allocation to DOE's Office of Environment, Safety, and Health. The prioritization exercise was used for further investigations and possible remediation at DOE's inactive waste sites that may produce long-term releases of contaminants to the environment. MEPAS uses mathematical algorithms to predict the potential for contaminant migration from a site to potential receptors using pathway analyses and calculates risk based on different exposure routes and durations. MEPAS considers approximately 20 contaminant transport scenarios and 19 exposure scenarios for organics, metals, radionuclides, and radioactive progeny. MEPAS also includes a comprehensive database containing model parameters for almost 400 contaminants. These parameters are used by the transport and exposure assessment components of MEPAS and reflect up-to-date revisions as suggested by both EPA and NRC.

RESRAD (DOE) addresses potential exposure via multiple pathways from buried radioactive waste or contaminated soil. This model is useful for computing dose to a critical population from onsite exposures. Specific exposure routes included in RESRAD are direct radiation, inhalation of contaminated soil, and ingestion of contaminated agricultural products. RESRAD relies on the use of predicted relationships between contaminant concentrations and the media involved in the exposure pathways.

PRESTO (EPA) addresses onsite exposures to radiation from shallow trenches. Like RESRAD, PRESTO does not use HEAST, making this model useful only for dose assessments. The hydrologic model includes precipitation, infiltration, leaching of waste, and migration of contaminants through the vadose zone into the groundwater. This model uses both physically based and empirical equations to compute flow and transport. In addition, PRESTO computes exposures resulting from inhalation, ingestion, and dermal contact and simulates transport of contaminants over long time periods (1,000 yr).

GENII (DOE) predicts radiation exposure through pathways that include surface water, soil, and air. Exposure routes include inhalation and ingestion of drinking water and aquatic food products.

RiskPro (General Sciences Corporation) addresses environmental transport of contaminants via groundwater, surface water, air, and vadose zone. The groundwater component of RiskPro allows for three-dimensional computation of transport. For surface water, a compartmental model that incorporates loss terms, adsorption to sediments, and erosion is used. For air transport, a Gaussian plume model is used that incorporates up to 49 combinations of conditions. The program uses a validated vadose zone transport model. In addition, RiskPro includes a chemical database with 20,000 chemicals, 1,000 of which have known parameter values.

MULTIMED (EPA) is a screening-level model that simulates contaminant transport from a waste disposal site into the environment. Modules that comprise the model include the following:

- Landfill
- Vadose zone flow and transport
- Aquifer flow and transport
- Surface water
- Air emissions
- Air dispersion.

In addition, MULTIMED has the ability to perform uncertainty analyses using a Monte Carlo approach.

PATHRAE (DOE) is a multiple pathway model used for assessing dose from onsite exposures to radionuclides.

GEOTOX (U.S. Army) was developed by Lawrence Livermore Laboratories for the U.S. Army's Biomedical Research and Development Laboratory. This model uses a compartmental approach to calculate uniform yet time-dependent concentrations for each compartment. Compartments considered include air (gas and particulate), soil (upper and lower), biota, groundwater, surface water, and sediments.

SMCM (UCLA, EPA) is a useful screening model for assessing the transport and fate of VOCs through multiple environmental media. This compartmental model estimates concentrations in soil, air, and water but does not consider metals or radionuclides. In this model, compartments comprising air, water, biota, and suspended particulates are assumed to be homogeneous; compartments representing soil and sediment are assumed to be nonhomogeneous (i.e., spatially varying). Contaminants are produced or degraded and transferred from one compartment to another. Contaminant movement into the atmosphere is considered to occur via diffusion.

5.1.2 Waste Management Models

For WM, a model survey was also conducted to determine which models were best suited for risk assessment of WM activities. In addition to the models surveyed for ER, the following models were also surveyed for WM:

- Radiological Assessment System for Consequence Analysis (RASCAL)
- Industrial Source Complex Short-Term (ISC2-ST)
- Industrial Source Complex Long-Term (ISC2-LT)
- CHEM-PLUS
- Disposal Unit Source Term (DUST)

- Dose Integrated Over Ten Thousand Years (DITTY)
- Prediction of Radiation Effects from Shallow Trench Operations (Presto).

GENII evaluates radionuclide exposures via routine contaminant releases during the treatment, storage, and disposal of waste. The computer code GENII is used to aid in the evaluation of exposures to contaminants since it is able to calculate doses from acute releases. GENII models dispersion for many transport media and exposure pathways, although the main transport medium for nonroutine events is atmospheric. GENII also estimates doses to individuals and populations. GENII is ideal for modeling exposures from nonroutine WM events since it is able to estimate both acute and chronic exposure. DITTY is the dose assessment code used by GENII.

RASCAL is a model developed to predict the dose received by individuals after a nuclear accident at a nuclear power plant. Given some basic user-entered data, the model predicts acute bone, thyroid, lung, and effective dose equivalent (EDE) received by the maximally exposed individual at various distances from the source up to 40 km (25 miles). This model was developed for the use of the NRC Inspectors who are evaluating the environment after a power plant accident. RASCAL considers radionuclides and models acute scenarios with both a straight-line plume and a puff model.

CHEM-PLUS is a hazard model for toxic vapor, fire, and explosion events. It is used to evaluate the consequence of a hazardous chemical discharge. The model includes heavy gas dispersion, two-phase flow and expansion, and improved flame jet and pool fire models. CHEM-PLUS estimates release rates and duration of discharge and liquid evaporation if actual data are unavailable and includes a database of over 300 chemicals.

ISC2 is an EPA air dispersion model that evaluates both short-term and long-term exposures to radionuclides and chemical contaminants in the air.

DUST is a model developed by Brookhaven National Laboratory that calculates source terms or flux of contaminants from engineered facilities. DITTY, the dose assessment code used by GENII, is used to calculate exposure.

PRESTO is a model that can evaluate the transport of contaminants in groundwater, surface water and air. It was used to calculate the cancer incidence for carcinogenic chemicals and the hazard index for non-carcinogenic chemicals from the atmospheric pathway.

5.2 Modeling Parameters

After the contaminants of concern and potential transport pathways have been identified, many modeling parameters are required, the number of parameters depending on the type of model chosen. Some of the key physical and environmental parameters are described in the following subsections. Many chemical-

specific values such as bioaccumulation values, octanol-water partition coefficients, and others are compiled for Superfund use in the Superfund Chemical Data Matrix and could be used as default modeling parameters if actual site data are unavailable.

5.2.1 Water Solubility

Water solubility refers to the maximum concentration of a contaminant that dissolves in a given amount of water and is measured in units of mg/L. The solubility of a contaminant is an indication of its ability to migrate through the environment and is influenced by such environmental conditions as temperature and pH. Highly water soluble contaminants are less absorbed to soil and are therefore rapidly leached from contaminated soil into both surface water and groundwater. This parameter is used to calculate volatilization. A highly soluble contaminant is less volatile than a contaminant of low solubility.

5.2.2 Vapor Pressure

Vapor pressure is a measure of the volatility of a contaminant and is measured in units of mm Hg. This parameter is useful in determining the rate of volatilization from contaminated soils and water bodies to the air pathway. Site characteristics such as temperature, wind speed and soil conditions, as well as adsorption and water solubility, affect volatilization rates. Contaminants with high vapor pressure that also have an affinity to soil or water are less likely to vaporize and become airborne.

5.2.3 Henry's Law Constant

This parameter considers molecular weight, solubility, and vapor pressure and serves as an indication of a contaminant's volatility in solution. When a contaminant is highly water-soluble in relation to its vapor pressure, the contaminant has a high Henry's Law constant and dissolves mainly in water. Thus, a high constant suggests inhalation as a potential exposure pathway. A contaminant with large vapor pressure in relation to its water solubility has a low Henry's Law constant and is likely to remain in water.

5.2.4 Octanol-water Partitioning Coefficient

The octanol-water partitioning coefficient (K_{ow}) characterizes the equilibrium partitioning of a chemical between octanol and water. The greater the K_{ow}, the more likely a chemical or radionuclide is to partition to octanol than to remain in water. Octanol is used as a surrogate for lipids (fat), and organisms tend to accumulate contaminants in the lipid portions of their tissues. Therefore, the K_{ow} predicts bioaccumulation

in biota. Contaminants with large K_{ow} values generally accumulate in biota and vegetation, adsorb onto soil, sediment, and vegetation, and transfer to humans through the food chain. Contaminants with small K_{ow} values tend to distribute in the air; therefore, exposure via the food chain is of less concern for contaminants with small K_{ow} values (EPA 1989a).

5.2.5 Carbon Partitioning Coefficient

The carbon matter partitioning coefficient (K_{oc}) characterizes the partitioning of a chemical between organic carbon and water. The K_{oc} is contaminant-specific and independent of soil properties. A high K_{oc} indicates that the contaminant bonds tightly to the soil, and less of the contaminant is available to move into groundwater or surface water. A contaminant with a low K_{oc}, however, has the potential to move into groundwater or surface water. Default values for K_{ow} and K_{oc} for organic compounds are listed in table 5.2–1 (Strenge and Peterson 1989). If K_{oc} values are unavailable, a K_{oc} can be estimated using K_{ow} as follows (Mills *et al.* 1985):

$$K_{oc} = (0.63)(K_{ow}), \qquad [5.1]$$

where K_{oc} = Organic carbon partitioning coefficient (mL/g)
K_{ow} = Octanol-water partitioning coefficient (mL/mL).

5.2.6 Soil-water Partitioning Coefficient

The K_{oc} is used to estimate the soil-water partitioning coefficient (K_d), which characterizes the extent of partitioning between soil or sediment and water (unadjusted for dependence upon organic carbon). If a contaminant has a high K_d, it is more likely to bind to soil or sediment than to remain in water. When K_d values are unavailable for organics, K_{oc} is used to estimate K_d as follows (Strenge and Peterson 1989):

$$K_d = 10^{-4} * K_{oc} * S_d , \qquad [5.2]$$

where K_{oc} = Organic carbon partitioning coefficient (mL/g)
S_d = Soil distribution coefficient (dimensionless).

S_d = 57.735 (% organic matter) + 2.0 (% clay) + 0.4 (% silt) + 0.005 (% sand). The K_d values for inorganics and radionuclides are dependent on soil pH and soil characteristics. These values are listed in table 5.2–2 according to soil pH range and soil composition (Strenge and Peterson 1989).

5.2.7 Environmental Transfer Factors

Environmental transfer parameters describe the amount of contaminant transferred from environmental media to edible foods such as aquatic foods and farm products. The contaminant concentration in fish and shellfish is related to the contaminant concentration in water through use of bioaccumulation factors. Soil-to-plant transfer factors and animal product transfer factors represent the contaminant concentration in food crops and animal products. Environmental transfer factors for chemicals and radionuclides are listed in chapter 8.

5.2.8 Transformation and Degradation Rates

Environmental decay rate constants are used to describe the transformation of radionuclides and chemicals into other contaminant species. Decay rate constants are measured in units of d^{-1} for radionuclides and chemicals in water, soil, and air. The environmental decay rate for water is the rate of contaminant removal from drinking water and farm products by degradation and radiological decay during distribution to consumers. For air, the environmental decay rate is assumed to be the rate of contaminant removal from plant surfaces by degradation and radiological decay. The decay rate for soil is the rate of contaminant removal from farm land by volatilization and radiological decay.

Table 5.2–1. Partitioning Coefficients for Organic Compounds

Constituents	K_{ow} (mL/mL)	K_{oc} (mL/g)
1,1-Dichloroethane	6.2E1	3E1
1,1,1-Trichloroethane	3.2E2	1.5E2
1,1,2,2-Tetrachloroethane	1.1E3	6.7E2
2-Methylnaphthalene	7.2E3	4.5E3
2-Methyl phenol	9.3E1	1.5E1
2,4-Dimethyl phenol	6.5E2	9.6E1
4-Methyl-2-Pentanone	5.3	1.9E1
4-Methyl Phenol	8.5E1	1.7E1
Acenaphthene	1E4	4.6E3
Acetone	5E1	2.2
Anthracene	2.8E4	1.4E4
Aroclor-1016	3.8E5	1.8E5
Aroclor-1242	1.3E4	6.3E3
Aroclor-1248	5.8E5	2.8E5
Aroclor-1254	1.1E6	5.3E5
Aroclor-1260	1.4E7	6.7E6
Benzene	1.3E2	8.3E1
Benzo(a)anthracene	4E5	1.4E6
Benzo(a)pyrene	1.2E6	5.5E6
Benzo(b)fluoranthene	1.2E6	5.5E5
Benzoic acid	7.41E1	4.6E1
Bis(2-ethylhexyl)phthalate	4.1E9	8.7E4
Carbon disulfide	1.0E2	5.4E1
Carbon tetrachloride	4.4E2	1.1E2
Chloroform	9.3E1	3.1E1
Chlordane	6E4	9.5E3

Table 5.2-1. Partitioning Coefficients for Organic Compounds

Constituents	K_{ow} (mL/mL)	K_{oc} (mL/g)
Chrysene	4.1E5	2E5
DDT	1.6E6	2.4E5
Di-n-octyl phthalate	7.4E9	3.6E9
Ethylbenzene	1.4E3	1.1E3
Fluoranthene	7.9E4	3.8E4
Fluorene	1.6E4	7.3E3
Ideno(1,2,3-cd)pyrene	3.2E6	1.6E6
Methylene chloride	2E1	8.8
Naphthalene	1.7E3	9.4E2
N-Nitrosodiphenyl amine	1.4E3	6.5E2
Pentachlorphenol	1E5	5.3E4
Phenol	2.9E1	1.4E1
Phenanthrene	2.9E4	1.4E4
Pyrene	2.1E5	3.8E4
Toluene	5.4E2	3E2
Vinyl Chloride	2.4E1	5.7E1

Source: Strenge and Peterson 1989.

Table 5.2–2. Soil-Water Partitioning Coefficients for Inorganics and Radionuclides

Constituents	pH Range and Soil Composition [a]								
	pH≥9			pH 5-9			pH≤5		
	<10%	10-30%	≥30%	<10%	10-30%	≥30%	<10%	10-30%	≥30%
Actinium	1E2	2.5E2	2E3	2.28E2	5.38E2	4.6E3	3	6	6
Aluminum	3.53E2	3.53E2	3.53E2	3.53E4	3.53E4	3.53E4	3.98E3	3.98E3	4.46E3
Americium	8.2	2E1	1E2	8.2E1	2E2	1E3	8.2E1	2E2	1E3
Antimony	0	1	1	2	6	1.59E1	2	5	1.59E1
Arsenic	6E-1	2	2	5.86	1.94E1	1.94E1	5.86	1.92E1	2.15E1
Asbestos	1E5	1E5	1E5	1E5	1E5	1E5	1E5	1E51E5	1E5
Barium	5.3E2	2.8E3	1.6E4	5.3E2	2.8E3	1.6E4	5.3E1	2.8E2	1.6E3
Beryllium	7	1.4E2	8E2	7E1	1.4E3	8E3	7	1.4E2	8E2
Borate	0	0	0	0	0	0	0	0	0
Boron	1.9E-1	1.3	1.3	1.9E-1	1.3	1.3	1.9E-1	1.3	1.3
Cadmium	3	4.29E1	1E2	1.49E1	4.23E2	5.67E2	3	4.29	1E2
Calcium Hypochlorite	0	0	0	0	0	0	0	0	0
Calcium Oxide	7E1	1.4E3	8E3	7E1	1.4E3	8E3	7	1.4E2	8E2
Carbon-14	0	0	0	0	0	0	0	0	0
Cement Dust	1E5	1E5	1E5	1E5	1E5	1E5	1E5	1E5	1E5
Cerium	1E2	2.5E2	2E3	2.28E2	5.38E2	4.6E3	3	6	6
Cesium	5.1E1	2.49E2	2.7E2	5.1E1	2.49E2	2.7E2	1E1	2.49E1	2.7E1
Chlorate	0	0	0	0	0	0	0	0	0
Chromium III	1E1	10E1	7.9E1	1.68E2	5.65E2	3.6E3	1E1	1E1	7.9E1
Chromium VI	1	1	7.9	1.68E1	5.65E1	3.6E2	1	1	7.9
Cobalt	1.94	8.81	2E2	1.94	8.81	2E2	2E-1	9E-1	2E-1
Copper	4.19	9.2	3.36E1	4.19E1	9.22E2	3.36E2	4.19	9.2	3.36E1
Curium	8.2	2E1	1E2	8.2E1	2E2	1E3	8.2E1	2E2	1E3
Europium	1E2	2.5E5	2E3	2.28E2	5.38E2	4.6E3	3	6	6
Fluoride	0	0	0	0	0	0	0	0	0
Hydrogen Fluoride	0	0	0	0	0	0	0	0	0
Iodine	0	0	0	0	0	0	0	0	0
Iron	1.5	1.5	1.5	1.5E1	1.5E1	1.5E1	1E1	1E1	1.29E1
Krypton-85	0	0	0	0	0	0	0	0	0
Lead	2.34E2	5.97E2	1.83E3	2.34E2	5.97E2	1.83E3	1E1	1E1	1.21E1
Lithium	0	2E-1	8E-1	0	2E-1	8E-1	0	0	0
Magnesium	7	1.4E2	8E2	7E1	1.4E3	8E3	7	1.4E2	8E2
Manganese	1.65E1	2.53E1	3.69E1	1.65E1	2.53E1	3.69E1	1.5	2.5	4
Mercury	3.22E2	5.8E2	5.28E3	3.22E2	5.8E2	5.28E3	3E1	6E1	5E2
Molybdenum	0	0	0	4E1	1.2E2	2.8E2	1E2	3E2	7E2
Neptunium-237	3	3	3	3	3	3	3	3	3
Nickel	1.22	5.86	6.5E1	1.2E1	5.86E1	6.5E2	1.2	5.86	6.5E1
Niobium	5	1E2	1E1	5E1	1E2	1E2	5E1	1E2	1E2
Nitrate	0	0	0	0	0	0	0	0	0
Nitric Acid	0	0	0	0	0	0	0	0	0
Nitrogen Dioxide	0	0	0	0	0	0	0	0	0
Palladium-107	4E-1	4	4E1	4E-1	4	4E1	4E-1	4	3.93E1

Table 5.2–2. Soil-Water Partitioning Coefficients for Inorganics and Radionuclides

Constituents	pH Range and Soil Composition [a]								
	pH≥9			pH 5-9			pH≤5		
	<10%	10-30%	≥30%	<10%	10-30%	≥30%	<10%	10-30%	≥30%
Actinium	1E2	2.5E2	2E3	2.28E2	5.38E2	4.6E3	3	6	6
Paper Fiber	1E5	1E5	1E5	1E5	1E5	1E5	1E5	1E5	1E5
Phosphate Ion	5	5	5	5E1	5E1	5E1	1E2	1E2	1E2
Plutonium	1E1	1E1	1.4E1	1E1	1E2	2.5E2	4	4	4.3E1
Polonium-210	5.9	1.49E1	1.49E1	5.9	1.49E1	1.49E1	5.91	1.49E1	1.49E1
Potassium Ion	0	2E-1	8E-1	0	2E-1	8E-1	0	0	0
Protactinium	0	5	5E1	0	5E1	5E2	0	5	5E1
Radium	2.43E1	1E2	1.24E2	2.43E1	1E2	1.24E2	2.43	1E2	1.24E2
Ruthenium	2.7E1	5E1	5E1	2.74E2	3.51E2	6.9E2	2.6E1	2.6E1	2.6E1
Samarium-151	1E2	2.5E2	2E3	2.28E2	5.38E2	4.6E3	3	6	6
Selenium	5.9	1.49E1	1.49E1	5.91	1.49E1	1.49E1	5.91	1.49E1	1.49E1
Silicate Ion	2	2	2	5	5	5	4	4	4
Silver	4E-1	4	4E1	4E-1	4	4E1	4E-1	4	4E1
Sodium Ion	0	2E-1	8E-1	0	2E-1	8E-1	0	2E-1	8E-1
Strontium	2.43E1	1E2	1.24E2	2.43E1	1E2	1.24E2	2.43	1E2	1.24E1
Sulfate	0	0	0	0	0	0	0	0	0
Sulfuric Acid	0	0	0	0	0	0	0	0	0
Sulphur-35	0	0	0	0	0	0	0	0	0
Technetium-99	3	2E1	2E1	3	2E1	2E1	3	2E1	2E1
Thallium	0	2E-1	8E-1	0	2E-1	8E-1	0	2E-1	8E-1
Thorium	4E1	6E1	1E2	1E2	5E2	2.7E3	4E1	6E1	1E2
Tin	2.5	5	5	5	1E1	1E1	2.5	5	5
Tin-126	2.5	5	5	5	1E1	1E1	2.5	5	5
Tritium	0	0	0	0	0	0	0	0	0
UO_2F_2	0	5	5E1	0	5E1	5E2	0	5	5E1
Uranium	0	5	5E1	0	5E1	5E2	0	5	5E1
Vanadium	5	1E2	1E1	5E1	1E2	1E2	5E1	1E2	1E2
Yttrium	1E2	2.5E2	2E3	2.28E2	5.38E2	4.6E3	3	6	6
Zinc Compounds	12.7	1.43E2	1.46E3	1.27E1	9.39E2	1.46E3	3	2.8E2	2.8E2
Zinc-65	12.7	1.43E2	1.46E3	1.27E1	9.39E2	1.46E3	3	2.8E2	2.8E2
Zirconium	5	5E1	1E2	5E1	5E2	1E3	5E1	5E2	1E3

Source: Strenge and Peterson 1989.

[a] Soil composition = total percent of clay, organic matter, and iron, and aluminum oxyhydroxides.

6. EXPOSURE ASSESSMENT

Exposure is defined as the contact of an organism with a chemical or physical agent. The magnitude of exposure is determined by measuring or estimating the amount of the agent available at exchange boundaries (i.e., the lungs and skin) during a specified time period. Exposure assessment is the determination or estimation (qualitative and quantitative) of the magnitude, frequency, duration, and route of such exposure with regard to both current and future conditions. The objective of the exposure assessment is to estimate the type and magnitude of exposures to contaminants of concern that are associated with a site or facility. The magnitude of exposure (i.e., the dose or intake of a contaminant) is the most important element since it is the intake or dose (the amount that is taken into the body) that is used to estimate risks. The exposure assessment follows the general principles outlined by EPA (1992a). While the Agency for Toxic Substances and Disease Registry (ATSDR) Public Health Assessment Guidance Manual follows another outline for presentation of health assessments, the format of RAGS was considered to be more advantageous for the programmatic element of this methodology. Following RAGS, the exposure assessment proceeds through the following steps:

- Characterization of exposure setting and potential receptors
- Identification of exposure pathways and routes
- Identification of exposure scenarios
- Quantification of exposure.

Before exposure can be determined, the release site's or treatment facility's exposure setting or physical characteristics must be determined. At the same time, the behavioral patterns of human populations (potential receptors) on or near the release site or TSD facility must be characterized. Particular emphasis should be placed on behavioral patterns that might affect any aspect of contaminant exposure. As discussed in chapter 2, physical characteristics of the release site or TSD facility that may be relevant to exposure assessment include climate, meteorology, geologic setting, vegetation, soil type, ground water hydrology, and location of and description of surface water. Population characteristics that are likely to affect exposure include location of current populations relative to the site or facility and identification of any subpopulations of concern (i.e., those that may have increased risk). The output of exposure setting characterization is a qualitative evaluation of the release site or treatment facility and the surrounding populations with emphasis on those characteristics that influence exposure.

The second step of exposure assessment is the identification of potential exposure pathways and routes. An exposure pathway describes the route a chemical or physical agent takes from its source to the exposed individual. A complete exposure pathway links the source (or release from a source), an exposure route, an exposure point, and a receptor. If the exposure point differs from the source, the exposure pathway also

must include a transport medium or media (i.e., environmental media through which contaminants are transported). Fate and transport modeling can be used to predict contaminant movement from sources to receptors and estimate exposure point concentrations. Exposure routes are the mechanisms by which receptors come into contact with contaminants. Major types of exposure routes are inhalation, ingestion, direct radiation, and dermal contact.

The third step of the exposure assessment is identification of exposure scenarios. An exposure scenario is a representation of an individual's or population's activities and the frequency and duration of those activities. Exposure scenarios include residential, agricultural, industrial, or recreational activities for both onsite and offsite populations.

The final step in the exposure assessment is exposure quantification. In this step, the receptors' intakes or doses of contaminants are quantified using EPA methods to determine the magnitude of exposure. For each exposure pathway and contaminant, quantifiable exposure estimates are produced, which are then used as input into the final stages of the risk assessment.

6.1 IDENTIFICATION OF EXPOSURE PATHWAYS

An exposure pathway is the course a chemical or physical agent takes from a source to an exposed organism. Exposure pathways involve the transport of gases, liquids, and particulate solids within a given medium and across the interfaces between air, water, and soil. The pathway describes a unique mechanism by which an individual or population is exposed to contaminants at a site or facility. For example, contaminants in surface soil may travel to groundwater. Individuals may then be exposed to the contaminants through the drinking water supply, inhalation of vapors, and absorption through the skin while showering. The exposure pathway for exposure via drinking water is: source → surface soil → groundwater → drinking water ingestion.

Exposure pathways may be of three types: potential, completed, or eliminated. A potential exposure pathway is an exposure pathway that is being considered for analysis. A potential pathway is based on the likelihood of the existence of exposure routes and linkages between environmental media. A complete exposure pathway must have the following elements:

- A source and mechanism of chemical release (i.e., source term)
- A transport medium (or media)
- An exposure route (e.g., ingestion, inhalation)
- An exposure point
- A receptor.

An eliminated pathway is an exposure pathway that has undergone preliminary analysis and has been found to be incomplete (i.e., one or more of the elements comprising a complete pathway is absent). Incomplete exposure pathways must be eliminated from the risk assessment, since incomplete exposure pathways cannot be evaluated. A conceptual model (CM) illustrates transport media and exposure route relationships at a site or facility, serving as a tool to determine whether or not exposure pathways are complete (see figure 6.1-1). A more detailed description of CMs is provided in section 6.1.3.

6.1.1 Identification of Transport Media

Transport media, or the environmental media through which contaminants travel, must be determined in order to identify exposure pathways. The following five transport media are considered in estimating health risks:

- Air
- Surface soil
- Groundwater
- Surface water
- Direct radiation.

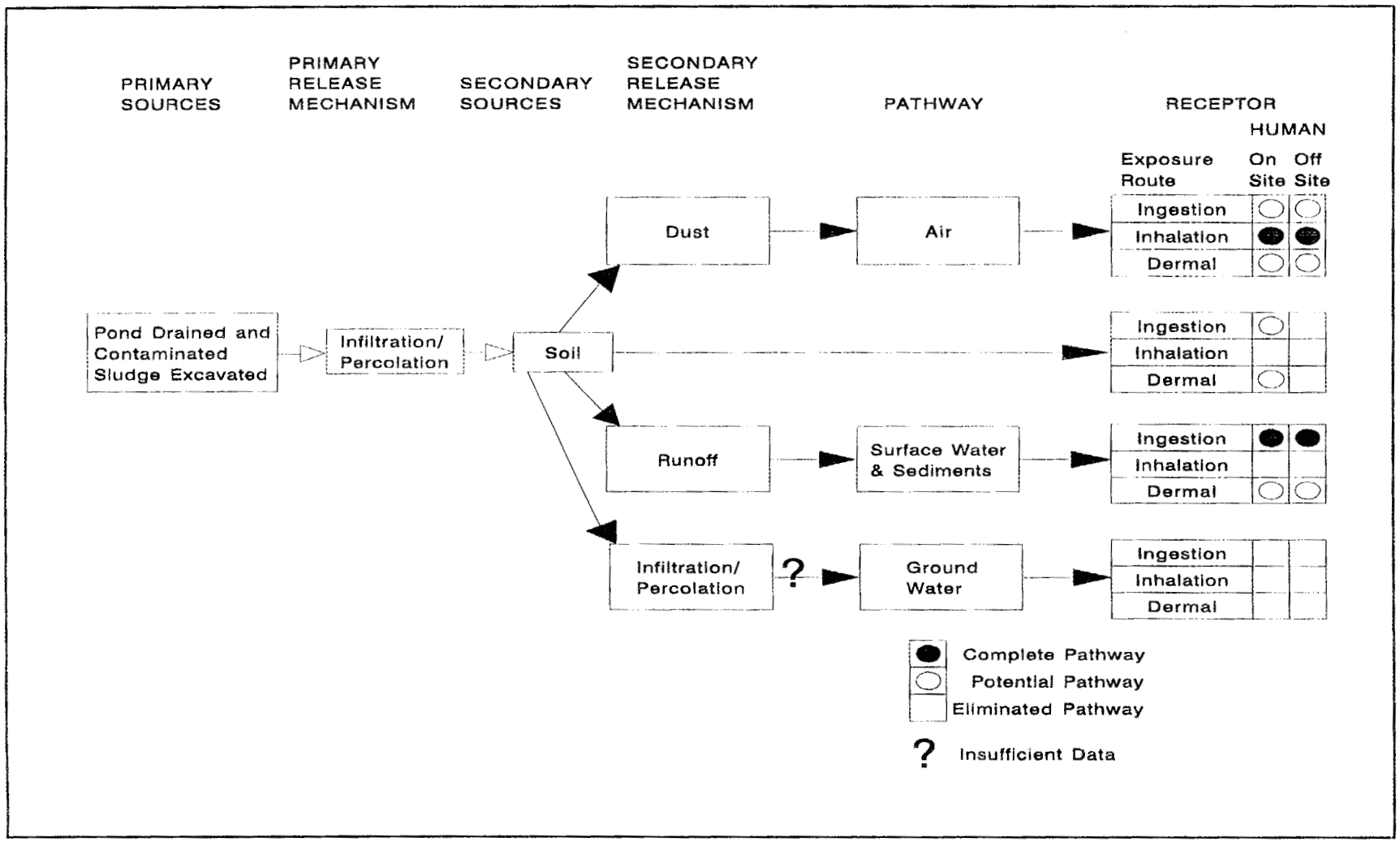

Figure 6.1–1. Representation of Conceptual Model.

The atmospheric transport medium may transport gaseous or particulate forms of contaminants through the air surrounding sites or TSD facilities. The airborne contaminant can be inhaled or ingested directly by an individual.

The surface soil transport medium may transport gaseous (via volatilization) or particulate forms of contaminants from the surface soil to the air where they can be inhaled, to subsurface soil and downward to ground water, or by overland runoff directly to surface water.

The surface water and groundwater transport media carry contaminants through water where they can be ingested directly via the ingestion exposure route. Contaminants in these two transport media may also continue through environmental media such as soil or crops (through irrigation), and groundwater may recharge surface water.

Direct radiation may result in exposure to photon-emitting radionuclides. Measured values of radionuclide concentrations in soil, water, or contaminated surfaces (e.g., buildings or equipment) are the sources of direct radiation exposure. This transport medium involves no transport per se because exposure is direct and occurs at the release site or facility location.

6.1.2 Identification of Exposure Routes

Exposure routes are the mechanisms by which receptors come into contact with contaminants. As illustrated by figure 6.1-2, contaminants are transported through environmental media to an exposure point of contact, whereby they are taken into organisms (i.e., in this case, members of the public or onsite employees). The exposure routes included in the human health risk evaluation are as follows:

- **Drinking water ingestion.** Contaminated drinking water may be ingested after it is transported via groundwater, surface, overland flow, or surface water. Factors may be applied to the water concentration that will account for purification of the water in a treatment plant.

- **Aquatic food ingestion (fish and invertebrates).** Aquatic food may be contaminated by contaminants transported via groundwater, overland flow, or surface water. Average daily intake of fish and shellfish is estimated using bioconcentration factors and average daily ingestion rates for aquatic foods. Aquatic food is contaminated from surface water sediment more so than from the water column because of higher residuals in sediment.

- **Food crop ingestion.** Crop concentrations are estimated using soil-to-plant transfer factors and air-to-plant transfer factors. Average daily intake is estimated using average daily ingestion rates for both root and leafy vegetables.

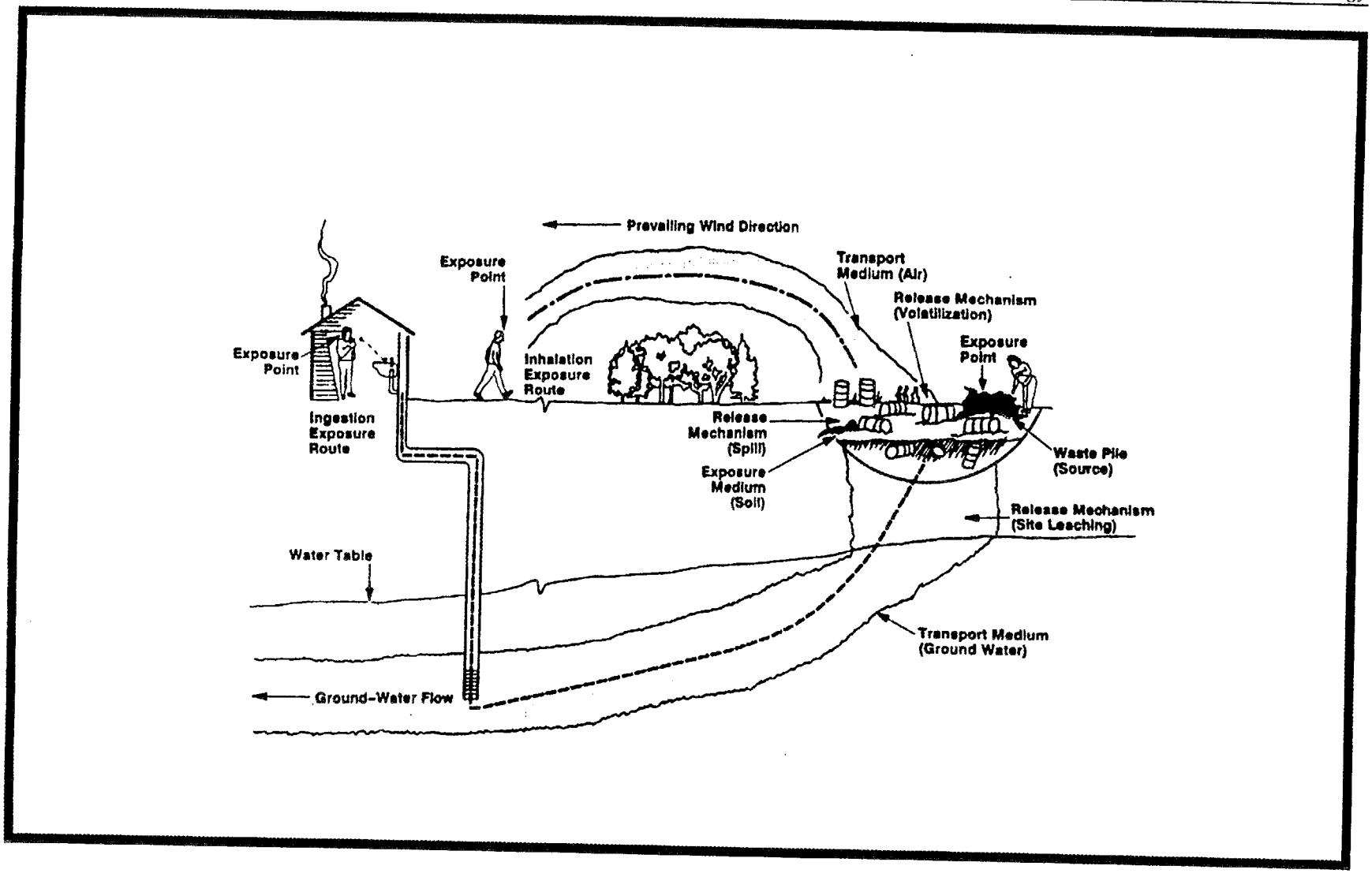

Figure 6.1-2. Illustration of Exposure Routes.

Source: EPA 1989a.

- **Animal product ingestion.** The concentration of contaminant in animal meat and milk is estimated using animal ingestion-to-animal product transfer factors. Average daily intake of exposed individuals is estimated using average daily ingestion rates for meat and milk.

- **Water immersion (domestic bathing and swimming).** Humans may be exposed to contaminated groundwater and surface water by being immersed. Dermal contact (for chemicals) and direct radiation are included for domestic bathing for both water transport media. Inhalation of volatile organics during showering with groundwater is also included. Exposure from swimming in contaminated water is considered for the surface water exposure pathway. For chemicals, an equivalent daily intake amount is estimated based on dermal contact time and absorption characteristics of the specific contaminant. For radiation exposures, the dose from immersion in water is estimated using dose conversion factors (DCF). If applicable, a contribution to radiation dose may also be included for recreational boating and shoreline fishing.

- **Soil ingestion.** Soil may be transported via air to receptors, where it may be ingested. Soil may also be ingested when deposited on crops or directly ingested by dermal contact. Contaminated soil is assumed to be ingested each day with the ingestion rate based on a lifetime average.

- **Inhalation.** Contaminants transported via air may be inhaled by receptors. The average daily intake is estimated using an average inhalation rate for the exposed population.

- **Direct radiation.** Exposure to radiation may occur externally or internally and is harmful to tissues. Each type of radiation differs in its physical characteristics and in its ability to harm biological tissue.

6.1.3 Conceptual Model

A CM is a diagram that illustrates transport media and exposure route scenario relationships at a site or facility. A CM is used to develop a conceptual understanding of a site's or facility's potential risk to human health and the environment. More importantly, a CM serves as a tool to help eliminate pathways from a risk assessment by qualitatively and conceptually evaluating whether exposure pathways are complete. The CM identifies which pathways to evaluate and data gaps that must be filled in order to evaluate a potential pathway. The CM should include the following information:

- **Known and suspected sources of contamination.** Open barrels or tanks, waste piles, and contaminated surface soil are examples of contamination sources. Sources should be identified for past, current, and future releases.

- **Types of contaminants and affected media.** The contaminants in sources or the environment at a site or facility and the media in which contaminants occur must be determined. A medium contaminated from a past release can be the contaminant source for other media; therefore, identifying affected past, present and future media is important. For example, soil contaminated from a previous spill could be a contaminant source for groundwater or surface water.

- **Known and potential routes of migration.** Environmental media may transport contaminants through the environment. Sources of contaminant migration include the air, surface soil, surface water, groundwater, and direct radiation. After a chemical is released into the environment, it may be (1) transported through water, sediment, or the air, (2) physically transformed by volatilization, or precipitation, (3) chemically transformed, or (4) biologically transformed.

- **Known or potential human and environmental receptors.** An exposure pathway must have a receptor to be complete. Any point of potential contact with contaminated media is an exposure point. After contaminated or potentially contaminated media have been identified, exposure points should be identified by determining if and where any of the potentially exposed populations can contact these media through an exposure route (such as ingestion, inhalation, or dermal contact). If a contaminant is released from a source into the air but has no potential receptor (nearby population), the pathway is of no potential concern to human health and can probably be excluded from the risk assessment.

Based on the above information, a CM summarizes exposure pathway information by identifying exposure populations and transport media, exposure points, and exposure routes. This information should be reviewed to determine the complete pathways that exist for a release site or TSD facility.

Although a complete exposure pathway must have a source and mechanism of contaminant release, a potential receptor, and an exposure route, the source itself can be an exposure point without a release to any other mechanism. In such cases, the exposure pathway consists of a source, an exposure point, and an exposure route. If a pathway does not meet these conditions, it is considered an incomplete pathway and should be eliminated from the assessment. When complete pathways are determined, those pathways that require further evaluation should be selected. All complete pathways should be selected for further evaluation unless sound justification can be made for their omission. Absence of data or insufficient data are often acceptable reasons for eliminating an exposure pathway from the evaluation; however, the impacts of omitting an exposure pathway from the risk assessment should still be addressed qualitatively. Based on such qualitative information, risk managers can then decide whether further sampling and data are warranted in a subsequent analysis. Accompanying a CM should be text that justifies why a pathway is eliminated, clearly stating the problem and the data needed to resolve it. Such text serves as a record of assumptions for each pathway modeled. Absence of data is symbolized by a "?" in figure 6.1-1.

6.2 IDENTIFICATION OF EXPOSURE SCENARIOS

An individual's activities and the frequency and duration of those activities make up the exposure scenario for a given exposure pathway and determine the magnitude of his/her exposure to contaminants and, consequently, the magnitude of risk. An exposure scenario consists of a set of associated exposure factor values, which provide numerical information (e.g., exposure duration, exposure frequency, consumption rates, inhalation rates) for calculating chemical intake or dose. Default exposure values are provided in appendix A. Exposure scenarios are the aspects of activity that can be used to calculate a dose or intake. Exposure can be calculated for activities that occur under normal or nonroutine conditions.

Within the context of the exposure scenario, the following information is integrated:

(1) the physical characteristics of an area (variables indicative of where and in what forms a contaminant is most likely to occur in the environment),

(2) the activity patterns and physical characteristics of potentially exposed individuals (variables that describe frequencies and durations of exposure), and

(3) chemical-specific information (contaminant concentrations in media, bioconcentration factors, and partitioning coefficients that characterize the individual contaminants) (EPA 1989a).

Exposure scenarios are often defined in terms of land use, both restricted and unrestricted land use, using varying levels of institutional controls. Institutional controls (e.g., erection and maintenance of fences with security guards, patrols, and warning signs) mitigate health risks by physically restricting land use at a site. According to EPA recommendations, unrestricted land use scenarios should be evaluated. These scenarios address exposure to individuals who may live on or near the site (i.e., residential scenario). The scenarios described here occur under either normal operations or nonroutine conditions.

6.2.1 Normal Operations

Normal operations constitute the day-to-day operations of WM facilities or ER sites. Different exposure scenarios (e.g., residential, industrial, agricultural, intruder) and parameters can apply during normal operations. A residential scenario is a situation where a population or an individual resides either on or offsite in a typical dwelling. An industrial scenario is a situation where a population or an individual works on or offsite but does not reside there or use the premises for showering or other residential activities. An agricultural scenario is a situation where a population or individual grows crops or raises livestock for consumption on or offsite. A recreational scenario is, for example, an individual living near a source in a residential area is assumed to be exposed to contaminants for different durations and frequencies (e.g.,

350 d/yr, 30 yr) than an individual who works near a source in an industrial area (e.g., 250 d/yr, 8 hr/d, 5 d/wk). An intruder scenario is a situation in which a hypothetical individual is chronically exposed to contaminated drilling wastes in an era of no institutional control. This methodology focuses on activities that occur during normal operations. The intake equations and parameters generally pertain to normal, chronic activities.

6.2.2 Nonroutine Events

The occurrence of natural disasters or catastrophic events constitute nonroutine events. Receptors for these situations generally include offsite residents and onsite employees. For nonroutine situations involving radionuclides, the acute portions of fate and transport models (if available) are used. To be most conservative, the wind direction, conditions, and population sector that would produce the worst risks is chosen as the sector to model for the event. For chemicals, fate and transport models such as ISC2-ST require wind directions. For nonroutine events, it is assumed that on average chemical releases last for one hour. As with radionuclides, the sector that would produce the worst case risks for each receptor evaluated is selected. From that worst case sector, the three most likely wind conditions are chosen and modeled. ISC yields the air concentration in an area for the particular sector chosen for the one hour span. The air concentration can then be used to estimate intakes and risks.

Some ER sites, such as buildings undergoing decommissioning and decontamination, have no current releases and do not produce risks. However, to assess future risks, catastrophic events or other probabilistic occurrences are often used to estimate potential future risks. For example, one might assume institutional controls lapse and the building begins a natural degradation process. The degradation of the building depends on the materials of which it is built. At some stage of this degradation process (or even before degradation begins), a natural disaster could occur (e.g., flooding, earthquake, tornado). The probability of such an occurrence has usually been studied and documented at individual installations. Depending on the conditions of the accident, a 100% immediate release (or some fraction thereof) of the building's contaminants could be assumed. This release would then serve as the mechanism for contaminants to reach potential receptors. The results of such a risk assessment should be presented along with an indication of the probability of the event's occurrence to put the risks of a 100% immediate release into perspective.

6.3 Quantification of Exposure

Exposure quantification provides the average individual dose for each contaminant. Both chemical and radionuclide doses are calculated for one pathway and contaminant at a time. For chemical contaminants, the dose is expressed as the amount of contaminant (per unit body weight) that is inhaled, ingested, or

absorbed by an average member of the population. For radioactive contaminants, the dose received from direct radiation is expressed as the effective dose equivalent (EDE) received from each contaminant. EDEs are used to normalize radiation doses and effects on a whole body basis rather than on an individually affected organ or tissue basis. For pathways other than direct radiation, intakes of radioactive contaminants are in units of activity (Ci) per unit time.

Chapters 7 through 11 document the methods used for exposure quantification for various exposure routes.

7. INGESTION EXPOSURE ROUTE

Individuals may be exposed to contaminants by ingesting contaminated water or food. By ingestion of groundwater or surface water used for drinking and bathing or by incidental ingestion of surface water while swimming, individuals may be exposed to contaminants in water. Exposure to contaminants may also occur if contaminated plants, aquatic foods, beef, or milk are ingested. The inadvertent ingestion of soil is also a potential route of exposure. The following sections discuss these water, food, and inadvertent soil ingestion exposure routes in more detail.

Unless otherwise indicated, all equations in this chapter are taken from Whelan et al.(1987).

7.1 INGESTION OF WATER

The following sections discuss ingestion of water exposure routes and provide the equations for calculating intakes. The ingestion of water exposure routes are listed below:

- Ingestion of contaminated groundwater and surface water used for drinking
- Ingestion of contaminated groundwater and surface water used for bathing
- Ingestion of contaminated surface water while swimming.

7.1.1 Domestic Water (Drinking and Bathing)

Exposure to contaminants via the domestic drinking water ingestion route may result from ground water or surface water transport media. The overland flow transport medium is included because it may contribute to contamination in surface water systems and, thus, may indirectly contribute to drinking water ingestion. The intake to an individual using a contaminated domestic water supply is calculated as follows:

$$D_w = IR * C_w * T_f * D_g * \exp(-\lambda_w * t_p), \qquad [7.1]$$

where D_w = Average individual intake from ingesting drinking water (mg/kg/d or pCi/d),

IR = Average daily drinking water ingestion rate for an individual (L/d); average daily domestic bathing water ingestion rate for an individual (L/d),

C_w = Average contaminant concentration in water before water treatment (mg/L or pCi/L),

T_f = Water treatment purification factor indicating fraction of contaminant remaining after treatment (dimensionless),

D_g = Average amount of chemical ingested per day related to daily dose per unit body weight (kg^{-1}) or ingestion dose conversion factor for radionuclide (dimensionless),

λ_w = Contaminant environmental degradation (or radiological decay) constant in water (d^{-1}),

t_p = Average time of contaminant transit through the water distribution system (d).

For radionuclides, the dose conversion factor (D_g) is used to represent the dose equivalent per intake. The water treatment purification factor accounts for removal of contaminants during water treatment at municipal water supply facilities. If no water treatment is performed, the purification factor is unity (1).

For incidental ingestion of domestic water while bathing, the same equation is used with the exception of the value for intake rate (IR). The ingestion of water during bathing is normally insignificant as compared with the ingestion of drinking water; however, for locations where water is used for bathing but not for drinking, the bathing dose may be significant. Each individual is assumed to bathe once each day.

7.1.2 Incidental Ingestion of Water While Swimming

The incidental ingestion of water while swimming is another route by which individuals may be exposed to contaminants. Average individual intake from exposure to ingestion of contaminants while swimming is calculated as follows:

$$D_{sw} = \frac{IR * C_w * ET * D_g}{EF}, \qquad [7.2]$$

where D_{sw} = Contaminant-specific average individual intake from incidental ingestion of water while swimming (mg/kg/d or pCi/d),

IR	=	Intake rate for recreational swimming (L/hr),
C_w	=	Average contaminant concentration in water (mg/L or pCi/L),
ET	=	Exposure time (hr/yr),
D_g	=	Average amount of chemical ingested per day related to daily dose per unit body weight (kg^{-1}) or ingestion dose conversion factor for radionuclide (dimensionless),
EF	=	d/yr.

The exposure factors for ingestion of domestic water, bathing water, and incidental ingestion of water while swimming are listed in appendix A.

7.2 Ingestion of Food

The following sections discuss the ingestion of food exposure routes and provide equations for calculating intakes. The exposure routes for ingesting food are the following:

- Ingestion of contaminated aquatic foods
- Ingestion of contaminated food crops
- Ingestion of contaminated animal products.

7.2.1 Aquatic Food

Two types of aquatic foods are considered for this exposure route: fish (representative of organisms living in free-flowing waters) and shellfish (representative of organisms living in, or feeding on, sediments). The contaminant concentration in these organisms is related to the contaminant concentration in water through use of bioaccumulation factors. The average individual intake from ingestion of aquatic foods is calculated using the following equation:

$$D_{aqf} = \sum_{f=1}^{n} [IR_f * C_w * B_f \exp(-\lambda_w * t_f)] * D_g, \qquad [7.3]$$

where D_{aqf} = Contaminant-specific average individual intake from ingestion of aquatic foods (mg/kg/d or pCi/d),

n	=	Number of aquatic foods considered (n = 2; fish and shellfish),
f	=	Index on aquatic food types (fish; shellfish),
IR_f	=	Average consumption rate of aquatic food *f* for individuals in the population (kg/d),
C_w	=	Average contaminant concentration in water (mg/L or pCi/L),
B_f	=	Bioaccumulation factor for fish or shellfish (L/kg),
λ_w	=	Contaminant environmental degradation (or radiological decay) constant in water (d^{-1}),
t_f	=	*f*-specific average time for decay from food harvest to consumption (d),
D_g	=	Average amount of chemical ingested per day related to daily dose per unit body weight (kg^{-1}) or ingestion dose conversion factor for radionuclide (dimensionless).

Bioaccumulation factors from models derived for use in radiological analysis are available. For chemical contaminants that behave differently from the elemental form, additional data must be obtained. Default correlations for estimating bioaccumulations are based on octanol-water partitioning coefficients. These correlations are taken from Lyman et al. (1982). The correlation equation for fish is based on Veith et al. (1980):

$$\log_{10}(B_{fish}) = 0.76 \log_{10}(K_{ow}) - 0.23, \qquad \textbf{[7.4]}$$

where B_{fish} = Bioaccumulation factor for fish (L/kg),

K_{ow} = Octanol-water partition coefficient.

The equation used for shellfish is similar to equation 7.4 and is taken from Southworth et al. (1978a,b):

$$\log_{10}(B_{shellfish}) = 0.819 \log_{10}(K_{ow}) - 1.146, \qquad \textbf{[7.5]}$$

where $B_{shellfish}$ = Bioaccumulation factor for shellfish (L/kg),

K_{ow} = Octanol-water partition coefficient.

Equations 7.4 and 7.5 represent only an order-of-magnitude estimate and should be used only when contaminant-specific data are not available. The exposure factors for aquatic food ingestion are presented in appendix A.

7.2.2 Food Crops

Both irrigation with contaminated water and direct deposition of atmospheric contaminants onto plants and soil can contaminate agricultural crops. Two food products associated with contaminated crop production are considered: leafy vegetables (representing plants such as lettuce for which the edible portions of the plant are above ground, exposed, and eaten directly with little processing) and other vegetables (representing all other crops—including fruit—for which directly deposited contaminants have a much smaller chance of being incorporated directly into the edible portion of the plant). The method that is used to estimate contaminant concentrations in the edible portions of the plant considers uptake through three pathways: direct deposition, absorption through the leaves from the atmosphere and absorption through the roots from the soil.

The contribution to plant contaminant concentration from direct deposition onto leaves at the time of harvest is calculated as follows:

$$C_p = D_c * T_{fp} * r * \frac{[1 - \exp(-\lambda_e t_p)]}{(\lambda_e Y_p)}, \qquad [7.6]$$

where C_p = Contaminant-specific, *p*-specific (leaf or root) concentration in the vegetable from deposition onto leaves (mg/kg or pCi/kg),

p = Index on vegetable type (1 = leafy; 2 = root or other vegetable),

D_c = Contaminant deposition rate from air or water onto farmlands (mg/m^2-d or pCi/m^2-d),

T_{fp} = *p*-specific translocation factor from plant surfaces to edible plant parts (dimensionless),

r = Fraction of deposition retained on plant surfaces (dimensionless),

λ_e = Contaminant effective weathering and decay constant(d^{-1}) ($\lambda_e = \lambda_a + 0.04951$) (0.04951 = Weathering decay constant corresponding to a half-time of 14 days [d^{-1}]),

λ_a = Contaminant environmental degradation and decay constant in air (d^{-1}),

t_p = Duration of growing season (60 days [NRC 1977]),

Y_p = *p*-specific crop yield for edible portion of plants (kg/m^2).

For the air deposition pathway, the contaminant deposition rate (D_{ca}) is calculated from the contaminant concentration in air and an average deposition velocity as follows:

$$D_{ca} = 86400 * C_a * V_d, \qquad \textbf{[7.7]}$$

where D_{ca} = Contaminant deposition rate in air (mg/m^2-d or pCi/m^2-d),

C_a = Contaminant concentration in air (mg/m^3 or pCi/m^3),

V_d = Deposition velocity (m/s),

86,400 = Unit conversion factor (s/d).

For the water pathways, the contaminant deposition rate (D_{cw}) is calculated from the irrigation rate (I_r) and the contaminant concentration in water as follows:

$$D_{cw} = C_w * \frac{I_r}{30}, \qquad \textbf{[7.8]}$$

where D_{cw} = Contaminant deposition rate in water ($mg/L\text{-}m^2$-d or $pCi/L\text{-}m^2$-d),

C_w = Concentration in water (mg/L or pCi/L),

I_r = Irrigation water application rate (L/m^2-mo),

30 = Units conversion factor (d/mo).

The contribution to contaminant concentration in plants from the root uptake pathway (C_r) is calculated as follows for air deposition pathways:

$$C_r = \frac{C_s * B_v}{S_d}, \qquad \textbf{[7.9]}$$

where C_r = Plant concentration from uptake through roots (mg/kg or pCi/kg),

C_s = Contaminant concentration in soil (mg/kg or pCi/kg),

B_v = Soil-to-plant transfer factor (dimensionless),

S_d = Area soil density (kg/m^2).

For inorganics, the B_r transfer factor for reproductive parts of the plant should be used. Values for this parameter can be found in Baes et al. (1984). The calculation of contaminant concentration in plants from contaminated water involves estimating the average soil concentration over the irrigation period (usually defined as the growing season for the site). The plant concentration at the time of harvest (C_h) is estimated as follows:

$$C_h = B_v * \frac{D_c}{S_d * \lambda_e} * [1 - \exp(-\lambda_e * t_p)], \qquad \textbf{[7.10]}$$

where C_h = Plant concentration at the time of harvest (mg/kg or pCi/kg),

B_v = Soil-to-plant transfer factor (dimensionless),

D_c = Contaminant-specific deposition rate from water onto farmlands (mg/m^2-d or pCi/m^2-d),

S_d = Site-specific soil density (kg/m^2),

λ_s = Contaminant-specific effective weathering and decay constant (d^{-1}) ($\lambda_e = \lambda + 0.04951$) (0.04951 = Weathering decay constant corresponding to a half-time of 14 days [d^{-1}] (Whelan et al. 1987),

t_p = Duration of growing season (60 days [NRC 1977]).

The soil-to-plant transfer factor (B_v) is available only for organics and radionuclides (Strenge and Peterson 1989). If a contaminant's transfer factor is not available, an estimate can be made based on the

correlation of Travis and Arms (1988), which is based on the relationship between transfer and the octanol-water partition coefficients:

$$\log (B_v) = 1.588 - 0.588 \log (K_{ow}), \qquad \textbf{[7.11]}$$

where B_v = Soil-to-plant transfer factor (dimensionless),

K_{ow} = Octanol-water partition coefficient.

The original Travis and Arms (1988) correlation is based on plant concentrations expressed as dry weight; however, the soil-to-plant transfer factors used in this evaluation are expressed as wet weight. Therefore, the correlation presented in the following equation was obtained by assuming that the dry weight concentration is four times the wet weight concentration (i.e., the plant is assumed to be 75% water) (Strenge and Peterson 1989):

$$\log (B_v) = 0.986 - 0.588 \log (K_{ow}), \qquad \textbf{[7.12]}$$

where B_v = Soil-to-plant transfer factor (mg/kg wet plant per mg/kg dry soil or pCi/kg wet plant per pCi/kg dry soil)

K_{ow} = Octanol-water partition coefficient.

Equation 7.12 is also an order-of-magnitude estimate and should only be used when contaminant-specific data are unavailable.

Equations 7.6 through 7.12 are used to estimate the contaminant concentrations in plants based on a farming cycle of one year. The contaminant concentrations in plants resulting from irrigation and from atmospheric deposition is estimated as 30 times the one-year value, which is based on the 30-yr residence. This estimate is appropriate because crop plants are harvested annually, and no contaminant accumulation occurs. However, for contributions to contaminant concentrations in plants via root uptake from soil, a correction must be made to account for contaminant accumulation in soil that results from repeated atmospheric deposition or irrigation water applications each year. This correction is made by applying a soil retention factor (S_f) to the root uptake plant concentration, which includes contribution to contamination of soils from previous years. The S_f is calculated as follows:

$$S_f = \frac{1}{10{,}500 * \lambda_s} \sum_{n=1}^{30} [1 - \exp(-\lambda_s * n * 350)], \qquad \textbf{[7.13]}$$

where S_f = Soil retention factor for 30 years of atmospheric deposition or irrigation water application (dimensionless),

λ_s = Environmental degradation and decay constant in soil (d^{-1}),

n = Index on year within the 30-yr period,

350 = d/yr,

24,500 = d/30 yr.

The average total plant concentration at the time of consumption (C_c) is then calculated as the sum of the contributions from direct deposition on leaves and soil uptake by roots (including a term from background soil concentration levels) as follows:

$$C_c = [C_p + (S_f * C_r) + (C_b * B_v)] * [\exp(-\lambda_w * t_{hc})], \qquad \textbf{[7.14]}$$

where C_c = Contaminant-specific, vegetable type-specific average concentration at time of consumption (mg/kg or pCi/kg),

C_p = Contaminant-specific, *p*-specific (leaf or root) concentration in the vegetable from deposition onto leaves (mg/kg or pCi/kg),

S_f = Soil retention factor for 30 years of atmospheric deposition or irrigation water application (dimensionless),

C_r = Plant concentration from uptake (mg/kg or pCi/kg),

C_b = Background soil concentration (mg/kg or pCi/kg),

B_v = Soil-to-plant transfer factor (dimensionless),

λ_w = Contaminant effective weathering and decay constant (d^{-1}) ($\lambda_w = \lambda_d + 0.04951$) (0.04951 = Weathering decay constant corresponding to a half-time of 14 days [d^{-1}]),

t_{hc} = Vegetable type-specific time between harvest and consumption (d).

Equation 7.14 is used for computing the average contaminant concentration for most contaminants. As described in chapter 9, however, a more realistic model is required for the contaminant tritium because it is associated more closely with water. A special equation is also used to estimate plant uptake of tritium from the water pathways.

The average individual dose (D_{fc}) from ingestion of agricultural crops is estimated from the plant contaminant concentration (as determined by equation 7.14) is calculated as follows:

$$D_{fc} = \sum_{p=1}^{2} IR * C_c * D_g, \qquad [7.15]$$

where D_{fc} = Contaminant-specific average individual intake from consumption of contaminated crops (mg/kg/d or pCi/d),

IR = Average daily consumption rate (kg/d),

C_c = Vegetable type-specific average concentration at time of consumption (mg/kg or pCi/kg),

D_g = Average amount of chemical ingested per day related to daily dose per unit body weight (kg^{-1}) or ingestion dose conversion factor for radionuclide (dimensionless).

The exposure factors for food crop ingestion are listed in appendix A.

7.2.3 Animal Products

Atmospheric deposition of contaminants onto feed crops and use of contaminated water to irrigate those crops can result in the ingestion of contaminated crops by animals. In addition, contaminated water can be used as part of livestock drinking water supply. Subsequent ingestion of contaminated animal products can then result in human exposure to contaminants. The two animal products considered in the human health risk evaluation methodology are cow's milk and meat.

In evaluating the contaminant concentration in milk and meat, it is assumed that the animals are fed crops containing contaminant levels defined by equation 7.14 (without consideration of the decay correction between harvest and consumption [the exponential term with t_{hc}] in that equation). The animal product concentration resulting from animal ingestion of contaminated feed is calculated as follows:

$$C_{mf} = C_c * F_m * f_f * IR_f * \exp(-\lambda_w * t_{hc}), \qquad [7.16]$$

where C_{mf} = Contaminant-specific, product-specific contaminant concentration from animal ingestion of contaminated feed (mg/kg or pCi/kg for meat; mg/L or pCi/L for milk),

m = Index on animal product (meat or milk),

C_c = Crop-specific contaminant concentration in feed crop used by the animal (equation 7.14),

F_m = Transfer coefficient that relates daily intake rate by an animal to the contaminant concentration in an edible animal product (mg/kg meat per mg/d or pCi/kg meat per pCi/d; mg/L milk per mg/d or pCi/L milk per pCi/d),

f_f = Fraction of animal feed that is contaminated (dimensionless),

IR_f = Intake rate of feed by the animal (68 kg/d for meat; 55 kg/d for milk),

λ_w = Environmental degradation and decay constant for contaminant in water (d^{-1}),

t_{hc} = Holding time between harvest or slaughter and consumption (20 days for meat; 4 days for milk).

In evaluating the contaminant concentration (C_c) from equation 7.14, some parameter values representative of animal feed production are used that differ from the vegetable production parameters for human consumption. The duration of growing season (t_p; equation 7.6) is set to 30 days in order to represent animal grazing habits. Also, the crop yield (Y_p; equation 7.6) is less (0.7 kg/m^2) for animal feed production.

The contribution to animal product contaminant concentration from animal ingestion of contaminated water is calculated as follows:

$$C_{mw} = C_w * F_m * f_w * IR_w * \exp(-\lambda_w * t_{hc}), \qquad [7.17]$$

where C_{mw} = Contaminant concentration in animal product (m) from animal ingestion of water (mg/kg or pCi/kg for meat; mg/L or pCi/L for milk),

C_w = Contaminant concentration in water (mg/L or pCi/L),

F_m = Contaminant transfer coefficient that relates daily intake rate by an animal to the contaminant concentration in an edible animal product (mg/kg meat per mg/d or pCi/kg meat per pCi/d; mg/L milk per mg/d or pCi/L milk per pCi/d),

f_w = Fraction of animal water that is contaminated (dimensionless),

IR_w = Intake rate of water by the animal (50 L/d for beef cattle; 60 L/d for milk cows),

λ_w = Environmental degradation and decay constant for contaminant in water (d^{-1}),

t_{hc} = Holding time between harvest or slaughter and consumption (20 days for meat; 4 days for milk).

As a conservative default, both types of animals are assumed to derive all of their drinking water from the contaminated source. Default values for chemical contaminants can be estimated from the octanol-water partition coefficients using expressions presented by Travis and Arms (1988).

For milk, the following expression is used:

$$\log (F_{milk}) = -8.10 + \log (K_{ow}), \qquad \textbf{[7.18]}$$

where F_{milk} = Contaminant transfer coefficient that relates daily intake rate by an animal to the contaminant concentration in milk (mg/L milk per mg/d or pCi/L milk per pCi/d),

K_{ow} = Octanol-water partition coefficient.

For meat, the transfer coefficient is estimated using the following expression:

$$\log (F_{meat}) = -7.6 + \log (K_{ow}), \qquad \textbf{[7.19]}$$

where F_{meat} = Contaminant transfer coefficient that relates daily intake rate by an animal to contaminant concentration in meat (mg/kg meat per mg/d or pCi/kg meat per pCi/d),

K_{ow} = Octanol-water partition coefficient.

The total contaminant concentration in the animal product (m) is the sum of the contributions from feed and water intake:

$$C_{TC} = C_{mw} + C_{mf}, \qquad [7.20]$$

where C_{TC} = Contaminant concentration in the animal product *m* (mg/kg or pCi/kg for meat; mg/L or pCi/L for milk),

C_{mw} = Contaminant concentration in animal product (m) from animal ingestion of water (mg/kg or pCi/kg for meat; mg/L or pCi/L for milk),

C_{mf} = Contaminant-specific, product-specific contaminant concentration from animal ingestion of contaminated feed (mg/kg or pCi/kg for meat; mg/L or pCi/L for milk).

The average individual dose (D_{ap}) is calculated from the animal product concentration (C_{TC}; equation 7.20) and the average consumption rate of the products:

$$D_{ap} = \sum_{m=1}^{n} IR_m * C_{TC} * D_g, \qquad [7.21]$$

where D_{ap} = Contaminant-specific average individual intake from ingestion of contaminated animal product *m* (mg/kg/d or pCi/d),

IR_m = Average daily consumption rate of animal product *m* (meat or milk) (0.26 kg/d for meat; 0.30 L/d for milk)(NRC 1977),

C_{TC} = Contaminant concentration in the animal product *m* (mg/kg or pCi/kg for meat; mg/L or pCi/L for milk),

D_g = Average amount of chemical ingested per day related to daily dose per unit body weight (kg^{-1}) or ingestion dose conversion factor (dimensionless).

Special equations, given in chapter 11, are used to estimate the tritium concentration in animal products and the average individual dose (D_{ap}) for tritium.

Beef cattle are assumed to be fed primarily on grain while milk cows are fed on grass. For air pathways, the concentration in animal drinking water is set to zero. Appendix A lists consumption values for animal product ingestion.

The following pages contain tables listing environmental transfer factors for organics and radionuclides. Bioaccumulation factors for fish and shellfish are listed in tables 7.2–1 and 7.2–2. Soil-to-plant transfer factors are given in tables 7.2–3 and 7.2–4. The animal product transfer factors (milk and meat) are listed in tables 7.2–5 and 7.2-6.

Table 7.2–1. Bioaccumulation Factors for Fish and Shellfish: Organics

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Acenaphthene	6.46E2	1.35E2
Acenaphthylene	3.01E2	7.66E1
Acetone	3.89E-1	4.57E-2
Acetonitrile	3.26E-1	3.78E-2
Acetophenone	1.1E1	1.7
Acrolein	3.44E2	7.26E-2
Acrylonitrile	4.8E1	1.15E-1
Acrylamide	1.8E-1	1.99E-2
Aldrin	3.14E3	1.57E3
Alk-Aryl Sulfonates	4.16E4	1.2E4
Alkoxy-me-choline	2.42E3	5.6E2
Allyl Alcohol	3.99E-1	4.7E-2
4-Aminobiphenol	7.46E1	1.35E1
Anthracene	1.42E3	3.15E2
PCBs	2.3E4	6.34E3
Aroclor 1016 (PCB)	1.7E4	2.65E3
Aroclor 1221 (PCB)	7.42E2	1.57E2
Aroclor 1232 (PCB)	1.60E2	2.99E1
Aroclor 1242 (PCB)	7.84E2	1.66E2
Aroclor 1248 (PCB)	1.4E4	3.73E3
Aroclor 1254 (PCB)	2.3E4	6.34E3
Aroclor 1260 (PCB)	1.6E5	5.12E4
Benzene	2.41E1	3.9
Benzidine	1.1E2	8.31E-1
Benzo(a)anthracene	1.17E4	2.76E3

Table 7.2-1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Benzo(a)pyrene	2.38E4	6.57E3
Benzo(b)fluoranthene	2.38E4	6.57E3
Benzo(k)fluoranthene	2.38E4	6.57E3
Benzoic Acid	1.55E1	2.43
Benzoquinone, P-	8.34E-1	1.04E-1
Bis(2-chloroethyl)	1.1E1	1.2
Bis(2-chloro-isopropyl)ether	2.32E1	3.75
Bis(2-ethylhexyl)phthalate	1.19E7	5.33E6
Bromodichloromethane	2.97E1	4.89
Bromomethane	4.73	6.74E-1
n-Butane	9.25E1	1.66E1
1-Butanol	2.19	2.94E-1
Chlordane	3.22E2	5.88E2
Chloraniline, P-	1.45E1	2.25
Chlorobenzene	6.45E2	1.51E1
Chlorobenzilate	1.58E3	3.53E2
Chloro-1,3-butadiene, 2-	1.33E1	2.05
Chlorodibromomethane	2.28E1	3.68
Chloroethane	7.19	1.06
Chloroform	1.85E1	2.93
Chloro-m-cresol, P-	1.34E2	2.47E1
Chloromethane(methy)	3.1	4.28E-1
Chloronaphthalene, 2-	6.46E2	1.35E2
Chlorophenol, 2-	6.4	4.35
Chloropropene(ally), 3-	2.47	3.36E-1
Chrysene	1.08E4	2.81E3
Cyclohexane	2.42E2	4.69E1
Cyclohexanone	2.43	3.29E-1
DDD	2.71E3	8.53E3
DDE	8.45E3	3.86E4
DDT	2.98E4	8.39E3
Diazinon	4.63E2	9.43E1
Dibenzol(a,h)anthracene	1.13E5	2.81E3
Dibromo-3-chloropropane, 1,2-	3.24E1	5.36
Dibromethane	4.98	7.13E-1

Table 7.2-1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Dichlorobenzene, 1,2-	8.9E1	6.37E1
Dichlorobenzene, 1,4-	6E1	6.34E1
Dichlorodifluoromethane	2.59E1	4.21
Dichloroethane, 1,1-	1.35E1	2.09
Dichloroethane, 1,2-	2	1.16
Dichloroethylene, 1,1-	1.47E1	2.3
Dichloroethylene, 1,2-	1.36	1.77E-1
Dichlorophenol, 2,4-	3.4E1	1.69E1
Dichlorophenol, 2,6-	3.4E1	2.39E-1
Dichloropropane, 1,2-	1.95E1	3.1
Dichlorobenzene, 1,3-	6.6E1	6.34E1
Dieldrin	4.87E3	4E2
Diethyl Phthalate	4.36E1	7.38
Dimethylbenzide	5.54	8E-1
Dimethyl Ether	7.02E-1	8.63E-2
Dimethylphenol, 2,4-	1.5E2	1.43E1
Dimethyl phthalate	5.7E1	1.35
Di-n-butyl Phthalate	1.07E4	2.77E3
Dinitro benzene, M-	1E1	1.52
Dinitro-o-cresol, 4,6-	6.64E1	1.16E1
Dinitrophenol, 2,4-	8.12	1.21
Dinitro toluene, 2,4-	1.95E1	3.1
Dimethyl Formamide	1.02E-1	1.08E-2
2,4-D	8.05E1	1.43E1
Dioctyl Adipate	4.93E5	1.73E5
Di-n-octylphthalate	1.87E7	8.66E6
Di-n-propyl nitrosamine	8.12	1.21
Dioxane, 1,4-	5.98E-1	7.26E-2
N-phenyl Benzenamine	2.69E2	5.25E1
Diphenlhydrazine, 1,2-	9.42E1	1.69E1
Disulfoton	6.7E2	1.4E2
Diuron	7.9E1	1.4E1
EDTA	6.63E-1	8.13E-2
Endosulfan I (Alpha)	2.94E2	5.78E1
Endosulfan II (Beta)	3.32E2	6.59E1

Table 7.2–1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Endrin	1.48E3	2.76E3
Ethane	1.4E1	2.17
Ethanol	3.42E-1	3.98E-2
Ethyl Acetate	2.11	2.83E-1
Ethyl benzene	1.46E2	2.71E1
Ethyl Ether	2.79	3.83E-1
Ethylene Glycol	2E-2	1.87E-3
Ethyl Methacrylate	1.76E1	2.77
Fluoranthene	3.12E3	7.36E2
Fluorene	7.13E2	1.96E2
Freon 113	1.9E2	3.61E1
Formaldehyde	1.09	1.38E-1
Fuel Oil #2	5.05E2	1.04E2
Furan	6.15	8.95E-1
Gasoline	2.41E1	3.9
Glyphosate	3.09E-3	2.49E-4
Beta-Hexachlorocyclohexane	5.42E2	1.12E2
Delta-Hexachlorocyclohexane	7.7E2	1.63E2
Gamma-Hexachlorocyclohexane	1.8E2	1.12E2
Heptachlor	1.3E3	2.87E2
Heptachlor Epoxide	6.73E1	1.18E1
Heptanes	1.29E3	2.85E2
Hexachlorobenzene	5.56E3	1.37E3
Hexachlorobutadiene	2.53E3	5.88E2
Hexachlorocyclopentadiene	2.9E1	1.35E2
Hexachlorodibenzo-p-dioxin	7.56E4	2.28E4
Hexachlorodibenzofurans	7.26E4	2.19E4
Hexachloroethane	1.84E3	4.18E2
Hexachlorophene	3.17E5	1.07E5
Hexanes	4.84E2	9.88E1
Hexanone, 2-	6.59	9.65E-1
Hydraulic Fluid	1.42E3	3.15E2
Hydrazine	5.36E-2	5.4E-3
Hydroxyquinoline	1.76E1	2.77
Ideno(1,2,3-cd)pyrene	5.13E4	1.5E4

Table 7.2–1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Isobutane	7.37E1	1.3E1
Isobutyl Alcohol	2.23	2.99E-1
Isopentane	1.72E2	3.24E1
Isophorone	3.07E1	7
Isopropyl Alcohol	6.42E-1	7.84E-2
Isosafrole	6.19E1	1.08E1
JP4 Jet Fuel	5.05E2	1.04E2
Kepone	1.95E1	3.1
Kerosene	5.05E2	1.04E2
Methanol	1.53E-1	1.67E-2
Methapyrilene	8.93E1	1.60E1
Methoxychlor	8.30E3	2.38E2
Methoxy-1-proponal, 2-	4.30E-1	5.09E-2
Methylacrlonitrile	1.52	1.98E-1
Methylcyclohexane	5.14E2	1.06E2
Methylcyclopentane	1.94E2	3.70E1
Methylene-Bis(2-chloroaniline), 4,4	5.81E2	1.20E2
Methylene Chloride	5.74	8.31E-1
Methyl Ethyl Ketone	9.51E-1	1.2E-1
Methyl naphthalene, 2-	5.05E2	1.04E2
Methyl-2-pentanone, 4-	2.08	2.78E-1
Methyl phenol, 2-	1.85E1	2.93
Methyl phenol, 4-	1.72E1	2.72
Methyl-2-propanethiol, 2-	1.58E1	2.48
Mineral Oil	1.42E3	3.15E2
Mineral Spirits	2.38E1	3.85
Motor Oil	1.42E3	3.15E2
Nonane	8.24E3	2.10E3
Naphthalene	1.68E2	3.16E1
Naphthopquinone, 1,4-	1.17E1	1.80
Naphthyl amine, 1-	2.20E1	3.53
Naphthyl amine, 2-	2.20E1	3.53
N-Dodecane	1.14E4	2.98E3
Nitrobenzene	1.50E1	2.34
Nitro-o-toluidine, 5-	1.82E1	2.88

Table 7.2-1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Nitrophenol, 4-	1.26E2	2.62
Nitrosdiethylamine, N-	1.36	1.77E-1
Nitrosdimethylamine, N-	8.50E-2	1.98E-2
Nitro-di-n-butylamine, N-	1.70E1	2.67
Nitrodosiphenylamine, N-	1.41E2	2.62E1
Nitrodosipropylamine, N-	7.99	1.19
Nitrosomorpholine, N-	2.73E-1	3.12E-2
Nitrosopoperidine, N-	2.50E-1	2.84E-2
Nitrosopyrrolidine, N-	9.21E-2	9.68E-3
No. 6 Fuel	5.05E2	1.04E2
Nitroaniline, O-	1.35E1	2.09
Nitroaniline, P-	6.70	9.81E-1
Octane	5.08E3	1.25E3
Orthene	1.32E-1	1.43E-2
Parathion	4.63E2	9.43E1
Pentachlorobenzene	3.40E3	1.27E3
Pentachloroethane	6.7E1	7.24E1
Pentachloronitrobenzene	8.17E3	2.08E3
Pentachlorophenol	3.72E3	8.89E2
Pentane, N-	2.22E2	4.26E1
Pentaerythrite Tetranitrate (PETN)	1.65	2.17E-1
Phenacetin	9.20	1.38
Phenanthrene	1.44E3	3.21E2
Phenol	7.57	1.12
Picoline	4.11	5.80E-1
Polyacrylamide	1.12E2	2.05E1
Polychlorinated Furans	7.59E4	2.30E4
Propane	3.66E1	6.12
Pyrene	2.80E3	1.63E3
Pyridine	1.87	2.48E-1
Resorcinol	2.39	3.23E-1
Rozol	8.74E3	2.23E3
Safrole	4.93E1	8.44
Styrene	1.03E2	1.86E1
Styrene-butadiene	7.37E2	1.56E2

Table 7.2-1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Tersan 1991	3.30E1	5.48
Tetrachlorobenzene, 1,2,4,5-	1.80E3	4.77E2
Tetrachloroethane, 1,1,1,2-	1.18E2	2.16E1
Tetrachlorethane, 1,1,2,2-	3.85E1	6.47
Tetra chlorophenol, 2,3,4,6-	7.70E2	1.63E2
Tetrachloroethylene	5.57E1	9.62
Tetraethyl Dithio Pyrophosphate	8.34E2	1.78E2
Tetrahydro Furan	1.32	1.70E-1
TNT	1.61E1	2.52
Toluene	6.99E1	1.23E1
Toluene Diisocyanate	3.30E2	6.55E1
Tordon 22K	3.64E1	6.08
Toxaphene	1.90E2	3.61E1
Tribromomethane	3.92E1	6.60
Tributyl Phosphate	6.34E2	1.32E2
Trichlorobenzene, 1,2,4-	1.09E3	2.38E2
Trichloroethane, 1,1,1-	9	7.97
Trichloroethane, 1,1,2-	3.90E1	7.53
Trichloroethylene	3.79E1	6.36
Trichloroheptafluorobutane	4.24E2	8.57E1
Trichloromethanethiol	4.93E1	8.44
Trichloromonofluormethane	4.93E1	8.44
Trichlorophenol, 2,4,5-	1.90E3	7.96E1
Trichlorophenol, 2,4,6-	2E1	1.06E2
Trichloropropane, 1,2,3-	1.75E3	3.96E2
Triethanolamine	2.76E-2	2.64E-3
Triethylene Glycol	1.55E-2	1.41E-3
Trimethylbenzene	2.34E2	4.52E1
Trimethylphenol, 2,4,6-	2.34E2	4.52E1
Tris(2,3-dibromopropyl) Phosphate	2.7	1.69E2
Velsicol	1.14E2	2.08E1
Vaponite	2.04	2.72E-1
Vinyl Acetate	1.34	1.73E-1
Vinyl chloride	6.59	9.65E-1
Xylene (mixed)	1.77E2	3.34E1

Table 7.2–1. Bioaccumulation Factors for Fish and Shellfish: Organics (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Xylene, M-	1.77E2	3.34E1
Xylene, O-	1.03E2	1.86E1
Xylene, P-	1.46E2	2.71E1

Source: Strenge and Peterson 1989. These parameters are used in equations 7.3 through 7.5.

Table 7.2–2. Bioaccumulation Factors for Fish and Shellfish: Radionuclides

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Actinium-225	2.5E1	1E3
Actinium-227	2.5E1	1E3
Americium-241	2.5E1	1E3
Americium-242	2.5E1	1E3
Americium-242M	2.5E1	1E3
Americium-243	2.5E1	1E3
Antimony-125	1	1E3
Beryllium-7	2	1E1
Carbon-14	4.6E3	9.1E3
Cerium-144	1	1E3
Cesium-134	2E3	1E2
Cesium-135	2E3	1E2
Cesium-137+D	2E3	1E2
Cobalt-57	5E1	2E2
Cobalt-60	5E1	2E2
Curium-242	2.5E1	1E3
Curium-244	2.5E1	1E3
Curium-245	2.5E1	1E3
Europium-152	2.5E1	1E3
Europium-154	2.5E1	1E3
Europium-155	2.5E1	1E3
Iodine-129	1.5E1	5
Iodine-131	1.5E1	5

Table 7.2–2. Bioaccumulation Factors for Fish and Shellfish: Radionuclides (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Iodine-135	4E2	7.5E1
Lead-210	1E2	1E2
Lead-212	1E2	1E2
Manganese-54	4E2	9E4
Neptunium-237	1E1	4E2
Nickel-56	1E2	1E2
Nickel-63	1E2	1E2
Niobium-93m	3E4	1E2
Niobium-95	3E4	1E2
Phosphorous-32	1E5	2E4
Palladium-107	1E1	3E1
Plutonium-238	3.5	1E2
Plutonium-239	3.5	1E2
Plutonium-240	3.5	1E2
Plutonium-241	3.5	1E2
Polonium-210	5E2	2E4
Potassium-40	1E3	8.3E2
Protactinium-231	1.1E1	1.1E2
Protactinium-233	1.1E1	1.1E2
Radium-223	5E1	2.5E2
Radium-225	5E1	2.5E2
Radium-226	5E1	2.5E2
Radon-220+D	1E2	1E2
Radon-222+D	1E2	1E2
Ruthenium-103	1E1	3E2
Ruthenium-106	1E1	3E2
Samarium-151	2.5E1	1E3
Selenium-79	1.7E2	1.7E2
Sodium-22	1E2	2E2
Sulphur-35	7.5E2	1E2
Strontium-89	3E1	1E2
Strontium-90	3E1	1E2
Technetium-99	1.5E1	5
Thorium-227	3E1	5E2
Thorium-228	3E1	5E2

Table 7.2–2. Bioaccumulation Factors for Fish and Shellfish: Radionuclides (continued)

Contaminant	Bioaccumulation Factor (B_f)	
	Fish	Shellfish
Thorium-229	3E1	5E2
Thorium-230	3E1	5E2
Thorium-232	3E1	5E2
Thorium-234	3E1	5E2
Tin-126	3E3	1E3
Tritium	1	1
Elemental Tritium	4	3.8E3
Uranium-233	2	6E1
Uranium-234	2	6E1
Uranium-235	2	6E1
Uranium-236	2	6E1
Uranium-238	2	6E1
Uranium-239	2	6E1
Yttrium-90	2.5E1	1E3
Zinc-65	2E3	1E5
Zirconium-93	3.3	6.7

Source: Strenge and Peterson 1989. These parameters are used in equations 7.3 through 7.5.

Table 7.2–3. Soil-to-Plant Transfer Factors: Organics

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Acenaphthene	4.72E-2
Acenaphthylene	7.04E-2
Acetone	1.33E1
Acetonitrile	1.52E1
Acetophenone	1.03
Acrolein	9.57
Acrylamide	6.94
Acrylonitrile	2.39E-1
Aldrin	8.36E-3
Alk-Aryl Sulfonates	1.99E-3

Table 7.2–3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Alkoxy-me-choline	1.73E-2
Allyl Alcohol	1.30E1
4-Aminobiphenol	2.39E-1
Aniline	2.77
Anthracene	2.59E-2
PCBs	3.12E-3
Aroclor 1016 (PCB)	5.77E-3
Aroclor 1221 (PCB)	4.25E-2
Aroclor 1232 (PCB)	1.37E-1
Aroclor 1242 (PCB)	4.07E-2
Aroclor 1248 (PCB)	4.54E-3
Aroclor 1254 (PCB)	3.12E-3
Aroclor 1260 (PCB)	7.14E-4
Benzene	5.76E-1
Benzidine	1.71
Benzo(a)anthracene	5.61E-3
Benzo(a)pyrene	3.04E-3
Benzo(b)fluoranthene	3.04E-3
Benzo(k)fluoranthene	3.04E-3
Benzoic Acid	8.04E-1
P-Benzoquinone	7.43
Bis(2-chloroethyl)	1.32
Bis(2-chloro-isopropyl)ether	5.92E-1
Bis(2-ethylhexyl)phthalate	2.69E-5
Bromodichloromethane	4.91E-1
Bromomethane	1.99
n-Butane	2.07E-1
1-Butanol	3.57
Chlordane	1.67E-2
P-Chloraniline	8.48E-1
Chlorobenzene	2.21E-1
Chlorobenzilate	2.39E-2
Chloro-1,3-butadiene, 2-	9.06E-1
Chlorodibromomethane	6.00E-1
Chloroethane	1.44
Chloroform	7.04E-1

Table 7.2-3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
P-chloro-m-cresol	1.56E-1
Chloromethane(methy)	2.74
Chloronaphthalene, 2-	4.72E-2
Chlorophenol, 2-	5.33E-1
Chloropropene, 3-	3.25
Chrysene	5.54E-3
Cyclohexane	9.96E-2
Cyclohexanone	3.29
DDD	2.53E-3
DDE	8.71E-4
DDT	2.56E-3
Diazinon	6.08E-2
Dibenzol(a,h)anthracene	5.54E-3
Dibromo-3-chloropropane, 1,2-	4.60E-1
Dibromethane	1.91
Dichlorobenzene, 1,2-	8.02E-2
Dichlorobenzene, 1,4-	8.04E-2
Dichlorodifluoromethane	5.45E-1
Dichloroethane, 1,1-	8.94E-1
Dichloroethane, 1,2-	1.35
Dichloroethylene, 1,1-	8.36E-1
Dichloroethylene, 1,2-	5.11
Dichlorophenol, 2,4-	2.04E-1
Dichlorophenol, 2,6-	4.13
Dichloropropane, 1,2-	6.76E-1
Dichloropropene, 1,3-	6.76E-1
Dichlorobenzene, 1,3-	8.04E-2
Dieldrin	9.19E-2
Diethyl Phthalate	3.67E-1
Dimethylbenzide, 3,3-	6.11E-1
Dimethyl Ether	8.47
Dimethylphenol, 2,4-	2.30E-1
Dimethylphthalate	1.21
Di-n-butyl phthalate	5.60E-3
Dinitrobenzene, M-	1.12
Dinitro-o-cresol, 4,6-	2.66E-1

Table 7.2–3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Dinitrophenol, 2,4-	1.32
Dinitro toluene, 2,4-	6.76E-1
Dimethyl Form amide	3.66E1
2,4-D	2.30E-1
Dioctyl Adipate	3.03E-4
Di-n-octylphthalate	1.91E-5
Di-n-propyl nitrosamine	1.32
Dioxane, 1,4-	9.57
N-Phenyl Benzenamine	9.19E-2
Diphenlhydrazine, 1,2-	2.04E-1
Disulfoton	4.59E-2
Diuron	2.33E-1
EDTA	8.84
Endosulfan	8.59E-2
Endrin	5.61E-3
Ethane	8.70E-1
Ethanol	1.46E1
Ethyl Acetate	3.67
Ethyl benzene	1.46E-1
Ethyl Ether	2.96
Ethylene Glycol	1.27E2
Ethyl Methacrylate	7.32E-1
Fluoranthene	1.43E-2
Fluorene	3.62E-2
Freon 113	1.20E-1
Formaldehyde	6.08
Fuel Oil #2	5.69E-2
Furan	1.63
Gasoline	5.76E-1
Glyphosate	5.25E2
Beta-Hexachlorocyclohexane	5.39E-2
Delta-Hexachlorocyclohexane	4.13E-2
Gamma-Hexachlorocyclohexane	5.39E-2
Heptachlor	2.77E-2
Heptachlor Epoxide	2.64E-1
Heptanes	2.79E-2

Table 7.2–3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Hexachlorobenzene	9.18E-3
Hexachlorobutadiene	1.67E-2
Hexachlorocyclopentadiene	5.39E-2
Hexachlorodibenzo-p-dioxin	4.72E-2
Hexachlorodibenzofurans	1.30E-3
Hexachloroethane	2.12E-2
Hexachlorophene	4.24E-4
Hexanes	5.88E-2
Hexanone, 2-	1.54
Hydraulic Fluid	2.59E-2
Hydrazine	5.99E1
Hydroxyquinoline	7.32E-1
Ideno(1,2,3-cd)pyrene	1.70E-3
Isobutane	2.46E-1
Isobutyl Alcohol	3.52
Isopentane	1.29E-1
Isophorone	4.78E-1
Isopropyl Alcohol	9.07
Isosafrole	2.81E-1
JP4 Jet Fuel	4.00E-2
Kepone	6.76E-1
Kerosene	5.69E-2
Methanol	2.70E1
Methapyrilene	2.12E-1
Methoxychlor	3.16E-2
Methoxy-1-proponal, 2-	1.23E1
Methylacrlonitrile	4.72
Methylcyclohexane	5.61E-2
Methylcyclopentane	1.18E-1
Methylene-Bis(2-chloroaniline), 4,4	5.11E-2
Methylene Chloride	1.71
Methylene Dithiocyanate	1.14E1
Methyl Ethyl Ketone	6.72
Methyl naphthalene, 2-	5.69E-2
Methyl-2-pentanone, 4-	3.71
Methyl phenol, 2-	7.04E-1

Table 7.2-3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Methyl phenol, 4-	7.42E-1
Methyl-2-propanethiol, 2-	7.93E-1
Mineral Oil	2.59E-2
Mineral Spirits	5.81E-1
Motor Oil	2.59E-2
Nonane	6.81E-3
Naphthalene	1.31E-1
Naphthopquinone, 1,4-	9.94E-1
Naphthyl amine, 1-	6.17E-1
Naphthyl amine, 2-	6.17E-1
N-Dodecane	5.32E-3
Nitrobenzene	8.25E-1
Nitro-o-toluidine, 5-	7.13E-1
Nitrophenol, 4-	7.62E-1
Nitrosdiethylamine, N-	5.11
Nitrosdimethylamine, N-	2.39E1
Nitro-di-n-butylamine,N-	7.52E-1
Nitrodosiphenylamine, N-	1.50E-1
Nitrodosipropylamine, N-	1.33
Nitrosomorpholine, N-	1.74E1
Nitrosopoperidine, N-	1.86E1
Nitrosopyrrolidine, N-	3.97E1
No. 6 Fuel	5.69E-2
Nitroaniline, O-	8.94E-1
Nitroaniline, P-	1.52
Octane	9.83E-3
Orthene	3.02E1
Parathion	6.08E-2
Pentachlorobenzene	9.68E-3
Pentachloroethane	7.32E-2
Pentachloronitrobenzene	6.85E-3
Pentachlorophenol	1.25E-2
Pentane, N-	1.06E-1
Pentaerythrite Tetranitrate (PETN)	4.42
Phenacetin	1.20
Phenanthrene	2.56E-2

Table 7.2-3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Phenol	1.39
Picoline	2.21
Polyacrylamide	1.79E-1
Polychlorinated Furans	1.26E-3
Propane	4.19E-1
Pyrene	8.15E-3
Pyridine	4.02
Resorcinol	3.34
Rozol	6.51E-3
Safrole	3.34E-1
Styrene	1.91E-1
Styrene-Butadiene	4.27E-2
Tersan 1991	4.53E-1
Tetrachlorobenzene, 1,2,4,5-	1.93E-2
Tetrachloroethane, 1,1,1,2-	1.72E-1
Tetrachlorethane, 1,1,2,2-	4.03E-1
Tetra chlorophenol, 2,3,4,6-	4.13E-2

Table 7.2–3. Soil-to-Plant Transfer Factors: Organics (continued)

Contaminant	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Tetrachloroethylene	3.04E-1
Tetraethyl Dithio Pyrophosphate	3.89E-2
Tetra hydro Furan	5.25
Trinitrotoluene	7.83E-1
Toluene	2.56E-1
Toluene Diisocyanate	7.86E-2
Tordon 22K	4.21E-1
Toxaphene	1.20E-1
Tribromomethane	3.97E-1
Tributyl Phosphate	4.78E-2
Trichlorobenzene, 1,2,4-	3.16E-2
Trichloroethane, 1,1,1-	3.48E-1
Trichloroethane, 1,1,2-	3.62E-1
Trichloroethylene	4.08E-1
Trichloroheptafluorobutane	6.50E-2
Trichloromethanethiol	3.34E-1
Trichloromonofluormethane	3.34E-1
Trichlorophenol, 2,4,5-	6.85E-2
Trichlorophenol, 2,4,6-	5.61E-2
Trichloropropane, 1,2,3-	2.21E-2
Triethanolamine	9.94E1
Triethylene Glycol	1.54E2
Trimethylbenzene	1.02E-1
Trimethylphenol, 2,4,6-	1.02E-1
Tris(2,3-dibromopropyl)phosphate	4.02E-2
Velsicol R or MDBA	1.77E-1
Vaponite	3.77
Vinyl Acetate	5.18
Vinyl Chloride	1.54
Xylene (mixed)	1.26E-1
Xylene, M-	1.26E-1
Xylene, O-	1.91E-1
Xylene, P-	1.46E-1

Source: Strenge and Peterson 1989. These parameters are used in equations 7.9 through 7.12 and 7.14.

Table 7.2–4. Soil-to-Plant Transfer Factors: Radionuclides

Radionuclides	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Actinium-225	2.5E-3
Actinium-227	2.5E-3
Americium-241	2.5E-4
Americium-242	2.5E-4
Americium-242M	2.5E-4
Americium-243	2.5E-4
Antimony-125	1.1E-2
Beryllium-7	4.7E-4
Cerium-144	5E-4
Cesium-134	2E-3
Cesium-135	2E-3
Cesium-137+D	2E-3
Cobalt-57	9.4E-3
Cobalt-60	9.4E-3
Curium-242	2.5E-3
Curium-243	2.5E-3
Curium-244	2.5E-3
Curium-245	2.5E-3
Europium-152	2.5E-3
Europium-154	2.5E-3
Europium-155	2.5E-3
Iodine-129	2E-2
Iodine-131	2E-2
Iodine-135	2E-2
Lead-210	6.8E-2
Lead-212	6.8E-2
Manganese-54	3E-2
Neptunium-237	2.5E-3
Nickel-56	1.9E-2
Nickel-63	1.9E-2
Niobium-93m	9.4E-3
Niobium-95	9.4E-3
Phosphorous-32	5E-1
Palladium-107	5
Plutonium-238	2.5E-4
Plutonium-239	2.5E-4

Table 7.2–4. Soil-to-Plant Transfer Factors: Radionuclides (continued)

Radionuclides	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Plutonium-240	2.5E-4
Plutonium-241	2.5E-4
Polonium-210	9E-3
Potassium-40	3.7E-1
Protactinium-231	2.5E-3
Protactinium-233	2.5E-3
Radium-223	1.4E-3
Radium-225	1.4E-3
Radium-226	1.4E-3
Radon-220+D	6.8E-2
Radon-222+D	6.8E-2
Ruthenium-103	1E-2
Ruthenium-106	1E-2
Samarium-151	2.5E-3
Selenium-79	1.3
Sodium-22	5E-2
Sulphur-35	5.9E-1
Strontium-89	2E-1
Strontium-90	2E-1
Technetium-99	2.5E-1
Thorium-227	4.2E-3
Thorium-228	4.2E-3
Thorium-229	4.2E-3
Thorium-230	4.2E-3
Thorium-232	4.2E-3
Thorium-234	4.2E-3
Tin-126	2.5E-3
Elemental Tritium	5.3
Uranium-233	2.5E-3
Uranium-234	2.5E-3
Uranium-235	2.5E-3
Uranium-236	2.5E-3
Uranium-238	2.5E-3
Uranium-239	2.5E-3
Yttrium-90	2.5E-3

Table 7.2–4. Soil-to-Plant Transfer Factors: Radionuclides (continued)

Radionuclides	Soil-to-Plant Transfer Factor (B_v) (pCi/kg wet plant per pCi/kg soil)
Zinc-65	4E-1
Zirconium-93	1.7E-4

Source: Strenge and Peterson 1989. These parameters are used in equations 7.9 through 7.12 and 7.14.

Table 7.2–5. Animal Product Transfer Factors: Organics

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Acenaphthene	2.51E-4	7.94E-5
Acenaphthylene	1.26E-4	3.98E-5
Acetone	1.46E-8	4.61E-9
Acetonitrile	1.16E-8	3.65E-9
Acetophenone	1.20E-6	3.80E-7
Acrolein	2.56E-8	8.10E-9
Acrylamide	4.47E-8	1.41E-8
Acrylonitrile	5.27E-9	1.67E-9
Aldrin	8.51E-2	2.40E-2
Alk-Aryl Sulfonates	6.03E-2	1.91E-2
Alkoxy-me-choline	1.43E-3	4.52E-4
Allyl Alcohol	1.51E-8	4.77E-9
4-Aminobiphenol	1.51E-5	4.79E-6
Aniline	2.19E-7	6.92E-8
Anthracene	7.08E-4	2.24E-4
PCBs	2.76E-2	8.74E-3
Aroclor 1016 (PCB)	9.55E-3	3.02E-3
Aroclor 1221 (PCB)	3.01E-4	9.53E-5
Aroclor 1232 (PCB)	3.99E-5	1.26E-5
Aroclor 1242 (PCB)	3.24E-4	1.02E-4
Aroclor 1248 (PCB)	1.44E-2	4.57E-3
Aroclor 1254 (PCB)	5.25E-2	1.12E-2
Aroclor 1260 (PCB)	3.54E-1	1.12E-1
Benzene	3.32E-6	1.05E-6

Table 7.2-5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Benzidine	5.02E-7	1.59E-7
Benzo(a)anthracene	1.00E-2	3.16E-3
Benzo(a)pyrene	2.89E-2	9.13E-3
Benzo(b)fluoranthene	2.89E-2	9.13E-3
Benzo(k)fluoranthene	2.89E-2	9.13E-3
Benzoic Acid	1.86E-6	5.89E-7
P-Benzoquinone	3.97E-8	1.26E-8
Bis(2-chloroethyl)	7.94E-7	2.51E-7
Bis(2-chloro-isopropyl)ether	3.16E-6	1.00E-6
Bis(2-ethylhexyl)phthalate	1.03E2	3.26E1
Bromodichloromethane	4.37E-6	1.38E-6
Bromomethane	3.89E-7	1.23E-7
n-Butane	1.95E-5	6.16E-6
1-Butanol	1.41E-7	4.46E-8
Chlordane	7.41E-3	3.72E-4
Chloroaniline, P-	1.70E-6	5.37E-7
Chlorobenzene	1.74E-5	5.50E-6
Chlorobenzilate	8.14E-4	2.57E-4
Chloro-1,3-butadiene, 2-	1.51E-6	4.79E-7
Chlorodibromomethane	3.09E-6	9.77E-7
Chloroethane	6.76E-7	2.14E-7
Chloroform	2.34E-6	7.41E-7
P-Chloro-m-cresol	3.16E-5	1.00E-5
Chloromethane(methy)	2.24E-7	7.08E-8
Chloronaphthalene, 2-	2.51E-4	7.94E-5
Chlorophenol, 2-	3.79E-6	1.20E-6
Chloropropene, 3-	1.66E-7	5.25E-8
Chrysene	1.02E-2	3.23E-3
Cyclohexane	6.91E-5	2.18E-5
Cyclohexanone	1.62E-7	5.13E-8
DDD	1.26E-2	3.02E-3
DDE	4.90E-2	9.55E-3
DDT	2.82E-2	1.07E-2
Diazinon	1.62E-4	5.13E-5

Table 7.2-5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Dibenzol(a,h)anthracene	1.02E-2	3.23E-3
Dibromo-3-chloropropane, 1,2-	4.90E-6	1.55E-6
Dibromethane	4.17E-7	1.32E-7
Dichlorobenzene, 1,2-	1.00E-4	3.18E-5
Dichlorobenzene, 1,4-	1.00E-4	3.16E-5
Dichlorodifluoromethane	3.64E-6	1.15E-6
Dichloroethane, 1,1-	1.55E-6	4.90E-7
Dichloroethane, 1,2-	7.59E-7	2.40E-7
Dichloroethylene, 1,1-	1.74E-6	5.50E-7
Dichloroethylene, 1,2-	7.59E-8	2.40E-8
Dichlorophenol, 2,4-	1.99E-5	6.31E-6
Dichlorophenol, 2,6-	1.10E-7	3.47E-8
Dichloropropane, 1,2-	2.51E-6	7.94E-7
Dichloropropene, 1,3-	2.51E-6	7.94E-7
Dichlorobenzene, 1,3-	1.00E-4	3.16E-5
Dieldrin	7.94E-3	1.07E-2
Diethyl Phthalate	1.82E-4	5.75E-5
Dimethylbenzide, 3,3-	2.99E-6	9.45E-7
Dimethyl Ether	3.16E-8	1.00E-8
Dimethyl phenol, 2,4-	1.62E-5	5.13E-6
Dimethylphthalate	9.12E-7	2.88E-7
Di-n-butyl Phthalate	1.00E-2	3.18E-3
Dinitro benzene, M-	1.05E-6	3.31E-7
Dinitro-o-cresol, 4,6-	1.26E-5	3.98E-7
Dinitrophenol, 2,4-	7.94E-7	2.51E-7
Dinitro toluene, 2,4-	2.51E-6	7.94E-7
Dimethyl Formamide	2.51E-9	7.94E-10
2,4-D	4.79E-6	7.59E-6
Dioctyl Adipate	1.56	4.79E-1
Di-n-octylphthalate	1.86E2	5.89E1
Di-n-propyl nitrosamine	7.94E-7	2.51E-7
Dioxane, 1,4-	2.56E-8	8.10E-9
N-Phenyl Benzenamine	7.94E-5	2.51E-5
Diphenlhydrazine, 1,2-	1.99E-5	6.31E-6
Disulfoton	2.64E-4	8.34E-5

Table 7.2–5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Diuron	1.58E-5	5.00E-6
EDTA	2.94E-8	9.29E-9
Endosulfan	2.19E-4	2.82E-5
Endrin	1.20E-2	1.74E-3
Ethane	1.62E-6	5.13E-7
Ethanol	1.23E-8	3.89E-9
Ethyl Acetate	1.35E-7	4.27E-8
Ethyl benzene	3.54E-5	1.12E-5
Ethyl Ether	1.95E-7	6.16E-8
Ethylene Glycol	2.94E-10	9.29E-11
Ethyl Methacrylate	2.19E-6	6.92E-7
Fluoranthene	1.99E-3	6.31E-4
Fluorene	3.97E-4	1.26E-4
Freon 113	5.02E-5	1.59E-5
Formaldehyde	5.63E-8	1.78E-8
Fuel Oil #2	1.82E-4	5.75E-5
Furan	5.50E-7	1.74E-7
Gasoline	3.32E-6	1.05E-6
Glyphosate	2.51E-11	7.94E-12
Beta-Hexachlorocyclohexane	1.99E-4	6.31E-5
Delta-Hexachlorocyclohexane	1.66E-2	2.51E-3
Gamma-Hexachlorocyclohexane	1.66E-2	2.51E-3
Heptachlor	1.55E-2	3.24E-3
Heptachlor Epoxide	7.94E-2	3.55E-2
Heptanes	6.25E-4	1.98E-4
Hexachlorobenzene	4.47E-2	8.51E-3
Hexachlorobutadiene	1.51E-3	4.79E-4
Hexachlorocyclopentadiene	2.51E-4	7.94E-5
Hexachlorodibenzo-p-dioxin	1.32E-1	4.17E-2
Hexachlorodibenzofurans	1.26E-1	3.97E-2
Hexachloroethane	1.00E-3	3.16E-4
Hexachlorophene	8.72E-1	2.76E-1
Hexanes	1.72E-4	5.43E-5
Hexanone, 2-	6.03E-7	1.91E-7
Hydraulic Fluid	9.80E-8	3.10E-8

Table 7.2–5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Hydrazine	1.07E-9	3.39E-10
Hydroxyquinoline	2.19E-6	6.92E-7
Ideno(1,2,3-cd)pyrene	7.94E-2	2.51E-2
Isobutane	1.44E-5	4.57E-6
Isobutyl Alcohol	1.44E-7	4.57E-8
Isopentane	4.40E-5	1.39E-5
Isophorone	4.57E-6	1.45E-6
Isopropyl Alcohol	2.81E-8	8.90E-9
Isosafrole	1.15E-5	3.63E-6
JP4 Jet Fuel	1.82E-4	5.75E-5
Kepone	2.51E-6	7.94E-7
Kerosene	1.82E-4	5.75E-5
Methanol	4.27E-9	1.35E-9
Methapyrilene	1.86E-5	5.89E-6
Methoxychlor	5.02E-4	1.48E-4
Methyxy-1-proponal, 2-	1.66E-8	5.25E-9
Methylacrlonitrile	8.72E-8	2.76E-8
Methylcyclohexane	1.86E-4	5.89E-5
Methylcyclopentane	5.17E-5	1.64E-5
Methylene-Bis(2-chloroaniline), 4,4	2.19E-4	6.92E-5
Methylene Chloride	5.02E-7	1.59E-7
Methylene Dithiocyanate	1.88E-8	5.96E-9
Methyl Ethyl Ketone	4.72E-8	1.49E-8
Methyl naphthalene, 2-	1.82E-4	5.75E-5
Methyl-2-Pentanone, 4-	1.32E-7	4.17E-8
Methyl phenol, 2-	2.34E-6	7.41E-7
Methyl phenol, 4-	2.14E-6	6.76E-7
Methyl-2-propanethiol, 2-	1.91E-6	6.03E-7
Mineral Oil	7.08E-4	2.24E-4
Mineral Spirits	3.27E-6	1.03E-6
Motor Oil	7.08E-4	2.24E-4
Nonane	7.16E-3	2.26E-3
Naphthalene	4.27E-5	2.00E-4
Naphthopquinone, 1,4-	1.29E-6	4.07E-7
Naphthyl amine, 1-	2.94E-6	9.29E-7

Table 7.2–5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Naphthyl amine, 2-	2.94E-6	9.29E-7
N-Dodecane	1.10E-2	3.47E-3
Nitrobenzene	1.78E-6	5.62E-7
Nitro-o-toluidine, 5-	2.29E-6	7.24E-7
Nitrophenol, 4-	2.04E-6	6.46E-7
Nitrosdiethylamine, N-	7.59E-8	2.40E-8
Nitrosdimethylamine, N-	5.25E-9	1.66E-9
Nitro-di-n-butylamine, N-	2.09E-6	6.61E-7
Nitrodosiphenylamine, N-	3.39E-5	1.07E-5
Nitrodosiporpylamine, N-	7.76E-7	2.45E-7
Nitrosomorpholine, N-	9.12E-9	2.88E-9
Nitrosopoperidine, N-	8.14E-9	2.57E-9
Nitrosopyrrolidine, N-	2.19E-9	6.92E-10
No. 6 Fuel	1.82E-4	5.75E-5
Nitroaniline, O-	1.55E-6	4.90E-7
Nitroanilne, P-	6.15E-7	1.95E-7
Octane	3.79E-3	1.20E-3
Orthene	3.52E-9	1.11E-9
Parathion	1.62E-4	5.13E-5
Pentachlorobenzene	3.89E-3	1.23E-3
Pentachloroethane	1.18E-4	3.72E-5
Pentachloronitrobenzene	7.08E-3	2.24E-3
Pentachlorophenol	2.51E-3	7.94E-4
Pentane, N-	6.15E-5	1.95E-5
Pentaerythrite Tetranitrate (PETN)	9.77E-8	3.09E-8
Phenacetin	9.34E-4	2.95E-4
Phenanthrene	9.34E-7	2.95E-7
Phenol	7.23E-7	2.29E-7
Picoline	3.24E-7	1.02E-7
Polyacrylamide	2.51E-5	7.94E-6
Polychlorinated Furans	1.33E-1	4.21E-2
Propane	5.75E-6	1.82E-6
Pyrene	5.25E-3	1.66E-3
Pyridine	1.15E-7	3.63E-8
Resorcinol	1.59E-7	5.01E-8

Table 7.2-5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Rozol	7.74E-3	2.45E-3
Safrole	8.52E-6	2.69E-6
Styrene	2.24E-5	7.08E-6
Styrene-Butadiene	2.99E-4	9.45E-5
Tersan 1991	5.02E-6	1.59E-6
Tetrachlorobenzene, 1,2,4,5-	1.18E-3	3.72E-4
Tetrachloroethane, 1,1,1,2-	6.15E-6	1.95E-6
Tetrachlorethane, 1,1,2,2-	6.15E-6	1.95E-6
Tetra chlorophenol, 2,3,4,6-	3.16E-4	1.00E-4
Tetrachloroethylene	1.00E-5	3.16E-6
Tetraethyl Dithio Pyrophosphate	3.52E-4	1.11E-4
Tetra hydro Furan	7.23E=8	2.29E-8
Trinitrotoluene	1.95E-6	6.16E-7
Toluene	1.35E-5	4.27E-6
Toluene Diisocyanate	1.04E-4	3.29E-5
Tordon 22K	5.70E-6	1.80E-6
Toxaphene	1.62E-3	6.31E-4
Tribromomethane	6.30E-6	1.99E-6
Tributyl Phosphate	2.45E-4	7.76E-5
Trichlorobenzene, 1,2,4-	5.02E-4	1.59E-4
Trichloroethane, 1,1,1-	7.94E-6	2.51E-6
Trichloroethane, 1,1,2-	7.41E-6	2.34E-6
Trichloroethylene	6.03E-6	1.91E-6
Trichloroheptafluorobutane	1.44E-4	4.57E-5
Trichloromethanethiol	8.52E-6	2.69E-6
Trichloromonofluormethane	8.52E-6	2.69E-6
Trichlorophenol, 2,4,5-	1.32E-4	4.17E-5
Trichlorophenol, 2,4,6-	1.86E-4	5.89E-5
Trichloropropane, 1,2,3-	9.34E-4	2.95E-4
Triethanolamine	4.47E-10	1.41E-10
Triethylene Glycol	2.09E-10	6.61E-11
Trimethylbenzene	6.61E-5	2.09E-5
Trimethylphenol, 2,4,6-	6.61E-5	2.09E-5
Tris(2,3-dibromopropyl)phosphate	3.32E-4	1.05E-4
Velsicol R or MDBA	2.56E-5	8.10E-6

Table 7.2–5. Animal Product Transfer Factors: Organics (continued)

Contaminant	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Vaponite	1.29E-7	4.07E-8
Vinyl Acetate	7.41E-8	2.34E-8
Vinyl Chloride	6.03E-7	1.91E-7
Xylene (mixed)	4.57E-5	1.45E-5
Xylene, M-	4.57E-5	1.45E-5
Xylene, O-	2.24E-5	7.08E-6
Xylene, P-	5.00E-2	6.00E-3

Source: Strenge and Peterson 1989. These parameters are used in equations 7.16 through 7.19.

Table 7.2–6. Animal Product Transfer Factors: Radionuclides

Element	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Actinium-225	5E-3	2.5E-6
Actinium-227	5E-3	2.5E-6
Americium-241	5E-3	2.5E-6
Americium-242	5E-3	2.5E-6
Americium-242M	5E-3	2.5E-6
Americium-243	5E-3	2.5E-6
Antimony-125	3E-3	7.5E-4
Beryllium-7	8E-4	2E-6
Cerium-144	1E-3	1E-5
Cesium-134	3E-2	5E-3
Cesium-135	3E-2	5E-3
Cesium-137+D	3E-2	5E-3
Cobalt-57	1E-3	5E-4
Cobalt-60	1E-3	5E-4
Curium-242	5E-3	2.5E-6
Curium-243	5E-3	2.5E-6
Curium-244	5E-3	2.5E-6
Curium-245	5E-3	2.5E-6
Europium-152	5E-3	2.5E-6
Europium-154	5E-3	2.5E-6

Table 7.2–6. Animal Product Transfer Factors: Radionuclides (continued)

Element	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Europium-155	5E-3	2.5E-6
Iodine-129	2E-2	1E-2
Iodine-131	2E-2	1E-2
Iodine-135	2E-2	1E-2
Lead-210	2.9E-4	1E-5
Lead-212	2.9E-4	1E-5
Manganese-54	5E-3	1E-4
Neptunium-237	5E-3	2.5E-6
Nickel-56	1E-3	3.4E-3
Nickel-63	1E-3	3.4E-3
Niobium-93m	5E-4	1.2E-3
Niobium-95	5E-4	1.2E-3
Phosphorous-32	5E-2	1.2E-2
Palladium-107	1E-3	5E-3
Plutonium-238	5E-3	2.5E-8
Plutonium-239	5E-3	2.5E-8
Plutonium-240	5E-3	2.5E-8
Plutonium-241	5E-3	2.5E-8
Polonium-210	1.2E-2	1.2E-4
Potassium-40	1.2E-2	1E-2
Protactinium-231	5E-3	2.5E-6
Protactinium-233	5E-3	2.5E-6
Radium-223	3.4E-2	2E-4
Radium-225	3.4E-2	2E-4
Radon-220+D	2.9E-4	1E-5
Radon-222+D	2.9E-4	1E-5
Ruthenium-103	1E-3	5E-7
Ruthenium-106	1E-3	5E-7
Samarium-151	5E-3	2.5E-6
Selenium-79	1	2.3E-2
Sodium-22	5E-2	4E-2
Sulphur-35	1E-1	1.8E-2
Strontium-89	3E-4	1.5E-3
Strontium-90	3E-4	1.5E-3
Technetium-99	4E-1	1E-2
Thorium-227	5E-3	2.5E-6

Table 7.2-6. Animal Product Transfer Factors: Radionuclides (continued)

Element	Transfer factors (F_m)	
	Beef (d/kg)	Milk (d/L)
Thorium-228	5E-3	2.5E-6
Thorium-230	5E-3	2.5E-6
Thorium-232	5E-3	2.5E-6
Thorium-234	5E-3	2.5E-6
Tin-126	8E-2	1.3E-3
Elemental Tritium	7.1E-8	2.2E-8
Uranium-233	3.4E-4	2.5E-6
Uranium-234	3.4E-4	2.5E-6
Uranium-235	3.4E-4	2.5E-6
Uranium-236	3.4E-4	2.5E-6
Uranium-238	3.4E-4	2.5E-6
Uranium-239	3.4E-4	2.5E-6
Yttrium-90	5E-3	5E-6
Zinc-65	5E-2	6E-3
Zirconium-93	5E-4	2.5E-6

Source: Strenge and Peterson 1989. These parameters are used in equations 7.16 through 7.19.

7.3 Ingestion of Soil

Soil ingestion can occur by the inadvertent consumption of soil on hands, food items, or objects. The population exposed to contaminants through soil ingestion can be identified by estimating the area of contaminant spread and then determining the population within that area. Both the use of and accessibility to the site or facility and surrounding areas must be considered when evaluating soil exposure pathways.

Employees at a facility or site can be exposed to soil contaminated directly during the manufacture, transport, storage, or disposal of contaminants. Ingestion of particulates (heavy metals and radionuclides) occurs when individuals are exposed to contaminants by wind-generated dust or by ingesting soil that is on their skin. The risk of ingesting hazardous particulate contaminants is minimized if workers are wearing protective clothing and/or equipment for the specific activity. Onsite employees may also be exposed to contaminated soil during working hours, depending on the type of employment. However, the ingestion of soil is likely to be insignificant for onsite employees.

The quantity of soil ingested by humans varies according to age. However, the entire population in a contaminated area may inadvertently ingest soil. The average individual dose from ingestion of soil is estimated as follows:

$$D_{si} = 10^{-3} * U_{ds} * C_{msi} * F_{ds} \left[\frac{1 - e^{\lambda_{di} * ED_{ds}}}{\lambda_{di} * AT_{dsi} * BW_{ds} * 365.25}\right], \qquad \textbf{[7.22]}$$

where D_{si} = Average daily dose for chemical pollutant *I* from ingestion of soil (mg/kg/d or pCi/d),

10^{-3} = units conversion factor (g/g),

U_{ds} = ingestion rate for soil (g/d or pCi/d),

C_{msi} = measured soil concentration for pollutant *I* (mg/kg or pCi/g),

F_{ds} = fraction of days per year that soil ingestion occurs (dimensionless),

λ_{di} = environmental loss rate constant for surface soil (d^{-1}),

ED_{ds} = exposure duration for the soil ingestion pathway (y),

AT_{dsi} = averaging time for pollutant *I* for the soil ingestion pathway (d),

BW_{ds} = body weight of exposed individual for the soil ingestion pathway (kg),

365.25 = conversion factor (d/yr).

The exposure factors for soil ingestion are summarized in appendix A.

8. INHALATION EXPOSURE ROUTE

Exposure to contaminants may also occur through inhalation. Exposure occurs, for instance, when volatile or particulate contaminants become airborne and are inhaled from the atmospheric transport pathway. Another inhalation pathway is the inhalation of airborne contaminants while showering. These inhalation exposure routes are discussed in the following sections.

Unless otherwise indicated, all equations in this chapter are taken from Whelan et al. (1987).

8.1 INHALATION OF AIRBORNE CONTAMINANTS IN ATMOSPHERE

The primary inhalation route is inhalation of airborne contaminants from the atmospheric transport medium. All individuals within 80 km (50 mi) of the installation or facility are considered to be potentially exposed through this medium. Preliminary analyses for the PEIS showed that exposures to the most exposed individual in the offsite population decrease as distance from the release site or facility increases; exposures beyond 50 mi are usually insignificant in comparison to those estimated within the 80 km (50-mi) radius. The average dose for inhalation of contaminated air (D_{at}) is calculated from the average individual ventilation rate (IR_a) and the average contaminant concentration in air (C_a):

$$D_{at} = IR * C_a * D_h, \qquad \textbf{[8.1]}$$

where D_{at} = Contaminant-specific average individual intake from breathing contaminated air (mg/kg/d or pCi/d),

IR = Average inhalation rate (20 m^3/d) (EPA 1989a),

C_a = Contaminant-specific average air concentration (mg/m^3 or pCi/m^3),

D_h = Average amount of chemical inhaled per day related to daily dose per unit body weight (kg^{-1}) or inhalation dose conversion factor for radionuclide (dimensionless).

8.2 INHALATION OF AIRBORNE CONTAMINANTS WHILE SHOWERING

Inhalation of contaminants may also occur while showering. This inhalation exposure route is considered only for the groundwater pathway because volatile compounds are not expected to remain in surface water for the time required to reach usage locations (i.e., receptors). Inhalation intake while showering requires an estimate of the amount of volatile contaminant released into the shower air. Showering represents a system that promotes release of volatiles from the water (i.e., high turbulence, high surface area, and small droplets); therefore, the concentration of contaminant in the shower air is assumed to be in equilibrium with the contaminant concentration in the water. The air concentration can be estimated using the Henry's Law Constant (Lyman et al. 1982) as follows:

$$C_{sa} = 10^3 * C_{sw} * \frac{H}{(R * T)}, \qquad \textbf{[8.2]}$$

where C_{sa} = Volatile contaminant concentration in shower air (mg/m^3),

10^3 = Unit conversion factor (L/m^3),

C_{sw} = Contaminant concentration in shower water (mg/L),

H = Henry's Law Constant (atm-m^3/mol),

R = Gas constant (8.2 x 10^{-5} atm-m^3/mol-K),

T = Average temperature of air-shower atmosphere (311°K or 100°F).

Equation 8.2 predicts relatively high air concentrations for highly volatile contaminants; therefore, a mass balance must be performed to ensure that the amount of contaminant predicted to be in the shower air is not greater than the total amount in the shower water. The mass balance is performed as follows:

$$C_{sa} * V_{sa} \leq C_{sw} * V_{sw}, \qquad \textbf{[8.3]}$$

where C_{sa} = Volatile contaminant concentration in shower (mg/m^3),

V_{sa} = Volume of air in shower stall (2 m^3),

C_{sw} = Contaminant concentration in shower water (mg/L),

V_{sw} = Volume of water used during shower (190 L)(approximately 50 gal).

Using the values in equation 8.3 in combination with equation 8.2 and solving for Henry's Law Constant, the following expression is obtained:

$$H \leq 2.4 * 10^{-3} \frac{atm-m^3}{mol}. \qquad \textbf{[8.4]}$$

The value of the Henry's Law Constant is, therefore, limited to a maximum value of 2.4E-3 in application of equation 8.2 to evaluate the shower air contaminant concentration. The total amount of pollutant inhaled during a shower is then estimated as follows:

$$D_{is} = IR * t_s * C_{sa} * P_f * D_h * \exp(-\lambda_w * t_t), \qquad \textbf{[8.5]}$$

where D_{is} = Contaminant-specific inhalation intake concentration while showering (mg/kg-d or pCi/d),

IR = Average inhalation rate for individuals (m^3/hr),

t_s = Average time spent showering per day (hr/d),

C_{sa} = Volatile contaminant-specific concentration in shower air (mg/m^3),

P_f = Water treatment purification factor (fraction of contaminant remaining after treatment)(dimensionless),

D_h = Average amount of chemical inhaled per day related to daily dose per unit body weight (kg^{-1}) or ingestion dose conversion factor for radionuclide (dimensionless),

λ_w = Contaminant-specific environmental degradation constant for contaminant in water (d^{-1}),

t_t = Average time of contaminant transit through water distribution system (d).

The exposure factors for the inhalation of contaminants in the atmosphere and inhalation of contaminants while showering are presented in appendix A.

9. DERMAL CONTACT EXPOSURE ROUTE

Individuals may be exposed to contaminants as a result of dermal contact with and absorption of contaminants (EPA 1992b). Exposure may result from dermal contact with contaminated soil or water. Dermal contact with water and soil is generally not considered for WM activities because surface water and groundwater are not involved. The following sections provide a discussion of the dermal contact exposure routes.

Unless otherwise indicated, all equations in this chapter are taken from Whelan et al. (1987).

9.1 DERMAL CONTACT WITH CONTAMINATED SOIL

Soil can become contaminated from various sources and activities. For example, soil contamination may occur during the manufacture, transport, storage, or disposal of contaminants. Then, the offsite population can be exposed if the soil is carried into a house or building from the surrounding area.

Absorption of contaminants may result from dermal contact with soil that is a contaminated source or becomes contaminated by atmospheric deposition or irrigation. Soil contact represents absorption of contaminants through the skin. The penetration of a contaminant through the skin is dependent on several parameters:

- Concentration available to skin
- Exposed skin surface area
- Condition of the skin
- Loss of contaminant from the skin by evaporation after exposure
- Chemical characteristics.

The effective uptake of contaminants from dermal contact with soil is likely to be insignificant compared with the inhalation or ingestion exposure routes. Therefore, calculations for dermal contact with contaminated soil are not presented in this methodology. Soil ingestion as a result of contact with contaminants on the hands is discussed in section 7.3.

9.2 DERMAL CONTACT WITH CONTAMINATED WATER

Contaminant uptake may also result from dermal contact with water contaminated from the groundwater or surface water transport media. A portion of the population potentially exposed to contaminated

groundwater or surface water includes individuals served by a water supply system that draws water from a contaminated source. Such individuals may be dermally exposed to contaminants while showering or bathing. Relative to ingestion of water, however, the dermal uptake of contaminants during bathing is assumed to be a minor dose. This assumption may be more accurate for showering than for bathing. Similar to incidental ingestion of water while bathing (section 7.1.1), the dermal exposure route is analyzed with the assumption that each person bathes once per day.

Individuals who may experience dermal exposure to surface water contaminants can also be identified by geographically-defined sources of recreational surface water such as contaminated rivers, lakes, and ponds. The exposed population includes the swimmers in such potentially contaminated waters, who can experience exposure to contaminants over a major proportion of their bodies.

The amount of contaminated material that comes into contact with the skin and the rate at which that material is absorbed are important factors in estimating the dermal intake. Exposure duration, event time and frequency are three necessary variables for the application of the dermal dose equations. The total daily absorption rate of contaminated water, corrected by the gastrointestinal tract absorption fraction, is calculated as follows:

$$DA_w = \frac{DA_{event} * A_{skin} * F_{event}}{BW * f_1}, \qquad \textbf{[9.1]}$$

where DA_w = Contaminant-specific daily intake via dermal absorption equivalent to oral intake (mg/kg/d),

DA_{event} = Dose absorbed per unit area per event (mg/cm^2 -event),

A_{skin} = Area of skin exposed to contaminated water (cm^2),

F_{event} = Frequency of events (events/day) (1 event/day for showering; 0.066 event/day for swimming),

BW = Body weight of exposed individual (kg),

f_1 = Fraction of material absorbed in passing through the GI tract (unitless).

The dose as computed by equation 9.1 is equivalent to oral exposure because the GI absorption correction has been applied. The equation is used for all contaminants except radionuclides. For radionuclides, the GI absorption fraction and body weight are not applied because radionuclide-specific dose conversion

factors for dermal intake are provided for the 70-kg (154 lbs) reference individual. The equation for radionuclides is as follows:

$$D_{rad} = DA_{event} * A_{skin} * F_{event} , \qquad [9.2]$$

where DA_{rad} = Daily intake via dermal absorption (pCi/d),

DA_{event} = Dose absorbed per unit area per event (pCi/cm^2 -event),

F_{event} = Frequency of events (events/day).

Specific equations are needed for evaluation of the dose absorbed per event for inorganic compounds and organic compounds. For inorganic compounds, the absorbed dose per unit area per event is evaluated as follows:

$$DA_{event} = K_p^w * C_w * t_{event}, \qquad [9.3]$$

where DA_{event} = Contaminant-specific intake from absorption per unit area per event (mg/cm^2 -event or pCi/cm^2 -event),

K_p^w = Permeability coefficient from water (cm/hr),

C_w = Concentration of chemical in water (mg/cm^3 or pCi/cm^3),

t_{event} = Duration of event (hr/event) (0.167 hr/event or 10 min for showering; 0.5 hr/event for swimming).

The dose absorbed (oral equivalent) per unit area per event is evaluated for organic chemicals using the permeability constant and the octanol-water partition coefficient. For permeability constants for organics not found in Strenge and Peterson (1989), the following equation, which correlates permeability constant with the octanol partition coefficient (K_{ow}) and molecular weight (MW), is used:

$$\log K_p = -2.73 + 0.71 \log K_{ow} - 0.0061(MW) \qquad [9.4]$$

For inorganics not found in Strenge and Peterson (1989), a skin permeability constant of 1E-3 is assumed.

The dose absorbed per event is calculated according to event duration. For event duration that is less than the time to reach steady state, the following equation is used to determine dose absorbed:

$$DA_{event} = 2 * K_p * C_w \sqrt{\frac{6 \tau t_{event}}{\pi}}, \qquad \textbf{[9.5]}$$

where DA_{event} = Contaminant-specific intake from absorption per unit area per event dependent on time to reach steady state (t*) (mg/cm^2-event or pCi/cm^2-event),

K_p = Permeability coefficient (cm/hr),

C_w = Concentration of chemical in water (mg/cm^3 or pCi/cm^3),

τ = Delay time (hr),

t_{event} = Duration of event (hr/event) (0.167 hr/event for showering; 0.5 hr/event for swimming).

For event duration that is greater than the time to reach steady state, the following equation is used to determine dose absorbed:

$$DA_{event} = K_p * C_w \left[\frac{t_{event}}{1 + B} + 2 \tau \left(\frac{1 + 3 B}{1 + B}\right)\right], \qquad \textbf{[9.6]}$$

where DA_{event} = Contaminant-specific intake from absorption per event dependent on time to reach steady state (mg/cm^2 -event or pCi/cm^2 -event),

K_p = Permeability coefficient (cm/hr),

C_w = Concentration of chemical in water (mg/cm^3 or pCi/cm^3),

t_{event} = Duration of event (hr/event) (0.167 hr/event for showering; 0.5 hr/event for swimming),

B = Octanol-water partitioning coefficient (K_{ow}) divided by 10^4.

The exposure factors for dermal exposure are listed in appendix A.

10. EXPOSURE TO RADIATION

Radioactive atoms undergo spontaneous nuclear transformations and release excess energy in the form of ionizing radiation. Such transformations are referred to as radioactive decay. As a result of the radioactive decay process, one element is transformed into another element. A radioactive species of a particular element is referred to as a radionuclide or radioisotope. Radiation is capable of transferring enough localized energy to atoms to remove electrons from the electric field of their nucleus, a process called ionization. This ionization or energy transfer can be harmful to living tissues. The type of ionizing radiation emitted by a particular radionuclide depends on the nature of the nuclear transformation and may include emissions of alpha particles, electrons (beta particles), and neutrons. Each of these transformations may be accompanied by emissions of photons (gamma radiations or x-rays). Each type of radiation differs in its physical characteristics and in its ability to harm biological tissue (EPA 1989a), as described below:

- **Alpha Particles** have a large mass of 4 (i.e., 2 neutrons and 2 protons in the nucleus) and a charge of 2, tending to ionize atoms quickly. Alpha particles will usually not penetrate an ordinary sheet of paper or the outer layer of skin. Alpha particles are a significant hazard only when taken into the body, where their energy is absorbed by a small volume of tissues.

- **Beta Particles** are high-speed electrons that are emitted over a continuous energy spectrum. These particles are smaller than alpha particles and transfer their energy to tissue at a lower rate. Unshielded beta sources are potentially harmful if the radiation has enough energy and is within a few centimeters of exposed skin surfaces. Beta sources shielded with certain metallic materials may produce low energy x-ray radiation, which may contribute to external radiation exposure. Beta particles deposit much less energy than alpha particles to small volumes of tissue and inflict less internal damage per unit of energy.

- **Gamma Radiations** are photons emitted from the nucleus of a radioactive atom. X-rays, identical in form to gamma rays, are photons emitted from electron transitions. Both are forms of electromagnetic radiation, and both have a high penetration ability and are of most concern as external radiation hazards.

- **Neutrons** are emitted during nuclear fission reactions, along with fission fragments and beta and gamma radiation. For radionuclides likely to be encountered at DOE or Superfund sites, the rate of spontaneous fission is minute and no significant neutron radiation is expected.

Quantities of radionuclides are typically expressed in terms of activity at a given time. The conventional unit of activity is the curie (Ci), which is the quantity of a given radionuclide in which 3.7×10^{10} atoms undergo spontaneous transformation each second. Environmental concentrations of radionuclides are often expressed in picoCuries (pCi), which is 10^{-12} Ci.

Ionizing radiation can cause adverse effects to tissues only when the energy released during radioactive decay is absorbed in tissue. The absorbed dose (D) is the mean energy imparted by ionizing radiation per unit mass of tissue. The conventional unit of absorbed dose is the rad. At low levels of exposure, absorbed dose is not sufficient to relate the amount of energy absorbed to the biological effect (e.g., cancer induction). For the same absorbed dose delivered at the same rate, some types of radiation may produce more pronounced biological effects than others. Because absorbed dose is not sufficient to relate the relative effectiveness of radiation to biological effects, the concept of dose equivalent (H) was developed. The H is defined as the absorbed dose modified by a quality factor that represents the biological effects of radiation from a radionuclide. The H was developed to normalize the unequal biological effects produced from equal absorbed doses of different types of radiation. The conventional unit of the dose equivalent is the rem. The dose equivalent is defined as follows:

$$H = D * Q, \qquad \textbf{[10.1]}$$

where H = Dose equivalent (rem),

D = Absorbed dose (rad),

Q = Quality factor (unitless).

Quality factors assigned by the International Commission on Radiological Protection (ICRP) (1987) include values of $Q=20$ for alpha particles, $Q=10$ for neutrons and protons, and $Q=1$ for beta particles, positrons, x-rays, and gamma rays. The Q values for neutrons vary with energy. For example, $Q=3$ for $E<10$ keV, and $Q=10$ for $E>10$ keV. Other values of Q are also appropriate if the energies are known more exactly. A Q value of 20, for example, means that if an equal amount of energy is absorbed, an alpha particle will inflict approximately 20 times more damage to biological tissue than a beta particle or gamma ray. While rad and rem are conventional units, the SI (international system) units of activity, absorbed dose, and dose equivalent are the Becquerel (Bq), Gray (Gy), and the Sievert (Sv), respectively. The relationship of these units is shown below:

1 Bq = 2.7×10^{-11} Ci
1 Ci = 3.7×10^{10} Bq
1 Gy = 1 joule/kg = 100 rad
1 rad = 0.01 Gy = 0.01 joule/kg

1 Sv = 100 rem.

When subjected to equal doses of radiation, organs and tissues have different cancer induction rates. To normalize radiation doses and effects on a whole body basis, the EDE is used. The EDE is defined as the weighted sum of the dose equivalents to different organs or tissues. Weighting factors are the ratios of risk for a given tissue to total risk for all tissues when the body is irradiated uniformly. The weighting factors for different organs or tissues are used in defining EDE are the following (ICRP 1977):

Gonads	0.25
Breast	0.15
Red marrow	0.12
Lungs	0.12
Thyroid	0.03
Bone surfaces	0.03
Remainder	0.3

The weighting factor for gonads represents stochastic risk of hereditary effects, but all other weighting factors represent stochastic risk of fatal cancers. The EDE can be defined as follows:

$$EDE = \sum W_T * DE_T , \qquad \textbf{[10.2]}$$

where EDE = Effective dose equivalent (rem),

W_T = Weighting factor for organ or tissue T,

DE = Dose equivalent to organ or tissue T (rem).

EDEs due to inhalation and ingestion may be determined from radionuclide concentrations in environmental media by multiplying the radionuclide concentration by the duration of exposure, intake per unit time, and the dose conversion factor (DCF), which represents the dose equivalent per unit intake. Internal DCFs are available from Federal Guidance Report No. 11. These DCFs give the 50-yr committed dose from an acute intake (section 10.1).

Although ICRP 60 (1990) is a more recent revision of the ICRP guidance that has recommended revised weighting factors for doses to various organs, ICRP 26 (1977) tissue weighting factors and risk values are still widely used. DOE has not adopted the ICRP 60 weighting factors for its use, and some of the models used to estimate risks (e.g., GENII) are organ-based and cannot use whole-body DCFs such as those in ICRP 60. However, the NRC and DOE both use ICRP 60 risk factors. To be consistent as far as possible

with federal guidance, this methodology uses ICRP 26 tissue weighting factors in conjunction with ICRP 60 risk factors. This combined use of ICRP guidance is generally accepted in the fields of industrial hygiene and health physics.

Radionuclide releases may occur by short-term releases and/or long-term releases, such as those releases that may occur from waste disposal sites. In an acute release to the biosphere, a member of the offsite or onsite population may be irradiated by drinking contaminated well or surface water, by eating fish or fresh vegetables contaminated by the water, or by being exposed to contamination (by irrigation, flooding, or atmospheric deposition) that may deposit on the ground and plants around the individual's home, resulting in a source of long-term exposure from a short-term release. The exposure route determines whether the received dose is external or internal. External radiation sources include contaminated air, water, and surfaces. Internal sources of radiation result from ingestion, inhalation, and absorption of radionuclides.

10.1 External Exposure

External exposure refers to the irradiation of a receptor by radiation sources external to the body. The most important modes of external exposure for radionuclides in the environment are the following:

- exposure to air containing gamma-emitting radionuclides
- immersion in or proximity to contaminated water
- exposure to contaminated surface soil or equipment surfaces.

Only radiations that penetrate the body surface (gamma and x-rays, beta, and neutron radiations) contribute significantly to external exposure, with gamma-emitters being the most prevalent. External radiation exposure to contaminated land surfaces is depicted in figure 10.1–1.

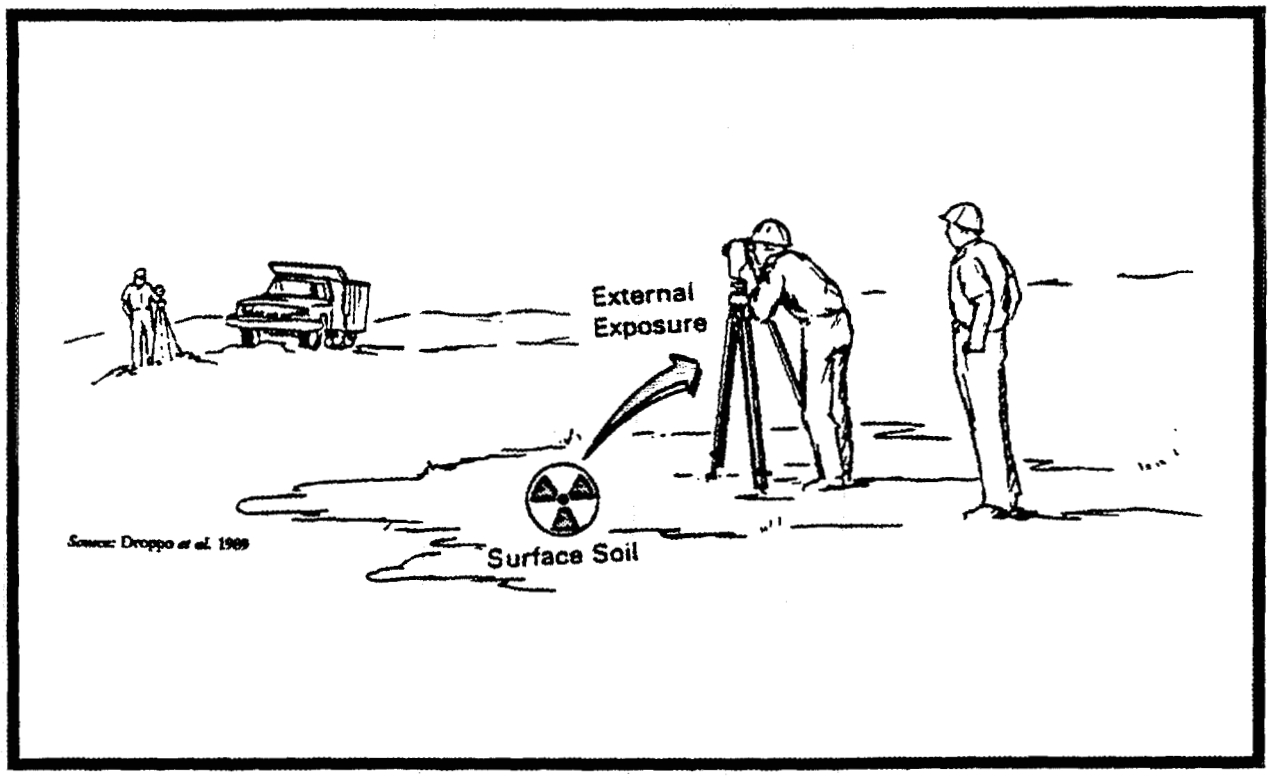

Figure 10.1-1. Depiction of exposure pathway involving direct radiation.

External radiation is considered for individuals exposed to land that is either contaminated by radionuclides or becomes contaminated by atmospheric deposition of radionuclides or to equipment surfaces that are contaminated by atmospheric deposition of radionuclides. External radiation is also considered for individuals involved in aquatic recreational activities associated with contaminated surface waters. Aquatic recreational activities include boating, swimming, and shoreline fishing and/or hiking. The radiation dose is calculated from the water concentration, equipment surface concentration, or the surface soil concentration, depending on which transport medium is being studied. For immersion in or proximity to water, the recreational dose is calculated as follows:

$$= 70 * [(D_{rf} * t_b) + t_{sw}] * C_w * D_{gw} + 70 * t_{sh} * C_{sd} * W * \qquad \textbf{[10.3]}$$

where $D_{rad,w}$ = EDE from external exposure during water immersion (rem/70 yr),

D_{rf} = Water immersion reduction factor (0.5 unitless),

t_b = Average boating time spent by an individual (12 hr/yr),

t_{sw} = Average swimming time spent by an individual (12 hr/yr),

C_w = Contaminant concentration in water (pCi/L),

D_{gw} = External dose conversion factor for radionuclide immersion in water (rem/hr per pCi/L),

t_{sh} = Average shoreline activity time spent by an individual (12 hr/yr),

C_{sd} = Average sediment concentration deposited on shoreline from contaminated water (pCi/m^2),

W = Shore-width factor to correct for finite size of shoreline (unitless) [river shoreline default - 0.2 (Dunster 1971)],

D_{gp} = External dose conversion factor for exposure to a contaminated plane of radionuclide (rem/hr per pCi/m^2).

The water immersion dose conversion factors represent the dose rate (rem/hr) received while totally immersed on contaminated water of unit concentration (pCi/L). However, the boating exposure scenario uses the immersion factors multiplied by a geometric correction factor of 0.5 to account for the fact that the boat is approximately on the surface of the water and occupants are exposed to half of the source strength.

The shore-width factor (W) represents the fraction of dose received from a given shoreline situation that may not be well represented as an infinite plane (for which dose conversion factors are defined). It is essentially a geometric correction to account for the finite size of shorelines.

The average radionuclide concentration in shoreline sediment (C_{sd}) is estimated from a code developed by Soldat et al. (1974) that relates water concentration (C_w) to sediment concentration following a long period of deposition. The equation estimates an effective surface contamination (C_{sd}) for use in calculating gamma exposure rates to persons standing on sediment:

$$C_{sd} = T_c * t_{\frac{1}{2}} * C_w * [1 - \exp(-\lambda_s * t_w)] \ , \qquad \textbf{[10.4]}$$

where C_{sd} = Radionuclide concentration in sediment (pCi/m^2),

T_c = Transfer constant from water to sediment (100 L/m^2/d),

$t_{1/2}$ = Radionuclide physical half-life (d),

C_w = Radionuclide concentration in water (pCi/L),

λ_s = Radionuclide environmental degradation and decay constant in soil (d^{-1}),

t_w = Length of time the shoreline sediment is exposed to contaminated water (d). Set to 35 years to represent the midpoint of the 70-yr evaluation period.

The value of the transfer constant (T_c) was derived for several radionuclides by using data obtained from an analysis of water and sediment samples taken from the Columbia River at Richland, Washington and at Tillamook Bay, Oregon, 75 km (43 miles) south of the river mouth (Nelson 1965; Toombs and Cutler 1968).

For the WM evaluation, the radiation dose from aquatic recreational activities as follows:

$$De_i = (0.5\ tb + ts) * Cw_i * Db_i + tf * Cd_i * W * Ds_i\ , \qquad \textbf{[10.5]}$$

where De_i = EDE from external exposure from aquatic recreational activities for radionuclide *I* (rem),

Cd_i = Sediment concentration of radionuclide *I*, deposited on shoreline from contaminated water (Ci/m^2),

tb = Time spent by an individual in boating (h),

tf = Time spent by an individual fishing or in shoreline activities (h),

ts = Time spent by an individual swimming (h),

Cw_i = Concentration in water (pCi/L),

Db_i = External dose conversion factor for radionuclide *I*, for immersion in water (rem/h per Ci/L),

Ds_i = External dose conversion factor for exposure to a contaminated plane of radionuclide *I* (rem/h per Ci/m^2),

W = Shore width factor to correct for finite size of shoreline (dimensionless).

The radionuclide sediment concentration (Cd_i) is calculated for acute releases as shown below:

$$Cd_i = 100 * T_i * E_w , \qquad \textbf{[10.6]}$$

where Cd_i = Sediment concentration of radionuclide *I*, deposited on shoreline from contaminated water (Ci/m^2),

100 = Transfer constant from water to sediment (L/m^2/d),

T_i = Physical half-life of the radionuclide (d),

E_w = Time-integrated water concentration (Ci sec/L).

For contaminated soil, the radiation dose received in one lifetime (i.e., 70 yr) is calculated from the average soil concentration (C_s) as a result of disposal activities or air deposition as follows:

$$D_{rad,s} = 70 * t_g * C_s * D_s , \qquad \textbf{[10.7]}$$

where $D_{rad,s}$ = EDE from exposure to contaminated soil (rem/70 yr),

70 = Units conversion factor (70 yr),

t_g = Average time of exposure to contaminated soil (8760 hr/yr [continuous exposure]),

C_s = Radionuclide concentration in soil (pCi/m^2),

D_s = Dose conversion factor (rem/pCi-yr).

For the WM evaluation, the radiation dose is calculated from the soil concentration as follows:

$$Dx_i = ts * Cs_i * Ds_i \,, \qquad \textbf{[10.8]}$$

where Dx_i = EDE individual from exposure to contaminated ground for radionuclide *I* (rem),

Cs_i = Soil concentration (Ci/m^2),

ts = Time of exposure to contaminated ground (h),

Ds_i = External dose conversion factor for exposure to a contaminated plane of radionuclide *I* (rem/h per Ci/m^2).

When actual exposure data are unknown or unavailable, computer codes or other more simplistic methods may be used to estimate the EDE rates attributable to direct radiation. Some of these methods include PATHRAE and PRESTO (EPA models for estimating exposure to low-level radiation), and MicroShield (Grove Engineering 1992).

Before doses from exposure to radiation from contaminated equipment or building surfaces (i.e., radiation with no transport medium) can be calculated, such information as number and type of shielding materials, source-to-receptor distances, and source geometry must be obtained. More sophisticated computer codes (such as MicroShield or ISOSHLD) can be used to account for these aspects of external radiation. Four of the main shielding types are as follows:

- Air gap shields
- Transition shields
- End clad and side clad shields
- Immersion shields.

An air gap is a shield region with air as the shielding material. Air gaps are usually present when shield thicknesses are less than the total source-to-receptor distance. Transition shields determine which shields are cylindrical or spherical. Spherical and cylindrical sources have shields of the same geometry, respectively. The transition between the inner spherical or cylindrical shield and the outer slab shields is called the transition shield. End side clad shields are used for cylindrical sources; they are materials that wrap around the source. Finally, immersion shields are required when a line of sight between the exposure point (or dose point) and part of the source is immersed in a medium separate from the specified shields. Immersion shields can exist for sources that are lines, cylinders, and horizontal areas (Grove Engineering 1992).

Although shielding provides protection from exposure to radiation, shielding materials affect the efficacy of the protection. Reduction and scattering of radiation between a source and exposure point are affected by all intervening materials. Shield materials determine the radiation attenuation and buildup characteristics used to calculate the dose rate (rad/hr) (Grove Engineering 1992). DE can be derived from dose rate by multiplying by duration of exposure and a radiation-specific quality factor. EDE can then be derived by weighting the sum of dose equivalents (DEs) to affected tissues. For photon radiation, the quality factor is one and absorbed dose is equal to DE.

The source geometry includes the distance and orientation between the source and exposure point, dimensions of the source region, and the dimensions, locations, and orientations of intervening shields (Grove Engineering 1992). Source-to-receptor distances vary among the receptors considered (e.g., on-site employees, off-site populations) and often with each remediation or waste treatment task.

10.2 Internal Exposure

Internal exposure refers to the irradiation of human tissues by radiations emitted by radionuclides that are ingested, inhaled, or absorbed. All types of radiation may contribute to internal exposure. For internal exposure, intakes of radionuclides commit the individual to receiving doses over future times, even with no further intakes, until the source of radiation (i.e., radionuclide) is removed from the body by biological elimination or radioactive decay. Internal doses to tissues are usually reported in terms of 50-yr committed dose equivalents, the total dose equivalent (received by the tissue) over the 50-yr period following the intake of a radionuclide. Internal dose conversion factors are usually calculated as 50-yr committed dose equivalents (CEDE) (i.e., dose equivalents received to age 70 following acute intake at age 20).

Internal radiation is considered for individuals exposed to radionuclides that are systematically incorporated into the body by ingestion, inhalation, or dermal absorption. The radiation dose is determined from the quantity of radionuclides ingested, inhaled, or absorbed, depending on which exposure route is being evaluated. Calculations for the ingestion, inhalation, and dermal exposure routes are provided in chapters 7, 8, and 9, respectively.

11. EXPOSURE TO TRITIUM

Hydrogen has three naturally occurring isotopes that have almost identical chemical properties and are expected to behave similarly in the environment. With atoms that weigh three times ordinary light hydrogen atoms, tritium (H^3) is a radioactive isotope of hydrogen. Tritium may be found anywhere other hydrogen isotopes are found. By virtue of its chemical composition and common chemical properties with other hydrogen isotopes, tritium is closely associated with water. Tritium can be found in water molecules or form radioactive hydrogen gas or steam. In fact, the movement of tritium is generally expected to be water coincident (Strenge and Peterson 1989). Tritium is considered separately from other contaminants in the risk evaluation because of its close association with water, which comprises 80% of the mass of some soft tissues.

In this methodology, special equations are used to estimate exposure to tritium not only because of its association with water but also because of the contaminant's behavior in the body. Ingested or inhaled radionuclides can become distributed in all parts of the body. When radionuclides are distributed in the body, they become *internal emitters*. A radionuclide entering the body usually follows certain metabolic pathways, and as a chemical element, seeks specific body organs. While iodine concentrates primarily in the thyroid, for example, radium and strontium concentrate primarily in the bones. The distribution of tritium, however, occurs throughout the entire body.

An internally deposited (i.e., ingested, inhaled, or dermally absorbed) radionuclide, such as tritium, may emit particles with a short range. If such particles are emitted, their energies will be absorbed into the tissues. The dose rate in the tissue can then be calculated from the activity concentration. Such is the case when an alpha or low-energy beta emitter, such as tritium, is embedded in tissue.

Tritium that is inhaled, ingested, or absorbed through the skin is assumed to become instantaneously and uniformly distributed throughout all the soft tissues in the body. While some tritium from tritiated water can become organically bound in the body, the ICRP assumes a single-exponential retention function for the body, which is based on tritiated water with a biological half-life of 10 days. Because of the unique characteristics of tritium, food crops and animal products that have been contaminated with tritium must be considered separately from those that have been contaminated by other radionuclides.

Unless otherwise noted, all equations in this chapter are taken from Whelan et al. (1987).

11.1 TRITIUM IN FOOD CROPS

While equation 7.14 is used for computing the average contaminant concentration for most contaminants, a special model is required for tritium. The concentration of tritium in plants is assumed to have the same specific activity as the contaminating media (air or water); therefore, the fractional content of hydrogen in the plant is used to estimate the tritium concentration in the food crop product. The concentration of tritium in vegetables from atmospheric deposition is calculated for air pathways as follows:

$$C_{ct} = 9 * C_a * \left(\frac{f_{hp}}{H_a}\right) , \qquad \textbf{[11.1]}$$

where C_{ct} = Tritium concentration in vegetables from atmospheric deposition (pCi/kg),

9 = Inverse of hydrogen mass fraction in water (kg H_20/kg H),

C_a = Tritium concentration in air (pCi/m^3),

f_{hp} = Vegetable type-specific total fraction of hydrogen in plant which results from plant uptake pathway (0.1 kg H/kg plant) (Napier et al. 1980),

H_a = Absolute humidity (0.008 kg H_20/m^3).

A similar equation is used to estimate plant uptake of tritium from the water pathways:

$$C_{ct} = 9 * C_w * f_{hp} , \qquad \textbf{[11.2]}$$

where C_{ct} = Tritium concentration in vegetables from water uptake (pCi/L),

C_w = Tritium concentration in water (pCi/L),

f_{hp} = Total fraction of hydrogen in plant which results from plant uptake pathway (0.1 kg H/kg plant) (Napier et al. 1980).

The dose (D_{fc}) from ingestion of agricultural crops is calculated using an equation that is similar to equation 7.14. In the special case of tritium, however, the value calculated for the tritium concentration in vegetables (C_{ct}) is used instead of the contaminant concentration in vegetables (C_c) in equation 7.14. The dose from ingestion of agricultural crops contaminated by tritium is calculated as follows:

$$D_{fc} = \sum_{p=1}^{2} IR * C_{ct} * D_g \,, \qquad [11.3]$$

where D_{fc} = EDE from consumption of crops contaminated by tritium (rem),

IR = Average daily consumption rate (leafy = 0.082 kg/d; other = 0.52 kg/d [NRC 1977]),

C_{ct} = Tritium concentration in vegetable at time of consumption (pCi/kg),

D_g = Ingestion dose conversion factor (rem pCi-yr).

11.2 Tritium in Animal Products

A special equation is also used to estimate the tritium concentration in animal products. The concentration of tritium is assumed to have the same specific activity as the total animal intake of feed and water. For air and water pathways, the animal product tritium concentration (C_T) is estimated as follows:

$$C_T = \frac{(C_{Tf} * IR_f) + (C_{Tw} * IR_w)}{(F_{hf} * IR_f) + (\frac{IR_w}{9})} * F_{hm} \,, \qquad [11.4]$$

where C_T = Animal product tritium concentration (pCi/kg),

C_{Tf} = Tritium concentration in animal feed (pCi/kg),

IR_f = Intake rate of feed by the animal (68 kg/d for meat; 55 kg/d for milk),

C_{Tw} = Tritium concentration in water used for animal drinking water (pCi/L),

IR_w = Intake rate of water by the animal (50 L/d for beef cattle; 60 L/d for milk cows),

F_{hm} = Fraction of hydrogen in animal product *m* (0.1 kg H/kg meat; 0.11 kg H/L milk)(Napier et al. 1980),

F_{hf} = Fraction of hydrogen in animal feed crop (0.068 kg H/kg meat; 0.1 kg H/kg milk)(Napier et al. 1980).

Beef cattle are assumed to be fed primarily on grain while milk cows are fed on grass. For air pathways, the concentration in animal drinking water is set to zero.

The dose (D_{ap}) for tritium is calculated using an equation similar to equation 7.21. However, in the special case of tritium, the value calculated for the animal product tritium concentration (C_T) is used instead of the contaminant concentration in the animal product (C_{TC}) in equation 7.20. The average individual dose for tritium is calculated as follows:

$$D_{ap} = \sum_{m=1}^{n} IR_m * C_T * D_g \,, \qquad \textbf{[11.5]}$$

where D_{ap} = EDE from ingestion of tritium contaminated animal product *m* (rem),

IR_m = Average daily consumption rate of animal product *m* (meat or milk) (0.26 kg/d for meat; 0.30 L/d for milk) (NRC 1977),

C_T = Tritium concentration in the animal product *m* (pCi/kg for meat; pCi/L for milk),

D_g = Ingestion dose conversion factor (rem/pCi-yr).

12. TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to evaluate the potential for contaminants of concern to cause adverse health effects in exposed individuals. Toxicity assessment also provides, where possible, an estimate of the relationship between the intake or dose of a contaminant and the increased likelihood and/or severity of adverse effects (EPA 1989a). The first step in the toxicity assessment involves characterizing the nature and strength of the dose-response relationship. The second step quantitatively evaluates the dose-response relationship, which is the relationship between the dose of contaminant received and the incidence and/or severity of adverse health effects in the exposed population. Based on this relationship, toxicity values for noncarcinogens and carcinogens are derived.

12.1 NONCARCINOGENIC SUBSTANCES

Noncarcinogenic effects resulting from exposure to chemicals are evaluated based on the RfD toxicity value. The RfD is an estimate of the individual daily exposure level for the human population that is likely to be without appreciable risk of harmful effects. The RfD is the amount of contaminant taken into the body (in units of mg of contaminant/kg body weight/day) that is not expected to result in adverse health effects. Reference doses are developed based on experimental thresholds such as No Observed Adverse Effect Levels (NOAELs) or Lowest Observed Adverse Effect Levels (LOAEL). This concept implies that there exists an intake threshold that must be reached before adverse effects occur.

The development of these RfDs is based on the period of exposure to a contaminant. Subchronic RfDs are associated with short-term exposures between two weeks and seven years. Such short-term exposures can result when a particular activity is performed for a limited number of years or when a chemical with a short half-life degrades to negligible concentrations within several months. Chronic RfDs are associated with long-term exposure to a contaminant, ranging from seven years to a lifetime (70 yr). Chronic RfDs are used in this methodology to evaluate potential noncarcinogenic effects. Acute RfDs are used to evaluate exposures of less than two weeks in duration (e.g., nonroutine events).

The RfDs also reflect routes of exposure (e.g., inhalation, dermal contact, ingestion). Ingestion exposure routes usually have documented RfDs, while inhalation routes may report reference values in terms of reference concentrations (RfCs). The RfC can be converted to an RfD using the following equation:

$$RfD = \frac{RfC * 20}{70}, \qquad \textbf{[12.1]}$$

where RfD = Reference dose (mg/kg-d),

RfC = Reference concentration (mg/m^3),

70 = Body weight (kg),

20 = Inhalation rate (m^3/d).

No RfDs are available for the dermal route of exposure. In some cases, noncarcinogenic effects associated with dermal exposure have been evaluated using an oral RfD, which provides a conservative estimate of effects. Most RfDs are expressed as the amount of substance administered per unit time and unit body weight, whereas exposure estimates for the dermal route are eventually expressed as absorbed doses. Thus, for dermal exposure, the oral toxicity value must be adjusted from an administered to an absorbed dose. When toxicity values are not available for a chemical, the chemical is usually evaluated only qualitatively in the risk assessment. In some cases, toxicity values can be developed for substances without toxicity values, as described in section 12.3.

12.2 CARCINOGENIC SUBSTANCES

Potential carcinogenic effects from chemicals are evaluated using chemical-specific cancer potency factors. Cancer potency factors represent an upper-bound estimate of the probability of a carcinogenic response per unit intake of a chemical over a lifetime (EPA 1992c). Cancer potency factors for chemicals are used to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a potential carcinogen. This probability is termed "cancer incidence" and includes the development of both fatal and nonfatal cancer.

Carcinogenic effects resulting from exposure to radionuclides and chemical carcinogens are assumed to have no threshold dose below which health effects are not observed (i.e., any exposure is associated with some degree of risk). Responses to carcinogens are less likely to show a threshold effect because cancer may be produced by an event in a single cell (EPA 1987). According to the EPA (1989a), cancer risk may be used as the sole basis for assessing the radiation related human health risks of a site containing radionuclides. Carcinogenic effects usually occur at levels significantly lower than those levels associated with systemic toxic effects; thus, cancer risk is usually the *predominant* adverse effect of radionuclides. In addition to cancer risks, this methodology uses adverse genetic effects as an endpoint for radionuclides, based on ICRP 60 (ICRP 1990) criteria.

In conjunction with the cancer potency factors used to evaluate chemical carcinogenic effects, EPA assigns a carcinogenic weight-of-evidence classification to each substance based on the strength of human and

animal evidence of carcinogenicity. These weight-of-evidence classifications determine the likelihood that a given contaminant is a carcinogen and are used in the toxicity assessment as a qualitative indicator of a contaminant's toxicity. All radionuclides are classified by EPA as Group A since radiation acts as a complete carcinogen in that it can both induce and promote cancer. The weights-of-evidence are listed below.

- **Group A: Human Carcinogen** (sufficient evidence of carcinogenicity in humans)
- **Group B: Probable Human Carcinogen** (B1- limited evidence of carcinogenicity in humans; B2- sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- **Group C: Possible Human Carcinogen** (limited evidence of carcinogenicity in animals and inadequate or lack of human data)
- **Group D: Not Classifiable as to Human Carcinogenicity** (inadequate or no evidence)
- **Group E: Evidence of Noncarcinogenicity in Humans** (no evidence of carcinogenicity in adequate studies)

Contaminants usually have documented cancer potency factors for each potential exposure pathway. However, the toxicity values reported for some pathways may be in terms of units of concentration in air or water (i.e., a unit risk value). The units of concentration in air can be converted to a cancer potency factor using the following equation:

$$CPF = \frac{CA * 20}{70}, \qquad \textbf{[12.2]}$$

where CPF = Cancer potency factor (mg/kg-d),

CA = Contaminant concentration in air (mg/m^3),

20 = Average air intake rate (m^3/d),

70 = Average adult body weight (kg).

The units of concentration in water can be converted to a cancer potency factor using the following equation:

$$CPF = \frac{CW * 2}{70}, \qquad \textbf{[12.3]}$$

where CPF = Cancer potency factor $(mg/kg/d)^{-1}$,

CW = Contaminant concentration in water (mg/L),

2 = Adult water intake rate (90% high) (L/d),

70 = Average adult body weight (kg).

In accordance with EPA policy, the latest available cancer potency factors and RfDs should be used to calculate risks using this or any other methodology.

Table 12.2-1 summarizes the endpoints (health effects) evaluated by this methodology. The endpoints for risk evaluations of radionuclides are cancer fatality (the probability of an individual dying from cancer), cancer incidence (the probability of developing fatal and nonfatal cancers), and adverse genetic effects. The toxicity values used to determine the risks from radionuclides are risk factors. Based on ICRP 60 (1990), the fatal risk factor for low dose rates over all tissues or organs is 5E-4 per rem lifetime dose (i.e., 70 yr); the cancer incidence risk factor is 1.7E-3 per rem lifetime dose; the genetic effects risk factor is 1E-4 per rem lifetime dose. These risk factors are used in this methodology to estimate cancer fatality, cancer incidence, and genetic effects risks for offsite and onsite populations. According to ICRP 60, the genetic effects incorporated into the risk factor estimate include gene mutations, gross chromosomal aberrations, and multifactorial effects (i.e., joint action of multiple genetic and environmental factors). The multifactorial effects include congenital abnormalities present at birth and common disorders of adult life.

Table 12.2-1. Summary of Health Effect Endpoints

Types of Contaminant	Public and Onsite Population Toxicity Values	Endpoints
Noncarcinogen	Chronic RfD	Hazard Index, Hazard Quotient
Chemical Carcinogen	Cancer Potency Factor	Cancer Incidence
Radionuclide	5E-4 per rem lifetime*	Cancer Fatality
Radionuclide	1.7E-3 per rem lifetime*	Cancer Incidence
Radionuclide	1E-4 per rem lifetime*	Genetic Effects

* *Source:* ICRP 1990.

12.3 Data Sources for Toxicity Values

IRIS, EPA's on-line toxicity value database, is the preferred source for numerical toxicity values and information. This system provides toxicity factors such as chemical-specific cancer potency factors, RfDs, supporting discussion, and references (EPA 1988). The information contained in IRIS has been reviewed and verified by EPA work groups. HEAST summarizes interim RfDs, cancer potency factors, and other toxicity information. If HEAST does not contain the necessary information, EPA criteria documents should be consulted. These criteria documents contain general toxicity information. Generally, if neither IRIS nor HEAST contain toxicity values, the chemical is not evaluated or is treated qualitatively because of the uncertainty and time involved in developing toxicity values. For the PEIS, some alternative methods are used for estimating toxicity values for contaminants whose values are unavailable in IRIS or HEAST. These methods are described elsewhere (ORNL 1995b).

For nonroutine event scenarios, acute toxicity values are required to address acute exposures. Nonroutine events may include fires and other nonroutine events that occur during TSD activities at a facility. EPA cancer potency factors for acute conditions are used. For the PEIS, Argonne National Laboratory (ANL) has developed acute RfC values for lethal substances (e.g., mustard gas) using the LC_{50} (acute lethal concentration to 50% of animals) approach. This approach may be appropriate for other similar applications for nonroutine event scenarios as well.

The LC_{50} is defined as the concentration of gas or vapor which, administered by continuous inhalation, causes death in half of the animals tested. The LC_{50} values are reduced by an uncertainty factor to account for possible greater sensitivity of humans than of the test species to the substance under consideration. Due to the uncertainties inherent in extrapolating from animal data and scaling from experimental exposure times to predicted event-specific exposure times, the risks estimated from these 50% lethality contours cannot be considered absolute indicators of expected fatalities in the event that these accidents would occur. Instead, the lethality contours are considered to represent reasonably conservative estimates of acute toxicity.

13. RISK CHARACTERIZATION

Toxicity and exposure assessments are integrated and summarized into qualitative and quantitative expressions of risk in the risk characterization process. Risk characterization requires interpretative and qualifying information that summarizes the uncertainties and assumptions used in calculating toxicity and exposure. The following sections describe the risk characterization methodology used to characterize health effects posed by carcinogens and noncarcinogens. The resulting risk estimates and hazards quantify the estimated potential risk to the population considered for each specific release site or treatment facility. Describing all known health effects for each contaminant present throughout the DOE complex is impossible, but such sources as ATSDR toxicological profiles, ICRP 60 (ICRP 1990), National Council for Radiological Protection (NCRP) guidance, and *Radiobiology for the Radiologist* (Hall 1988) provide information about the health effects of radiation and chemicals.

13.1 QUANTIFICATION OF CARCINOGENIC RISK

The quantification of carcinogenic risk differs for chemical carcinogens and radionuclide carcinogens. The differences in estimating risks for each carcinogen are discussed in the sections below.

13.1.1 Chemical Carcinogens

For chemical carcinogens, risks are estimated as the likelihood of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen (i.e., incremental or excess individual lifetime cancer risk). The cancer potency factor converts daily intakes averaged over a lifetime of exposure, as derived from the exposure assessment, to the estimated incremental lifetime risk of an individual developing cancer. Generally, the dose-response relationship is assumed to be linear in the low-dose portion of the multistage model dose-response curve. The methodology is based on the assumption that no transformation or degradation of chemical contaminants occurs. Cancer potency factors can be used to estimate cancer incidence risk from chemical contaminants as follows:

$$Risk = CDI * CPF, \qquad [13.1]$$

where Risk = Probability of cancer incidence (dimensionless),

CDI = Chronic daily intake averaged over 70 yr (mg/kg/day),

CPF = Cancer potency factor (mg/kg-day).

Risk is generally expressed using one significant figure (e.g., 2E-7), risks to individuals are interpreted as the probability of an individual developing cancer over his/her lifetime as a result of exposure to contaminants originating from a given site or facility. When population risks are estimated, cancer incidence is expressed as the rate at which cancers develop, not as a probability. Equation 13.1 is only valid at low risk levels (i.e., below estimated risks of 1E-2, the range of risks usually applicable for Superfund site risks). At risks above this level, a standard one-hit equation can be used to estimate risks (EPA 1989a). This methodology does not consider this one-hit equation, however, because it is assumed that 1.E-6 is the risk level that triggers the need for remediation. Since any risk above 1.E-6 will be flagged as a concern, it is not necessary, especially in a screening-level analysis, to refine the calculations of risk estimates higher than 1.E-2.

Risks less than 1.E-6 are considered acceptable by EPA. Cancer incidence resulting from risks below 1.E-6 cannot be distinguished from the normal cancer rate in an exposed population. Risks ranging from 1.E-4 to 1.E-6 require an investigation of remedial alternatives to determine if risks can be reduced to the 1.E-6 level.

13.1.2 Radionuclide Carcinogens

Estimating cancer risk from radionuclides assumes that dose conversion factors (rem/pCi) have been used to convert from intake (pCi) to dose, resulting in EDEs (rem). The EDE is then multiplied by a risk factor for estimating cancer risk per EDE. The following equation can be used to estimate fatal cancer risk or cancer incidence for an individual:

$$Risk = RF * EDE, \qquad \textbf{[13.2]}$$

where R = Probability of cancer fatality or incidence due to exposure to radiation from a radionuclide (dimensionless),

RF = Cancer fatality or incidence risk factor (5E-4 per rem lifetime dose for fatality, 1.7E-3 per rem lifetime dose for incidence, and 1E-4 per rem lifetime dose for genetic effects),

EDE = Total effective dose equivalent (rem).

When population risks are estimated, incidence and fatality risks are expressed in terms of the rate at which cancers develop or the number of fatalities. Although risks from exposure to chemicals and radionuclides

can be quantified, the models, assumptions, and data used to estimate chemical risks are different from those used to estimate radionuclide risks. One important difference is how the cancer toxicity values (i.e., cancer potency factors versus risks factors) were developed. For both radionuclides and chemicals, cancer toxicity values are obtained by extrapolation from experimental and epidemiological data. For radionuclides, human epidemiological data form the basis of the extrapolation, while for many chemical carcinogens, laboratory experiments are the primary basis for the extrapolation. Another even more fundamental difference between the two is that cancer potency factors for chemical carcinogens generally represent an upper-bound or 95th percent confidence limit value, while radionuclide risk factors are best estimate values.

Estimation of human health risk from exposure to carcinogens is based on population risk and maximum individual risk, measured in cancer-related fatalities and incidence (for radionuclides) and cancer incidence (for chemical carcinogens). Most-exposed individual (MEI) risks are probabilities of developing or dying from cancer, but population risks are actual numbers of cancer deaths or incidence. For ER activities, depending on fate and transport models chosen, major health effects are quantified for a consecutive 7,000-10,000 yr. For WM activities, major health effects are usually quantified for a consecutive 10,000 yr. Seventy years is assumed to represent a typical individual's lifetime and is multiplied by 100 lifetimes to obtain a duration of 7,000 yr. These durations are chosen to address long time periods. The longer period for WM was chosen to be consistent with performance assessments of TSD facilities. The difference in time periods examined should not greatly affect the risk estimates; the difference between risks at 7,000 yr versus risks at 10,000 yr is not significant.

13.2 Quantification of Noncarcinogenic Hazards

Adverse human health effects associated with exposure to noncarcinogens or carcinogenic substances that exhibit noncarcinogenic effects are estimated differently than carcinogenic risk. The exposure level over a period of time is compared with an RfD derived for a similar exposure period. This ratio is known as the Hazard Quotient and is calculated as follows:

$$HQ = \frac{D}{RfD}, \qquad \textbf{[13.3]}$$

where HQ = Hazard quotient (unitless),

D = Exposure level or intake (mg/kg/d),

RfD = Reference dose (mg/kg-d).

In equation 13.3, E and RfD are expressed in the same units and represent the same exposure period. If the hazard quotient exceeds unity, the possibility exists for systemic toxic effects. The hazard quotient (HQ) is an indication that effects may occur and is not a statistical probability.

Since hazard quotients from noncarcinogens and individual risk estimates from carcinogens and radionuclides cannot be combined to form an overall individual risk estimate, the resulting risk estimate from the human health evaluation is reported in two different formats: maximum individual risk values (for chemical and radionuclide carcinogens) and hazard quotients (for noncarcinogens). Similar to carcinogens and radionuclides, health effect estimates from noncarcinogens are based on consecutive average 70-yr time periods for long time periods (i.e., 7,000 yr or 10,000 yr).

13.3 Risk Estimates for Multiple Substances

At most sites or facilities, the risk from simultaneous exposure to many hazardous substances must be estimated. The following equation estimates the incremental individual lifetime cancer fatality and cancer incidence risks for simultaneous exposure to several carcinogens. Despite the differences between chemical and radiological lifetime cancer risk estimation methods, cumulative risks attributable to multiple contaminants or multiple pathways still must be considered. Radiological fatalities must be reported separately and not summed with cancer incidence. Equation 13.4 does not consider synergism of contaminants. Synergy is a complicated issue that requires a toxicologist's site-specific evaluation. For a screening-level or installation-wide analysis, the level of detail required to evaluate synergy is unwarranted.

$$Risk_T = \sum Risk_i, \qquad \textbf{[13.4]}$$

where $Risk_T$ = Total cancer risk,

$Risk_i$ = Risk estimate for the ith substance.

Human health effects resulting from exposure to several noncarcinogens are addressed by a hazard index (HI) approach. The hazard index is equal to the sum of the hazard quotients. If the hazard index exceeds unity (1), there may be concern for potential health effects. The hazard index is derived from the following equation:

$$Hazard\ Index = \frac{E_1}{RfD_1} + \frac{E_2}{RfD_2} + \ldots + \frac{E_i}{RfD_i}, \qquad \textbf{[13.5]}$$

where Ei = Daily intake for the ith toxicant (mg/kg/d),

RfD_i = Reference dose for the ith toxicant (mg/kg/d).

In equation 13.5, E and RfD are expressed in the same units and represent the same exposure period.

13.4 Combining Risks Across Exposure Pathways

The total exposure of an individual or population to various chemicals equals the sum of the exposures by all pathways. For each pathway, the risk estimates and hazard indices have been developed for a particular exposure point and time period; they do not necessarily apply to other locations or time periods. Risks from two pathways are not summed unless the same individual (or population) is predicted to be exposed via both pathways at the same exposure point (or area) within the same 70-yr period. If it is considered appropriate to sum risks and hazard indices across pathways, cancer risks for each exposure pathway contributing to exposure of the same individual or population should be added, as follows:

$$Total\ Cancer\ Risk = Risk(EP_1) + Risk(EP_2) + \ldots + Risk(EP_i), \qquad [13.6]$$

where Risk(EP) is the risk from a given exposure pathway in a given 70-yr time period.

Noncarcinogenic effects from several exposure pathways can be estimated by separately calculating the total hazard index for each exposure duration (i.e., chronic, subchronic, and shorter-term). Total Hazard Index can be calculated using the following equation:

$$Total\ HI = HI(EP_1) + HI(EP_2) + \ldots + HI(EP_l), \qquad [13.7]$$

where HI(EP) is the hazard index from a given exposure pathway. Limitations associated with the hazard index are that (1) hazard quotients are combined for substances with RfDs based on critical effects of various toxicological significance, (2) RfDs of varying levels of confidence might be combined, and (3) combining many compounds that do not produce the same type of effect could overestimate the potential for effects. If a HI greater than unity (1) results from a risk evaluation, then a chemical-specific evaluation must be conducted to determine whether hazards from multiple noncarcinogenic substances are a concern. For screening-level analyses, however, the hazard index approach is appropriate (EPA 1989a), and additional chemical-specific analysis may be inappropriate.

13.5 EXPRESSIONS OF "RISK"

Noncarcinogenic health effects, chemical and radiological cancer incidence, and radiological fatality risks are not combined because their derivations are incompatible and they are based on different toxicity factors (cancer potency factors, risk factors, and RfDs). Health effects from exposure to carcinogens are expressed in terms of the probability of an individual obtaining cancer in a lifetime, while health effects from exposure to noncarcinogens are expressed in terms of the possibility for systemic toxic effects to occur or not to occur rather than a probability.

In this methodology, cancer risks are expressed in terms of maximum individual risk and population risk for radionuclides and carcinogenic chemicals. Both maximum individual and population risks are calculated independently for each exposure pathway and route. Hence, the risks are the highest risks calculated for contact with a contaminant through each exposure pathway and route. For example, the reported risk for inhalation could be the risk calculated for the person(s) living downwind and closest to the source, while the risk for drinking water for the same source could be attributed to a different group of people receiving water from an intake nearest a contaminant release to surface water. The equations described in Section 13.1 are used to estimate maximum individual risk for chemicals and radionuclides.

For noncarcinogens, health effects are expressed in terms of a hazard index, the sum of the hazard quotients for each noncarcinogenic contaminant. As with chemical carcinogens and radionuclides, hazard indices are calculated independently for each exposure pathway and route. Equation 13.7 is used to estimate the hazard index for noncarcinogens.

13.6 UNCERTAINTIES IN RISK ASSESSMENT

Uncertainties in the risk assessment are documented along with the final risk estimates to ensure that results are placed in proper perspective. The results of any risk assessment are not fully probabilistic estimates of risk, rather they are conditional estimates based on multiple assumptions about exposure, toxicity, and other variables. Therefore, uncertainties should be evaluated at each step of the risk assessment. These uncertainties should be documented to identify additional data collection needs and to determine which uncertainties contribute significantly to the overall uncertainty of the assessment. To reduce the uncertainty of the final risk estimate, assumptions and parameters are defined more precisely and considered further. Sources of uncertainty in risk assessment include the following (EPA 1989a):

- Definition of the physical setting
- Model applicability and assumptions

- Fate, transport, and exposure parameters
- Toxicity and risk characterization.

Defining the physical setting often requires judgements and assumptions. Within the definition of the physical setting, uncertainties about current and future land uses, potential current and future exposure pathways, and contaminants of concern are identified. The sources and quality of land use information, including the likelihood of occurrence for future land uses and exposure pathways, are described. A rationale for including or excluding each exposure pathway in the risk assessment is documented through the use of a conceptual model. Also, the reasons for and possible consequences of excluding a contaminant from the assessment are reported with the final risk results.

The applicability of the fate and transport models used in risk calculation and their mathematical formulations are also evaluated for uncertainties. The models are evaluated for their ability to approximate relationships among site-specific environmental conditions. Key assumptions used in the models are documented along with a discussion of their potential impacts on final risk estimates. The potential impacts of each assumption on risk estimates are reported in terms of direction (i.e., overestimated or underestimated risk) and magnitude (e.g., order of magnitude).

Uncertainties may also occur in fate, transport, and exposure parameter values. These uncertainties are discussed, and a rationale for their selection is provided. In addition, the potential magnitude and direction of bias (i.e., overestimation or underestimation of risk) resulting from assumptions and parameter values are discussed. Model uncertainty may result from the general limitations of mathematical models in trying to simulate an infinitely complex process using a finite number of variables. Model uncertainty may also result from the inappropriate application of a model to a particular scenario. Worst-case assumptions can be made where model uncertainty is high.

Scenario uncertainty may result from a generalized or incorrect conceptualization of a release and exposure scenario. More specifically, scenario uncertainty may result from generalized assumptions concerning the amount of contaminants released, the spatial distribution of potential receptors, and the intake parameters considered for the receptors. Worst-case scenarios are often used where scenario uncertainty is high to ensure a conservative estimate of risk.

Parameter uncertainty may result from sampling errors, variability, and/or the use of generic data. The fate and transport codes used to establish the unit risk database require large amounts of data, including meteorological measurements, hydrogeologic settings, and release parameters. Where possible, actual data are used, but generic data are often substituted where site-specific data are unavailable. Worst-case values are often used where parameter uncertainty is high to ensure conservative estimates of risk.

In addition, toxicity and risk characterization uncertainties are evaluated. Toxicity assessment uncertainties include potential for human toxicity, potential interactions from multiple chemicals, and derivation of

toxicity (e.g., human or animal data, duration of study, and any special considerations). If a contaminant is excluded from the risk assessment due to inadequate toxicity information, the contaminant's potential health effects and possible consequences of its exclusion are qualitatively documented.

For programmatic evaluations such as the PEIS, many uncertainties merely result in systematic error and may not affect the comparison of relative risks as a whole. Other uncertainties may be specific to a particular site. In either case, a qualitative and/or quantitative assessment of uncertainties should be incorporated into any presentation of risks. A qualitative analysis assesses the general accuracy and acceptability of data, algorithms, and assumptions used in the risk assessment and may also provide an indication of the direction of error associated with the risk estimates (i.e., overestimate or underestimate). A quantitative analysis provides an estimation of both the direction and magnitude of potential errors associated with the risk estimates.

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APPENDIX A: EXPOSURE FACTORS

Inconsistencies among exposure assumptions can arise from the following cases:

- Risk assessors use exposure factors derived from site-specific data.
- Risk assessors use their best professional to choose from a range of exposure factors published in open literature.
- Risk assessors make assumptions and choose exposure factor values based on extremely limited data.

To ensure consistency in the public and on-site population methodology, the exposure factors listed in table A.1-1 have been established for use in the intake and exposure equations. These values are based on EPA values in *RAGS Supplemental Guidance: Standard Default Exposure Factors* (EPA 1991b), unless otherwise indicated in table A.1-1. Site-specific parameter values should be obtained whenever possible; however, EPA-recommended values are used in the absence of site-specific data to ensure consistency among models and exposure scenarios.

Table A.1–1. Exposure Factors for the Offsite and Onsite Population Health Risk Evaluation

Exposure Factor Description	Exposure Factor Value[a]
Exposure duration (years)	30 yr[b] (to reflect time spent at one residence)
Exposure frequency (days/yr)	350 d/yr
Averaging time (years)	70 yr (carcinogens) 30 yr (noncarcinogens)[c]
Daily water intake (L)	2 L/d (adult)[d] 2 L/d (child)[d]
Daily soil and dust intake (mg)	100 mg/d (adult) 200 mg/d (child)
Daily inhalation intake (m^3)	20 m^3/d (adult - total) 15 m^3/d (adult - indoor)
Daily produce consumption from contaminated area (g)	80 g/d (leafy and other vegetables)
Daily finfish consumption from contaminated area (g)	54 g/d (finfish - recreational) 2.7 g/d (shellfish)[d]
Daily animal product consumption from contaminated area (g)	75 g/d (beef) 300 g/d (milk)
Body mass (kg)	15 kg (child 1-6) 70 (adult 7-71)
Skin surface area (cm^2)	19,400 cm^2 (adult male)[e]
Annual rate of incidental ingestion of surface water while swimming (L/yr)	0.91 L/yr[d]
Duration of showering (min/d)	12 min/d[d]

[a] *Source*: EPA (Environmental Protection Agency). 1991b. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors. Interim Final. EPA Office of Emergency and Remedial Response. OSWER DIR: 9285.6-03 Washington, D.C.

[b] For ingestion of soil and dust, duration is 6 yr (child 1-6) and 24 yr (adult 7-31), for a total of 30 yr.

[c] For exposure durations less than 30 yr, averaging time = exposure duration.

[d] *Source*: EPA (Environmental Protection Agency). 1989a. Risk Assessment Guidance for Superfund, (Vol. I) Human Health Evaluation Manual (PART A), Interim Final. EPA/540/1-89/002. Office of Emergency and Remedial Response. Washington, D.C.

[e] *Source*: Droppo, J. G. Jr., D. L. Strenge, J.W. Buck, B.L. Hoopes, R.D. Brockhaus, M.B. Walter and G.Whelan. 1989. Multimedia Environmental Pollutant Assessment System (MEPAS) Application Guidance. Volume I: User's Guide. Pacific Northwest Laboratory. PNL-7216 Vil. 1/UC-602,630.