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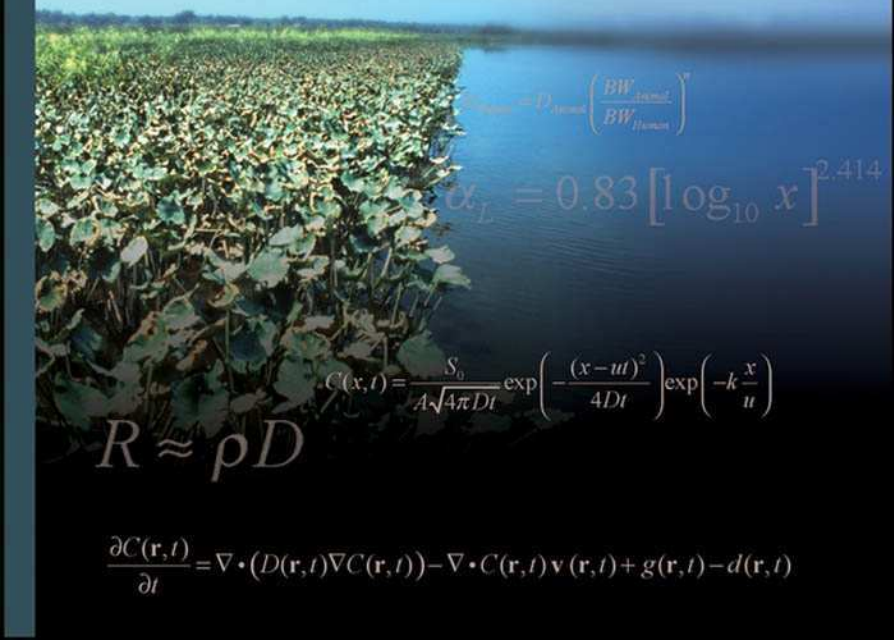
QUANTITATIVE ENVIRONMENTAL RISK ANALYSIS FOR HUMAN HEALTH



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QUANTITATIVE ENVIRONMENTAL RISK ANALYSIS FOR HUMAN HEALTH

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This book is dedicated to our wives:

Pam (Fjeld)

Wendy (Eisenberg)

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PREFACE

Environmental risk analysis for human health is the systematic analytical process of assessing, managing, and communicating the risk to human health from contaminants released to or contained in the environment in which humans live. It is a discipline central to the development of environmental regulations and the demonstration of compliance with those regulations. The goal of the book is to provide both the methods that are commonly used in environmental risk analysis and the underlying scientific basis for these methods. Although the text covers all three of the activities involved in environmental risk analysis (risk assessment, risk management, and risk communication), the focus is on environmental risk assessment, especially the computational aspects.

The book is designed for both academic and professional audiences. It may be used to instruct graduate students and advanced undergraduates with a background in a quantitative science or engineering. Practitioners may find the book useful for gaining an understanding of the science and methods outside their specialty. To make the text as accessible as possible, we presume no prior knowledge of environmental processes or environmental modeling, although we do expect readers to have a working knowledge of the fundamentals of physical science and mathematics through vector calculus, including some knowledge of statistics.

Development of a textbook on environmental risk analysis is a challenging undertaking. Environmental risk analysis encompasses a variety of diverse technical disciplines, including surface water hydrology, groundwater hydrology, air dispersion meteorology, chemical process engineering, toxicology, health physics, decision analysis, and risk communication, to name a few. Each of these disciplines is a separate field of technical study, often with individual academic curricula and professional certification. A significant challenge in developing the book has been choosing the appropriate degree of depth and detail for each of these many technical disciplines. Our approach is to provide enough information for each discipline so that the reader can develop an understanding of its role in the overall analysis, its methods, and significant uncertainties. Because the treatment of each specialty is limited, practitioners are likely to seek more focused texts for their particular specialty.

Certain perspectives on environmental risk analysis have shaped the treatment:

1. Most environmental risk analyses require a completely integrated approach to be successful.
2. The risk analysis is driven by the questions asked and the nature of the system—a single approach does not fit all.
3. Quantitative analysis is a useful tool, but analysts, reviewers, and managers should understand the limitations and uncertainties of the analysis.

4. Although risk assessment is the main focus of the book, risk communication, risk management, and regulatory requirements are essential features of most risk analyses and have a significant impact on virtually all technical aspects of the analysis.

Several unifying principles are used to address these perspectives and to assist in organizing the text:

1. The paradigm for the risk assessment calculation is four sequential steps (release assessment, transport assessment, exposure assessment, and consequence assessment) in which the output of one step provides the input to the next.
2. The contaminant transport equation and its solutions may be used to model a wide variety of environmental systems by choosing model aspects and conditions appropriate to the system.
3. The characterization of human health consequences as either deterministic or stochastic, as is commonly done in health physics, is extended to include both chemical and radioactive contaminants, thereby providing a unified basis for describing and quantifying human health consequences.
4. Both qualitative and quantitative uncertainties are important at every step of the analysis.

The book has its origins in class notes for a risk assessment course taught since the mid-1980s in the Department of Environmental Engineering and Science at Clemson University. These evolved into a set of instructional modules prepared for the U.S. Department of Energy and published in 1998. These modules were subsequently used at Clemson University and for six semesters of instruction in the Professional Master of Engineering Program at the University of Maryland. The book represents a significant enhancement and update of the original modules and has benefited from extensive classroom experience.

The overall organization of the book is as follows: Chapter 1 is an overview of environmental risk analysis and environmental risk assessment, Chapter 2 describes the modeling process and fundamentals of environmental models, Chapters 3 through 11 are concerned with environmental risk assessment, Chapter 12 deals with uncertainty and sensitivity analysis, Chapter 13 covers risk communication, Chapter 14 describes methods of risk management, and Chapter 15 presents environmental laws and regulations. Since a four-step paradigm is used for the risk assessment calculation, the risk assessment chapters are organized as follows: Chapter 3, release assessment; Chapter 4, generic transport; Chapters 5 to 8, surface water, groundwater, atmospheric, and food chain transport, respectively; Chapter 9, exposure assessment; and Chapters 10 and 11, basic human toxicology and dose–response; respectively. Much of the material presented in Chapters 2 through 11 is in the form of deterministic quantitative relationships. There are exceptions to this practice; for example, Chapter 3 contains an abbreviated treatment of probabilistic methods used for analyzing releases. For historical, pedagogical, and practical reasons, probabilistic methods are not described substantially until Chapter 12.

This approach allows treatment of the various disciplines in a simplified, largely deterministic fashion conducive to instruction at this level.

The book is designed to allow flexible approaches to instruction. We recognize that some readers will benefit from certain mathematical treatments, and some will not. To accommodate varying degrees of facility with mathematics, the book is structured to facilitate passing up mathematically demanding parts without interrupting the orderly presentation of material. Thus, selected sidebars, examples, and problems with heavy mathematical content can be skipped without seriously affecting the reader's ability to proceed through the remainder of the book. Similarly, Chapter 12, Chapter 14, or both may be omitted in a one-semester course. Our experience is that readers who have stronger backgrounds in mathematics have a greater appreciation for, and accrue greater benefits from, using the contaminant transport equation as a unifying theoretical basis for most of the mathematical models that are used in risk calculations. Consequently, the instructor must decide whether the material in Chapter 4 is appropriate for a given class. To fit the course into a single semester, some chapters will probably need to be skipped, depending on the course focus. For instructors wishing to emphasize the overall environmental risk analysis process, Chapters 13, 14, and probably 15 are essential; however, one or more of the environmental transport chapters (Chapters 5, 6, 7, or 8) could be omitted. For instructors wishing to emphasize the risk assessment calculation, all or parts of Chapters 13, 14, or 15 could be omitted.

We are indebted to the many people who have contributed to the book. We thank Sandra Clipp for her invaluable help in preparing the manuscript, Debbie Falta for checking the examples and assisting in preparation of the solutions manual, Rachael Williams for her careful review of Chapters 1 through 9, graduate students at Clemson University and in the Professional Master of Engineering Program at the University of Maryland for valuable comments and corrections, Mary Shirley for her assistance with the figures, and Tom Overcamp for his review of the atmospheric transport chapter. Thanks are also extended to Kevin Farley, David Hoel, Owen Hoffman, Tom Kirchner, Frank Parker, Art Rood, and Linda Wennerberg, who reviewed a set of educational modules that served as a precursor to the book. We also want to thank Jerry E. and Harriet Calvert Dempsey for financial support through their endowment to Clemson University.

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1 Introduction

Environmental risk analysis for human health is a systematic analytical process for assessing, managing, and communicating the risk to human health from contaminants released to or contained in the environment in which humans live. Environmental risk analysis encompasses a broad variety of disciplines and endeavors, including natural sciences such as geology, meteorology, hydrology, and ecology, which describe the natural environment in which contaminants migrate; biological sciences such as physiology, toxicology, anatomy, and cell biology, which describe the interaction and response of humans to environmental toxins; physical sciences such as physics and chemistry, which describe how contaminants migrate in natural systems; and decision and social sciences, which provide methods for making rational decisions and for communicating with stakeholders throughout the risk analysis process.

A well-established paradigm for risk analysis is that it is comprised of (1) risk assessment, (2) risk management, and (3) risk communication (ACS 1998). Most of this book addresses the environmental risk assessment component of environmental risk analysis. However, most environmental risk assessments are performed to answer a question or resolve an issue, such as: Is it safe for a proposed chemical plant to operate in this location? Because the *issue* drives the scope, depth, technical content, cost, and schedule of the risk assessment, we also address the risk management and risk communication components of environmental risk analysis.

Much of the material presented in Chapters 2 through 11 is in the form of deterministic quantitative relationships. There are exceptions to this practice; for example, Chapter 3 (release assessment) contains an abbreviated treatment of probabilistic methods used for analyzing releases. Probabilistic methods are not introduced until Chapter 12 (uncertainty analysis). There are historical, pedagogical, and practical reasons for this approach. Historically, environmental risk assessment has used deterministic methods to estimate impacts on (i.e., "risks" to) exposed persons. Currently, many regulatory compliance requirements are of a deterministic nature. Because environmental risk analysis involves a blend of so many separate disciplines, an introductory textbook such as this best treats these disciplines in a simplified, largely deterministic fashion. To keep the book to a reasonable size, it is virtually impossible to treat each discipline probabilistically. Also, many probabilistic risk assessments are conducted using a probabilistic driver to repeat a deterministic calculation using different input parameter values.

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

1.1 RISK ANALYSIS

According to the Society for Risk Analysis (SRA 2003), **risk analysis** is

> a detailed examination including risk assessment, risk evaluation, and risk management alternatives, performed to understand the nature of unwanted, negative consequences to human life, health, property, or the environment. . . .

The Food and Drug Administration (FDA 2002) definition of risk analysis focuses on activities accomplished by its components:

> Risk analysis is a tool to enhance the scientific basis of regulatory decisions. It includes risk assessment, risk management and risk communication activities. Each component has unique responsibilities: Risk assessment provides information on the extent and characteristics of the risk attributed to a hazard. Risk management includes the activities undertaken to control the hazard. Risk communication involves an exchange of information and opinion concerning risk and risk-related factors among the risk assessors, risk managers, and other interested parties.

Given that the three-part paradigm for risk analysis is chosen from the very large universe of risk analysis paradigms, it is important to clarify the functions of the three components (shown schematically in Figure 1.1). Risk analysis is the overall

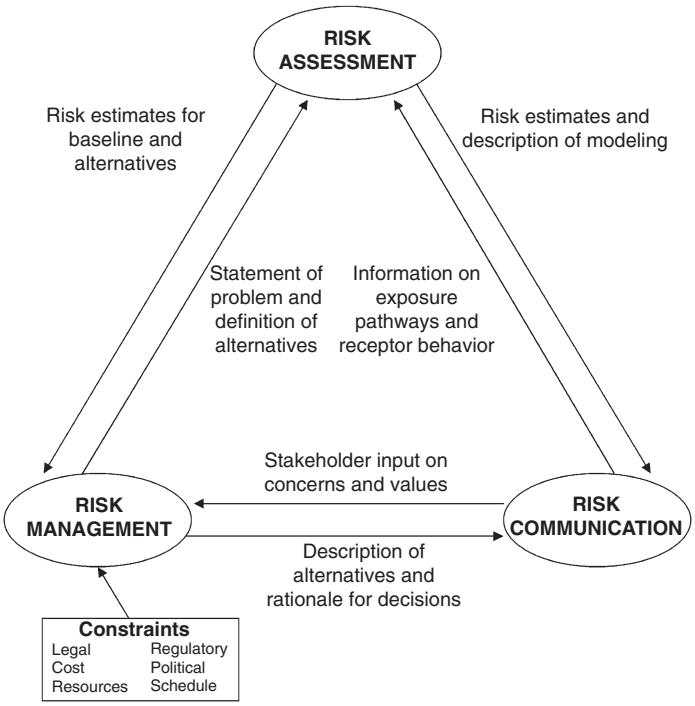


Figure 1.1 Relationships among the three components of risk analysis: risk assessment, risk management, and risk communication.

activity and each component—risk assessment, risk management, and risk communication—is a subsidiary activity required to accomplish the overall goal. It should be recognized that an environmental risk analysis is most often conducted by an entity with the responsibility and authority to make a decision; most frequently, the entity (i.e., the risk manager) is a government agency. The distinction between risk assessment and risk management has been stated succinctly as follows: "Risk assessment is the use of the factual base to define health effects of exposure of individuals or populations to hazardous materials and situations. Risk management is the process of weighing policy alternatives and selecting the most appropriate regulatory action, integrating the results of risk assessment with engineering data and social, economic, and political concerns to reach a decision" (NAS–NRC 1983). More recently, the Presidential/Congressional Commission on Risk Assessment and Risk Management (1997) defined risk management as "the process of identifying, evaluating, selecting, and implementing actions to reduce risk to human health and ecosystems," and risk assessment as the process of "considering the nature, likelihood, and severity of adverse effects on human health or the environment." In both of these documents, environmental risk assessment refers to the technical process through which quantitative estimates of risk are obtained, whereas **environmental risk management** refers to the broader process of balancing risks, costs, and social values. In this book, **environmental risk assessment** is defined as the process of making a quantitative estimate of the human health risks resulting from the release or potential release of contaminants to the environment. Environmental risk management considers both the technical results of an environmental risk assessment and the economic, social, legal, cultural, ethical, and political considerations that must be taken into account when making decisions in a broad societal context.

Risk communication refers to interactions among stakeholders, risk assessors, and risk managers. The objectives, often mandated by law, procedures, or good practices, are to assure that important issues are identified for analysis and to facilitate stakeholder understanding of the risk management decisions. Effective risk communication enhances the acceptance of risk analysis by inviting stakeholders to become involved in the analysis process and by assuring that stakeholder concerns are considered. Good risk communication requires both effective transmission and reception of information; it is not merely a means for presenting the results of a risk analysis to stakeholders.

Although in this book we adopt the idea that risk analysis is comprised of risk assessment, risk management, and risk communication, there has been a trend to blur the boundaries between these activities. For example, the American Society of Mechanical Engineers recently stated (ASME 2002): "It has been common practice among practitioners of risk analysis to make distinctions among the various 'phases' of risk analysis (e.g., risk assessment, management, communication). These distinctions are not useful in the overall debate. In attempting to develop a broad consensus on methodology, all aspects of the process should be integrated." Another view essentially incorporates risk assessment and risk communication into risk management. The Presidential/Congressional Commission on Risk Assessment and Risk Management (1997) articulates a six-stage risk management framework: "(1) Define the problem and put it in context; (2) analyze the risks associated with the problem in context; (3) examine options for addressing the risks; (4) make

decisions about which options to implement; (5) take actions to implement the decisions; (6) conduct an evaluation of the actions."

Furthermore, all stages are to engage stakeholders, and iterations are to be performed as warranted by new information. Nevertheless, other approaches recognize that close association of risk management and risk assessment has the potential for undermining the objectivity of the risk assessment. For example, the U.N. Food and Agriculture Organization states (FAO 2003): "There should be a functional separation of risk assessment and risk management in order to ensure the scientific integrity of the risk assessment, to avoid confusion over the functions to be performed by risk assessors and risk managers, and to reduce any conflict of interest. However, it is recognized that risk analysis is an iterative process, and interaction between risk managers and risk assessors is essential for practical application." The three-component paradigm for risk analysis is adopted in this book in part to make the explanation and understanding of these components easier. Even though a linear, one-pass approach is presented here, in practice, risk analysis usually requires significant communication and feedback among components (some are indicated in Figure 1.1) and multiple iterations within each component and for the entire process.

For brevity, in this book the terms environmental risk analysis, environmental risk assessment, environmental risk management, and environmental risk communication are often shortened to risk analysis, risk assessment, risk management, and risk communication, respectively. In so doing, the possibility of confusion is recognized, as there are other types of risk (e.g., financial, political, technological, programmatic) that have nothing to do with human health or environmental contaminants. The reader is cautioned to use these abbreviated forms with care whenever there may be ambiguity about the meaning.

1.2 RISK

Defining risk is a challenging problem. Physical scientists may tend to prefer a quantitative view of risk, and social scientists may favor inclusion of qualitative social and psychological elements in defining risk. Psychological and sociological studies have shown that a person's perception of risk can be affected by a myriad of objective and subjective factors. A quantitative approach to defining risk as appropriate for quantitative analysis has been adopted. However, as discussed in Chapters 13 and 14, the subjective and qualitative elements of risk are of great importance in the broader context of risk communication and risk management. Thus, it is appropriate to recognize the practical limitation of any particular quantitative definition that an analyst may use in performing an assessment.

A general definition of **risk** is: "the probability that a substance or situation will produce harm under specified conditions" (Presidential/Congressional Commission on Risk Assessment and Risk Management 1997). Under this definition, risk is a combination of (1) the probability that an adverse event will occur (such as a specific disease or type of injury) and (2) the consequences of the adverse event. Another definition of risk is "the potential for realization of unwanted, adverse consequences to human life, health, property, or the environment; estimation of risk is usually based on the expected value of the conditional probability of the

event occurring times the consequence of the event given that it has occurred" (SRA 2003).

The definitions above imply a two-dimensional construct that includes (1) the probability of an adverse event (i.e., a hazard) and (2) the consequences of the event. A **hazard** is a potential source of danger; and hazards are a normal part of everyday experience, ranging from the familiar (the electrical energy in household outlets or an automobile accident) to the exotic (the existence of undiscovered viruses or a meteorite falling from the sky). The distinction between hazard and risk is stated succinctly by the Presidential/Congressional Commission on Risk Assessment and Risk Management (1997) as: "Risk encompasses impacts on public health and on the environment, and arises from exposure and hazard. Risk does not exist if exposure to a harmful substance or situation does not or will not occur. Hazard is determined by whether a particular substance or situation has the potential to cause harmful effects."

For each hazard, there is a chance or likelihood, which is expressed as a probability, of contacting or experiencing the hazard. For example, earthquakes are a natural hazard that may cause injury, death, and property damage. The consequence for human health can range from no injury to death, depending on the severity of the earthquake, distance from the epicenter, and other factors. Quantification of risks such as this are expressed through a risk curve. The ordinate (*y*-axis) of a risk curve is exceedance probability or exceedance frequency, which is the probability or frequency that the severity of the effect exceeds the corresponding value on the abscissa (*x*-axis). A risk curve for earthquake-caused fatalities is presented in Figure 1.2. The ordinate is the number of earthquakes per year that cause the number of deaths exceeding the value given on the abscissa. For example,

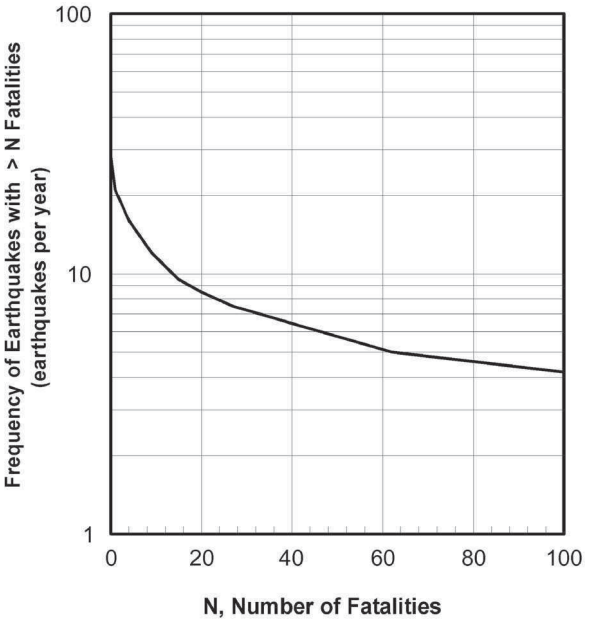


Figure 1.2 Risk curve for earthquake-caused fatalities. (From USGS 1997.)

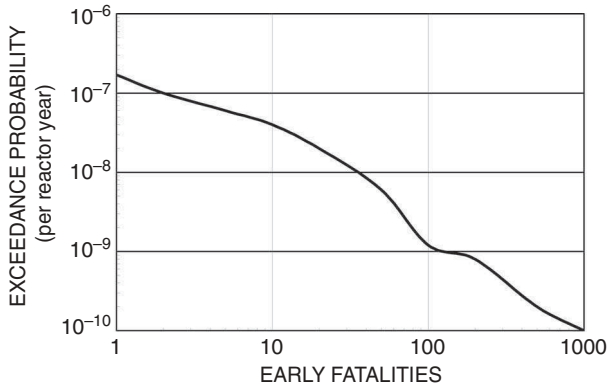


Figure 1.3 Risk curve for early fatalities as a result of a nuclear reactor accident. Results are for Unit 1 of the Surrey Power Station near Williamsburg, Virginia. (From NRC 1991.)

the number of earthquakes causing more than 100 deaths is approximately 4 per year. Another exceedance curve is shown in Figure 1.3 for nuclear reactor accidents. The abscissa is the number of fatalities occurring as a result of a reactor accident, and the ordinate is the corresponding exceedance probability. For example, the probability per year of an accident occurring and causing more than 100 fatalities is approximately 10^{-9}. Thus, the probability of an accident occurring and causing more than 100 fatalities during 40 years of operation would be approximately 4×10^{-8}.

► **Example 1.1**

Use Figure 1.3 to find the following:

(a) The probability of more than 10 fatalities in one year of reactor operation.
(b) The probability of more than 10 fatalities in 40 years of reactor operation.
(c) The probability of one or fewer fatalities in 40 years of reactor operation.

Solution

(a) Reading from the graph, the exceedance probability per year corresponding to more than 10 deaths is approximately 4×10^{-8}.
(b) As stated, the probability of more than 10 deaths per year of operation is 4×10^{-8}. Thus, the probability for 40 years of reactor operation is approximately $(40\,\text{yr})(4 \times 10^{-8}\,\text{yr}^{-1}) = 1.6 \times 10^{-6}$.
(c) From the graph, the probability of more than one fatalities is approximately $1.6 \times 10^{-7}\,\text{yr}^{-1}$. Thus, the probability of one or more fatalities for 40 years of reactor operation is $(40\,\text{yr})(1.6 \times 10^{-7}\,\text{yr}^{-1}) = 6.4 \times 10^{-6}$. The probability of less than one fatality is then $1 - 0.0000064 = 0.9999936$.

The two-dimensional definition of risk clearly articulates that risk may be thought of as a particular *un*desirable outcome and the probability of that outcome. Clearly, any particular outcome is uncertain; however, low-consequence outcomes typically have a probability of occurrence close to 1. In the context of environmental risk analysis for human health, the probability represented in the risk curve may reflect uncertainty due to a variety of factors, either individually or together: (1) the occurrence of some event that could initiate an environmental release, (2) the probability of a release given an initiating event, (3) the likelihood that a contaminant would migrate to a particular location, (4) the likelihood that a person would be exposed at that location, and (5) the probability, given an exposure, that a person would respond with a particular level of injury. Variability of risk in space, time, and across a population is considered in Chapter 12. For some problems in environmental risk analysis, either the health impact or its probability of occurrence may be "degenerate" or "trivial"; that is, probabilities may be zero or one and health impacts may be zero. However, even in these degenerate cases, the risk paradigm may be used.

Another approach (Kaplan and Garrick 1981) defines risk as a triple (sometimes called the Kaplan–Garrick **risk triple**):

$$R_i = \langle S_i, P_i, C_i \rangle \tag{1.1}$$

where S_i is the scenario i, P_i the probability of scenario i, and C_i the consequence of scenario i. In this construct the scenario represents what can happen (or the set of conditions), the probability represents how likely it is, and the consequence represents the impacts. This mathematically robust definition of risk has the advantage of directly representing a commonsense understanding of the concept; for example, the definition promulgated by the Presidential/Congressional Commission on Risk Assessment and Risk Management (1997) of risk as the "probability that a substance or situation will produce harm under specified conditions" reflects the quantitative definition succinctly.

Although Eq. 1.1 represents the consequence C_i as a scalar quantity, it is sometimes useful to consider it to be a vector with various components. For example, the accidental release of a contaminant from an industrial facility could cause different classes of consequences, such as injury, prompt death, latent cancer fatality, and genetic damage. Although each of these is a human health impact, they are qualitatively different. Different scenarios may produce a different distribution of consequences among these categories. Another common partitioning of consequences is to separate health effects among the general public from those among workers at a facility. The distribution of consequences among categories can become especially important when evaluating alternative risk management strategies. For example, some strategies for reducing public consequences may produce unacceptably high consequences for workers. This type of trade-off is discussed in more depth in Chapter 14.

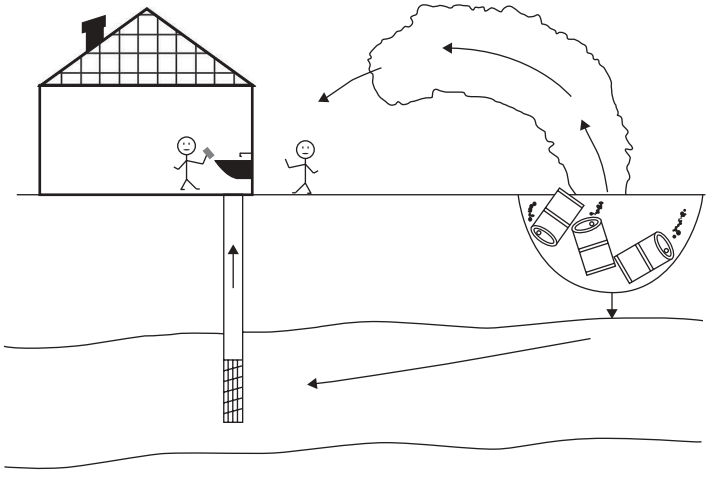
Based on the above, environmental risk as used in this book is the risk triple. The scenario represents the conditions of contaminant release, contaminant transport, and human exposure; the probability is the probability of the scenario; and the consequence is the health effect (more generally, the consequence would include impacts on human health, ecological effects, and aesthetic effects).

1.3 CONTAMINANTS IN THE ENVIRONMENT

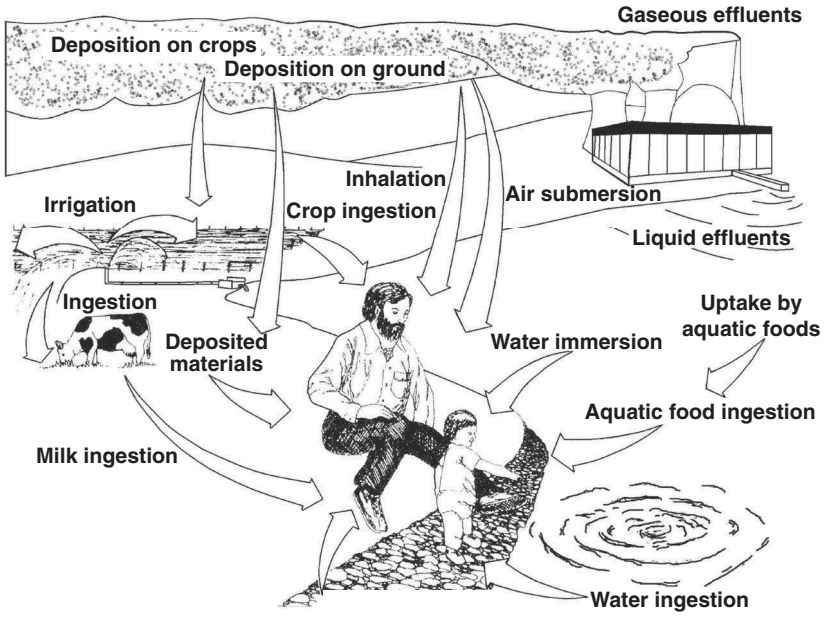
In the context of environmental risk assessment, a contaminant can be defined as a substance in the environment that is capable of causing adverse human health, ecological, or aesthetic effects. Recognizing that virtually any element or compound in sufficient quantity is capable of causing harm, identification of specific substances as contaminants requires the exercise of judgment. The Environmental Protection Agency (EPA) defines a **contaminant** as "any physical, chemical, biological, or radiological substance or matter that has an adverse effect on air, water, or soil" (EPA 2005). Environmental contaminants can be the result of either natural processes or human activities. Examples of naturally occurring contaminants include airborne particulate matter and gases from volcanic activity or forest fires; waterborne metals such as arsenic, mercury, or uranium decay products due to leaching from soil; and aflatoxin B1 in grains due to mycotoxin-producing molds. Examples of anthropogenic contaminants include ozone and related photochemical oxidants in air due to emissions from internal combustion engines, chlorinated hydrocarbons in air and water from the use of pesticides and herbicides, and radionuclides such as hydrogen-3, cesium-137, and plutonium-239 from nuclear reactors.

Many anthropogenic contaminants are routinely released to the environment because they either serve some useful purpose (such as protecting crops against insects or disease) or they are the by-products of an activity (such as the generation of electricity) that society considers to be beneficial. Others are released accidentally as a result of equipment failure, human error, or a natural phenomenon (such as a flood or earthquake). The conceptualizations in Figure 1.4 illustrate the release, transport, and human exposure of contaminants from (a) buried wastes and (b) an operating facility, both of which are typical scenarios encountered in environmental risk assessment. Risk assessment scenarios generally have the following elements: an actual or potential source of a contaminant, mechanisms for the release of the contaminant to the environment, environmental pathways through which the contaminant is transported and transformed, routes or mechanisms of exposure to humans or other receptors, and the possibility of an adverse human health, ecological, or aesthetic effect.

There exist plentiful historical examples for which contaminant releases resulted in documented adverse human health or ecological effects (Table 1.1). These range from the classic water pollution example in which John Snow traced cholera to a contaminated well in nineteenth-century London (Snow 1855) to the induction of thyroid cancer in children in Belarus and the Ukraine as a result of radionuclides released in the Chernobyl accident in 1986 (UNSCEAR 2000). With examples such as those in Table 1.1 in mind, questions then arise when contaminants are found in environmental media (e.g., pharmaceuticals and endocrine-disrupting chemicals in water, fine particles in urban atmospheres, or pesticides and herbicides in food) or when permits are sought for certain types of facilities (e.g., for hazardous waste incinerators, radioactive waste disposal sites, or chemical manufacturing facilities). Do these pose a threat? The intuitive answer can range from alarm to indifference, either of which may be appropriate but neither of which is defensible without a systematic informed evaluation. Such an evaluation is achieved by an environmental risk assessment, which may generally be defined as the process of making a



(*a*)



(*b*)

Figure 1.4 Human exposures due to routine releases of environmental contaminants: (*a*) buried waste (adapted from EPA 1989); (*b*) facility release (adapted from DOE 1978).

quantitative estimate of the human health, ecological, or aesthetic effects of the release or potential release of contaminants to the environment. It is a systematic process for obtaining an objective estimate of the risk posed by environmental contaminants. Contaminant effects may be considered to be in three broad classes: effects on human health, such as cancer or systemic disease; impacts on ecosystems, such as loss of species or decreased species diversity; and adverse impacts on

TABLE 1.1 Examples of Contaminant Releases Resulting in Adverse Human Health or Ecological Impacts

Location	Date	Contaminant	Effect	Reference
London	1852	Human waste	Cholera	Snow 1855
Ducktown, TN	1900s	SO_2 from a smelter	Death of vegetation	Wagner 1971
Donora, PA	1948	SO_2 and particulate matter from various industries	20 immediate deaths; 5910 cases of respiratory distress in a population of 14,000	Waldbott 1978
Minimata, Japan	1950s	Methyl mercury	Dead fish, birds, and cats; nervous disorders and birth defects in humans	CERHR 2006
Seveso, Italy	1976	Dioxin	Chloracne, death of farm animals, high female/male birth ratio	CDC 2006
Bhopal, India	1984	Methyl isocyanate released in an accident at a chemical plant	3800 immediate deaths; other effects (lungs, eyes, stillbirths) in 170,000 survivors	EPA 1986
Ukraine and Belarus	1986	Radioactivity released from the Chernobyl accident	31 immediate deaths; increased thyroid cancer in children	UNSCEAR 2000
Sweden and northeastern United States	Present	Acid rain due to oxides of nitrogen and sulfur in the atmosphere from combustion of fossil fuels	Widespread damage to forest ecosystems and freshwater fish habitats	Lloyd 2001

aesthetic qualities of the natural environment, such as reduced visibility due to air pollution or odors from industrial operations. Conceptually, the risk assessment framework presented in this book for human health effects could be extended to ecological and aesthetic effects, but a description of the implementation of such an approach is beyond our scope.

1.4 USES OF ENVIRONMENTAL RISK ASSESSMENT

There are various reasons for performing environmental risk assessments, most of which serve one of the following generic purposes: risk management for an existing or proposed facility, development of regulations, demonstration of compliance with regulations, litigation, or scientific inquiry. In practice, most risk assessments are performed for the purpose of risk management or to demonstrate regulatory compliance. As introduced in Section 1.1 and covered in detail in Chapter 14, risk assessment is only one component of a larger risk management process, which is usually conducted in a regulatory context. At both the federal and state levels, there is an abundance of environmental regulation with broad policy goals (e.g., protection of human health or the environment) which either implies or is interpreted by

regulators to imply that risk assessment is required. For example, the Comprehensive Environmental Response, Compensation and Liability Act (42 U.S.C. 9601 et seq.; CERCLA or "Superfund") stipulates that hazardous waste cleanup levels must assure "protection of human health and the environment" against contaminants that "will, or may reasonably be anticipated to cause" certain adverse effects. Another risk management context in which risk assessments are needed is to support remediation programs for complex contaminated sites. For example, the Department of Energy's environmental management program lists the elimination of urgent risks and risk reduction as two of its objectives. Risk assessments are used to evaluate the level of risk posed by contaminated sites, to identify sites that pose urgent risks, to establish cleanup priorities, and to determine the reduction of risk that can be obtained through remediation. Another practical application of risk assessment is in regulatory compliance. The operator of a proposed facility might be required to perform a risk assessment either to show compliance with numerical regulatory requirements or to provide a regulatory agency with evidence that the facility will not result in harm to public health or the environment. On a smaller scale, a person might want to estimate the risk to herself or to a family member, due to lead in drinking water, mercury in fish, or fine particles in the atmosphere.

▶ Example 1.2

In 1985, the EPA established the first set of risk-based standards for volatile organic compounds in drinking water. These standards were applied to eight compounds, five of which were considered to be carcinogens. The concentration limits that were established at that time yielded lifetime cancer probabilities that ranged from 2×10^{-6} (for TCE) to 1×10^{-4} (for 1,1-dichlorobenzene). These risk estimates were based on the consumption of 2 L of water per day for 70 years.

Sometimes, risk assessments are undertaken to determine if a problem exists that requires a response. Such risk assessments are usually conducted as part of a risk analysis that includes risk management and risk communication. Such studies may be conducted or sponsored by regulatory agencies to determine if some sort of regulatory action is required. Examples include those above, in which there is concern over the impact of a given instance of environmental contamination or the potential impact of an industrial plant or waste disposal facility. This also includes retrospective risk assessments in which an attempt is made to estimate the risks posed by historical contaminant releases from a facility. If a reasonable case is made that historical releases caused significant harm, compensation may then be paid to those affected.

■ Fernald Risk Assessment

The Fernald Feed Materials Production Center (FMPC), part of the U.S. Department of Energy's nuclear weapons production complex, operated from 1951 to

1988. The FMPC mainly produced uranium metal at a 1000-acre site located about 15 miles northwest of Cincinnati, Ohio. During operations, radioactive material was released from the site into the air from waste material stored in two large silos and from waste burned or buried in pits and incinerators. Increased risks of cancer in the population near the facility resulting from radioactive material releases from the FMPC were estimated by two risk assessments; phase I (CDC 1998) addressed lung cancer and phase II (CDC 1999) addressed kidney cancer, female breast cancer, bone cancer, and leukemia. The phase I study estimated a median lifetime dose of 0.45 Sv (sievert), principally from inhalation of radon decay products, which was estimated to produce an excess of 85 lung cancer deaths in an exposed population of about 50,000. When some types of uncertainties are considered, the estimated doses ranged from 0.12 to 1.74 Sv, corresponding to an estimated number of excess lung cancer deaths of 25 to 309. This implies an increase of 1 to 12% in cancer incidence from 1951 to 2088 in the exposed population. The phase II study estimated upper bound incidences of various cancer types resulting from exposure to releases from the FMPC. Radiation doses were estimated for hypothetical individuals who ate contaminated foodstuffs (vegetables, fish, milk, eggs), breathed contaminated air, and resided on contaminated soil. The upper bound cancer estimates were 23 for leukemia, 4 for kidney cancer, 3 for female breast cancer, and 4 for bone cancer in a population estimated at 46,000.

In implementing legislative initiatives, regulatory agencies frequently use risk assessment to develop limits on contaminant concentrations in air or water that meet a numerical risk goal. For example, the Clean Air Act (42 U.S.C. 7401 et seq.) requires the EPA to issue ambient standards sufficient to "protect the public health with an adequate margin of safety," issue standards for sources of hazardous pollutants which are "known or anticipated to cause adverse effects," and set supplemental emission standards if it is found that the standards do not provide an "ample margin of safety" (for known and potential carcinogens, generally defined as a 1 in 10,000 to 1 in 1 million lifetime chance of cancer). Similarly, the Atomic Energy Act (42 U.S.C. 2011 et seq.) stipulates the formulation of standards for the "protection of the public health, safety, and the environment" from radiation hazards.

Suits ("toxic torts") may be brought alleging that a given effect (e.g., cancer, birth defect, mental disorder) occurred as a result of exposure to a given substance. Risk assessments can be used by either defendants or plaintiffs to support their side of a case. Risk assessments have been used in cases involving radiation hazards, dioxin, Agent Orange, and volatile organic compounds, to name a few. Risk assessments can be used to provide weight of evidence that a toxic response may or may not be due to the exposure in question and thus may or may not be eligible for redress under the law. The scientific inquiry purpose of risk assessment is frequently tied to an investigation of new or alternative methods of analysis. Another issue for scientific inquiry is the investigation of contaminants or impacts not previously considered in a regulatory context, which could be significant.

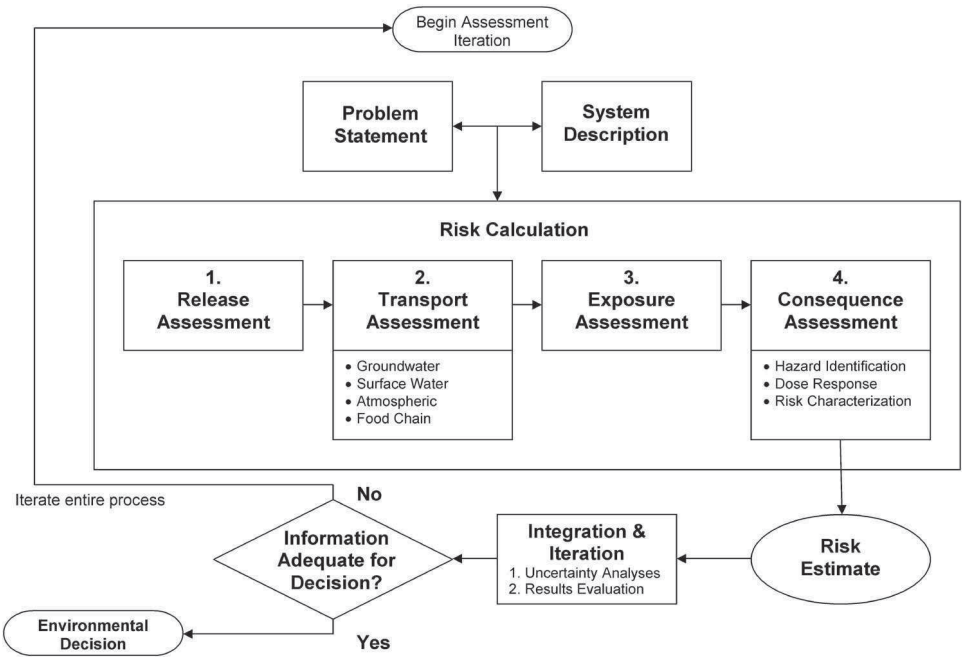


Figure 1.5 Risk assessment process.

1.5 RISK ASSESSMENT PROCESS

The overall risk assessment process consists of four major components: problem statement, system description, risk calculation, and integration and iteration (Figure 1.5). The focus of this book is on the risk calculation component, which is the computational core of the overall process. However, this computational core depends greatly on other parts of the risk assessment process, particularly the problem statement and system description. Also, during the final step of integration and iteration a decision is made to determine whether the assessment is complete and adequate or whether certain aspects need to be revisited. In reality, the risk assessment process may be much more complicated and nonlinear, with multiple iterations (Morgan and Henrion 1990, Sec. 3.8.8).

1.5.1 Problem Statement

Virtually all risk assessments are performed to answer a question. Even risk assessments pursuing scientific inquiry have a hypothesis to be tested, consistent with the scientific method. The question asked has a great influence on the scope, level of detail, and focus of the risk assessment, including the time scale, the spatial scale, the contaminants considered, the endpoint of the assessment (the measure of risk or impact), the persons at risk, and the treatment of uncertainty. For example, retrospective assessments of doses and risks, called dose reconstructions, have been performed to determine whether previous operations at former DOE weapons

facilities were harmful to the adjacent population. Some dose reconstructions have been directed toward an entire population within broad geographical boundaries; others have been directed to populations of special concern, such as nearby Native American groups. Dose reconstructions are focused on the releases of radioactive and chemical contaminants from the site but are not concerned with releases from other sources or contaminants already present in the air, water, and soil. Dose reconstructions usually consider uncertainties, so both the average dose and risk may be reported as well as their ranges.

In addition, the statement of the problem might implicitly or explicitly mandate certain assumptions or methods. For example, the EPA uses the concept of "maximally exposed individual" in several regulatory applications. A maximally exposed individual is defined as "the single individual with the highest exposure in a given population" and is used synonymously with the worst-case or bounding estimate. The concept is found in regulations for high-level nuclear waste and Superfund sites, where an upper limit on the dose or risk to the maximally exposed individual is prescribed. In practice the analyst may limit the assessment by choosing the nearest accessible location to the site as the location for the maximally exposed individual and by using pessimistic values for variables associated with environmental transport and uptake of contaminants. In this fashion, calculations at multiple locations and for multiple values of a large number of variables are avoided.

1.5.2 System Description

The system description includes qualitative and quantitative information about physical processes in the system, the time scales of interest, and the geometry and physical configuration of the system. The system description provides key information for the risk calculation component of the risk assessment, including the release form, the temporal character of the releases, transport mechanisms and transport media, biota at the site, land-use characteristics, human activities in the vicinity, and toxicological characteristics of the contaminants of concern. From this information the analyst can formulate a conceptual model for each step of the risk calculation. For example, a dose reconstruction (CDC 2005) was performed for the Savannah River Site, a DOE facility used to manufacture material for nuclear weapons. Although several instances of groundwater contamination on the site had been documented, the dose reconstruction did not consider radionuclide migration by the groundwater pathway because the contaminated groundwater moves so slowly that it had not yet migrated past the site boundary. However, since air and surface water releases were well documented, the conceptual model included migration in the air and in the Savannah River.

1.5.3 Risk Calculation

When applied to human health effects, the objective of the risk calculation component of the risk assessment process is to produce a quantitative estimate of human health risk due to the release of a contaminant to the environment. The process for making this estimate of health effects can be formulated in different ways. In this book it is presented as four sequential steps (Figure 1.6): release

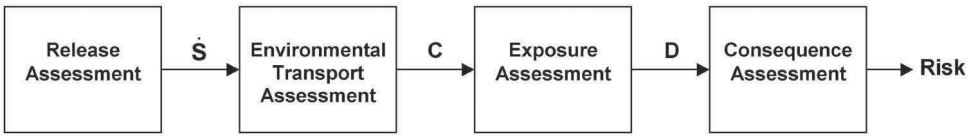


Figure 1.6 Risk calculation component of the risk assessment process.

assessment, transport assessment, exposure assessment, and consequence assessment. Each step has a qualitative component and a quantitative component. Qualitative components are those that do not result from calculations: for example, identification of contaminants or of potentially exposed populations. The quantitative output of each step is the input to a subsequent step, ultimately leading to a quantitative estimate of health risk. Each step and the quantitative results are described below.

Traditionally, the risk calculation step in the overall risk assessment process has been denoted by the term "risk assessment". However, as noted, the actual process of assessing risk involves more than just the risk calculation itself. The risk calculation component of the process can be formulated in various ways; the end result of each is a quantitative estimate of health risk. The American Association of Engineering Societies (AAES 1996) casts the risk calculation step in a fashion similar to that presented in this book. They specify the following three steps: source assessment, exposure assessment, and effects assessment. In the AAES formalism, risk characterization is a separate step that combines the results of effects assessment with risk assessment policy. In 1983, the National Academy of Sciences (NAS–NRC 1983) cast risk assessment in terms of the following four steps: hazard identification, dose–response assessment, exposure assessment, and risk characterization. The EPA has modified this slightly for the baseline risk assessments they require for the CERCLA process. The EPA baseline risk assessment (EPA 1989) consists of data collection and evaluation, exposure assessment, toxicity assessment, and risk characterization, as shown in Figure 1.7. An important perspective provided by the NAS and EPA formulations is that the dose–response assessment (NAS) or toxicity assessment (EPA) depends primarily on the contaminant, its form, and to a lesser degree the nature of the exposed population (e.g., age, gender). Therefore, generic toxicity data may be obtained independently from site investigations and may be used at a variety of sites. This is significant for organizing significant amounts of work to accomplish the baseline and other risk assessments. The linear sequence of four calculational steps adopted in this book is intended for use by the risk analyst, who will produce an estimate of health risk by executing the sequence of steps. That being said, it is important to point out again that in practice there may be iterations within or among steps or iterations with other elements of the overall risk assessment process.

1.5.3.1 Release Assessment Release assessment is identification of contaminants and quantitative estimation of release probabilities and release rates into the environment. Contaminant identification is accomplished by direct measurement of inventories or effluents, process knowledge, and an audit of facility records. For convenience, in this book contaminants are grouped into five discrete categories:

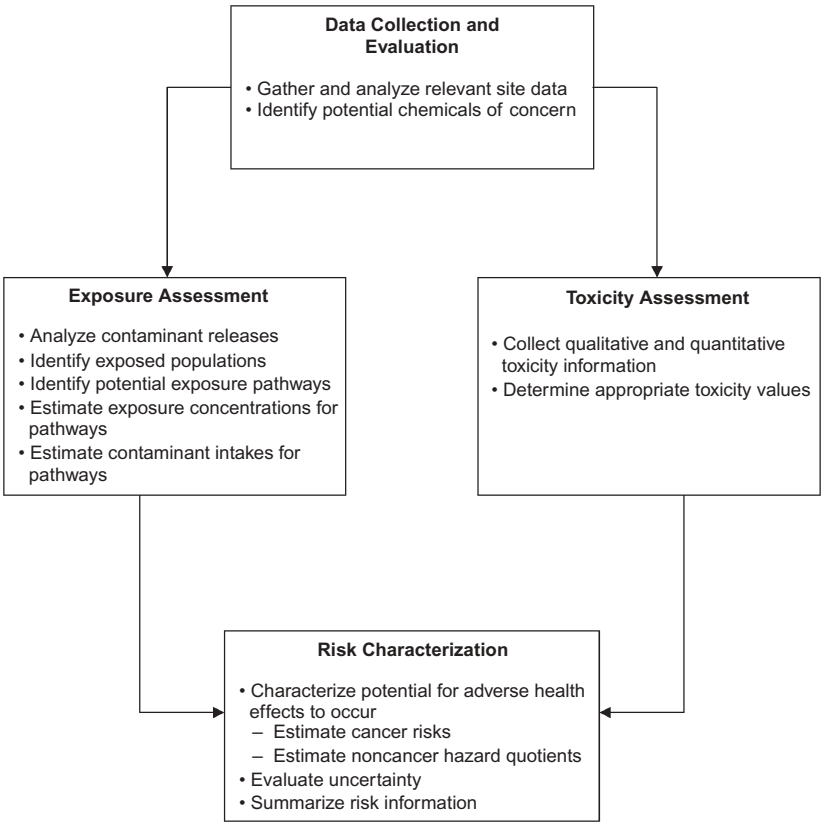


Figure 1.7 EPA and NAS formulations of the risk calculation component of the risk assessment process.

(1) organic compounds such as TCE, PCE, and CCl_4; (2) inorganic compounds such as SO_2 and NO_x; (3) metals such as lead, mercury, and chromium; (4) radionuclides such as ^{3}H, ^{90}Sr, ^{137}Cs, and ^{239}Pu; and (5) miscellaneous contaminants such as particulate matter, asbestos, and pathogens. Contaminant identification also includes the physicochemical form of the contaminants and the environmental media—atmosphere, soils, groundwater, and surface water—into which the contaminants are released.

The quantitative result of release assessment is **contaminant emission rate**, $\dot{S}$, which is the amount of contaminant released per unit time. For chemical contaminants, the emission rate is contaminant mass per unit time [M/T]; for radiological contaminants, it is the amount of radioactivity per unit time [activity/T]. The emission rate may have both a spatial and a temporal dependence, and it can result from either normal facility operation or an accident. Accidental releases occur as the result of an unlikely event (such as an earthquake, tornado, or fire) or a sequence of unlikely events (such as a series of component failures possibly combined with human error). Emission rate may be estimated either from direct measurement of emissions, from models based on process knowledge, or from a combination of the two.

1.5.3.2 Transport Assessment Transport assessment is (1) identification of the pathways (such as those illustrated in Figure 1.4) through which the contaminants move and are transformed by physical, chemical, and biological processes in the environment, and (2) estimation of **contaminant concentration**, C, in air, water, soil, and food at specific locations in time and space. As in release assessment, transport assessment may be conducted either by direct measurement or by the use of predictive models for the movement of contaminants through environmental media.

For some problems, such as a preexisting waste disposal site, it might be possible to determine contaminant concentrations through a network of field measurements. These concentration measurements could then be used to estimate exposures. More commonly, the concentrations must be based on transport models because measurements are either not practical (e.g., concentrations are below detectable limits, the area of consideration is too large) or not possible (e.g., future concentrations from existing or planned facilities are needed). The contaminant transport problem is complex because of the inherent complexity of environmental systems. In addition to the physical processes that govern transport in air and water; any of a number of chemical and biological processes may also be important. These processes are not always well understood, and they can depend on many factors. These factors, in turn, may be poorly understood or highly variable. Nonetheless, by combining and interfacing empirical data for processes that are poorly understood with mathematical theory for processes that are well understood, it is possible to develop models for predicting contaminant concentrations in air, water, and food. However, it must be remembered that a model is an idealization, so the adequacy with which the model represents the important aspects of the environmental system is usually an issue.

1.5.3.3 Exposure Assessment Human exposure assessment consists of (1) identification of exposed populations (receptors) and exposure routes, and (2) estimation of the rate at which humans are exposed to the contaminant. The quantitative result is an estimate of contaminant dose or dose rate to members of the exposed population. Human exposure can occur via a number of pathways. The most significant from an environmental contamination perspective include ingestion, inhalation, dermal absorption, and in the case of radioactivity, exposure due to contaminants located outside the human body. Ingestion can include the consumption of contaminated food or water and from the accidental ingestion of water or soil. Inhaled contaminants may be present in either gaseous form or as suspended particulate matter. Dermal absorption can arise from immersion in contaminated air or water or as a result of physical contact with contaminated soil.

For chemical contaminants, exposure is commonly quantified by the **average daily dose rate**, $\bar{D}$, which is the mass of contaminant taken into the body per unit body weight per unit time [M(c)/(M(body)/T]. The integrated dose, D [M(c)/M(body)], is used to quantify short-term exposures. For radiological contaminants, the dose measure is either **equivalent dose** or **effective dose**, and the integrated dose is used for all exposures, both short and long term.

1.5.3.4 Consequence Assessment In general, consequence assessment encompasses adverse aesthetic, ecological, and human health effects. In this book the

focus is on human health effects; and consequence assessment is identification of the types of health effects that can be caused by a contaminant and a quantitative estimate of the probability and/or severity of those effects. For purposes of contemporary human health risk assessment, it is convenient to define two broad health effect categories: deterministic and stochastic. Although these categories are addressed subsequently in some detail, they are introduced here because of their importance in the risk assessment process. **Deterministic effects** are those for which the severity is a function of dose. They typically occur only if an individual tolerance threshold is exceeded, and they display an increasing severity as the dose is increased above the threshold. Lead is a contaminant that causes deterministic effects. It affects the brain, and as the amount of lead in the brain increases, the degree of mental impairment increases. **Stochastic effect**s are those for which the probability is a function of dose. The effect is binary, that is, it either does or does not occur, and the severity is independent of the dose. The induction of cancer as a result of exposure to chemicals or radiation is the most widely analyzed stochastic effect in health risk assessments, although inherited effects also fall into the stochastic category. Benzene is known to cause leukemia in humans, but an exposure to benzene does not always result in leukemia. However, as the dose increases, the probability of contracting leukemia increases as well. Some risk agents induce both deterministic and stochastic effects. For example, in addition to the risk of leukemia, benzene reduces the number of all three types of blood cells and at very high concentrations damages the central nervous system.

The utility of the stochastic versus deterministic distinction lies in the metrics that are used to characterize health risk. The metric for stochastic effects is the **fractional response**, which is the probability of incidence of a binary effect. The metric for deterministic effects is a **margin of safety**, which is a comparison of the calculated dose to a dose that is considered to be safe. The deterministic/stochastic distinction presented here is similar to, but more general than, the noncarcinogenic/carcinogenic scheme used by the EPA. Stochastic effects are not limited to cancer but can include other binary effects, such as inherited abnormalities and some teratogenic effects (e.g., deformed or missing limbs).

1.5.4 Integration and Iteration

During the entire process of risk assessment, it is important to assure that the different parts of the analysis are integrated. For example, releases to both air and water may be important for a particular assessment. The assessment must model the transport of these releases, human exposure to contaminated media, and the response of the humans to the resulting dose. In addition, it may be important to model significant transfer from one medium to another. For example, a volatile contaminant released to water may provide a significant source of contamination for air. Another aspect of integration is the consistency of assumptions and choices for variable values. If it is assumed that all of the contamination released ends up in a small pond, it is incompatible to assume that a community of hundreds of thousands of people uses the pond for their entire water supply. If the assessment has assumed a significant annual rainfall (say, 3000 mm/yr) for purposes of

calculating deposition of sulfur dioxide onto the land surface, it would normally be incompatible to assume a high rate of irrigation from nearby surface water bodies.

When the results are obtained from the four-step risk calculation process, those results should be evaluated in the context of the problem statement. If the questions posed by the problem statement are not answered adequately, the assessment process needs to be iterated (i.e., repeated) to provide an adequate response. This usually means the scope or level of detail (or both) needs to be adjusted. For example, if doses and risks for a particular site are computed based on average adult characteristics but the problem statement asks for the risks to the entire population, including sensitive individuals, the assessment scope must be expanded to include children, the elderly, and the infirm.

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PROBLEMS

1.1 **(a)** Give an example of actual contaminant releases that have resulted in documented adverse (**i**) human health, (**ii**) ecological, and (**iii**) aesthetic effects. Give the setting and identify the following: source of the contaminant release, the environmental transport pathway(s), route of exposure, and the adverse effect.

(b) Give an example of a contaminant release in which the human health, ecological, or aesthetic effects are an open question.

1.2 Use the earthquake risk curve in Figure 1.2 to determine the following:

(a) The number of earthquakes each year that result in more than 60 fatalities.

(b) The number of earthquakes each year that result in 21 to 60 fatalities.

1.3 **(a)** Given the risk curve in Figure 1.8 for the risk per year of early fatalities as a result of an accident at a nuclear power plant, find the following: (**i**) the probability of more than 100 fatalities in a given year as a result of an accident; (**ii**) the probability of one or less than one fatality in a given year as a result of an accident.

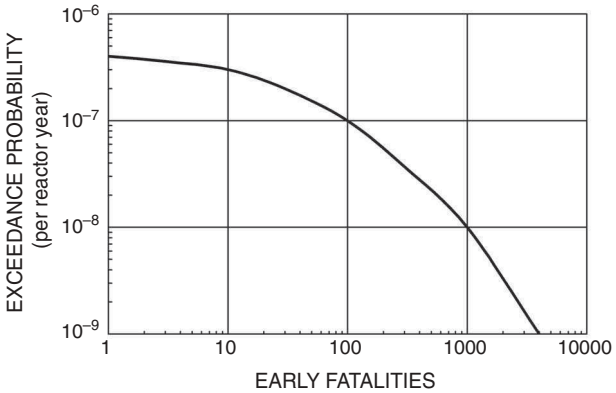

Figure 1.8 Risk curve for Problem 1.3.

(b) If the risk curve in Figure 1.8 were to apply to each of the 100 nuclear power reactors in the United States, what is the probability of one or more fatalities as a result of accidents during the 40-year operating life of the reactors?

1.4 A risk curve for fatalities as a result of train accidents in the Eurotunnel between France and England is shown in Figure 1.9. The abscissa is the number of fatalities and the ordinate is the exceedance frequency per year (i.e., the probability per year that an accident will occur that results in more than N fatalities). From this curve find the following:

(a) The probability of an accident resulting in more than one fatality.

(b) The probability of an accident resulting in more than 10 fatalities.

(c) The probability of an accident resulting in 21 to 30 fatalities (i.e., more than 20 but fewer than 31).

(d) If the exceedance frequency is constant over time, how often will there be an accident involving (**i**) more than one fatality and (**ii**) more than 10 fatalities.

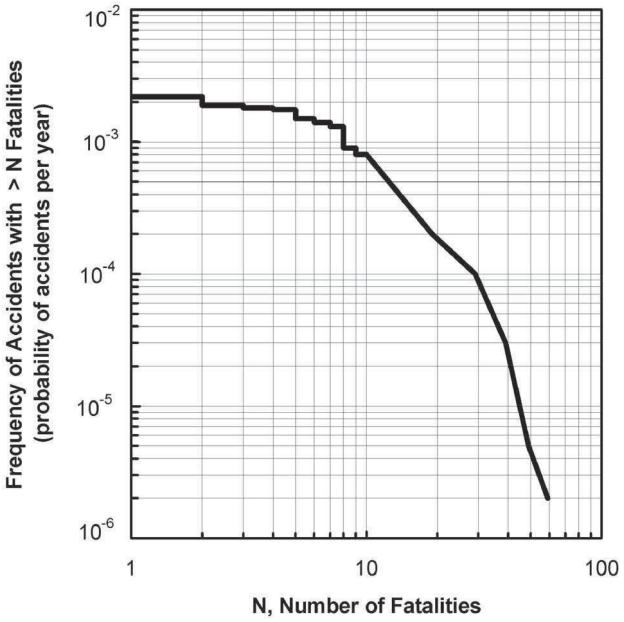

Figure 1.9 Risk curve for fatal accidents in the Eurotunnel between France and England for Problem 1.4. (Data from Evans and Verlander 1997.)

2 Fundamental Aspects of Environmental Modeling

2.1 INTRODUCTION

With few exceptions, computational models are central to environmental risk assessment and analysis. However, the modeling process involves more than obtaining an equation from a textbook. The development, implementation, and quality assurance of a model is achieved through several steps, each of which requires the application of technical knowledge and judgment. Also, in some settings, modeling is done in phases, with refinements being made at each successive phase. Various aspects of this generic modeling process are described in Section 2.2.

In Sections 2.3 and 2.4 we introduce the computational models commonly encountered in risk assessment practice. The fundamental physical basis for most of the models rests in the principle of the conservation of mass. The corresponding mathematical descriptions can be developed heuristically, as is done in introductory texts in environmental science and engineering, or rigorously, through a differential equation referred to here as the contaminant transport equation. A heuristic derivation is presented in Section 2.2, and a rigorous mathematical derivation is presented in Section 2.4.

2.2 MODELING PROCESS

2.2.1 Model Development

The choice and application of models to represent environmental systems can be a complex and formidable task that must be approached deliberately and systematically. Presented in Figure 2.1 is a generic process (Morgan et al. 1990) for quantitative modeling and analysis that can be applied to risk assessment. The problem statement and system description set the stage for the analysis and provide the basis for a conceptual model. The conceptual model is implemented through mathematical and computational models. The resulting numerical values are then evaluated to determine if they address the problem statement.

2.2.1.1 Problem Statement The importance to the process of the problem statement cannot be understated. As noted in Section 1.5.1, it has a profound effect "on

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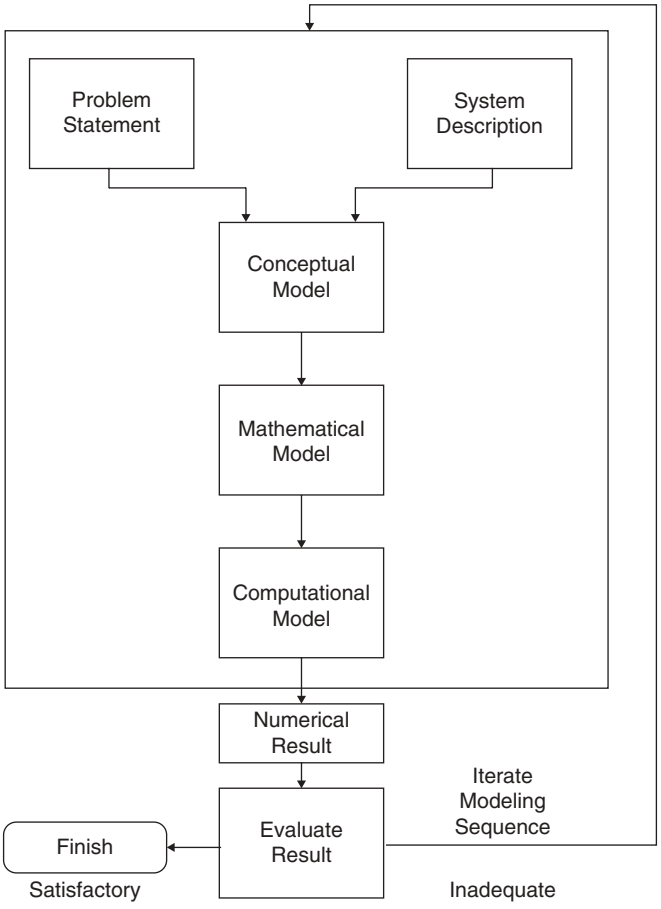

Figure 2.1 Process for model development and application.

the scope, level of detail, and focus of the risk assessment, including: the time scale, the spatial scale, the contaminants considered, the end point of the assessment (the measure of risk or impact), the persons at risk, and the treatment of uncertainty." For example, if the question to be answered about a facility releasing a contaminant to the environment is, "What is the dose to a hypothetical maximally exposed individual?", the model would need to consider a single person living at a particular location and engaging in activities that would maximize exposure to the contaminant. On the other hand, if the question to be answered is "What is the distribution of doses to the surrounding population?", the model would need to consider persons living at various locations around the site and engaging in a range of behaviors. Thus, the most important function of the problem statement is to specify the assessment measure for the model. The **assessment measure** is the quantitative result of the risk calculation. However, the problem statement specifies more than just the physical quantity to be computed (e.g., dose, concentration); it also specifies the conditions under which the quantity is calculated (dose to a person living 24 hours

a day at the facility boundary, annual average concentration in a town 3 km to the northeast). Depending on the context, the assessment measure may also be termed a performance measure or computational endpoint. Because the problem frequently is to determine how well the system performs relative to a quantitative criterion, the term "assessment measure" is used elsewhere in the book and is synonymous with computational endpoint and performance measure. Sometimes it is possible to make measurements that are relevant to a risk assessment. If field measurements of the assessment measure are available, they can be compared to the predictions of a model and thus be used for model validation. For example, if contaminant concentration in forage in an air–grass–cow–human food chain can be measured, it could be used to evaluate the model for the uptake of the contaminant by grass.

2.2.1.2 System Description Many problems in engineering and physical science are well defined and the description of the system is very clear, so much so that this aspect of modeling is usually not given very much attention. For example, if the problem were to determine the stress in an angle bracket supporting a weight, the system description would include the dimensions of the bracket, the weight of the bracket, the weight to be supported, and the physical properties of the material from which the bracket is made. For environmental systems, the situation is often more complex and not fully defined. Frequently, the configuration of the system is unknown or poorly known. For example, if the problem is migration of a contaminant over several kilometers in a groundwater system, knowledge of the stratigraphy of the system (the layering of rock and soil through which the groundwater flows in the subsurface) may be based on very few core samples measured over the area of interest. Consequently, the effort to determine the characteristics of the environmental system may be significant. This effort is termed "site investigation" in the U.S. EPA Superfund program and "site characterization" in programs managing nuclear waste or sites contaminated by radionuclides. The general objective is to characterize the system: to identify the contaminants present; their chemical and physical forms; their transport pathways in the environment; the various chemical, physical, and biological processes that may be active; the manner in which various environmental compartments are connected; and the characteristics and behavior of the exposed individuals or populations.

2.2.1.3 Conceptual Model The main modeling sequence is comprised of three closely related but significantly different models (Mercer and Faust 1980): the conceptual model, the mathematical model, and the computational model. The **conceptual model** is an abstraction of the various physical, chemical, and biological processes that affect the behavior of the contaminant in the system. Although it is developed from both the problem statement and the system description, it is unusual for the conceptual model to completely reflect the detail and complexity contained in the system description. This is because such detail is usually not needed to respond to the problem statement and frequently leads to an intractable mathematical model, one that requires process descriptions and/or transport parameters which are not available. The conceptual model is an idea that has no physical manifestation. Rather, it is an integrated concept that characterizes the following (Eisenberg et al. 1999):

- System geometry (size and shape) or structure (how various parts of the system are connected or related)
- Dimensionality (one, two, or three dimensions)
- Time dependence (steady-state or transient)
- Initial and boundary conditions
- Applicable conservation principles (e.g., mass, momentum, energy)
- Applicable constitutive relations (e.g., dependence of solubility on temperature, dependence of sorption capacity on pH)
- Significant processes
- Input parameters
- Assessment measures (i.e., computational endpoints or performance measures)

The goal of modeling is to predict the assessment measure with sufficient precision and accuracy that the question posed in the problem statement can be answered. Specification of choices for each of these model facets leads to a unique conceptual model. A different set of choices will yield a different conceptual model. Some possible conceptual models will not be suitable for a given problem statement and system description. For example, as discussed in Chapter 7, air dispersion with an inversion layer aloft may be modeled as a two-dimensional process for larger downwind distances, but in general, air dispersion from a point source is modeled as a three-dimensional process. Some authors refer to these choices as modeling assumptions, but this terminology is avoided in this book, to avoid any implication that a choice made for a conceptual model is equivalent to asserting that the real system behaves in that manner. An important aspect of documenting the conceptual model is to specify the rationale behind the choice for each of these items.

2.2.1.4 Mathematical Model The **mathematical model** is a mathematical representation of the conceptual model which permits calculation of the assessment measures. In general, the physical, chemical, and biological processes affecting the behavior of an environmental system can be expressed mathematically through a set of coupled partial differential equations (or integrodifferential equations) for the conservation of mass, energy, and momentum. In the context of environmental risk assessment, the equations for conservation of mass are the most important and most relevant. These equations must be solved in conjunction with the mathematical statement of the initial and boundary conditions included in the conceptual model. In many cases, additional constitutive relations must be used, as specified in the conceptual model.

An environmental system can often be approximated as a number of interconnected compartments. Each compartment can be approximated by a relatively simple conceptual model that leads to a mathematical model with a closed-form analytical solution, and the output of one compartment is coupled mathematically to be the input to another. Such an approach is employed extensively in this book in an attempt to capture the essential behavior of the contaminant in the system and the effect of the key variables on that behavior.

2.2.1.5 Computational Model The problem statement in risk assessment almost always poses a quantitative question such as:

- Is the concentration of DDT below 1 ppb?
- Is the average daily dose of arsenic below 2 mg/(kg·d)
- What is the cancer risk?

Consequently, it is necessary to convert the mathematical model into a form, the **computational model,** which can produce a quantitative result. Computational models for simple problems or for screening-level analyses may be implemented with a calculator or a spreadsheet; more complex scenarios often require the use of computer codes to account for multiple processes or heterogeneous media. For closed-form analytical mathematical models, numerical values for input variables substituted into the equation yield the quantitative result. For complex mathematical models, numerical methods may be required. For example, solutions of the contaminant transport equation in complex geometries are often obtained using finite-difference or finite-element techniques.

2.2.1.6 Result Evaluation: Decision on Iteration The assessment measure must be evaluated to determine if it answers the original statement of the problem. Either the numerical result responds successfully to the original question, or it does not. If it does, the analysis may stop, except for periodic reexaminations, as appropriate. If it does not, several corrective options are open, depending on how the model failed to respond successfully to the question. These options include:

- Gather additional site data or scientific information so that the system description can be refined.
- Revise the question asked so that the scope and focus of the modeling requirements are sharpened and more responsive.
- Revise the scope and attributes of the model (conceptual, mathematical, and numerical) so that the computed results respond directly to the question asked.

Thus, the modeling process may be iterative rather than linear.

2.2.2 Modeling Assurance

Modeling assurance refers to measures designed to enhance the level of confidence that can be placed in model predictions. The primary assurance techniques are verification and validation (Figure 2.2). Given the ubiquitous use of computers and computer software in risk assessment, special importance is attached to assuring that mistakes are not made. **Verification** is the process of assuring that the mathematical model is accurately translated into the computational model. The computational model could be a computer code written in a language such as Fortran or C++, a symbolic mathematical package, or a spreadsheet. One approach is to configure the computational model to solve a reference problem. Frequently, these reference problems are formulated in such a way (simple geometries, boundary

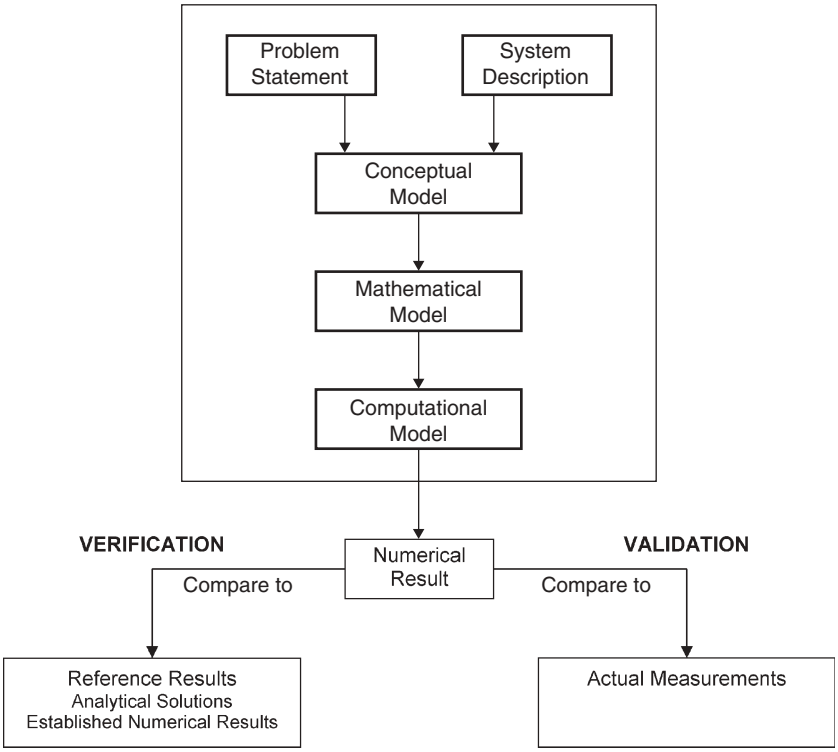

Figure 2.2 Modeling assurance.

conditions, and initial conditions) that they have analytical closed-form solutions. If the output of the computational model agrees with the analytical solution, it provides a level of assurance that the computational model correctly embodies the mathematical model. However, this does not mean that a different problem set might not disclose some previously unidentified error in the computational model. Another approach to verification is to compare results for a reference problem using the computational model to the results for the same problem using a different computational model that has been tested extensively. This method of verification is sometimes called **benchmarking** or **intercomparison**. To assure quality in the development of software, several very general, well-developed approaches [e.g., capability maturity model integration (Software Engineering Institute 2002)] are available for application to environmental risk assessment.

A more stringent test of the modeling process is **validation**, which is a comparison of the predictions of the computational model to actual field measurements. This is the classic approach in engineering to model assurance. If model predictions and measured responses of the system agree, it provides a degree of confidence in the conceptual, mathematical, and computational models. If the predictions and measured responses do not agree, further investigation is required to determine at which step(s) in the modeling process an error has been made. If the computational model has been verified and an error still surfaces during validation, the error is likely to have occurred in the formation of the conceptual model or, possibly, the mathematical model.

For some risk assessment models, such as those associated with groundwater flow and contaminant migration in the subsurface, another step, known as calibration, is sometimes included in the model development process. **Calibration** refers to the adjustment of transport parameters so that predictions of the model match observations. For example, many groundwater models are comprised of interconnected discrete nodes or elements, each with unique flow or transport characteristics. Calibration is performed manually (trial and error) or through mathematical optimization methods. The latter is the inverse problem (de Marsily 1986), in which selected parameter values are adjusted to provide a best fit to measured field values. To validate models calibrated in this way, data sets different from those used for calibration must be used for comparison to the model predictions.

One approach to modeling assurance that is not reflected in Figure 2.2 is **peer review**, which as the name suggests, is a critical review of the model by a panel of experts. The U.S. EPA has issued guidelines for performing peer review on environmental models (Browner 1994; Dearfield and Flaak 2000). Peer review not only seeks to assure the correctness of the mathematical model, but attempts to assure that the conceptual model is well matched to the characteristics of the system modeled and to the requirements of the analysis.

2.2.3 Environmental Modeling in Phases

Environmental modeling is frequently conducted in phases, proceeding from simple, conservative models using generic and/or conservative data to more realistic (and necessarily more complex) models using more site-specific, less conservative data. A **conservative model** is one that is formulated to overestimate risk. This is accomplished through the conceptual model choices, mathematical approximations, and parameter values that are used to develop the model. The initial phase of modeling, referred to as **screening**, is focused on aspects that have the greatest impact on the quantitative assessment measure. For example, for a facility releasing a variety of contaminants to air and surface water, screening calculations can identify which contaminants (**risk drivers**) and which environmental pathways (**critical pathways**) are most significant from the standpoint of yielding risks. Characterization of the release (i.e., amounts, timing, physical and chemical forms) and the environmental pathways (i.e., surface or subsurface hydrology, meteorological data, foods grown and consumed) may then be focused on the risk drivers and the critical pathways.

Many technical and regulatory organizations have articulated screening procedures for particular problems (EPA 1989, 1992; NCRP 1996; IAEA 2001). All screening methods share the same general structure, which is a progression from generic and conservative models and parameter values to site-specific and realistic models and parameter values. Illustrated in Table 2.1 are four phases of a typical screening procedure.

2.3 PHYSICAL AND MATHEMATICAL BASIS FOR RISK ASSESSMENT MODELS

This section is organized around three important points regarding most of the models encountered in environmental risk assessment: that they are based on mass

TABLE 2.1 Characteristics of Various Phases in a Tiered Approach to Environmental Assessment[a]

Screening Phase	Modeling			Parameters
	Transport	Exposure	Consequence	
1	No dilution	Generic single pathway	Generic	Conservative and generic
2	Generic advection–dispersion	Generic multiple pathways	Generic	Conservative and generic
3	Generic advection–dispersion	Multiple pathways, all site specific	Generic	Mainly conservative and generic; some site-specific data
4	Generic advection–dispersion	Multiple pathways, all site specific	Generic	All realistic and/or site specific
5	Site-specific advection–dispersion	Multiple pathways, all site specific	Risk factors specific to population demographics	All realistic and/or site specific

Source: Data from NCRP 1996; EPA 2001; IAEA 2001.

[a] In general, as the level of the assessment advances, the models and parameter choices move from conservative and generic toward realistic and site specific.

conservation (Section 2.3.1), that there are three models that appear frequently (Section 2.3.2), and that all of the models based on mass conservation can be derived from a single differential equation, the contaminant transport equation (Section 2.3.4).

2.3.1 Mass Balances

2.3.1.1 The System In the context of mathematical models based on conservation laws (i.e., energy, mass, and momentum), a **system** is a useful concept. It is defined as "a collection of matter, parts, and/or components which are included inside a specified, often arbitrary, boundary" (Shearer et al. 1971). For a complete, continuous conceptual boundary in space, such as a sphere, everything inside the sphere is the system and everything outside the sphere is not. In risk assessment, a system can be a specific environmental compartment such as suspended sediment in a body of surface water, a particular type of vegetation, or an organ in the human body. To account for more complexity, a system can also consist of several interconnected subsystems. For example, a lake may be represented as a system with the aqueous phase, suspended sediment, and bottom sediment as three subsystems.

In developing mathematical models of contaminant transport, it is convenient to think in terms of a fixed volume in space, known as a **control volume**[1] in many engineering disciplines. The control volume may be infinitesimal or finite in size.

[1] Some authors extend the concept of control volume to a Lagrangian formulation, in which case the control volume changes position with time.

Mass, momentum, and energy may be transported through the boundaries of the control volume, or they may be produced or consumed by processes operating within the control volume. By accounting for all flows, sources, and sinks, balances can be derived to describe momentum, energy, and mass within the system. For assessing risks from contaminants released to the environment, mass balances are used more widely than momentum and energy balances, which accounts for the emphasis here.

2.3.1.2 Conservation of Mass and the Mass Balance Equation The principle of **mass conservation** is fundamental in the sciences and engineering. Simply stated, matter can be neither created nor destroyed; it can only be transformed from one form into another. However, as articulated in Einstein's special theory of relativity, matter can be converted into energy and energy into matter. But the conversion of mass to energy that occurs in the decay of radioactive contaminants can be neglected in the context of mass conservation in environmental systems. That notwithstanding, the transformation that takes place in radioactive decay is extremely important in considering the fate of a radioactive contaminant. For example, the decay of ^{137}Cs to stable ^{137}Ba represents the removal of ^{137}Cs from the system.

The principle of mass conservation can be illustrated with the aid of Figure 2.3, which depicts a control volume of a fluid containing contaminants. Mass conservation applies to each constituent in the system (the contaminants and the fluid) as well as the total mass. The mass of each contaminant inside the control volume may change due to transport across the system boundary, generation inside, or destruction inside. In this context, generation and destruction refer to a given contaminant. For example, destruction of an organic compound such as trichloroethylene (C_2HCl_3) by anaerobic bacteria would decrease its mass and increase the masses of H^+, CO_2, Cl^-, and $C_2Cl_2H_2$ (dichloroethene).

A corollary of mass conservation is the mass balance equation. For the control volume in Figure 2.3, the mass balance may be stated verbally as (Henry and Heinke 1996; Hemond and Fechner-Levy 2000; Bird et al. 2002)

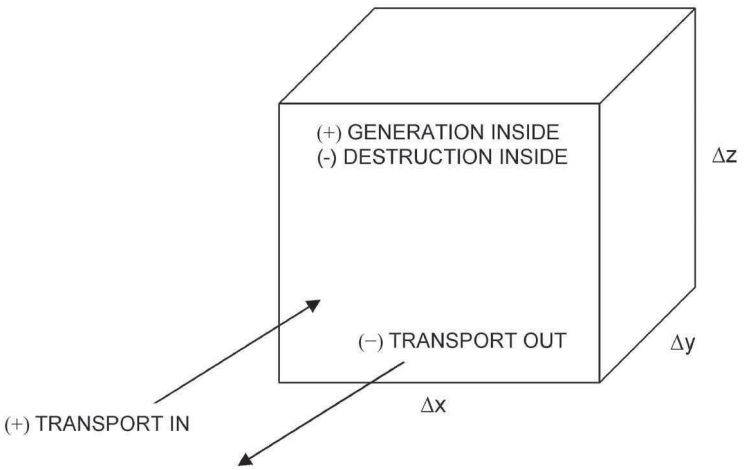

Figure 2.3 Mass conservation in a control volume.

$$\begin{bmatrix}\text{change}\\ \text{in stored}\\ \text{mass}\end{bmatrix}=\begin{bmatrix}\text{mass}\\ \text{transported}\\ \text{in}\end{bmatrix}-\begin{bmatrix}\text{mass}\\ \text{transported}\\ \text{out}\end{bmatrix}+\begin{bmatrix}\text{mass}\\ \text{generated}\\ \text{from sources}\end{bmatrix}-\begin{bmatrix}\text{mass}\\ \text{destroyed}\\ \text{by sinks}\end{bmatrix} \quad (2.1)$$

In many environmental applications, the equation for the time rate of change of mass is more useful. It is obtained by taking the time derivative of each term in Eq. 2.1, yielding

$$\begin{bmatrix}\text{time rate of}\\ \text{change of}\\ \text{stored mass}\end{bmatrix}=\begin{bmatrix}\text{rate of}\\ \text{mass input}\end{bmatrix}-\begin{bmatrix}\text{rate of}\\ \text{mass output}\end{bmatrix}+\begin{bmatrix}\text{mass}\\ \text{generation}\\ \text{rate}\end{bmatrix}-\begin{bmatrix}\text{mass}\\ \text{destruction}\\ \text{rate}\end{bmatrix} \quad (2.2)$$

A special form of Eq. 2.2 obtains when the time rate of change of stored mass is zero and the rates on the right-hand side are constant. The system is then at steady state (i.e., there is no change of mass with time). This often occurs when a sufficient amount of time has passed to allow transients to die away. For steady-state systems, the rate of input plus generation must equal the rate of output plus destruction.

2.3.1.3 Contaminant Concentration and Contaminant Flux In environmental risk assessment, contaminants are a small constituent contained within another medium. For example, contaminants may be present in fluids (e.g., air, surface water, groundwater, blood) or in solid or semisolid matter (e.g., vegetation, animal products, soil, human organs). The amount of contaminant in a particular medium is expressed by the concentration, C [M/V or M(c)/M(medium)]. **Contaminant concentration** is the amount of the contaminant per unit volume or per unit mass of the medium. Since there are numerous ways to characterize the amount of the contaminant and the units of the medium, concentration units depend on the contaminant and the medium.

■ Units of Contaminant Concentration

Water For water, the preferred unit is mass per unit volume, or

$$C_w = \frac{\text{contaminant mass [mg(c)]}}{\text{volume of water (L)}}$$

Occasionally, the concentration may be expressed as a mass ratio, typically parts per million by mass (ppm) or parts per trillion by mass (ppt). The mass ratio is converted to concentration by multiplying by the water density:

$$C_w\ [\text{mg(c)/L}] = \text{ppm by mass}\ [\text{g(c)}/10^6\,\text{g(w)}] \times \rho_w\ [\text{g(w)/m}^3] \times 10^3\ [\text{mg(c)/g(c)}] \times 10^{-3}\,\text{m}^3/\text{L}$$

Air For air, the preferred units are contaminant mass per unit volume of air at standard temperature and pressure:

$$C_a = \frac{\text{contaminant mass}\,[\mu\text{g(c)}]}{\text{volume of air}\,(\text{m}^3)\,\text{at 25°C and 1 atm}}$$

Concentrations of gaseous contaminants are sometimes specified in regulations or reported in the literature as a volume ratio (or its equivalent, the partial pressure):

$$C_a^{\text{VR}}(\text{ppm-volume}) = \frac{\text{contaminant volume}\,(\text{m}^3)}{\text{volume of air}\,(\text{m}^3)\,\text{at 25°C and 1 atm}} \times 10^6$$

Mass concentration is related to volume ratio by

$$CV_a^{\text{VR}}(\text{ppm-volume}) = \frac{C_a[\mu\text{g(c)}/\text{m}^3\text{(a)}]}{\rho_c[\text{g(c)}/\text{m}^3\text{(c)}]} \times 10^{-6}[\text{g(c)}/\mu\text{g(c)}] \times 10^6$$

where ρ_c is contaminant density at 25°C and 1 atm. Contaminant density is hypothetical, in that it is the density of the pure contaminant. It can be calculated using the ideal gas law,

$$\rho_c = \frac{P \cdot \text{MW}}{RT}$$

where MW is the molecular weight of the contaminant (g/mol), P = 1 atm, T = 293 K, and R = 8.314 (J · K/g mol). Substituting these values into the equation above yields

$$C_a\,[\mu\text{g(c)}/\text{m}^3\text{(a)}] = 40.9\text{MW} \cdot C_{\text{a}}^{\text{VR}}\,(\text{ppm-volume})$$

Solid Solid concentrations are expressed as mass of contaminant per mass of solid medium (soil, vegetation, flesh, etc):

$$C_s = \frac{\text{contaminant mass}\,[\text{mg(c)}]}{\text{solid mass}\,[\text{kg(s)}]}$$

It is important to distinguish between concentrations expressed in terms of dry weight of the solid (with water removed) or in terms of fresh weight (including water). Symbolically, these are represented, respectively, by

$$C_s(\text{dry}) = \frac{\text{contaminant mass}\,[\text{mg(c)}]}{\text{dry solid mass}\,[\text{kg(s, dry)}]}$$

and

$$C_s(\text{wet}) = \frac{\text{contaminant mass}\,[\text{mg(c)}]}{\text{fresh solid mass}\,[\text{kg(s, wet)}]}$$

Radionuclides The amount of a given radionuclide in a sample is expressed by a special quantity called **activity.** Physically, activity is the expected number of

decays from a collection of atoms per unit time. The radioactive decay process is first order, and the rate of decay (i.e., the activity) is given by

$$A = \lambda N$$

where λ is a radionuclide decay constant (s^{-1}) and N is the number of atoms of the radionuclide. The decay constant is related to the half-life, $t_{1/2}$, by

$$\lambda = \frac{\ln 2}{t_{1/2}}$$

Physically, the half-life is the time required for the number of radioactive atoms in a sample to decrease by a factor of 2.

The SI unit of activity is the becquerel (Bq), where 1 Bq = 1 decay/s. The traditional unit of radioactivity is the curie (Ci):

$$1\,\text{Ci} = 3.7 \times 10^{10}\ \text{decays/s} = 3.7 \times 10^{10}\,\text{Bq}$$

For a given radionuclide, there is a one-to-one correspondence between mass and activity:

$$A(\text{Bq}) = \lambda N(\text{decays/s}) = \lambda\left(\text{s}^{-1}\right) \cdot m(\text{g}) \frac{N_0(\text{atoms/mol})}{\text{AW}(\text{g/mol})}$$

where N_0 is Avogadro's constant, 6.022×10^{23} atoms/mol, and AW is the atomic weight of the radionuclide. The reason that radioactive material is quantified by activity (i.e., decay rate) is because the harm posed by radioactive material comes from the radiation (i.e., gamma rays, beta particles, alpha particles, etc.) emitted in the decay process. The emission rate of radiation is directly proportional to activity. Radionuclide concentrations are expressed in a fashion analogous to that for chemical contaminants, with contaminant mass being replaced by activity:

$$C_w = \frac{\text{radionuclide activity (Bq)}}{\text{volume of water (L)}}$$

$$C_a = \frac{\text{radionuclide activity (Bq)}}{\text{volume of air at 25°C and 1atm}\left(\text{m}^3\right)}$$

$$C_s = \frac{\text{radionuclide activity (Bq)}}{\text{solid mass [kg(s)]}}$$

The total mass of contaminant in an incompressible fluid medium is concentration times volume. Thus, the contaminant mass balance of Eq. 2.2 can also be expressed in terms of concentration by dividing both sides by the volume of the medium. This yields an equation for each contaminant of interest:

$$\begin{bmatrix}\text{time rate of}\\ \text{change of}\\ \text{concentration}\end{bmatrix} = \begin{bmatrix}\text{rate of}\\ \text{mass input}\\ \text{per unit}\\ \text{volume}\end{bmatrix} - \begin{bmatrix}\text{rate of}\\ \text{mass output}\\ \text{per unit}\\ \text{volume}\end{bmatrix} + \begin{bmatrix}\text{generation}\\ \text{rate}\\ \text{per unit}\\ \text{volume}\end{bmatrix} - \begin{bmatrix}\text{destruction}\\ \text{rate}\\ \text{per unit}\\ \text{volume}\end{bmatrix} \tag{2.3}$$

The transport of contaminants dissolved or suspended in fluids is usually dominated by the bulk flow of the fluid.[2] This transport is quantified by the advective flux, which in one dimension is given by

$$j = Cu \tag{2.4}$$

where j is the advective flux [M/L^{2}T], C the contaminant concentration in the fluid, and u the velocity of the fluid [L/T].

Contaminant flux is useful in that it provides a way to calculate the rate at which contaminant is transported across a surface due to advection. For simple one-dimensional problems such as rivers or pipes in which the direction of fluid flow is perpendicular to the flow area and fluid velocity and contaminant concentration are uniform across the flow area,

$$\dot{M} = jA \tag{2.5}$$

where $\dot{M}$ is the rate at which contaminant mass crosses area A. Substituting Eq. 2.4 into Eq. 2.5 yields

$$\dot{M} = CuA = CQ \tag{2.6}$$

where Q is the volumetric flow rate of the fluid [L^3/T].

▶ Example 2.1

An industrial plant discharges waste into a river at a flow rate of 0.5 m^3/s. Upstream from the waste discharge, the volumetric flow rate of the river is 10 m^3/s. The principal contaminant in the waste stream is arsenic at a concentration of 100 µg/L. Arsenic is present in the watershed, and the arsenic concentration upstream from the waste discharge is 1.5 µg/L. If the waste stream becomes completely mixed with the river water, what is the concentration of arsenic in the river downstream from the waste discharge?

Solution To solve this problem using the tools presented, it is first necessary to define the control volume as illustrated in Figure 2.4. It is bounded on the top and bottom, respectively, by the upper surface and bed of the river, bounded on the sides by the banks of the river (and the imaginary extension of the bank across the waste discharge), bounded upstream by a cross section of the river at the upstream edge of the waste discharge, and bounded downstream from the waste discharge by a cross section at some arbitrary location A.

[2] The two processes responsible for contaminant transport across the surface of a control volume are advection and dispersion. These are discussed in further detail in Section 2.4.1.

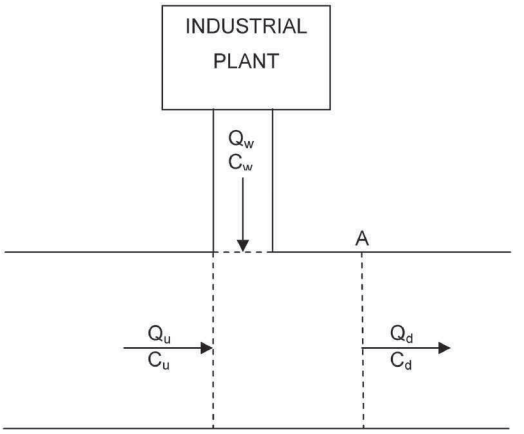

Figure 2.4 Control volume for the problem in Example 2.1.

There are two components of interest in this problem: water and arsenic. They both enter the system by flow across the upstream cross section of the river and by the waste discharge. They exit the system by flow across the downstream cross section of the river. There is no generation or destruction of either water or arsenic in the system. Furthermore, since the inflow of water and arsenic is balanced by outflows, the system is at steady state and Eq. 2.2 becomes

$$0 = \text{rate of mass input} - \text{rate of mass output}$$

For water this becomes

$$0 = Q_u\rho_u + Q_w\rho_w - Q_d\rho_d$$

Rearranging yields

$$Q_u\rho_u + Q_w\rho_w = Q_d\rho_d$$

where Q is the volumetric flow rate of the water, ρ the water density, and u, w, and d represent upstream, waste, and downstream, respectively. Since the density of the water at all three stations may be taken to be equal, the material balance for water becomes

$$Q_u + Q_w = Q_d$$

Since $Q_u = 10\,\text{m}^3/\text{s}$ and $Q_w = 0.5\,\text{m}^3/\text{s}$, $Q_d = 10.5\,\text{m}^3/\text{s}$.

For arsenic the mass balance is

$$Q_uC_u + Q_wC_w = Q_dC_d$$

where C is the concentration of arsenic and the subscripts are the same as above. Solving for C_d gives

$$C_d = \frac{Q_u C_u + Q_w C_w}{Q_d}$$
$$= \frac{(10\,\mathrm{m^3/s})(1.5\,\mu\mathrm{g/L} + 0.5\,\mathrm{m^3/s})(100\,\mu\mathrm{g/L})}{10.5\,\mathrm{m^3/s}}$$
$$= 6.2\,\mu\mathrm{g/L}$$

Note that 77% of the arsenic downstream is due to waste discharge and 23% comes from upstream sources.

A conservative calculation of the downstream concentration can be made by neglecting the contribution of the waste stream to the water flow rate downstream and approximating the downstream flow rate as being equal to the upstream flow rate. The resulting estimate of the downstream concentration is 6.5 µg/L.

2.3.2 Simple Models

Three relatively simple models for contaminant concentration appear frequently throughout the risk assessment process. They are presented here because of their importance and because students new to the field may not be familiar with them. They will be seen again in Section 2.4 in the context of the general transport equation.

2.3.2.1 First-Order Removal The kinetics of chemical reactions is often expressed as a power function of the concentration:

$$\frac{dC}{dt} = \pm kC^n \tag{2.7}$$

where k is a rate constant $[T^{-1}C^{1-n}]$ and n is the reaction order. If $n = 0$, the reaction is zeroth order; if $n = 1$, it is first order; if $n = 2$, it is second order; and so on. Many processes in risk assessment, not just chemical reactions, are either inherently first order or they are approximated as being first order. Physically, a first-order process is one for which the time rate of change of contaminant concentration (or mass) is proportional to the concentration (or mass) present. The radioactive decay process and some chemical reactions are rigorously first order. Other processes, such as the removal of contaminants from plants and animals, leaching of contaminants from buried wastes, degradation of organic contaminants in the environment, settling of contaminants sorbed to particulate matter, and so on, are typically approximated as being first order. Also, it is shown in Example 2.6 that the decrease in contaminant concentration in a well-mixed compartment, due to removal with the fluid leaving the compartment, can be represented as a first-order process.

The differential equation that forms the basis for the **first-order removal model** is Eq. 2.7 with $n = 1$:

$$\frac{dC}{dt} = -kC \tag{2.8}$$

where k is a first-order rate constant for the removal process. Equation 2.8 can be solved by rearranging,

$$\frac{dC}{C} = -kdt \tag{2.9}$$

and integrating. If the contaminant concentration at $t = 0$ is C_0, the result is

$$C(t) = C_0 \exp(-kt) \tag{2.10}$$

Thus, the contaminant concentration decreases exponentially with time as a result of first-order removal. This time dependence is sometimes characterized by the half-life, $t_{1/2}$, which is the time required for contaminant mass or, equivalently, contaminant concentration to decrease by a factor of 2. Letting $C(t_{1/2}) = C_0/2$ in Eq. 2.10 and solving for $t_{1/2}$ yields

$$t_{1/2} = \frac{\ln 2}{k} \tag{2.11}$$

The ratio $C(t)/C_0$ is 0.5 after one half-life, 0.25 after two half-lives, 0.125 after three half-lives, and so on. It takes a little less than seven half-lives for the concentration to decrease by a factor of 100 and about 10 half-lives for it to decrease by a factor of 1000.

► **Example 2.2**

Tetrachloroethylene (C_2Cl_4), also known as PCE, is a solvent that was used extensively in the past to remove grease from metal machine parts. PCE wastes were often disposed in open seepage basins. Consider an industrial site in which the PCE concentration in soil below an old seepage basin is 750 mg(c)/kg(s). The PCE is degraded by naturally occurring bacteria at the site, and the process can be approximated as first order with a half-life of 400 days.

(a) Assuming that bacterial degradation is the dominant process affecting PCE concentration in the soil, calculate the concentration 5 years from now.
(b) The degradation process is actually a transformation process in which one of the Cl atoms in the molecule is replaced by an H atom, yielding the degradation product C_2HCl_3, which is another contaminant, known as trichloroethylene or TCE. What is the concentration of TCE after the 5-year period?
(c) Perform a mass balance for the system.

Solution

(a) The time dependence of the concentration is given by Eq. 2.10:

$$C(t) = C_0 \exp(-kt)$$

The first-order rate constant, k, is obtained by rearranging Eq. 2.11:

$$t_{1/2} = \frac{\ln 2}{k} \Rightarrow k = \frac{\ln 2}{t_{1/2}} = \frac{\ln 2}{400\,\text{d}} = 1.73 \times 10^{-3}\,\text{d}^{-1}$$

Substituting yields

$$\text{C}(5\,\text{yr}) = [750\,\text{mg}(\text{C}_2\text{Cl}_4)/\text{kg(s)}]\ \exp[(-0.00173\,\text{d}^{-1})\ (5\,\text{yr})\ (365\,\text{d/yr})] = 32\,\text{mg}(\text{C}_2\text{Cl}_4)/\text{kg(s)}$$

(b) A mole of C_2HCl_3 is produced every time a mole of C_2Cl_4 degrades. During the 5-year period, the mass of C_2Cl_4 that degrades in each kilogram of soil is 750 – 32 = 718 mg. The corresponding number of moles is

$$\frac{718\,\text{mg}(\text{C}_2\text{Cl}_4)}{[165.8\,\text{g}(\text{C}_2\text{Cl}_4)/\text{mol}(\text{C}_2\text{Cl}_4)][10^3\,\text{mg}(\text{C}_2\text{Cl}_4)/\text{g}(\text{C}_2\text{Cl}_4)]} = 4.33 \times 10^{-3}\,\text{mol}\ \text{C}_2\text{Cl}_4$$

Thus, 4.33×10^{-3} mol of C_2HCl_3 is produced per kilogram of soil. Multiplying by the molecular weight of C_2HCl_3 yields the mass concentration of C_2HCl_3:

$$[4.33 \times 10^{-3}\,\text{mol}(\text{C}_2\text{HCl}_3)/\text{kg(s)}]\ [131.4\,\text{g}\ (\text{C}_2\text{HCl}_3)/\text{mol}(\text{C}_2\text{HCl}_3)]\ [10^3\,\text{mg}(\text{C}_2\text{HCl}_3)/\text{g}(\text{C}_2\text{HCl}_3)] = 569\,\text{mg}\ (\text{C}_2\text{HCl}_3)/\text{kg(s)}$$

(c) The mass balance can be verified in a variety of ways. One way is to account for the 750 mg of C_2Cl_4 originally in each kilogram of soil. After 5 years, 32 mg of C_2Cl_4 and 569 mg of the degradation product, C_2HCl_3, remained. The remaining mass (750 – 32 – 569 = 149 mg) is the difference between the mass of 4.33×10^{-3} mol of Cl (153 mg) and the replacement mass of 4.33×10^{-3} mol of H (4 mg).

2.3.2.2 Constant-Source First-Order Removal The **constant-source first-order removal model** is used for systems in which the contaminant is either being produced in, or is entering, an environmental compartment at a constant rate and the removal rate is first order. Examples include buildings (constant generation rate inside with venting to the exterior), well-mixed ponds or lakes (constant emission rate into the pond from either a stream or an end-of-pipe source, and first-order removal by sedimentation, degradation, etc.), vegetation (constant deposition on foliage and first-order removal by weathering, degradation, etc.), and animals (constant uptake by foraging contaminated food and first-order biological removal by excretion, metabolism, etc.).

The model can be obtained by rearranging Eq. 2.3 and expressing the time rate of change of contaminant concentration as the difference between sources (the first and third terms on the right-hand side) and sinks (the second and fourth terms). Constant source means that transport into the compartment, production within the

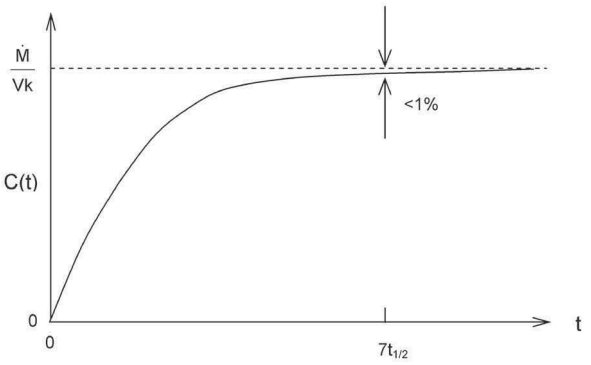

Figure 2.5 Concentration vs. time for a constant-source first-order removal model.

compartment, or their sum does not change with time. First-order removal means transport out of the compartment, destruction within the compartment, or their sum is first order. For a compartment of volume, V, this is represented by

$$\frac{dC(t)}{dt} = \frac{\dot{M}}{V} - kC(t) \tag{2.12}$$

where $\dot{M}$ is the rate of mass transport into or generation within the compartment. The solution for a compartment in which the concentration initially is zero $[C(0) = 0]$ is

$$C(t) = \frac{\dot{M}}{Vk}[1 - \exp(-kt)] \tag{2.13}$$

Equation 2.13 is displayed graphically in Figure 2.5, where contaminant concentration is plotted as a function of time. Initially, the concentration increases linearly with time, but the rate of increase declines continually. This occurs because the contaminant removal rate, $kC(t)$, increases with increasing concentration and gradually approaches the generation rate, which is not changing. As a consequence, $dC(t)/dt$ asymptotically approaches zero, and the concentration asymptotically approaches a steady-state value, $C_{ss} = \dot{M}/Vk$. The concentration reaches 1% of its steady-state value within seven half-lives. If the initial concentration is nonzero, the same steady-state concentration results, but the time required to reach steady state depends on the half-life and the initial concentration.

► Example 2.3

A defective gas-fueled space heater is unwisely operated in a room 8 ft high by 12 ft wide by 20 ft long. The heater generates CO, a toxic combustion product, at the rate of 100 g/h. Although the room is closed, it is ventilated at a relatively typical

air exchange rate of $0.5\,h^{-1}$. Air exchange rate is a term used by heating and air-conditioning engineers which is equivalent to the first-order rate constant for contaminant removal due to the infiltration of fresh, uncontaminated air into a room and the corresponding exhaust of contaminated air to the outside. It is equal to the volumetric flow rate into and out of a room divided by the volume of the ventilated space.

(a) What would be the maximum concentration of CO if steady state were reached?
(b) What is the concentration of CO in the room after 2 hours?
(c) There are no regulatory standards for carbon monoxide levels in the home. The Environmental Protection Agency limit on outdoor exposures is approximately $10\,mg/m^3$. The concentration obtained is considerably higher than this. If the heater were to be turned off after 2 hours, how long would it take to reach this level?

Solution

(a) The constant-source first-order removal model (Eq. 2.13) can be applied to this problem:

$$C(t) = \frac{\dot{M}}{Vk}[1 - \exp(-kt)]$$

The steady-state concentration is given by the leading term. The volume is $V = (8\,ft)(12\,ft)(20\,ft) = (1920\,ft^3)(0.02832\,m^3/ft^3) = 54.4\,m^3$. Substituting gives

$$\frac{100\,g/h}{(54.4\,m^3)(0.5\,h^{-1})} = 3.68\,g/m^3 = 3680\,mg/m^3$$

(b)
$$C(2\,h) = 3680[1 - \exp(-0.5\,h^{-1})(2\,h)]\,mg/m^3$$
$$= 2330\,mg/m^3$$

(c) When the heater is turned off, there is no longer a source of carbon monoxide, and Eq. 2.12 reduces to Eq. 2.8 for first-order removal. The solution is Eq. 2.10:

$$C(t) = C_0 \exp(-kt)$$

Solving for time gives

$$t = -\frac{1}{k} \ln \frac{C(t)}{C_0}$$

and substituting values yields

$$t = -\frac{1}{k}\ln\frac{C(t)}{C_0} = -\frac{1}{0.5\,\text{h}^{-1}}\ln\frac{10}{2330}$$
$$= 10.9\,\text{h}$$

Clearly, if someone knew the hazard, they should have opened all the doors and windows in the room and used a fan to enhance clearance of the CO. Mathematically, this would have increased the removal rate constant (see Problem 2.7).

2.3.2.3 Instantaneous Partitioning In risk assessment there are a number of important physicochemical processes in which a contaminant undergoes dynamic exchange between two different media. A conceptual model applicable to these systems is shown in Figure 2.6, which illustrates contaminant partitioning between compartments A and B. The kinetics are illustrated in Figure 2.7 for a contaminant introduced into compartment A at $t = 0$. As time passes, the concentration in A decreases (Figure 2.7*a*) and the concentration in B increases (Figure 2.7*b*) until equilibrium is achieved and the concentrations stabilize.

Contaminant partitioning between the two compartments is described by a **partition factor**, PF (Figure 2.7*c*), which is given by

$$\text{PF} = \frac{C_\text{B}}{C_\text{A}} \tag{2.14}$$

If the time required to reach equilibrium is small compared to the time scale of interest, partitioning is approximated as occurring instantaneously; and the partition factor becomes the ratio of the equilibrium concentrations:

$$\text{PF}_e = \frac{C_{\text{B},e}}{C_{\text{A},e}} \tag{2.15}$$

where the subscript e refers to equilibrium. This is the **instantaneous partitioning model**. It is used to describe a wide variety of environmental systems, including the sorption of contaminants in aqueous solution to rock, soil, and sediment; the uptake of contaminants by plants from soil; the uptake of contaminants by aquatic animals (fish, mollusks) from water; and the exchange of a contaminant between air and raindrops.

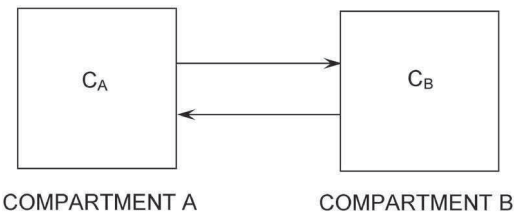

Figure 2.6 Instantaneous partitioning between two compartments.

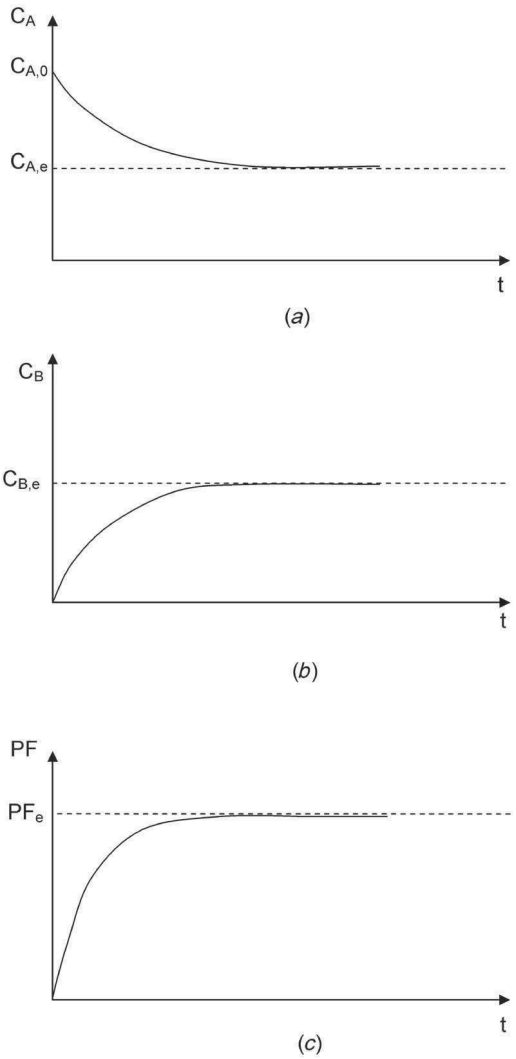

Figure 2.7 Partitioning of a contaminant between two compartments. At $t = 0$, a contaminant is introduced into compartment A, at which time it begins to partition between compartments A and B.

▶ Example 2.4

The mercury concentration in a reservoir is 50 μg(c)/L. The partition factor for uptake by fish is 3.2 L/kg(f). What is the mercury concentration in fish?

Solution The partition factor for partitioning between aquatic animals and water is

TABLE 2.2 Partition Factors in Environmental Risk Assessment

Name	Use	Symbol
Distribution coefficient	Partitioning of contaminant between the aqueous and solid phases in surface water and groundwater	K_D
Bioconcentration factor		
Fish	Uptake of a contaminant by an aquatic animal from the aqueous phase	B_F
Vegetation	Uptake of a contaminant by vegetation from contaminated soil	B_V
Henry's law constant	Partitioning of a contaminant between the gaseous and aqueous phases	H
Organic carbon–water partition coefficient	Partitioning of an organic contaminant between water and natural organic matter	K_{oc}

$$\mathrm{PF[L/kg(f)]} = \frac{C_f[\mathrm{mg(c)/kg(f)}]}{C_w[\mathrm{mg(c)/L}]}$$

Rearranging to obtain concentration in fish, we have

$$C_f = \mathrm{PF} \cdot C_w$$

Inserting numerical values gives us

$$\begin{aligned} C_f &= \mathrm{PF} \cdot C_w \\ &= [3.2\,\mathrm{L/kg(f)}][50\,\mu\mathrm{g(c)/L}] \\ &= 160\,\mu\mathrm{g(c)/kg(f)} \end{aligned}$$

The name given to the partition factor and its units vary, depending on the context in which it is being applied. The various names and uses of partition factors in environmental risk assessment are included in Table 2.2. They are described in more detail in subsequent chapters.

2.4 CONTAMINANT TRANSPORT EQUATION

Many of the models used in risk assessment can be derived from a single differential equation which, at its basis, is a mass balance expression applied to a moving fluid. This equation is known variously as the advection–dispersion equation, the reactive transport equation, and the contaminant transport equation. The latter term is adopted here. The contaminant transport equation can be applied to a wide range of contaminant transport processes, ranging from generation and release to migration through environmental media to movement through the human body. Consequently, it provides a unified basis for development of many of the models employed in quantitative risk assessment.

2.4.1 Transport Processes

The contaminant transport equation is a mass balance equation that applies specifically to a species (i.e., a contaminant) which is a relatively minor component (in terms of mass) suspended or dissolved within a medium such as air, water, or a body fluid. The equation takes into account the processes that can cause the amount of contaminant contained in a control volume of a medium to change. As illustrated in Figure 2.3, the processes are (1) transport across the surface (either from the outside to the inside, or vice versa), (2) generation inside the control volume, or (3) destruction inside the control volume.

There are two processes that can cause a contaminant to be transported across the surface of a control volume: advection and dispersion. **Advection** is the transport of a contaminant with the mean flow of the fluid. **Dispersion** is a general term that is used to describe a variety of processes that cause contaminant movement to deviate from the mean flow path of the fluid. These processes include molecular diffusion, turbulent diffusion, and advective heterogeneities. **Molecular diffusion** is the spreading of a contaminant due to the random motion of the molecules of the medium and the contaminant. It might be important, for example, in the migration of contaminants in very slowly moving groundwater, or in a slightly different context, in the migration of contaminants within a soil particle following sorption on the surface. **Turbulent diffusion** is the spreading of a contaminant due to turbulence of the medium. It is responsible for the spreading of a contaminant plume in the atmosphere and contributes to spreading in streams and rivers. The term **advective heterogeneities** refers to contaminant spreading that occurs as a result of nonuniform velocity distributions of the medium. For example, in a channel (i.e., a river or stream) the fluid velocity is zero at the interface between the water and the sides and bottom of the channel. Thus, contaminant molecules near the sides and bottom travel slower than do those near the center, resulting in spreading of the contaminant in the direction of flow. Advective heterogeneities also contribute to dispersion in the subsurface, for a variety of reasons (discussed in Chapter 6).

Advection and dispersion are referred to as **conservative processes** because they do not result in a change in total contaminant mass. Generation and destruction, on the other hand, are **nonconservative processes**.

2.4.2 Derivation of the Contaminant Transport Equation

The **contaminant transport equation** is the constitutive transport equation from fluid mechanics applied to a contaminant that is intimately mixed with the fluid through dissolution, entrainment, or suspension but whose concentration is too low to affect fluid flow. The equation is basically a modification of Eq. 2.3 to account explicitly for advection and dispersion as the mechanisms for transport into and out of a control volume. In word form,

$$\begin{bmatrix}\text{time rate of}\\ \text{change of}\\ \text{concentration}\end{bmatrix} = \begin{bmatrix}\text{net rate of}\\ \text{increase due}\\ \text{to dispersion}\end{bmatrix} + \begin{bmatrix}\text{net rate of}\\ \text{increase due}\\ \text{to advection}\end{bmatrix} + \begin{bmatrix}\text{generation}\\ \text{rate}\end{bmatrix} - \begin{bmatrix}\text{destruction}\\ \text{rate}\end{bmatrix} \tag{2.16}$$

The units of each term in the equation are contaminant mass per unit volume of suspending medium (air, water, blood, etc.) per unit time [M/(L^{3}T)]. The first two terms on the right-hand side of Eq. 2.16 represent the conservative processes, and the last two represent the nonconservative processes. Derivation of the mathematical form of the equation begins with the **contaminant continuity equation** (Logan 1999)[3]:

$$\frac{dC(\mathbf{r},t)}{dt} = -\nabla \cdot \mathbf{j}(\mathbf{r},t) + g(\mathbf{r},t) - d(\mathbf{r},t) \tag{2.17}$$

where $\mathbf{j}(\mathbf{r},t)$ is contaminant flux, $g(\mathbf{r},t)$ is contaminant generation rate per unit volume, and $d(\mathbf{r},t)$ is contaminant destruction rate per unit volume. In Eq. 2.17, the first term captures both advective and dispersive transport across the surface of the volume element. Treating advective and dispersive transport separately yields

$$\frac{dC(\mathbf{r},t)}{dt} = -\nabla \cdot \mathbf{j}^{\mathrm{D}}(\mathbf{r},t) - \nabla \cdot \mathbf{j}^{\mathrm{A}}(\mathbf{r},t) + g(\mathbf{r},t) - d(\mathbf{r},t) \tag{2.18}$$

where $\mathbf{j}^{\mathrm{D}}(\mathbf{r},t)$ is **dispersive flux** and $\mathbf{j}^{\mathrm{A}}(\mathbf{r},t)$ is **advective flux**. Because Eq. 2.18 has two dependent variables, contaminant concentration and contaminant flux, it cannot be solved without a relationship between them. For advection the relationship is

$$\mathbf{j}^{\mathrm{A}}(\mathbf{r},t) = C(\mathbf{r},t)\mathbf{v}(\mathbf{r},t) \tag{2.19}$$

where $\mathbf{v}$ is the fluid velocity vector. For dispersion, the relationship is the following approximation:

$$\mathbf{j}^{\mathrm{D}}(\mathbf{r},t) \approx -D\nabla C(\mathbf{r},t) \tag{2.20}$$

where D is the **dispersion coefficient**[4] and

$$D = D^{\mathrm{DM}} + D^{\mathrm{DT}} + D^{\mathrm{DH}} \tag{2.21}$$

where D^{DM} is the **molecular diffusion coefficient**, D^{DT} the **turbulent diffusivity**, and D^{DH} the **mechanical dispersion coefficient**. Each of these has units of [L^2/T].

■ Contaminant Concentration and Contaminant Flux: Formalized Treatment

Contaminant concentration and contaminant flux are defined in Section 2.3.1.3. More rigorous and formalized definitions are needed in deriving and developing a physical understanding of the contaminant continuity equation and the contami-

[3] Logan uses the term general transport equation to refer to a generic continuity equation which can be applied to either the fluid medium or the contaminant contained within the fluid.

[4] Rigorously, the dispersion coefficient is a tensor. However, in environmental applications, values for the off-diagonal components are rarely, if ever, used. Thus, we neglect them in this book.

nant transport equation. **Contaminant concentration** is defined in terms of a control volume dV as follows: $C(\mathbf{r},t)dV$ [or equivalently, $C(x,y,z,t)dx\,dy\,dz$] is the amount of contaminant in dV at time t, where dV is located at $\mathbf{r}$ (Figure 2.8). The units of contaminant concentration are mass/volume. **Contaminant flux**, $\mathbf{j}(\mathbf{r},t)$, is a vector quantity and thus has an associated direction. The definition of contaminant flux is the net rate per unit area at which the contaminant flows across a plane perpendicular to the principal direction of the flux vector, and the units of contaminant flux are mass/(area·time). By itself the flux is not particularly useful from a physical standpoint because it provides information on contaminant transport in only one direction. A more useful physical quantity is $\mathbf{j}(\mathbf{r},t)\cdot\mathbf{n}dA$, which is the net rate of contaminant mass transport in the direction $\mathbf{n}$.

Contaminant flux, if known, allows calculation of contaminant transport across a surface. For example, environmental regulators in New York might want to determine the net rate at which airborne SO_2 comes into the state across its east–west border with Pennsylvania, which can be calculated if the contaminant flux vector is known everywhere along the border between the two states (Figure 2.9). The x-axis is placed along the east–west border between the two states and its origin is at the Lake Erie shoreline (neglecting the small panhandle at the northwestern corner of Pennsylvania). The origin of the z-axis is at ground level, and the z-axis is perpendicular to the ground. The distance along the east–west border is L, and the maximum vertical extent of the contamination is H. The net rate of flow across an arbitrary area in the xz plane is $\mathbf{j}(x,z,t)\cdot\mathbf{i}_y dxdz$, where $\mathbf{i}_y$ is the unit vector in the y direction. The total rate of flow across the border, $\dot{M}$, is obtained by integrating over the entire area of interest:

$$\dot{M} = \int_{z=0}^{H}\int_{x=0}^{L}\mathbf{j}(x,z,t)\cdot\mathbf{i}_y\,dxdz$$

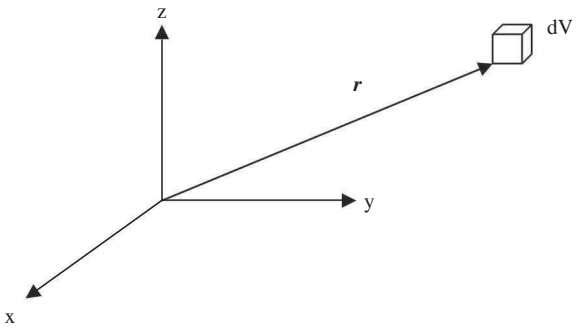

Figure 2.8 $C(\mathbf{r},t)dV$ is the amount of contaminant in dV at time t.

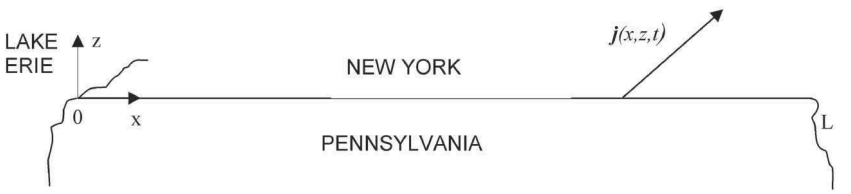

Figure 2.9 Border between Pennsylvania and New York.

Substituting Eqs. 2.19 and 2.20 into Eq. 2.18 yields the contaminant transport equation:

$$\frac{\partial C(\mathbf{r},t)}{\partial t} = \nabla \cdot (D(\mathbf{r},t)\nabla C(\mathbf{r},t)) - \nabla \cdot C(\mathbf{r},t)\mathbf{v}(\mathbf{r},t) + g(\mathbf{r},t) - d(\mathbf{r},t) \tag{2.22}$$

where $C(\mathbf{r},t)$ = contaminant concentration
$\mathbf{r}$ = position vector
t = time
$\mathbf{v}(\mathbf{r},t)$ = fluid velocity vector
$= \mathbf{v}(x,y,z,t) = v(x,y,z,t)\mathbf{i}_x + u(x,y,z,t)\mathbf{i}_y + w(x,y,z,t)\mathbf{i}_z$
$D(\mathbf{r},t)$ = dispersion coefficient
$g(\mathbf{r},t)$ = contaminant generation rate density
$d(\mathbf{r},t)$ = contaminant destruction rate density

As noted previously, Eq. 2.22 represents a unified approach to environmental transport in that many of the models used in this book can be derived from it. For example, it can be used to obtain the simple zero-dimensional compartmental models presented in Sections 2.3.2 and 2.4.3. At the same time, it can be used to obtain complex three-dimensional models used for groundwater, surface water, and atmospheric transport. This unified approach is possible not only because of the common use of compartmental models for many environmental processes, but more important, because advection and dispersion are the primary determinants of contaminant transport in risk assessment applications. Consequently, the spatial and temporal behaviors of contaminant concentration in response to a source are similar regardless of the transport medium. These similarities across media are often masked because of differences in the terminology and the methodology for describing and estimating the transport parameters in the various systems. The unified approach provides a common theoretical basis for the development of transport models. It also simplifies environmental transport analysis in that the mechanics of solving the transport equation and the form of the solutions are often independent of the medium or the specific transport pathway. Thus, the same general solution can often be applied to more than one system. The differences from system to system lie in specifying transport parameters such as the dispersion coefficient or partition factor, in establishing the conditions under which the solution is applicable, and in the general importance and specific nature of nonconservative processes.

It is important to remember that Eq. 2.22 and its solutions are simple approximations for complex natural processes. In addition, when applied to a given transport problem, the accuracy of predictions based on the contaminant transport equation depends on the completeness of the conceptual model (i.e., inclusion of all relevant processes and pathways), the ability of the contaminant transport equation to approximate the transport processes, and the accuracy of transport parameters (such as dispersion coefficients, reaction-rate constants, partition coefficients, etc.) used to quantify specific processes.

2.4.3 Zero-Dimensional Solutions of the Contaminant Transport Equation

Several zero-dimensional solutions of the contaminant transport equation are derived to illustrate how it can be used as the fundamental basis for most mathe-

matical models of contaminant transport in risk assessment. In this section, the contaminant transport equation is used to derive the zero-dimensional (with respect to space) models that were developed in Sections 2.3.1 and 2.3.2 from a heuristic mass balance approach. In Chapter 4, the contaminant transport equation is used to derive the more complex one-, two-, and three-dimensional environmental transport models.

2.4.3.1 Homogeneous Compartments A useful mathematical construct for modeling purposes is the **environmental compartment**, which is either a distinct component such as a lake, a cow, or the human body; or a distinct subcomponent within a larger component, such as the bottom sediment in a lake, cow's milk, or a specific body organ such as the liver. A **homogeneous compartment** is one in which the contaminant concentration does not vary spatially. This condition can be met in practice when mixing within the compartment is rapid relative to movement into or out of the compartment. Homogeneous compartments are either advective or nonadvective.

Nonadvective Compartments A **nonadvective homogeneous compartment** is one in which contaminant movement across a surface can be approximated as occurring in the absence of fluid flow. Examples are the incorporation of a gaseous contaminant such as SO_2 into raindrops, the transfer of a bloodborne contaminant such as lead across the blood–brain barrier, or the uptake of a soil contaminant such as ^{137}Cs into vegetation through the root system. In some nonadvective compartments the contaminant might actually be carried by a fluid medium, but the flow rate is negligible. Examples are contaminant uptake by fish from contaminated water or by humans from the consumption of contaminated food.

For a nonadvective compartment, the dispersion and advection terms in Eq. 2.22 are zero, and the equation reduces to

$$\frac{dC(t)}{dt} = g(t) - d(t) \tag{2.23}$$

where the generation and destruction terms include processes that occur inside the compartment as well as nonadvective transfer into and out of the compartment. Each of the three simple models introduced in Section 2.3.2 is frequently applied to nonadvective compartments and can be derived from Eq. 2.23.

The first-order removal model (Section 2.3.2.1) is obtained by letting $g(t) = 0$ and $d(t) = -kC(t)$. The resulting differential equation is Eq. 2.8, and the solution for $C(0) = C_0$ is Eq. 2.10. The constant-source first-order removal model (Section 2.3.2.2) is obtained by letting $g(t) = g$, yielding

$$\frac{dC(t)}{dt} = g - kC(t) \tag{2.24}$$

Equation 2.24 is identical to Eq. 2.12 except that $\dot{M}/V$ is replaced by g. Whereas $\dot{M}/V$ includes both transport into the compartment and generation inside, g accounts only for generation inside. The solution for $C(0) = 0$ is identical to Eq. 2.13, with $\dot{M}/V$ replaced by g.

The dynamic exchange of a contaminant between two nonadvective compartments which was illustrated conceptually in Figure 2.6 and graphically in Figure 2.7 is described mathematically by Eq. 2.23. Substituting appropriate rate expressions for generation and destruction would yield the transient behavior. However, as noted in Section 2.3.2.3, when the time required to reach equilibrium between the two compartments is small compared to the time scale of interest, the transient period can be neglected and partitioning can be approximated as occurring instantaneously through the instantaneous partitioning model (Eq. 2.14). Mathematically, the instantaneous partitioning model is obtained by solving Eq. 2.23 and letting $t \to \infty$ or, equivalently, setting $dC/dt = 0$. The latter is illustrated in Example 2.5.

▶ Example 2.5

The dynamic partitioning of a contaminant between the aqueous and solid phases of a surface water or groundwater system can be conceptualized as simultaneous adsorption and desorption. The adsorption and desorption rates can be approximated as first-order processes, with the adsorption rate being proportional to aqueous-phase concentration and the desorption rate being proportional to solid-phase concentration. The conceptual model is illustrated in Figure 2.10.

The adsorption rate is $k_1 C_w$, where k_1 is the first-order rate constant for adsorption, C_w is the contaminant concentration in the aqueous phase (taken to be independent of C_s and time), and the desorption rate is $k_2 C_s$, where k_2 is a first-order rate constant for desorption and C_s is contaminant concentration in the solid phase. The differential equation for solid-phase concentration thus becomes

$$\frac{dC_s}{dt} = k_1 C_w - k_2 C_s$$

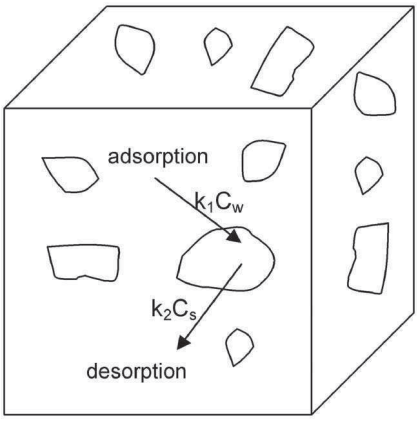

Figure 2.10 Dynamic partitioning of a contaminant between the solid and aqueous phases.

At equilibrium, $dC_S/dt = 0$ and

$$C_s = \frac{k_1}{k_2} C_w \quad \text{or} \quad \frac{k_1}{k_2} = \frac{C_s}{C_w}$$

This equation is equivalent to the equilibrium partitioning equation, where the equilibrium partitioning factor is

$$\text{PF}_e = \frac{k_1}{k_2}$$

Advective Compartments An **advective homogeneous compartment** is a homogeneous compartment in which the contaminant is carried into and out of the compartment by the fluid. In the process engineering literature, it is known as a continuous-flow stirred tank reactor (CFSTR) or a continuously stirred tank reactor (CSTR). A generalized advective compartment is presented in Figure 2.11. The advective flow rate into and out of the compartment is Q [L^3/T], the contaminant concentration in the influent fluid is C_1, and the initial contaminant concentration in the compartment is zero (i.e., $C_0 = 0$). The compartment volume, V, is constant. For this problem, the advective term in Eq. 2.22 [$-\nabla \cdot C(\mathbf{r},t)\mathbf{v}$] reduces to (see Example 2.7)

$$\frac{QC_1}{V} - \frac{QC(t)}{V} \tag{2.25}$$

and the governing differential equation for $C(t)$ takes on the form

$$\frac{dC(t)}{dt} = \frac{C_1 Q}{V} - \frac{C(t)Q}{V} + g(t) - d(t) \tag{2.26}$$

Equation 2.26 could also be formulated from the intuitive mass balance expressed in Eq. 2.3. The first term on the right-hand side of Eq. 2.26 is the rate at which the contaminant enters the compartment due to advection, the second is the rate

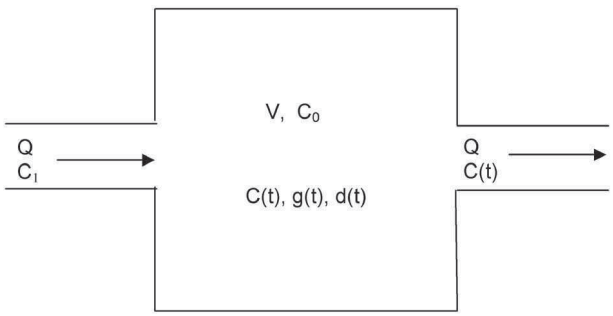

Figure 2.11 Advective compartment.

at which the contaminant leaves the compartment due to advection, the third term is the rate at which the contaminant is generated inside the compartment, and the fourth term is the rate at which the contaminant is destroyed inside the compartment.

▶ Example 2.6

A reservoir formed by damming a river has a volume of $2 \times 10^7\,\text{m}^3$. The river flowing into the reservoir has a volumetric flow rate of $8 \times 10^5\,\text{m}^3/\text{yr}$, and it is contaminated with vinyl chloride. In the reservoir, vinyl chloride undergoes first-order degradation with a first-order rate constant of $0.1\,\text{yr}^{-1}$. If the concentration of vinyl chloride in the river flowing into the reservoir is $25\,\mu\text{g/L}$, what is the concentration in the reservoir after 2 years and at steady state?

Solution The conceptual model for this problem is similar to Figure 2.11, except that there is no internal generation of vinyl chloride and destruction is first order. Thus, Eq. 2.26 reduces to

$$\frac{dC(t)}{dt} = \frac{C_1 Q}{V} - \frac{C(t)Q}{V} - kC(t)$$

Mathematically, this is the same as Eq. 2.24, with g replaced by C_1Q/V and k replaced by $k + Q/V$, where Q/V can be interpreted as a first-order rate constant for volumetric removal associated with fluid flow. The solution is

$$C(t) = \frac{C_1 Q}{V(k + Q/V)}\{1 - \exp[-(k + Q/V)t]\}$$

and the transient behavior of contaminant concentration is analogous to that depicted in Figure 2.5, where $t_{1/2} = \ln 2/(k + Q/V)$. Substituting numerical values gives us

$$\begin{aligned} C(t) &= \frac{(25\,\mu\text{g/L})(8 \times 10^5)}{2 \times 10^7\,\text{m}^3\left[0.1\,\text{yr}^{-1} + (8 \times 10^5\,\text{m}^3/\text{yr})/(2 \times 10^7\,\text{m}^3)\right]} \\ &\quad \left\{1 - \exp\left[-\left(0.1\,\text{yr}^{-1} + \frac{8 \times 10^5\,\text{m}^3/\text{yr}}{2 \times 10^7\,\text{m}^3}\right)t\right]\right\} \\ &= 7.14[1 - \exp(-0.14t)] \end{aligned}$$

Letting $t = 2\,\text{yr}$ and $t = \infty$, the concentrations at 2 years and at steady state are, respectively,

$$\begin{aligned} C(2\,\text{yr}) &= 7.14\{1 - \exp[(-0.14\,\text{yr}^{-1})(2\text{yr})]\} \\ &= 1.74\,\mu\text{g/L} \\ C(\infty) &= 7.14[1 - \exp(-\infty)] \\ &= 7.14\,\mu\text{g/L} \end{aligned}$$

► **Example 2.7** *Derivation of Eq. 2.26 from the Contaminant Transport Equation*

The contaminant transport equation is the fundamental basis for most mathematical models used in environmental risk assessment, and it can be used to derive both simple and complex models. Equation 2.26 is an example of a simple mathematical model that can be developed using the mass balance principles introduced in Section 2.3.1.2. It can also be derived from the contaminant transport equation, Eq. 2.22, as follows.

In a homogeneous compartment, the concentration is uniform spatially. Since dispersion does not occur unless there is a concentration gradient, the dispersion term in Eq. 2.22 is zero. This leaves

$$\frac{\partial C(\mathbf{r},t)}{\partial t} = -\nabla \cdot C(\mathbf{r},t)\mathbf{v} + g(\mathbf{r},t) - d(\mathbf{r},t)$$

Integrating over the volume of the compartment yields

$$\iiint_V \frac{\partial C(\mathbf{r},t)}{\partial t} dV = \iiint_V -\nabla \cdot C(\mathbf{r},t)\,\mathrm{v} dV + \iiint_V g(\mathbf{r},t)\,dV - \iiint_V d(\mathbf{r},t)\,dV$$

For a homogeneous compartment the average concentration in the compartment is

$$C(t) = \frac{1}{V}\iiint_V C(\mathbf{r},t)\,dV$$

Then the left-hand side (LHS) can be simplified as follows:

$$\iiint_V \frac{\partial C(\mathbf{r},t)}{\partial t} dV = \frac{\partial}{\partial t}\iiint_V C(\mathbf{r},t)\,dV = \frac{\partial}{\partial t}[VC(t)] = V\frac{dC(t)}{dt}$$

Similarly, the generation and degradation terms may be averaged over the volume, $g(\mathbf{r},t) \rightarrow g(t)$ and $d(\mathbf{r},t) \rightarrow d(t)$, and the second and third terms on the right-hand side (RHS) can be simplified to:

$$\iiint_V g(t)\,dV - \iiint_V d(t)\,dV = g(t)\iiint_V dV - d(t)\iiint_V dV = g(t)V - d(t)V$$

The advection term can be transformed into a more useful form by invoking Gauss's divergence theorem,

$$\iiint_V -\nabla \cdot C(\mathbf{r},t)\mathbf{v} dV = -\oiint_A C(\mathbf{r},t)\mathbf{v} \cdot \mathbf{n}\,dA$$

where **n** is the outward-pointing unit normal. This equation is basically a mathematical way of expressing a contaminant mass balance due to advection. The LHS is the rate of change of contaminant mass in the compartment due to advection,

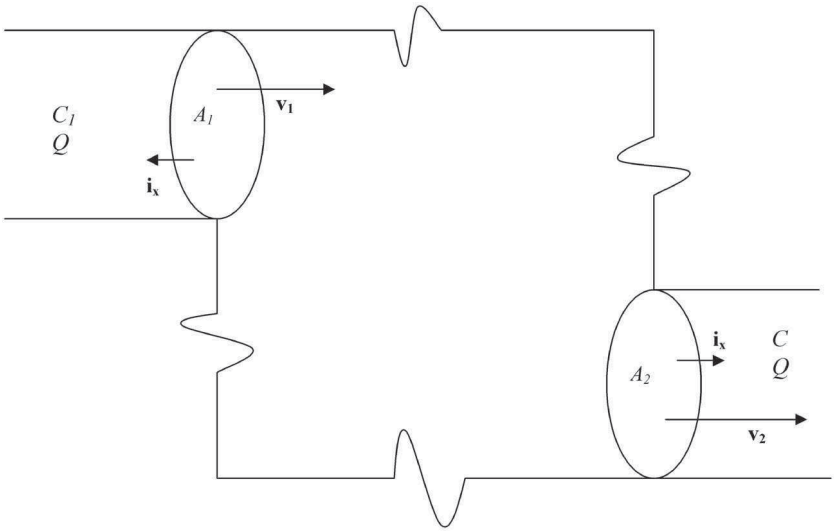

Figure 2.12 Modification of the advective compartment illustration for the purpose of deriving Eq. 2.26. Inlet and outlet are magnified.

and the RHS is the net rate of flow into the compartment (indicated by the negative sign in front of the integral). The application of this equation to the advective compartment in Figure 2.11 is facilitated by expanding the inlet and outlet as in Figure 2.12.

Because $\mathbf{v}$ is zero everywhere except at the inlet and outlet, the surface integral reduces to an integral over A_1 and A_2:

$$-\oiint_A C(\mathbf{r},t)\mathbf{v}\cdot\mathbf{i}dA = -\left[C_1\iint_{A_1}\mathbf{v}_1\cdot\mathbf{i}_x dA + C(t)\iint_{A_2}\mathbf{v}_2\cdot\mathbf{i}_x dA\right]$$
$$= -[C_1 v_1\cos\theta_1 A_1 + C(t)v_2\cos\theta_2 A_2]$$

where θ_1 is the angle between $\mathbf{v}_1$ and the unit vector normal to A_1, which is 180°, and θ_2 is the angle between $\mathbf{v}_2$ and the unit vector normal to A_2, which is 0°. Thus,

$$-[-C_1v_1A_1 + C(t)v_2A_2] = C_1v_1A_1 - C(t)v_2A_2 = C_1Q - C(t)Q$$

and

$$-\oiint_A C(\mathbf{r},t)\mathbf{v}\cdot\mathbf{i}dA = C_1Q - C(t)Q$$

Substituting all of the simplified forms of the integrals and dividing by V yields

$$\frac{dC(t)}{dt} = \frac{C_iQ}{V} - \frac{C(t)Q}{V} + g(t) - d(t)$$

which is identical to Eq. 2.26.

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PROBLEMS

2.1 A small stream with a flow rate of 0.1 m^3/s empties into a river that has a flow rate of 2 m^3/s. The stream is affected by mining operations and is contaminated with arsenic at a concentration of 50 mg/L. The river is not affected

by mining and has an arsenic concentration of 0.03 mg/L upstream from the small stream. What is the arsenic concentration in the river downstream from the stream?

2.2 Consider an electroplating facility that discharges to a river liquid wastes containing chromium. The effluent flow rate is 0.05 m^3/s and the flow rate of the river is 5 m^3/s. If the concentration of chromium is not allowed to exceed 100 μg/L, what is the maximum allowable concentration of chromium in the effluent?

2.3 Consider a contaminant having a concentration C_0 at $t = 0$ which undergoes first-order degradation. Generate a table of C/C_0 as a function of time expressed as the number of half-lives up to a maximum of 10.

2.4 What is the concentration in air (mg/m^3) corresponding to 75 ppm (by volume) of H_2S?

2.5 Cobalt-60 is a radioactive form of cobalt that has a half-life of 5.2 yr. It is produced in nuclear reactors as the result of neutron activation of ^{59}Co and ^{60}Ni, and it is a major constituent of low-level radioactive waste. The current inventory of ^{60}Co in a trench at a low-level waste disposal facility is 10^{12} Bq.

(a) What is the mass of ^{60}Co in the trench?

(b) What will the inventory be 100 years from now (in Bq)?

2.6 In the Chernobyl nuclear accident, approximately 10^6 Ci (3.7×10^{16} Bq) of ^{137}Cs was released to the atmosphere. Calculate the mass that was released. The half-life of ^{137}Cs is 30 yr and its atomic weight is 137 g/mol.

2.7 The concentration of ethylene dibromide (EDB) in an aquifer is 100 mg/L. Biodegradation of the contaminant can be approximated as a first-order process with a rate constant of 0.02 yr^{-1}. How long will it take for the concentration to decrease to 20 mg/L?

2.8 The pesticide carbaryl (the active ingredient in Sevin) undergoes photolysis when exposed to sunlight. Presented in the table are data from laboratory tests in which a simulated surface water was contaminated with carbaryl and exposed to the sun. Estimate a first-order rate constant for photolysis from these data.

Time (days)	Carbaryl (mg/L)	Time (days)	Carbaryl (mg/L)
0	148	20	52
2	137	50	15
5	113	100	1.1
10	94		

2.9 Consider the problem in Example 2.3. What would the removal rate constant need to be to keep the steady-state concentration below 10 mg/m^3?

2.10 Solve the differential equation in Example 2.6 using the Laplace transform technique (see Appendix A).

2.11 Consider the constant-source first-order removal model. Show that the steady-state concentration is $\dot{M}/Vk$ in two different but equivalent ways:

(a) Set $dC(t)/dt = 0$ in Eq. 2.12 and solve for C_{ss}.

(b) Take $\lim_{t\to\infty} C(t)$ for $C(t)$ given by Eq. 2.13.

2.12 In some states the legal intoxication limit is 0.8 g of ethanol per liter of body fluid. Use the constant-source first-order removal model to calculate the alcohol content in a person who over the course of 2 hours consumes four cans (355 mL/can) of beer that has an ethanol content of 6 g per 100 mL. The removal rate constant is $0.3\,h^{-1}$ and the person's volume of body fluids is 40 L. Does the concentration exceed the limit? If so, how long will it take for the concentration to decrease to the limit?

2.13 Consider the constant-source first-order removal model when the initial concentration is nonzero [i.e., $C(0) = C_0$].

(a) Using Laplace transforms, solve the differential equation for $C(t)$.

(b) Find the steady-state concentration, C_{ss}.

(c) Sketch $C(t)$ vs. t for (**i**) $C_0 > C_{ss}$ and (**ii**) $C_0 < C_{ss}$.

(d) Determine the time required for the concentration to come within 1% of the steady-state concentration.

2.14 A risk assessment is to be performed for a proposed incinerator. One of the pathways to be analyzed is atmospheric transport of lead (Pb) and subsequent uptake by crops at a large truck farm located near the proposed site, as illustrated in Figure 2.13. The following expression for the lead flux vector (including both the advective and dispersive components) is obtained by fitting a curve to the predictions of an atmospheric dispersion model:

$$\mathbf{j}_{Pb}(x,y,0) = 30(x - y^2)e^{-2x}\mathbf{i}_x + 3(2x - y^2)e^{-x}\mathbf{i}_y + 5(y^2 - x)\mathrm{e}^{-(x/2)}\mathbf{i}_z$$

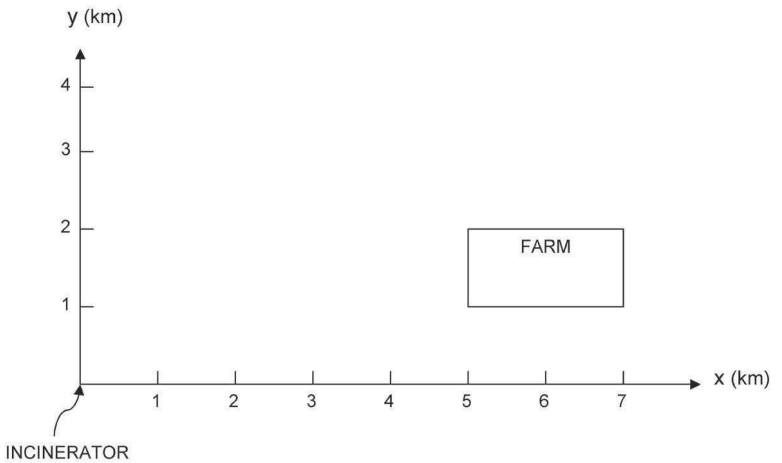

Figure 2.13 Location of the farm in Problem 2.14.

where $\mathbf{j}_{Pb}$ has units of mg/km$^2 \cdot$s and x and y are in kilometers. (Unit conversions are contained within each term.) The equation above is valid for $|y| < \sqrt{x}$. Using the flux vector, find the lead deposition rate (in mg/s) on the farm.

2.15 The two countries Smokylvania and Envirostan share a border, and they have a long-standing history of conflict, resulting from fundamental differences in societal values. Smokylvania is a heavily industrialized country whose people value material goods and a high standard of living. Envirostan is a pastoral country whose people value a clean environment and a high quality of life. Envirostan is downwind from Smokylvania, and its government wants the government of Smokylvania to impose stricter controls on atmospheric releases from its industrial facilities. The government of Smokylvania does not want to burden its industries with environmental controls the government considers to be unnecessary.

A risk assessment is to be performed to determine if emissions from Smokylvania are affecting the environment in Envirostan. One of the contaminants of potential concern is SO_2. The problem is to determine the emission rate (kg/s) of SO_2 from Smokylvania into Envirostan. The wind velocity vector and SO_2 concentration along the 100-km border between the two countries are

$$\bar{\mathbf{v}} = 10\frac{3}{5}\mathbf{i}_x + \frac{4}{5}\mathbf{i}_y \qquad \text{m/s}$$

and

$$C(x,0,z) = (100x - x^2)\mathrm{e}^{-(z/1000)} \qquad \text{mg(c)}/\text{m}^3$$

The x-axis lies along the border between the two countries and the z-axis is the vertical direction. The y-axis is positive in Envirostan. Unit conversions are contained within each term of the expressions.

2.16 Consider an ecosystem covering an area of 1 km^2 consisting of the compartments specified in Table 2.3. Consider 1 kg of a contaminant that is introduced into the ecosystem. The partition coefficients for the contaminants are as follows:

$$\mathrm{PF}_{aw} = 10\frac{\text{mg(c)}/\text{m}^3\text{(a)}}{\text{mg(c)}/\text{L(w)}} \qquad \mathrm{PF}_{sw} = 100\frac{\text{mg(c)}/\text{kg(s)}}{\text{mg(c)}/\text{L(w)}}$$

$$\mathrm{PF}_{bw} = 100\frac{\text{mg(c)}/\text{kg(b)}}{\text{mg(c)}/\text{L(w)}} \qquad \mathrm{PF}_{fw} = 1000\frac{\text{mg(c)}/\text{kg(f)}}{\text{mg(c)}/\text{L(w)}}$$

(a) Find C_w.

(b) Fill in the blanks in Table 2.4.

TABLE 2.3 Compartment Data for Problem 2.16

Compartment	Dimensions	Mass Density
Air (a)	$1000\,m \times 1000\,m \times 1000\,m$	$1.3\,kg/m^3$
Soil (s)	$1000\,m \times 800\,m \times 0.125\,m$	$1500\,kg/m^3$
Water (w)	$1000\,m \times 200\,m \times 5\,m$	$1000\,kg/m^3$
Bottom sediment (b)	$1000\,m \times 200\,m \times 0.05\,m$	$1500\,kg/m^3$
Fish (f)	1 ppm by volume in water	$500\,kg/m^3$

TABLE 2.4 Results of Calculations for Problem 2.16

Compartment	Concentration [$mg(c)/m^3$ or mg(c)/kg or mg(c)/L]	Fractional Inventory
Air		
Soil		
Water		
Sediment		
Fish		

3 Release Assessment

3.1 INTRODUCTION

The ultimate objective of the release assessment process is to determine the contaminant **emission rate**, which is the amount emitted into the environment per unit time (M/T for chemical contaminants; activity[1]/T for radiological contaminants). Release assessment consists of (1) the qualitative identification of potential contaminants and their physicochemical form, and (2) the quantitative characterization of the spatial and temporal behavior of the contaminant emission rate.

Release assessment is based on direct measurement, process knowledge, or a combination of the two. **Direct measurement** of emissions is probably the most reliable means of characterizing contaminant releases to the environment. However, measurement is often either not possible, as is the case for retrospective and prospective releases, or not practical, as when instrumentation is not available to monitor emissions. The alternative is to apply process knowledge. In the context of release assessment, **process knowledge** refers to knowledge of the various processes responsible for contaminant generation and release at a site or facility. It may be obtained from process descriptions in the open literature, blueprints and flowcharts of the facility, interviews with long-time workers, information from facility engineers and scientists, and consultation with outside experts. Even if measurement data are available, process knowledge is needed to formulate the conceptual model of release.

3.2 CONCEPTUAL MODEL

The conceptual model of release includes contaminant inputs and generation processes, contaminant release mechanisms, and contaminant release routes. These, in turn, involve a variety of physical, chemical, and biological processes. For example, a conceptual model of the release of gaseous effluent from a coal-fired power plant would include the characteristics of the coal, chemical and physical processes responsible for the production of gaseous and particulate contaminants in the combustion process, subsequent gas-phase reactions and physical removal processes that alter the contaminant composition, and contaminant removal from gaseous effluent by air pollution control devices. A conceptual model of contami-

[1] The quantification of radioactivity was explained in Chapter 2.

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nant release from buried waste might include characterization of the wastes and the waste containers, processes for the degradation of waste containers, penetration of rainwater through the trench cap, leaching- or solubility-limited dissolution of contaminants from the exposed waste, chemical and microbial transformation of waste constituents, and transport of water through the liner and backfill in the bottom of the trench. Process knowledge of this type can be used to identify the important contaminants and the major processes that can lead to their release. Consequently, development of a conceptual model is typically a multidisciplinary effort requiring expertise in areas such as physical chemistry, process engineering, microbiology, organic chemistry, air pollution control, geochemistry, soil physics, and environmental engineering.

The conceptual model for release is one component of a larger, comprehensive conceptual model that must be developed for each risk assessment. The comprehensive model includes all four components of the risk calculation: release, transport, exposure, and consequence. In the comprehensive model, the conceptual model for release provides the source term for the conceptual model for transport through environmental media. The development of conceptual and mathematical models of environmental transport is the subject of Chapters 4 through 8.

▶ Example 3.1

Process knowledge can be used to estimate the atmospheric release rate of SO_2 from a coal-fired power plant. This is accomplished by first developing a conceptual model for SO_2 generation and release. From process knowledge it is known that SO_2 is produced when coal containing sulfur undergoes combustion in the boiler. The gaseous effluents from the boiler are passed through a scrubber, which removes some of the SO_2, leaving the remainder to be released to the atmosphere through a stack. These processes are represented in the conceptual model depicted in Figure 3.1, which provides the basis for the following equation for calculating the emission rate of SO_2:

$$\dot{S}_{SO_2} = \dot{m}_{coal} f_s f_{ox} r(1 - \varepsilon)$$

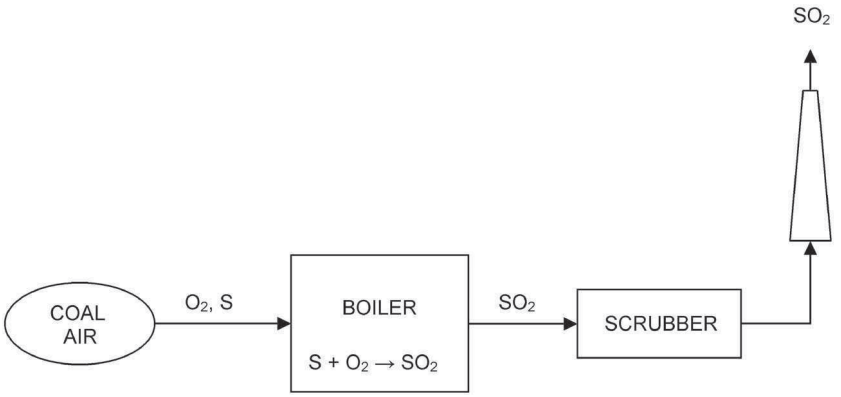

Figure 3.1 Model for the calculation of the SO_2 emission rate in Example 3.1.

where $\dot{S}_{SO_2}$ is the emission rate of SO_2 [kg(SO_2)/s], $\dot{m}_{coal}$ the rate at which coal is fed to the boiler [kg(coal)/s], f_s the fraction (by weight) of sulfur in the coal [kg(S)/kg(coal)], f_{ox} the fraction of sulfur oxidized to SO_2 in the combustion process, r the ratio of molecular weights of SO_2 to S [MW(SO_2)/MW(S)], and ε the efficiency of the scrubber for removing SO_2.

3.3 CONTAMINANT IDENTIFICATION

Once the conceptual model is developed, it is possible to identify contaminants that are or may be released to the environment. The goal is to identify all contaminants posing a potential for human health effects. For example, the investigation of an abandoned hazardous waste site under Superfund is concerned with any substance present at the site that may lead to deleterious effects. Sometimes regulations are more specific, with the scope being limited to a single contaminant or group of contaminants dictated by the regulatory framework. A manufacturer interested in producing a new pesticide may only be required to examine the risk posed by that substance in the environment; or a regulatory permit may contain release limits for a specified class of chemicals, with the risk assessment conducted only on the regulated compounds. Thus, contaminant identification cannot proceed until the risk assessment framework has been established and the goals are understood.

Presented in Table 3.1 is a list of important contaminants, their principal source or use, and health effects of potential concern. For purposes of contemporary human health risk assessment in the United States, it is convenient to group these contaminants into five general categories: organic compounds, metals, inorganic gases, radionuclides, and others. Most organic compounds of environmental concern, such as solvents, insecticides, herbicides, and chemical intermediates, are produced because they serve a particular useful purpose. Some, however, are either a by-product of producing a specific organic compound or a degradation product. Although metals occur naturally in the environment, human activities often produce elevated concentrations, resulting in potential effects on humans or ecosystems. The inorganic gases considered in this book are those that represent a human health threat, which is often through accidental, short-term releases. Inorganic gases linked to ecological impacts or climate change are not considered here. There are both anthropogenic and naturally occurring radionuclides of concern. Human-made radionuclides are often segregated into nuclear reactor fission products, nuclear reactor activation products, and transuranics. It is impossible to capture all contaminants of potential concern in a small number of discrete categories, and those that do not fit into one of the other four categories are included in "other." The contaminants listed in Table 3.1, plus some additional contaminants of interest, are separated into these categories in Table 3.2.

Specification of the physicochemical form of a contaminant can be important in the characterization of a release, since the form of a contaminant can affect its transport and its effects. For example, chromium(VI) is more toxic than chromium(III); the mobility of most metals in the subsurface depends to a large degree on the metal's oxidation state; PCBs have different chemical forms known as congeners, and toxicity is congener dependent; and some radionuclides pose a

TABLE 3.1 Common Environmental Contaminants in the United States

Contaminant	CAS[b]	Source or Use	Potential Human Health Effects[a] (ATSDR 2005)
Aflatoxin B1	001402-68-2	Fungal product in various crops	Acute: abdominal pain, vomiting, pulmonary edema, convulsions, death Cancer: liver
Aldrin/dieldrin	000309-00-2 000060-57-1	Banned insecticides	Acute: headache, dizziness, nausea, death
Arsenic	007440-38-2	Wood preservative, pesticide	Acute (inh): respiratory irritation Acute (ing): nausea, abnormal heart rhythm, death Chronic: skin discoloration, swelling Cancer: lung, bladder, liver, kidney, prostate
Benzene	000071-43-2	Industrial intermediate	Acute: drowsiness, dizziness, unconsciousness Chronic: anemia Cancer: leukemia
Cadmium	007440-43-9	In batteries, alloys; electroplating	Acute (inh): lung damage, kidney damage, death Acute (ing): stomach irritation, diarrhea Chronic: bone disorders, kidney damage
Carbon tetrachloride	000056-23-5	Many uses in past, most uses banned today	Acute: liver, kidney, and nervous system damage
^{137}Cs	007440-46-2	Nuclear reactor fission product, industrial and medical uses	Acute: radiation sickness Cancer: radiation-induced cancers
Chlordane	000057-74-9	Banned insecticide	Acute: digestive system, liver, and nervous system damage; headache, irritability, confusion, weakness, vision problems, vomiting, stomach cramps, diarrhea, jaundice
Chlorine	007782-50-5	Bleach for paper and cloth	Acute (inh): irritation of eyes, skin, and respiratory tract; pulmonary edema, death
Chloroform	000067-66-3	Industrial intermediate	Acute (inh): dizziness, fatigue, headache Chronic: liver and kidney damage
Chromium(VI)	018540-29-9	Steel manufacture, plating	Acute (inh): irritation of nose Acute (ing): stomach upsets and ulcers, convulsions, kidney and liver damage, death
^{60}Co		Nuclear reactor activation product, industrial and medical uses	Acute: radiation sickness Cancer: radiation-induced cancers

(*Continued*)

TABLE 3.1 *Continued*

Contaminant	CAS[b]	Source or Use	Potential Human Health Effects[a] (ATSDR 2005)
Creosote	008001-58-9	Wood preservative	Acute (inh): irritation of respiratory tract Acute (ing): skin irritation, eye burns, convulsions, unconsciousness, death Chronic (skin): skin damage Cancer: skin, scrotum
DDT, DDD, DDE	000050-29-3 000072-55-9 000072-54-8	Banned pesticide	Acute: tremors and seizures Cancer: probable human carcinogen
Ethylene dibromide (EDB)	000106-93-4	Pesticide, gasoline additive in the past	Acute (inh): reproductive effects Acute (ing): skin irritation, stomach ulcers Cancer: probable human carcinogen
Heptachlor/heptachlor epoxide	000076-44-8 001024-57-3	Insecticide	Acute: dizziness, confusion, convulsions
Hexachlorocyclohexanes (lindane)	000058-89-9	Insecticide	Acute: blood disorders, dizziness, headache, seizures, changes in the levels of sex hormones Cancer: probable human carcinogen
^{3}H (tritium)		Nuclear reactor fission or activation product	Cancer: radiation-induced cancers
Hydrogen sulfide	007783-06-4	Various natural and human-made sources	Acute (inh): irritation of respiratory tract, death Chronic (inh): eye irritation, headache, fatigue
^{131}I	007553-56-2	Nuclear reactor fission product	Acute: radiation sickness Cancer: thyroid
$^{85}Kr/^{88}Kr$		Nuclear reactor fission product	Acute: radiation sickness Cancer: radiation-induced cancers
Lead	007439-92-1	In batteries, solder, paint, gasoline	Acute and chronic: nervous system, kidney, and reproductive system damage
Mercury	007439-97-6	Chemical catalyst; in thermometers, dental fillings, batteries	Acute and chronic: impaired neurological development, especially in fetuses, infants, and children; kidney damage, respiratory failure, death
Methylene chloride	000075-09-2	Industrial solvent, paint solvent	Acute (inh): neurological damage Acute (skin): burns to skin and eyes
Methyl isocyanate	000624-83-9	Intermediate in manufacture of pesticides and plastics	Acute (inh): eye and throat irritation, severe eye and lung damage, death
Nitrogen oxides	010102-43-9 010102-44-0	High-temperature combustion	Acute (inh): irritation of respiratory tract
Particulate matter		Combustion of coal	Acute and chronic: respiratory problems, death

TABLE 3.1 *Continued*

Contaminant	CAS[b]	Source or Use	Potential Human Health Effects[a] (ATSDR 2005)
Plutonium isotopes	007440-07-5	Nuclear activation product	Cancer: lung, liver, and bone in animals
Polychlorinated biphenyls (PCBs)		Insulator for capacitors	Acute (skin): acnelike skin conditions Acute (ing): liver damage, neurobehavioral and immunological changes in children Cancer: probable human carcinogen
Polycyclic aromatic hydrocarbons (PAHs)		Incomplete combustion	Acute and chronic: reproductive and immunological effects in animals Cancer: probable human carcinogen
^{226}Ra		Naturally occurring	Cancer: bone
^{222}Rn		Naturally occurring	Cancer: lung
^{90}Sr	007440-24-6	Nuclear reactor fission product	Acute: anemia, impaired bone growth in children Cancer: radiation-induced cancers
Sulfur dioxide	007446-09-5	Combustion of coal	Acute: irritation of respiratory tract, respiratory distress
Tetrachloroethylene (PCE)	000127-18-4	Dry cleaning, metal degreasing	Acute: dizziness, headaches, sleepiness, confusion, nausea, difficulty speaking and walking, unconsciousness, death Cancer: probable human carcinogen
Thorium	000440-29-1	Naturally occurring	Chronic: liver disease, blood disease Cancer: lung (inh), pancreas, leukemia, liver, spleen
Trichloroethylene (TCE)	000079-01-6	Solvent for cleaning metal parts	Acute: nervous system effects, liver and lung damage, abnormal heartbeat, coma Cancer: probable human carcinogen
Uranium/uranium isotopes	007440-61-1	Naturally occurring; fuel for nuclear reactors	Chronic: kidney disease (from chemical toxicity)
Vinyl chloride	000075-01-4	Intermediate in manufacture of plastics	Acute: dizziness, sleepiness, unconsciousness, death Chronic: liver damage, immune reactions, nerve damage Cancer: liver
^{138}Xe		Short-lived nuclear reactor fission product gas	Acute: radiation sickness

[a] inh, inhaled; ing, ingested.
[b] The CAS is a unique 9-digit number assigned to chemical substances by the American Chemical Abstracts Service.

TABLE 3.2 Generic Contaminant Categories

Category	Specific Contaminants
Organic compounds	Aldrin/dieldrin, benzene, carbon tetrachloride, chlordane, chloroform, coal tar creosote, DDT, DDD, DDE, dioxin, EDB, heptachlor/heptachlor epoxide, lindane, methylene chloride, methyl isocyanate, PAHs, PCBs, PCE, TCE, vinyl chloride
Metals	Arsenic, barium, beryllium, cadmium, lead, mercury, nickel, selenium, silver, zinc
Inorganic gases	Cl_2, CO, HCl, HF, H_2S
Radionuclides	Fission products: ^{3}H, ^{85}Kr, ^{90}Sr, ^{131}I, ^{137}Cs Activation products: ^{14}C, ^{55}Fe, $^{58,60}Co$, ^{63}Ni, ^{134}Cs Transuranics: ^{237}Np, $^{238,239}Pu$, ^{241}Am, ^{244}Cm Naturally occurring: ^{40}K, ^{226}Ra, ^{222}Rn, ^{232}Th, ^{235}U, ^{238}U
Other	Viral and bacterial pathogens, particulate matter, asbestos, genetically engineered organisms, nanoparticles

much greater inhalation hazard than an ingestion hazard and are thus of greater concern in the form of respirable-sized particulate matter. In addition, many chemicals are transformed in the environment, and the fate of a contaminant is highly dependent on the initial chemical form.

An additional component of contaminant identification derives from the complex and iterative nature of a risk assessment. In many situations, a large number of contaminants may be present. It is generally not possible to perform a detailed assessment of all possible contaminants. In such cases, it may be necessary to identify the most important contaminants and reduce the total number to be analyzed through a screening analysis. As mentioned in Chapter 2, screening calculations are accomplished using simple models and highly conservative assumptions. Contaminants that pose minimal risks even after the application of conservative assumptions may be neglected. Alternatively, contaminants may be grouped on the basis of their environmental mobility and their toxicity, and a single indicator or surrogate contaminant may be substituted for each group. For example, a variety of organic solvents may be present at a hazardous waste site. If, however, these all have similar physical and toxicological properties, the entire inventory of solvents may be modeled as a single representative constituent, thus reducing the computational burden.

3.4 EMISSION-RATE QUANTIFICATION

In general, the complete quantitative characterization of contaminant release would consist of the probability, magnitude, and temporal and spatial dependence of the emission rate. The emission rate, which is obtained by measurement, process knowledge, or some combination, is frequently represented by relatively simple approximations. Ideally, characterization of the emission would be accomplished by direct measurement. Direct measurement could be used, for example, for the analysis of routine releases of easily measured contaminants from known emission points. Indirect environmental measurements are another possibility. The

measurements might be of the contaminant itself, the effects of the contaminant, or reaction–degradation products of the contaminant. Quantification of the release could possibly be achieved by back calculation through an environmental transport model. However, if there are multiple potential sources or if the contaminant is not measured directly, identification through environmental measurements becomes problematic.

For many problems, such as preoperational assessments of proposed facilities, assessments of hypothetical accidents, or retrospective risk assessments of historical releases, measurements are either not possible or not practical; therefore, it is necessary to develop a conceptual, mathematical, and computational model of contaminant emission. For the coal-fired power plant in Example 3.1, the conceptual model for the release of gaseous effluent would consider the composition of the coal, the rate at which coal is combusted, the rate at which gaseous and particulate contaminants are produced in the combustion process, the rate at which subsequent gas-phase reactions and physical removal processes occur, and the efficiency of air pollution control devices. The mathematical model, a material mass balance considering the factors and processes of the conceptual model, would provide mathematical expressions for the emission rates of the various gaseous effluents. The computational model would calculate these emission rates under a variety of release scenarios.

In some risk assessment problems, especially those involving waste disposal sites, the contaminant inventory is a critical part of the mathematical model for the emission rate. An **inventory** is an accounting of the total amount of the contaminant in a facility. It is obtained by conducting a mass balance on the contaminant: that is, the inventory is the difference between contaminant additions and subtractions through emissions and degradation. Example 3.2 illustrates the relationship between emission rate and inventory. The terms "source" and "source term" are often used in the context of release assessment; sometimes they refer to inventory and sometimes to emission rate.

▶ Example 3.2

A waste site receives a contaminant in waste at a constant rate, $\dot{M}$ (kg/yr). The contaminant inventory is reduced by two processes, degradation of the contaminant and leaching of the contaminant from the wastes. Degradation is first order with rate constant k (yr^{-1}). Although leaching of the contaminant from the site depends on variables that include the infiltration rate of water, the physicochemical form of the waste, and contaminant sorption to backfill, among others, it is commonly approximated as a first-order process. The leaching rate constant λ (yr^{-1}) is the fraction of the inventory that leaches from the site per year. The mathematical model for contaminant inventory, I, is an application of the constant-source first-order removal model:

$$\frac{dI}{dt} = \dot{M} - kI - \lambda I$$

The solution for $I(0) = 0$ is

$$I(t) = \frac{\dot{M}}{k+\lambda}\{1 - \exp[-(k+\lambda)t]\}$$

The emission rate is λI, or

$$\dot{S}(t) = \frac{\lambda \dot{M}}{k+\lambda}\{1 - \exp[-(k+\lambda)t]\}$$

3.4.1 Release Probability

The determination of release probability is unnecessary for problems such as the impact of routine permitted discharges or of analyses conducted specifically to assess the effects of a given release; but it is typically very important for accidental releases. Accidental contaminant releases can occur as the result of an unlikely external event (such as an earthquake, tornado, or airplane crash) or a sequence of unlikely internal events (such as a series of component failures or human error in a chemical or nuclear power plant). The severity of an accident can vary widely, and the release that occurs as a result of an accident can vary widely as well. Since accidental releases are probabilistic by nature, their variability is commonly characterized by an exceedance probability plot similar to the risk curves introduced in Chapter 1. An example is shown in Figure 3.2 for the mass of contaminant released in a hypothetical accident. Release curves of this type are interpreted in a fashion similar to that used for risk curves. For example, the probability of a release exceeding 10 units is about 3×10^{-5}, and the probability of exceeding 10,000 units is a little more than 10^{-8}.

Plots such as these are generated by a process formally known as probabilistic risk assessment (PRA) (Rasmussen 1990; Kumamoto and Henley 1996).

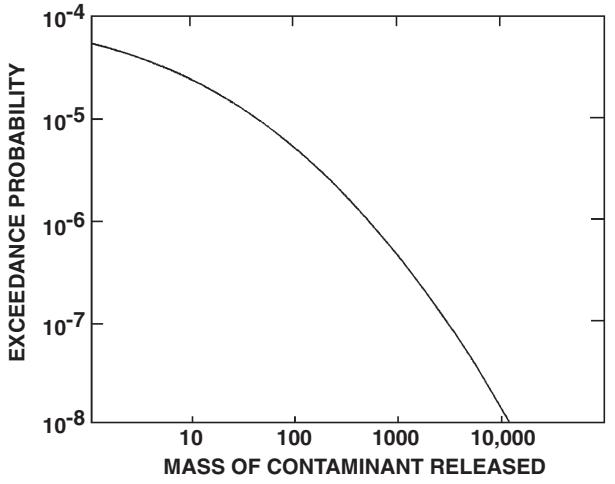

Figure 3.2 Exceedance probability for the mass of a contaminant released in an industrial accident.

Rasmussen identifies three approaches used in probabilistic risk assessments: actuarial methods, fault trees, and event trees.

3.4.1.1 Actuarial Methods Actuarial methods can sometimes be used when assessing the risks posed by commonplace events for which sufficient statistical data are available on the frequency of the events of interest. For example, compilations of data from death certificates indicate that the rates of death from injuries per 100,000 people by category are: motor vehicle, 15.3; firearm, 10.5; poison, 9.2; fall, 5.9; suffocation, 4.4 (Miniño et al. 2006). In applying actuarial data, care must be exercised to ensure that the underlying conditions are similar to the situation modeled. For example, in recent years added safety features incorporated into automobile designs have reduced the fatality rate from automobile accidents. Rates based on older data are no longer applicable.

The other limitation of actuarial methods is simply the lack of statistical data on rare events. For example, there have been very few accidents involving the release of very large quantities of chemical or radioactive contaminants. Consequently, the database is too small to provide reliable estimates of the probability of very large releases. In these cases, probabilistic methods relying on fault trees or event trees are typically used.

3.4.1.2 Fault Trees A **fault tree** is a logic diagram that depicts all of the possible ways in which a particular system failure (top event) can occur. In the environmental risk assessment context, the system failure is an accidental release of a contaminant. The fault tree for such a release is constructed by identifying the combinations of individual events, typically component failures, that can lead to the release. Starting with the contaminant release, the events are traced backward through various levels. Failure at one level results from a single event or series of events at a lower level. The events are linked through logical operators, called gates. If a series of events at a given level must all occur for the resulting higher-level event to occur, they are connected by an "AND" gate. If any of the events are sufficient for the resultant higher-level event to occur, they are connected by an "OR" gate. In general, systems containing AND gates at critical junctures are safer than systems containing OR gates in similar positions because multiple failures of systems or components must occur to have a system failure; with an OR gate, only one component or system need fail. Each tier of subsystem failures may be analyzed further in terms of more fundamental failures; this process may continue to increasingly more fundamental failures (e.g., component failures, parts failures) or fundamental events (initiating events). However, fault trees are usually "trimmed" at a reasonable level that is suitable for the problem being solved. By tracing events back to basic causes, a tree is generated that shows all possible events leading to the undesired outcome. The fault tree provides a logical relationship between the state of the top event (present or not) and the states of the more fundamental events in the system. This logical relationship may also be represented by an equivalent Boolean expression in which the state of the fundamental events is designated by a Boolean variable that can take on values of 1 or 0, representing the occurrence or nonoccurrence of the fundamental event. Furthermore, frequency of occurrence information for various events can be used in conjunction with the Boolean expressions derived to estimate the probability of the top event.

► Example 3.3

Anhydrous ammonia, commonly used as a feedstock for making fertilizer, may be synthesized on-site or it may be supplied in bulk by barges or rail tank cars. A common feature of most plants is tank storage of ammonia. Periodically, the storage tank must be refilled with ammonia produced on-site or shipped from off-site. A system for refilling a storage tank is shown in Figure 3.3. An operator switches on a pump to transfer ammonia from outside the facility into the storage tank. The operator receives a continuous reading from a sensor indicating the level of ammonia in the tank. Under normal operating conditions, the operator shuts off the pump when the ammonia reaches the desired level. As a safety measure, a second sensor, mounted near the top of the tank, indicates when the tank is almost completely full of liquid. This sensor switches a relay that shuts off the pump automatically when the high level is reached. A fault tree depicting the possible failure of this system is presented in Figure 3.4. The top event is a higher than acceptable pressure of ammonia in the storage tank ("ammonia storage tank overpressure"). Generally, this results from overfilling the tank and can lead to tank rupture, with concomitant release of ammonia liquid and gas to the environment. The AND gate feeding the top event indicates that both second-tier events must occur for the top event to occur. In this case the two second-tier events are (1) failure of the operator to shut off the fill pump AND (2) failure of the backup safety system to shut off the pump. Because this system has an AND gate at this critical juncture, it is safer than a similar system containing an OR gate in this position, all other things equal. The failure, "operator fails to shut off pump," may be caused by either of two more fundamental failures: (1) the operator fails to deactivate the power switch for the

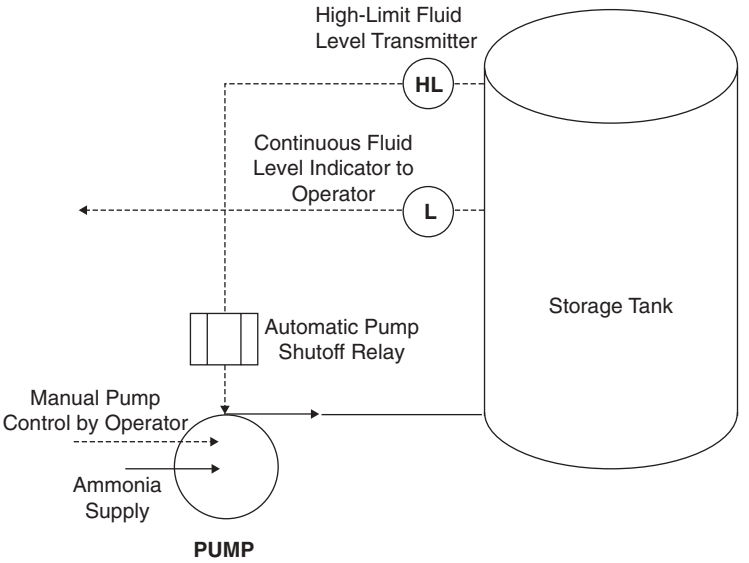

Figure 3.3 System for refilling the anhydrous ammonia storage tank in Example 3.3.

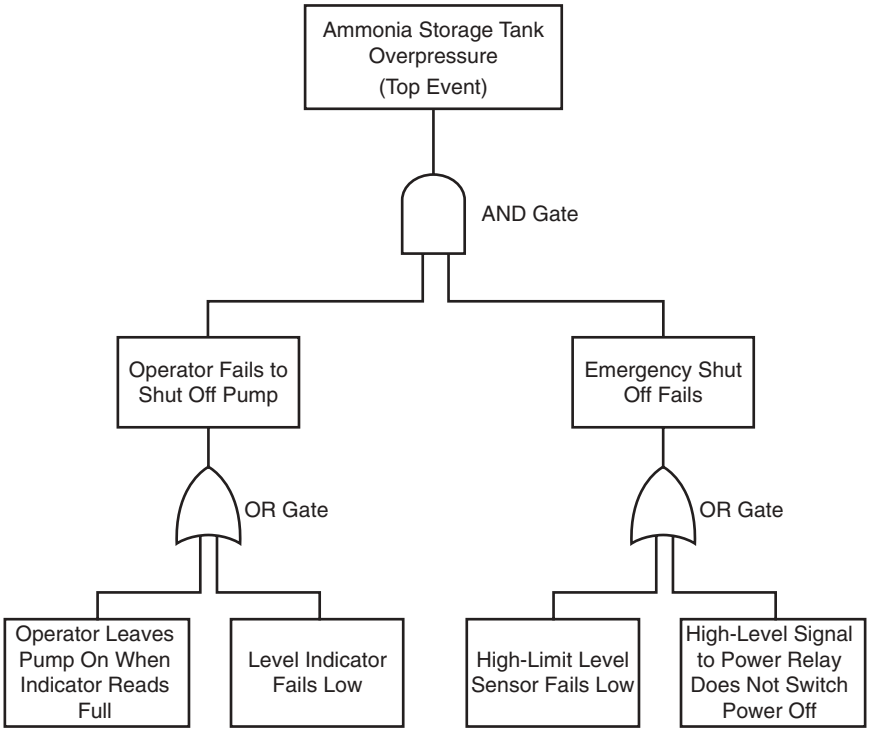

Figure 3.4 Fault tree for the release of anhydrous ammonia from a storage tank.

pump, even though the level gauge indicates a full tank, OR (2) the level gauge malfunctions and reads a lower level, even though the tank is full. The intermediate failure, "emergency shutoff fails," may be caused by either of two more fundamental failures: (1) the high-limit level sensor fails and indicates a low liquid level regardless of the actual liquid level, OR (2) the relay-operated cutoff switch maintains power to the pump, even though a high-liquid-level signal is received.

3.4.1.3 Event Trees An **event tree** is a logic diagram that identifies and quantifies the possible outcomes of a single initiating event. Event trees are commonly used to identify the spectrum of outcomes that can result from a given initiating event. Different outcomes are possible because preventive or mitigating measures may or may not be effective in eliminating or reducing a release. Each branch point in an event tree shows these two possible outcomes; the usual convention in event trees is to show failure as the lower branch and success as the upper branch. At the end of the tree, the outcome for each path through the tree is provided. Frequently, the conditional probability of each branch, at each node, is provided. The probability of success or failure for a branch point may be obtained from reliability data for a particular piece of equipment, from physical modeling, from a fault-tree

analysis, from expert judgment, or from a combination of methods. The quantification of probabilities at each branch in the tree permits calculation of the probability of each outcome through basic rules of probability theory. The completed event tree summarizes succinctly the various outcomes possible, the probability of occurrence of each outcome, and the sequence of events leading to that outcome. By combining the probability and the magnitude of the release for each branch of the event tree, a release exceedance curve similar to that in Figure 3.2 can be generated.

▶ Example 3.4

Consideration of the ammonia storage tank can be extended to illustrate an event tree. A modified system schematic and an associated event tree are shown in Figures 3.5 and 3.6, respectively. For this event tree the initiating event is refilling the ammonia storage tank, a routine operational event. Four systems are in place to prevent or mitigate potential adverse consequences that can arise from the initiating event: (1) the system to prevent overfilling the tank (analyzed in the fault tree example); (2) a pressure-operated relief valve, intended to open and relieve excess pressure when the set-point pressure is exceeded; (3) the tank's ability to resist a certain degree of overpressure without bursting; and (4) a secondary

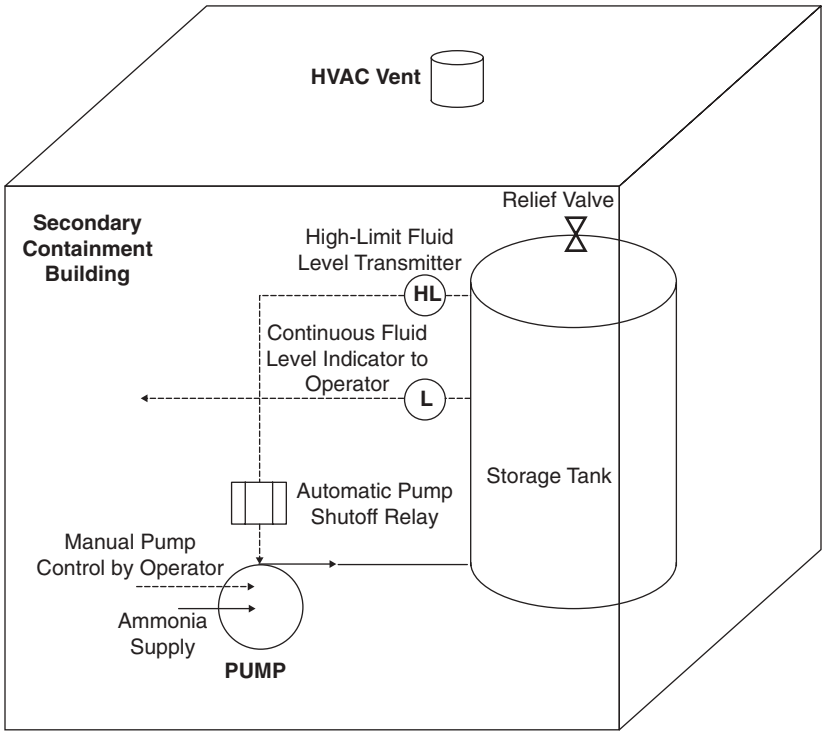

Figure 3.5 System for refilling an anhydrous ammonia storage tank.

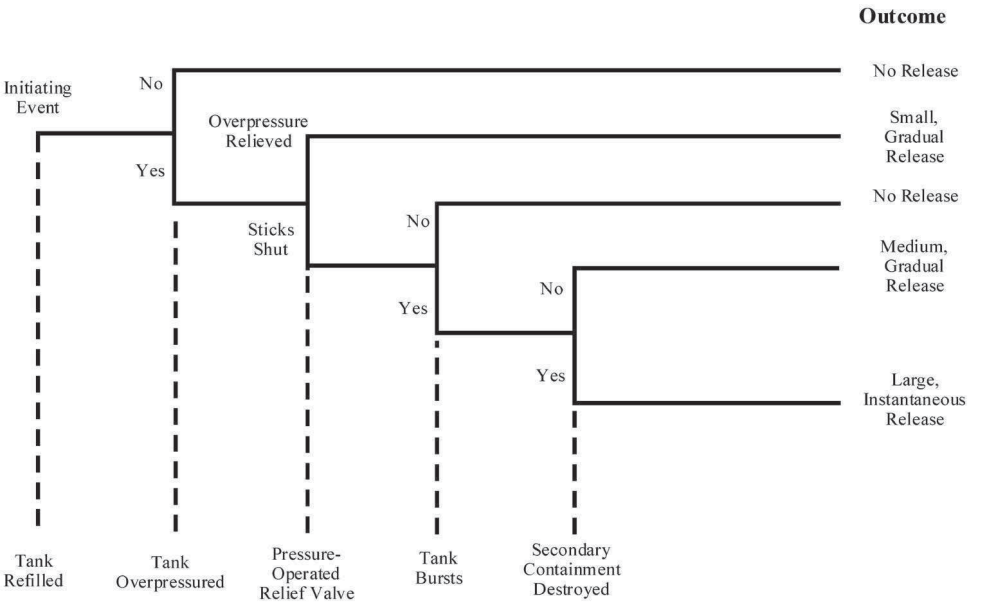

Figure 3.6 Event tree for the release of anhydrous ammonia from a storage tank.

containment building with a roof vent to allow any ammonia releases in the building to be vented gradually to the environment, if the building survives a tank bursting. For this example, the probabilities of success or failure at each branch point would probably be obtained by different methods. For example, actuarial data might be used for the failure rate of the pressure-operated relief valve, physical modeling might be used to determine the burst probability of the tank and survival probability of the secondary containment, and a fault tree such as that in Figure 3.4 might be used to determine the probability of tank overfill.

The event tree differs from the fault tree in that a fault tree answers the question "How can a specified release (particular event) occur?", and an event tree answers the question "For a specified initiating event, what releases may result and what are their likelihoods?" Whereas fault trees trace contributing events from the release backward, event trees trace events forward from the initiating event to the spectrum of releases. A fault tree can be used to analyze each branch in an event tree, thus allowing identification of the component failures and human errors that lead to the success or failure of a mitigating feature.

3.4.2 Contaminant Emission Rate

In general, the spatial and temporal dependence of a contaminant release are quantified by the **specific emission rate**, $\tilde{S}(\mathbf{r},t)$, which is mass emitted per unit volume per unit time. Since many of the closed-form analytical contaminant transport models are, by necessity, based on very simple spatial and temporal functions,

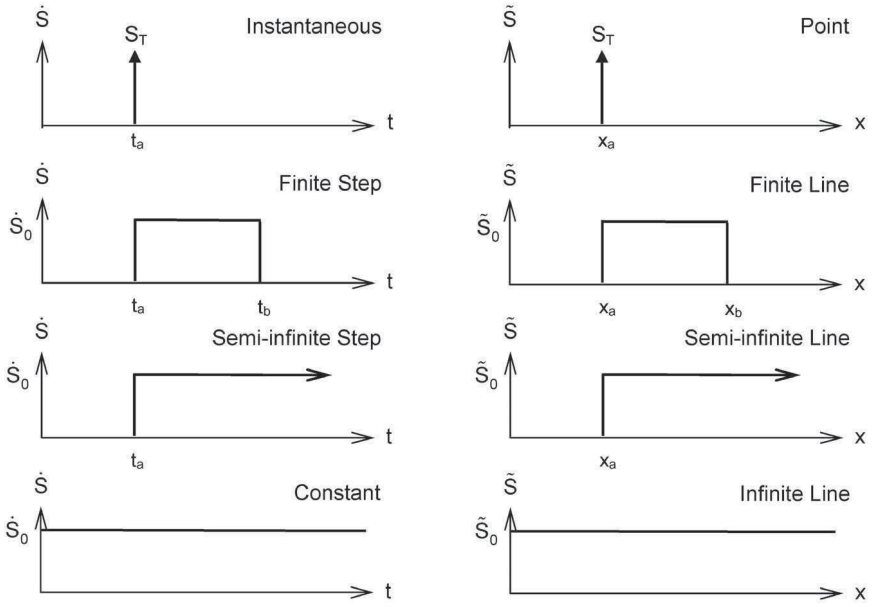

Figure 3.7 Emission-rate approximations.

actual emission rates are often replaced by simple approximations. Presented in Figure 3.7 are common temporal and spatial approximations. An emission occurring over a relatively short period of time may be represented by an **instantaneous approximation**, and one occurring over the course of a few days may be represented by a **finite-step approximation**. Similarly, for spatial dependence, an emission to the atmosphere from a stack or to a surface water from a process sewer outfall might be represented by a **point source approximation**, and an emission of atmospheric pollutants from a highway or leaching of wastes buried in a long, shallow trench might be represented by a **finite line source approximation**.

The approximations are formed such that the total amount of contaminant released under the approximation is equal to the actual amount released (i.e., mass is conserved). Mathematically, this is expressed as

$$S_T = \int_V \int_t \dot{\tilde{S}}_{\text{app}}(\mathbf{r},t)\, d\mathbf{r}dt = \int_V \int_t \dot{\tilde{S}}_{\text{act}}(\mathbf{r},t)\, d\mathbf{r}dt \tag{3.1}$$

where S_T is the total mass of contaminant released to the environment, $\tilde{\dot{S}}_{\text{app}}(\mathbf{r},t)$ the approximate specific emission rate, and $\tilde{\dot{S}}_{\text{act}}(\mathbf{r},t)$ the actual specific emission rate. In many risk assessments, the spatial dependence of the specific emission rate can be adequately approximated as a point in space, and the release is characterized by the **emission rate**, $\dot{S}(t)$, which is the amount of contaminant released per unit time. Emission-rate approximations are formulated analogously to the specific emission-rate approximations: that is,

$$S_T = \int_t \dot{S}_{\text{app}}(t)\, dt = \int_t \dot{S}_{\text{act}}(t)\, dt \tag{3.2}$$

TABLE 3.3 Common Emission-Rate Approximations[a]

Approximation	Analytical Form
Temporal Approximations	
Instantaneous at $t = t_a$	$\dot{S}_{\text{app}}(t) = S_T\delta(t - t_a)$
Semi-infinite step beginning at $t = t_a$	$\dot{S}_{\text{app}}(t) = \dot{S}_0 h(t - t_a)$
Finite step between $t = t_a$ and $t = t_b$	$\dot{S}_{\text{app}}(t) = \dfrac{S_T}{t_b - t_a}[h(t - t_a) - h(t - t_b)]$
Constant	$\dot{S}_{\text{app}}(t) = \dot{S}_0$
Spatial Approximations	
Point at $x = x_a$	$\tilde{S}_{\text{app}}(x) = S_T\delta(x - x_a)$
Line between x_a and x_b	$\tilde{S}_{\text{app}}(x) = \dfrac{S_T}{x_b - x_a}[h(x - x_a) - h(x - x_b)]$

[a] S_T, total mass emitted; $\dot{S}_0$, mass emitted per unit time; $\tilde{S}_0$, mass emitted per unit length; $h(t - t')$, Heaviside step function; $\delta(x - x')$, Dirac delta function.

The mathematical forms of some of the emission-rate approximations shown in Figure 3.7 are given in Table 3.3. Two special functions, the Dirac delta function and the Heaviside step function, are utilized in these approximations. They are described in Appendix A. The approximations in Table 3.3 are used as source terms in Chapters 4 to 7 to obtain analytical solutions to the contaminant transport equation. These analytical solutions are then used to illustrate the physicochemical aspects of contaminant transport in a generic environmental system: surface water, groundwater, and air.

▶ Example 3.5

Consider the actual emission rate shown in Figure 3.8 and represented mathematically as

$$\dot{S}_{\text{act}}(t) = 5t\exp\left(-\frac{t}{2}\right) \qquad \text{kg/h}$$

where t is in hours and the constant, 5, has units of kg/h^2. The total mass of contaminant released, S_T, is obtained by integrating the emission rate over the time of the release: in this case, from zero to infinity.

$$S_T = \int_0^\infty \dot{S}_{\text{act}}(t)\,dt \Rightarrow S_T = \int_0^\infty 5t\exp\left(-\frac{t}{2}\right)dt$$

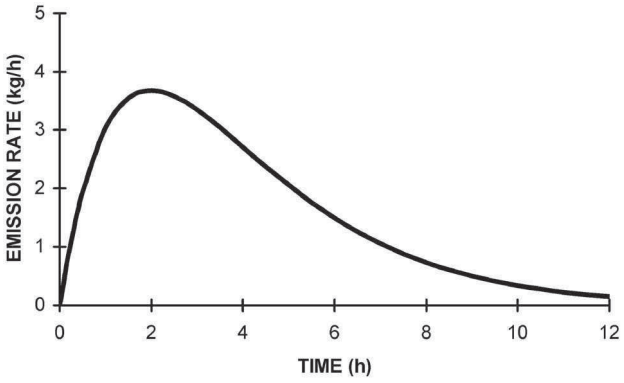

Figure 3.8 Actual emission rate for Example 3.5.

Integration by parts (or alternatively, finding the integral in a table of integrals) yields

$$S_T = -10\exp\left(-\frac{t}{2}\right)(t+2)\Big|_0^{\infty} = 20\,\text{kg}$$

Selecting an emission-rate approximation for a release is problem specific. For this problem, two approximations are illustrated here. The first is an instantaneous release at the time of the maximum emission rate. The maximum is determined by differentiating the emission rate with respect to time, setting the resulting expression equal to zero, and solving for $t_{\max}$:

$$\frac{dS_{\text{act}}}{dt} = \frac{d[5t\exp(-t/2)]}{dt} = 5\exp\left(-\frac{t}{2}\right) - \frac{5}{2}t\exp\left(-\frac{t}{2}\right) = 0$$

Solving for t yields $t_{\max} = 2\,\text{h}$. Thus,

$$S_{\text{app}}(t) = 20\delta(t - 2\,\text{h})\text{kg}$$

The second approximation is a finite step beginning at $t = 0$ and ending at $t = 4\,\text{h}$:

$$\dot{S}_{\text{app}}(t) = \dot{S}_0[h(t) - h(t - 4\,\text{h})]\text{kg/h}$$

Applying Eq. 3.2 to obtain $\dot{S}_0$ gives us

$$\int \dot{S}_0\,[h(t) - h(t-4)]dt = \int \dot{S}_{\text{act}}\,(t)\,dt$$

The right-hand side of the equation is the total mass emitted, which from the above is 20 kg. The left-hand side is solved as follows:

$$\int_0^{\infty} \dot{S}_0\,[h(t) - h(t-4)]dt = \dot{S}_0 \int_0^{\infty} [h(t) - h(t-4)]dt = \dot{S}_0 \int_0^{4} dt = 4\dot{S}_0$$

From above, the right-hand side is the total mass of contaminant released: $4\dot{S}_0 = 20$ or $\dot{S}_0 = 5\,\text{kg/h}$. Thus,

$$\dot{S}_{\text{app}}(t) = 5[h(t) - h(t - 4\,\text{h})]\text{kg/h}$$

The temporal dependence of an emission can be approximated as multiple instantaneous or finite-step emissions of differing magnitudes. This is illustrated in Example 3.6, where the actual emission rate in Example 3.5 is approximated as five finite-step emissions.

► **Example 3.6**

The emission rate in Example 3.5 is to be approximated as a series of five finite steps between 0 and 10, each step with a duration of 2 hours. The analytical expression for the emission-rate approximation is

$$\dot{S}_{\text{app}}(t = \dot{S}_1[h(t) - h(t-2)] + \dot{S}_2[h(t-2) - h(t-4)] + \dot{S}_3[h(t-4) - h(t-6)] + \dot{S}_4[h(t-6) - h(t-8)] + \dot{S}_5[h(t-8) - h(t-10)]\text{kg/h}$$

The magnitudes of the first four finite steps are assigned so that the actual total mass emitted during each 2-hour period is conserved:

$$\dot{S}_1 = \frac{\int_0^2 \dot{S}_{\text{act}}\,dt}{2\text{h}} = 2.64\text{kg/h}$$

$$\dot{S}_2 = \frac{\int_2^4 \dot{S}_{\text{act}}\,dt}{2\text{h}} = 3.29\text{kg/h}$$

$$\dot{S}_3 = \frac{\int_4^6 \dot{S}_{\text{act}}\,dt}{2\text{h}} = 2.07\text{kg/h}$$

$$\dot{S}_4 = \frac{\int_6^8 \dot{S}_{\text{act}}\,dt}{2\text{h}} = 1.08\text{kg/h}$$

The magnitude of the fifth time step, which begins at 8 hours and ends at 10 hours, is assigned to account for all of the mass emitted after 8 hours:

$$\dot{S}_5 = \frac{\int_8^\infty \dot{S}_{\text{act}}\,dt}{2\,\text{h}} = 0.92\text{kg/h}$$

The emission-rate approximation is compared to the actual emission rate in Figure 3.9.

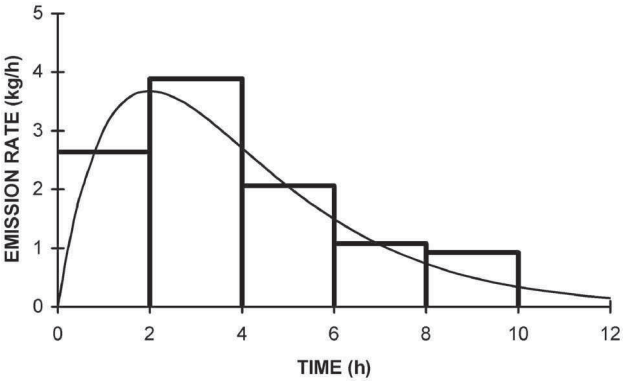

Figure 3.9 Comparison of the emission-rate approximation in Example 3.5 to the actual emission rate.

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ADDITIONAL READING

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PROBLEMS

3.1 Consider a coal-fired steam-electric plant that burns 90 kg of coal per second. The coal has a heating value of 12,000 Btu/lb (1 Btu = 1054 J), an ash content of 8%, and a sulfur content of 2.5%. Thirty percent of the ash remains in the boiler, and the remainder becomes entrained in the exhaust gases. All of the sulfur is converted to SO_2. The plant is equipped with an electrostatic precipitator that has a particulate removal efficiency of 99.5% and an SO_2

scrubber that has an SO_2 removal efficiency of 80%. The thermal efficiency of the power plant is 39%.

(a) What is the electrical output of the plant [in MW(e)]?

(b) What is the SO_2 emission rate (in kg/s) to the atmosphere and to the liquid effluent from the scrubber?

(c) What is the emission rate of particulate matter (in kg/s) to the atmosphere?

3.2 Consider the following emission rate for the accidental release of a contaminant to the atmosphere:

$$\dot{S}_{\text{act}}(t) = 12t - 4t^2 \qquad 0 \le t \le 3$$

where $\dot{S}_{\text{act}}(t)$ is in kg/h and t is in hours.

(a) What is the total mass released?

(b) At what time does the maximum emission rate occur?

(c) Write equations for the following emission-rate approximations: (**i**) instantaneous emission at $t = 1.5$ h, and (**ii**) finite-step emission from $t = 0$ to $t = 3$.

(d) Sketch the actual emission rate, the instantaneous approximation, and the finite-step approximation.

3.3 Consider the following actual emission rate:

$$\dot{S}_{\text{act}}(t) = \begin{cases} 5t & 0 \le t \le 4 \\ 0 & t > 4 \end{cases}$$

where $\dot{S}_{\text{act}}(t)$ is in kg/h and t is in hours.

(a) Sketch $\dot{S}_{\text{act}}$ vs. t.

(b) Find the total mass of contaminant released.

(c) Write the mathematical expression for each of the following emission-rate approximations: (**i**) finite-step emission from $t = 0$ to $t = 4$ h, and (**ii**) instantaneous emission at $t = 2$ h.

(d) Sketch the actual emission rate, the instantaneous approximation, and the finite-step approximation.

3.4 Consider the following emission rate for the accidental release of a contaminant to the atmosphere:

$$\dot{S}_{\text{act}}(t) = 8t \exp\left(\frac{-t^2}{4}\right)$$

where $\dot{S}_{\text{act}}(t)$ is in kg/h and t is in hours.

(a) What is the maximum emission rate, and when does it occur?

(b) What is the total mass of contaminant released? What fraction of the total is released in the 1- to 2-h period?

(c) Write the mathematical expression for each of the following approximations to the emission rate.

(i) Constant, finite step for the 0- to 4-hour period.

(ii) Constant, finite step for a period of 1 to 2 hours.

(iii) Instantaneous release at the time of the maximum emission rate.

(iv) Two contiguous finite steps. The first segment is from 0 to 4 hours, and the second is from 4 to 8 hours. In the segment from 0 to 4 hours, include all of the contaminant released during that period. In the segment from 4 to 8 hours, include all of the contaminant released between 4 hours and infinity.

(d) On a single graph, sketch the actual emission rate and each of the four approximations.

3.5 Consider the following emission rate for the accidental release of a contaminant to the atmosphere:

$$\dot{S}_{\text{act}}(t) = 30t \exp\left(\frac{-t}{10}\right)$$

where $\dot{S}_{\text{act}}(t)$ is in kg/h and t is in hours.

(a) Sketch $\dot{S}_{\text{act}}(t)$ vs. t.

(b) What is the maximum emission rate, and when does it occur?

(c) What is the total mass of contaminant released? What fraction of the total is released in the first 50 hours?

(d) Write the expression for each of the following approximations to the emission rate. Add (i) and (ii) to the sketch in part (a).

(i) Constant, finite step for the 0- to 50-hour period.

(ii) Constant, finite step for a period of 5 to 20 hours.

(iii) Instantaneous release at the time of the maximum emission rate.

3.6 Construct a fault tree for a flat tire. A flat tire can occur as a result of either a mechanical failure or an operational mishap. Operational mishaps include hitting objects in the roadway and running into an off-road object such as a curb or the edge of a culvert. Mechanical failure can result from a combination of high tire pressure, high tire temperature, and excessive tire wear.

4 Environmental Transport Theory

4.1 INTRODUCTION

Typical environmental transport pathways were illustrated in Figure 1.4. These pathways and others of importance are represented generically in Figure 4.1 in terms of discrete environmental compartments. The objective of environmental transport analysis is to estimate contaminant concentrations in these compartments. For some problems, such as a preexisting waste disposal site or an operating industrial facility, concentrations may be determined directly by measurement. However, such measurements are only possible for estimating the effects of current or past releases, and they only provide information for a given instant in time at a given set of locations. Thus, even if data are available, they may not be sufficient for a comprehensive risk evaluation. More typically, measurements are either not practical or not possible, and risk evaluations must be based solely on concentrations predicted using environmental transport models.

Environmental transport analysis consists of two overarching tasks. The first is to identify the important transport pathways and the physical, chemical, and biological processes that play an important role in contaminant behavior in those pathways. The second is to incorporate the pathways and processes into conceptual, mathematical, and computational models that are used to make quantitative estimates of contaminant concentrations in air, food, and water. The specific objective of the calculation is dictated by the measurement and assessment endpoints for the problem. This might be contaminant concentration at a given time and location, the time and magnitude of the maximum concentration at a specified point of compliance, or the location at which the contaminant exceeds a specified concentration.

The contaminant transport problem is complex because of the inherent complexity of environmental systems. In addition to the fluid flow processes that govern transport in air and water, other physical processes may be important as well as chemical and biological processes. These processes are not always well understood, and they can depend on many factors. These factors, in turn, may be poorly understood or highly variable. Nonetheless, by combining and interfacing empirical data for processes that are poorly understood with mathematical theory for processes that are well understood, it is possible to develop models for predicting contaminant concentrations in air, water, and food. Even though the study of environmental

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

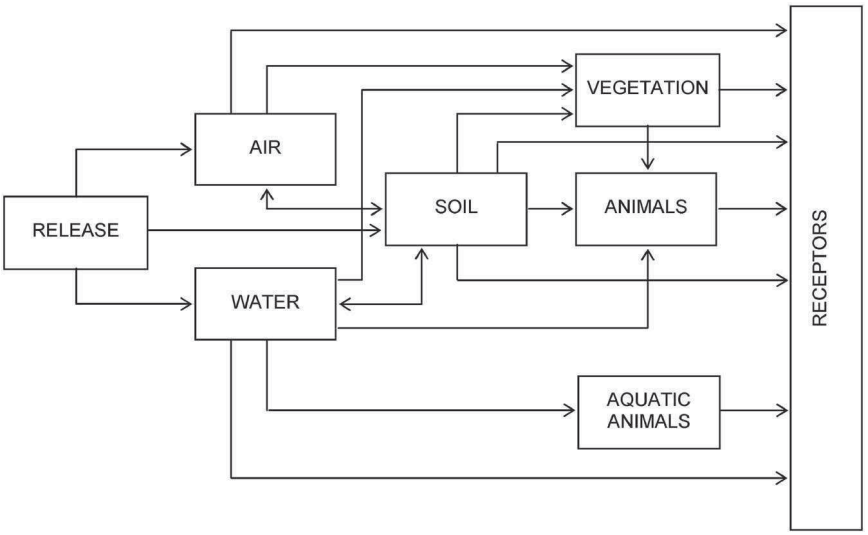

Figure 4.1 Generic environmental pathways and compartments.

transport processes is spread across many disciplines, the contaminant transport equation (Eq. 2.22) represents a unifying theoretical basis for the various mathematical models that are used today. The zero-dimensional compartmental models (first-order removal, constant-source first-order removal, and instantaneous partitioning) presented in Chapter 2 have applications in each of the four steps (release, environmental transport, exposure, consequences) of the risk calculation process. The one-, two-, and three-dimensional models developed in the present chapter, on the other hand, are limited primarily to environmental transport applications (although, in theory, they could be applied to biological transport as well). The solutions developed in this chapter are generic in that they can be applied to air, surface water, or groundwater transport problems. As pointed out in Chapter 2, this unified approach is possible because advection and dispersion are the primary determinants of contaminant transport in environmental media, and the spatial and temporal behaviors of contaminant concentration in response to a source are similar regardless of the transport medium. Medium-specific models for transport in surface water, groundwater, the atmosphere, and the food chain are developed in Chapters 5 to 8, respectively.

The contaminant transport equation is the means through which the various transport processes can be taken into account in a quantitative way. The sophistication of the mathematical description of a process depends on the level of mechanistic understanding of the process or the degree to which the process has been characterized empirically. Complex but poorly understood or poorly characterized processes are typically represented by very simple models. Conversely, well-understood or well-characterized processes generally have more complex or more rigorous mathematical descriptions.

4.2 ONE-DIMENSIONAL SOLUTIONS OF THE CONTAMINANT TRANSPORT EQUATION

A common environmental transport problem involves the release of a contaminant from an industrial facility to a river or stream, as illustrated in Figure 4.2*a*. The one-dimensional approximation for this problem is illustrated in Figure 4.2*b*. In a **one-dimensional approximation**, the contaminant is distributed uniformly across the channel, the fluid flow and dispersion are uniform and constant in time and space, and the x-axis is oriented to be parallel to the direction of advective flow. The concentration is a function of x and t only, and Eq. 2.22 reduces to

$$\frac{\partial C(x,t)}{\partial t} = D\frac{\partial^2 C(x,t)}{\partial x^2} - u\frac{\partial C(x,t)}{\partial x} + g(x,t) - d(x,t) \tag{4.1}$$

Presented in Table 4.1 are advection and advection–dispersion solutions to Eq. 4.1 for a point release with first-order degradation during transport [$d(x,t) = kC(x,t)$] for each of the four temporal approximations given in Table 3.3. Although this collection represents only a very small subset of the myriad of possible advection and advection–dispersion approximations [a comprehensive collection can be found in van Genuchten and Alves (1982)], they illustrate physical and mathematical aspects of environmental transport that are representative of a much broader range of problems. Physically, they provide insight into the spatial and temporal dependence of contaminant concentration in response to releases. Mathematically, they contain functions and arguments that appear frequently in environmental transport models. Further, the Laplace transform technique used to obtain the solutions in Table 4.1 from Eq. 4.1 is the one used by van Genuchten and Alves (1982) to assemble their collection of solutions.

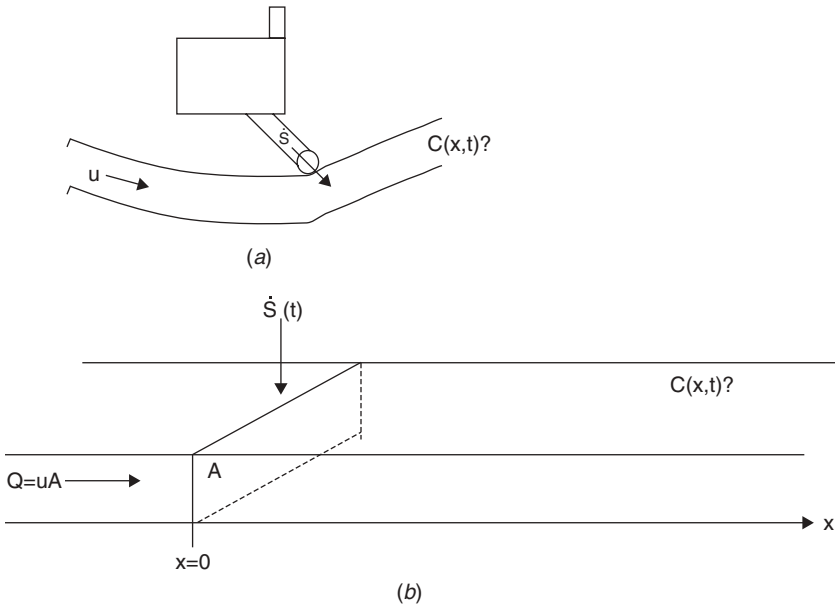

Figure 4.2 One-dimensional environmental transport approximation.

TABLE 4.1 Solutions to the One-Dimensional Transport Equation

Emission Rate	Analytical Expression	Equation Number
	Advection Solutions	
Instantaneous, $S_0\delta(t)$	$C(x,t)=\frac{S_0}{Q}\delta\left(t-\frac{x}{u}\right)\exp\left(-k\frac{x}{u}\right)$	(4.2)
Semi-infinite, $\dot{S}_0 h(t)$	$C(x,t)=\frac{\dot{S}_0}{Q}h\left(t-\frac{x}{u}\right)\exp\left(-k\frac{x}{u}\right)$	(4.3)
Finite step, $\dot{S}_0[h(t\ -h(t-T)]$	$C(x,t)=\frac{\dot{S}_0}{Q}\left[h\left(t-\frac{x}{u}\right)-h\left(t-T-\frac{x}{u}\right)\right]\exp\left(-k\frac{x}{u}\right)$	(4.4)
Constant, $\dot{S}_0$	$C(x)=\frac{\dot{S}_0}{Q}\exp\left(-k\frac{x}{u}\right)$	(4.5)
	Advection–Dispersion Solutions	
Instantaneous, $S_0\delta(t)$	$C(x,t)=\frac{S_0}{A\sqrt{4\pi Dt}}\exp\left[\frac{-(x-ut)^2}{4Dt}\right]\exp(-kt)$	(4.6)
Semi-infinite, $S_0h(t)$	$C(x,t)=\frac{\dot{S}_0}{2Q}\left[\operatorname{erfc}\left(\frac{x-ut}{\sqrt{4Dt}}\right)+\exp\left(\frac{ux}{D}\right)\operatorname{erfc}\left(\frac{x+ut}{\sqrt{4Dt}}\right)\right]\exp\left(-k\frac{x}{u}\right)$	(4.7)
Finite step, $\dot{S}_0[h(t\ -h(t-T)]$	$C(x,t)=\frac{\dot{S}_0}{2Q}[g(t)-h(t-T)g(t-T)]\exp\left(-k\frac{x}{u}\right)$ $g(t')=\left[\operatorname{erfc}\left(\frac{x-ut'}{\sqrt{4Dt'}}\right)+\exp\left(\frac{ux}{D}\right)\operatorname{erfc}\left(\frac{x+ut'}{\sqrt{4Dt'}}\right)\right]$	(4.8)
Constant, $\dot{S}_0$	$C(x)=\frac{\dot{S}_0}{Q}\exp\left(-k\frac{x}{u}\right)$	(4.9)

This book is intended for a broad audience, some of whom would benefit from the details of solving Eq. 4.1 to obtain analytical solutions, and some of whom would not. For this reason, only the analytical solutions and relevant discussion of the spatial and temporal behavior of contaminant concentration are included herein. Solutions of the differential equation (4.1) are reserved for the sidebars and the problems at the end of the chapter.

4.2.1 One-Dimensional Advection

The simplest one-dimensional problems are those for which dispersion can be neglected and advection is the only conservative transport process of importance. The first problem to be considered is a semi-infinite emission rate with first-order contaminant degradation between the source and the receptor. The solution (Eq. 4.3 in Table 4.1) is

$$C(x,t) = \frac{\dot{S}_0}{Q} h\left(t - \frac{x}{u}\right) \exp\left(-k\frac{x}{u}\right) \tag{4.10}$$

Graphical depictions of one-dimensional time-dependent solutions such as Eq. 4.10 are in two forms, concentration vs. distance (at a given time) and concentration vs. time (at a given location). A **concentration vs. distance plot** is a snapshot of the spatial extent of concentration at a fixed time. A **concentration vs. time plot** shows the temporal behavior of contaminant concentration at a fixed location (i.e., the response at a fixed sampling location). Equation 4.10 is plotted in Figure 4.3. In Figure 4.3*a*, C vs. x is shown at times t_1 and t_2, where $t_2 > t_1$. Mathematically, the concentration is $(\dot{S}_0/Q)\exp[-k(x/u)]$ for $t - x/u \geq 0$ or, equivalently, for $x \leq ut$; and it is zero otherwise. Thus, at time t_1, the leading edge of the contaminant front has reached a distance $x = ut_1$, where the concentration drops to zero. In the absence of degradation ($k = 0$), the concentration remains constant at $\dot{S}_0/Q$ between the release at $x = 0$ and the leading edge of the front at ut_1. At the greater time t_2, the contaminant front has traveled a greater distance, ut_2. This makes sense physically as the C vs. x "snapshot" captures the progress of the contamination as it moves downstream. If there is degradation between the source and the receptor (the curves for $k > 0$ in Figure 4.3), the progress of the contaminant front is the same, but the concentration decreases with increasing distance according to $\exp[-(k/u)\mathrm{x}]$.

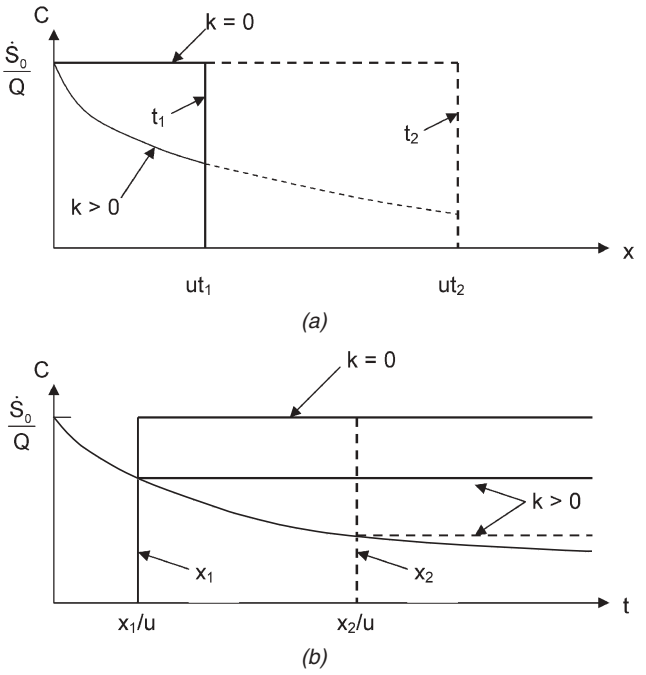

Figure 4.3 Concentration profiles for one-dimensional advection of a point, semi-infinite step release without degradation ($k = 0$) and with degradation ($k > 0$): (*a*) concentration vs. distance at t_1 and t_2, where $t_1 < t_2$; (*b*) concentration vs. time at x_1 and x_2, where $x_1 < x_2$.

The C vs. t plots are presented in Figure 4.3*b* at two locations, x_1 and x_2, where $x_2 > x_1$. Mathematically, the concentration is $(\dot{S}_0/Q)\exp[-k(x/u)]$ for $t - x/u \geq 0$ or, equivalently, $t \geq x/u$; and it is zero for $t < x/u$. At location x_1 the contaminant first appears at time x_1/u, which is the travel time from the source to location x_1. In the absence of degradation, the concentration then remains constant at $\dot{S}_0/Q$. At location x_2 the concentration is the same, but it arrives later, at time x_2/u. If degradation is occurring while the contaminant is in transit, the concentration is lowered by a factor of $\exp[-k(x_1/u)]$ at x_1 and $\exp[-k(x_2/u)]$ at x_2. The time variable can also be viewed as the contaminant travel time, and the exponential line in Figure 4.3*b* shows the effect of degradation as a function of contaminant travel time.

■ Laplace Solution of One-Dimensional Advection of a Semi-Infinite Release[1]

Laplace transforms are a useful technique for solving initial value problems of this type. The technique is illustrated in Appendix A for a time-dependent problem. The technique can be extended to contaminant environmental transport problems in time and one spatial dimension through the following procedure:

1. Simplify the contaminant transport equation (Eq. 4.1) for the problem being addressed and specify the starting values.
2. Take the Laplace transform of both sides of the equation with respect to both x and t.
3. Solve for the transformed concentration, $\overline{\overline{C}}(p,s)$.
4. Take the inverse transform of $\overline{\overline{C}}(p,s)$ to obtain $C(x,t)$. The inverse transforms may be taken in either order.
5. Reconcile the mathematical solution with physical insight.

This procedure is now applied to one-dimensional advection for a semi-infinite emission rate.

Step 1. There are two ways to account for the release. One is to incorporate it into an initial condition (see Problem 4.6). The other, which is done here, is to include it in the generation term,

$$g(x,t) = \frac{\dot{S}_0 \delta(x) h(t)}{A}$$

Equation 4.1 thus becomes

$$\frac{\partial C(x,t)}{\partial t} = -u\frac{\partial C(x,t)}{\partial x} + \frac{\dot{S}_0 \delta(x) h(t)}{A} - kC(x,t)$$

with the starting values $C(x,0) = 0$ and $C(0,t) = 0$.

[1] For some readers, the mechanics of obtaining solutions to the contaminant transport equation are not important. Those readers may wish to skip the portions of the text and the problems devoted to the solution technique and focus their attention on the solutions and the corresponding physical interpretations.

Step 2. Taking the Laplace transform with respect to time and moving all quantities that do not depend on time outside the Laplace operator yields

$$\mathcal{L}_t\left[\frac{\partial C(x,t)}{\partial t}\right] = -u\mathcal{L}_t\left[\frac{\partial C(x,t)}{\partial x}\right] + \frac{\dot{S}_0\delta(x)}{A}\mathcal{L}_t[h(t)] - k\mathcal{L}_t C(x,t)$$

which becomes

$$s\bar{C}(x,s) - C(x,0) = -u\frac{d\bar{C}(x,s)}{dx} + \frac{\dot{S}_0\delta(x)}{As} - k\bar{C}(x,s)$$

Applying the starting value $C(x,0) = 0$ and rearranging yields a differential equation in x:

$$\frac{d\bar{C}(x,s)}{dx} + \frac{s+k}{u}\bar{C}(x,s) = \frac{\dot{S}_0\delta(x)}{uAs}$$

Since there is a bounding condition on C at $x = 0$, the Laplace transform technique can be used for the x dimension just as it was for the t dimension. Taking the Laplace transform with respect to x and moving all quantities independent of x outside the Laplace operator gives us

$$\mathcal{L}_x\frac{d\bar{C}(x,s)}{dx} + \frac{s+k}{u}\mathcal{L}_x\bar{C}(x,s) = \frac{\dot{S}_0}{uAs}\mathcal{L}_x[\delta(x)]$$

Noting that $\mathcal{L}_x[\delta(x-a)] = \exp(-ap) \Rightarrow \mathcal{L}_x[\delta(x-0) = \exp(0) = 1$, this becomes

$$p\bar{\bar{C}}(p,s) - C(0,s) + \frac{s+k}{u}\bar{\bar{C}}(p,s) = \frac{\dot{S}_0}{uAs}$$

where the double overbars indicate a transform with respect to both t and x.

Step 3. Applying the starting value, $C(0,t) = 0$, and solving for $\bar{\bar{C}}(p,s)$ yields

$$\bar{\bar{C}}(p,s) = \frac{\dot{S}_0}{uAs[p + (s+k)/u]}$$

Step 4. Taking the inverse transform of $\bar{\bar{C}}(p,s)$ with respect to p yields

$$\bar{C}(x,s) = \mathcal{L}_p^{-1}\left[\bar{\bar{C}}(p,s)\right] = \frac{\dot{S}_0}{uAs}\mathcal{L}_p^{-1}\frac{1}{p + (s+k)/u}$$

From tables of Laplace transforms (Appendix A),

$$\mathcal{L}_s^{-1}\left[\frac{1}{s-a}\right] = \exp(at) \Rightarrow \mathcal{L}_p^{-1}\left[\frac{1}{p-a}\right] = \exp(ax)$$

Here,

$$a = -\frac{s+k}{u} \quad \text{and} \quad \mathcal{L}_p^{-1}\left[\frac{1}{p-(s+k)/u}\right] = \exp\left(-\frac{s+k}{u}x\right)$$

yielding

$$\bar{C}(x,s) = \frac{\dot{S}_0}{uAs}\exp\left[-(s+k)\frac{x}{u}\right]$$

Taking the inverse transform of $\bar{C}(x,s)$ with respect to s yields

$$C(x,t) = \mathcal{L}_s^{-1}\left[\bar{C}(x,s)\right] = \mathcal{L}_s^{-1}\left[\frac{\dot{S}_0}{uA}\frac{\exp[-(s+k)x/u]}{s}\right] = \frac{\dot{S}_0}{uA}\exp\left(-k\frac{x}{u}\right)\mathcal{L}_s^{-1}\left[\exp\left(e^{-sx/u}\right)\frac{1}{s}\right]$$

From tables of Laplace transforms (Appendix A), $\mathcal{L}^{-1}[\exp(-as)F(s)] = h(t-a)f(t-a)$. Here, $a = x/u \Rightarrow \mathrm{h}(t-a) = h(t-x/u)$ and $F(s) = 1/s \Rightarrow f(t) = t^0 \Rightarrow f(t-a) = (t-a)^0 = 1$. Thus,

$$C(x,t) = \frac{\dot{S}_0}{uA}h\left(t-\frac{x}{u}\right)\exp\left(-k\frac{x}{u}\right) = \frac{\dot{S}_0}{Q}h\left(t-\frac{x}{u}\right)\exp\left(-k\frac{x}{u}\right)$$

which is identical to Eq. 4.10.

Step 5. See Figure 4.3 and the accompanying explanation in Section 4.2.1.

► Example 4.1

At $t = 0$ a contaminant is released as a semi-infinite step into an environmental stream (either air, surface water, or groundwater) which is to be modeled using the one-dimensional advective approximation. The emission rate is 0.05 g/s, the flow rate of the stream is 25 m^3/s, and the cross-sectional area of the stream is 100 m^2.

(a) How long does it take for the contaminant to reach a receptor located 500 m from the emission?
(b) What is the contaminant concentration at the receptor at $t = 1500$ s, $t = 2000$ s, and $t = 2500$ s?
(c) Repeat part (b) for a degradation rate constant of 0.0005 s^{-1}.
(d) After a long time, what are the concentrations at $x = 0$, $x = 1000$ m, and $x = 2000$ m?

Solution

(a) The travel time due to advection is $t = x/u$. The flow velocity is calculated by

$$u = \frac{Q}{A} = \frac{25\,\mathrm{m^3/s}}{100\,\mathrm{m^2}} = 0.25\,\mathrm{m/s}$$

Thus,

$$t = \frac{500\,\mathrm{m}}{0.25\,\mathrm{m/s}} = 2000\,\mathrm{s}$$

(b) The solution for the semi-infinite step is Eq. 4.3 (also Eq. 4.10),

$$C(x,t) = \frac{\dot{S}_0}{Q} h\left(t - \frac{x}{u}\right) \exp\left(-k\frac{x}{u}\right)$$

For $k = 0$, this becomes

$$C(x,t) = \frac{0.05\,\mathrm{g/s}}{25\,\mathrm{m^3/s}} h\left(t - \frac{x}{u}\right) = 0.002h\left(t - \frac{x}{u}\right)\mathrm{g/m^3} = 2h\left(t - \frac{x}{u}\right)\mathrm{mg/m^3}$$

Substituting for t gives us

$$C(1000\,\mathrm{m},\ 1500\,\mathrm{s}) = 2h(1500\,\mathrm{s} - 2000\,\mathrm{s})\,\mathrm{mg/m^3} = 2h(-500\,\mathrm{s})\,\mathrm{mg/m^3} = 0$$
$$C(1000\,\mathrm{m},\ 2000\,\mathrm{s}) = 2h(2000\,\mathrm{s} - 2000\,\mathrm{s})\,\mathrm{mg/m^3} = 2h(0\,\mathrm{s})\,\mathrm{mg/m^3} = 2\,\mathrm{mg/m^3}$$
$$C(1000\,\mathrm{m},\ 2500\,\mathrm{s}) = 2h(2500\,\mathrm{s} - 2000\,\mathrm{s})\,\mathrm{mg/m^3} = 2h(500\,\mathrm{s})\,\mathrm{mg/m^3} = 2\,\mathrm{mg/m^3}$$

(c) The solution now becomes

$$C(x,t) = \frac{\dot{S}_0}{Q} h\left(t - \frac{x}{u}\right) \exp\left(-k\frac{x}{u}\right) = 2h(t - 2000)\exp\left[\left(-0.0005\,\mathrm{s^{-1}}\right)\left(\frac{500\,\mathrm{m}}{0.25\,\mathrm{m/s}}\right)\right]$$
$$= 0.74(t - 2000)\,\mathrm{mg/m^3}$$

Substituting for time as in part (b) gives us

$$C(1000\,\mathrm{m},\ 1500\,\mathrm{s}) = 0$$
$$C(1000\,\mathrm{m},\ 2000\,\mathrm{s}) = 0.74\,\mathrm{mg/m^3}$$
$$C(1000\,\mathrm{m},\ 2500\,\mathrm{s}) = 0.74\,\mathrm{mg/m^3}$$

(d) For large t, $h(t - x/u) = 1$ and

$$C(x,t) = C(x) = \frac{\dot{S}_0}{Q} \exp\left(-\frac{k}{u}x\right) = 2\exp\left(-2.0 \times 10^{-3} x\right)$$

Substituting for x yields

$$C(0) = 2\exp(0) = 2\,\mathrm{mg/m^3}$$
$$C(1000) = 2\exp[(-2.0 \times 10^{-3})\ (1000)] = 0.27\,\mathrm{mg/m^3}$$
$$C(2000) = 2\exp[(-2.0 \times 10^{-3})\ (2000)] = 0.037\,\mathrm{mg/m^3}$$

4.2.2 One-Dimensional Advection and Dispersion

To understand the combined effects of advection and dispersion, it is instructive to begin with a point, instantaneous release. The solution (Eq. 4.6 in Table 4.1) to the contaminant transport equation for this problem is

$$C(x,t) = \frac{S_0}{A\sqrt{4\pi Dt}} \exp\left(-\frac{(x-ut)^2}{4Dt}\right) \exp\left(-k\frac{x}{u}\right) \tag{4.11}$$

Presented in Figure 4.4*a* is concentration vs. distance at t_1 and t_2, where $t_1 > t_2$ for an instantaneous release. The concentration follows a Gaussian distribution in space. As advection carries the contaminant away from the source at $x = 0$, it spreads due to dispersion. The peak of the distribution travels at the same speed as the fluid, and at any given time, t is located at $x = ut$. The degree of spreading is quantified by the standard deviation of contaminant concentration, which increases with time (and equivalently, with distance) according to

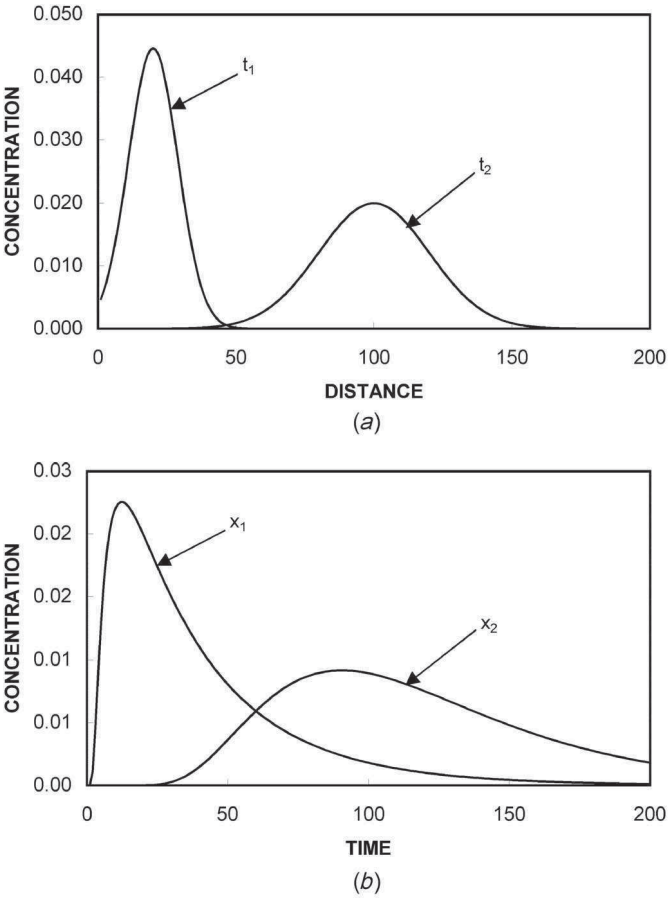

Figure 4.4 Concentration profiles for one-dimensional advection–dispersion of a point, instantaneous release without degradation ($k = 0$): (*a*) concentration vs. distance at t_1 and t_2 where $t_1 < t_2$; (*b*) concentration vs. time at x_1 and x_2 where $x_1 < x_2$.

$$\sigma = \sqrt{2Dt} = \sqrt{2D\frac{x}{u}} \tag{4.12}$$

Approximately 68% of the contaminant is contained within one standard deviation [i.e., locations between $(ut - \sigma)$ and $(ut + \sigma)$], and approximately 95% is contained within two standard deviations.

Concentration vs. time at a given location for the point, instantaneous release is shown in Figure 4.4*b*. Concentration vs. time is asymmetric, characterized by a rapid rise and a more gradual decline. The gradual decline of concentration is referred to as tailing and the portion of the curve to the right of the peak concentration is called the **dispersion tail**. The tail occurs because dispersion and advection are acting in concert before the peak arrives and in opposition after it leaves.

■ Dispersion Tail

In 1972 there was an accident at the Savannah River Site in South Carolina which resulted in the release of 155×10^{12} Bq of tritium to a stream that empties into the Savannah River. Measurements were obtained approximately 15 and 100 miles downriver from the point where the stream enters the river. Presented in Figure 4.5 is concentration vs. time at these locations. From the graph it is possible to see the dispersion tail and the movement of the contaminant due to the combined effect of advection and dispersion.

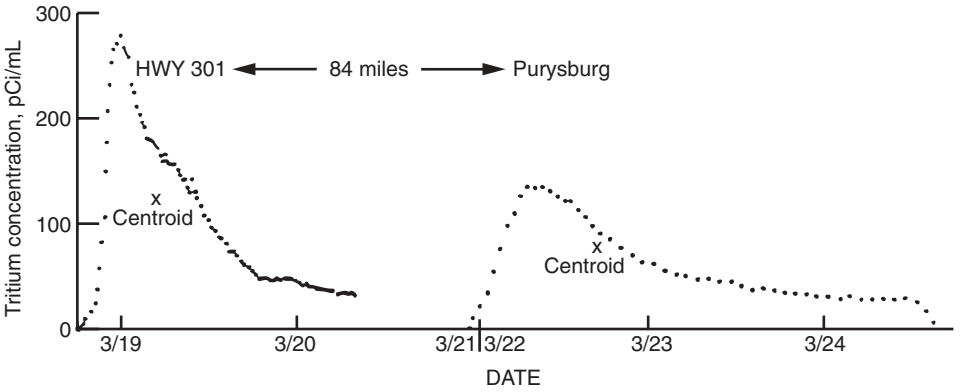

Figure 4.5 Concentration vs. time measurements of tritium in the Savannah River at two downstream locations following an emission from the Savannah River Site. (From Murphy et al. 1993.)

The relative importance of advection compared to dispersion at a given location is quantified by the **Peclet number**, Pe, which is given by $\text{Pe} = ux/D$. High Peclet numbers indicate that advection is dominant, and low Peclet numbers indicate that dispersion is dominant. In Figure 4.4, where neither advection nor dispersion

dominates, the Peclet number is 50. The time at which the peak concentration occurs is obtained by taking the derivative of Eq. 4.11 with respect to time, setting it equal to zero, and solving for t. The result is

$$t^* = \frac{-D + \sqrt{D^2 + u^2 x^2}}{u^2} \tag{4.13}$$

where t^* is the time at which the concentration is a maximum.

■ Laplace Solution for Advection and Dispersion of a Point, Instantaneous Release

Step 1. For a point, instantaneous source of mass S_0,

$$g(x,t) = \frac{S_0 \delta(x) \delta(t)}{A}$$

and Eq. 4.1 becomes

$$\frac{\partial C(x,t)}{\partial t} = D\frac{\partial^2 C(x,t)}{\partial x^2} - u\frac{\partial C(x,t)}{\partial x} + \frac{S_0 \delta(x)\delta(t)}{A} - kC(x,t)$$

with the starting values $C(x,0) = 0$ and $\partial C(x,0)/\partial x = 0$.

Step 2. Taking the Laplace transform with respect to time and moving all quantities that do not depend on time outside the Laplace operator gives

$$\mathcal{L}_t\left[\frac{\partial C(x,t)}{\partial t}\right] = D\mathcal{L}_t\frac{\partial^2 C(x,t)}{\partial x^2} - u\mathcal{L}_t\frac{\partial C(x,t)}{\partial x} + \frac{S_0\delta(x)}{A}\mathcal{L}_t[\delta(t)] - k\mathcal{L}_t C(x,t)$$

which becomes

$$s\bar{C}(x,s) - C(x,0) = D\frac{d^2\bar{C}(x,s)}{dx^2} - u\frac{d\bar{C}(x,s)}{dx} + \frac{S_0\delta(x)}{A} - k\bar{C}(x,s)$$

Applying the starting value, $C(x,0^-) = 0$, and rearranging yields a differential equation in x.

$$\frac{d^2\bar{C}(x,s)}{dx^2} - \frac{u}{D}\frac{d\bar{C}(x,s)}{dx} - \frac{(s+k)}{D}\bar{C}(x,s) = -\frac{\dot{S}_0\delta(x)}{DA}$$

This equation can be solved by the variation of parameters method (Hildebrand 1976). The solution is of the form

$$\bar{C}(x) = I(x) + U_1(x) + U_2(x)$$

where $\bar{C}(x,s)$ has been replaced by $\bar{C}(x)$ for simplicity, where $U_1(x) = A_1 \exp(r_1 x)$, $U_2(x) = A_2 \exp(r_2 x)$, and r_1 and r_2 are the solutions of

$$r^2 - \frac{u}{D}r - \frac{s+k}{D} = 0, \quad \text{or}$$

$$r_1 = \frac{u}{2D} + \frac{1}{2}\sqrt{\frac{u^2}{D^2} + \frac{4(s+k)}{D}}; r_2 = \frac{u}{2D} - \frac{1}{2}\sqrt{\frac{u^2}{D^2} + \frac{4(s+k)}{D}}$$

and where

$$I = \int^x H(\xi)\frac{[U_1(\xi)U_2(x) - U_2(\xi)U_1(x)]}{W[U_1(\xi), U_2(\xi)]}d\xi$$

Substituting

$$H(\xi) = -\frac{S_0\delta(\xi)}{DA} \text{ and } W[U_1(\xi), U_2(\xi)] = U_1(\xi)\frac{\partial U_2(\xi)}{\partial \xi} - U_2(\xi)\frac{\partial U_1(\xi)}{\partial \xi}$$

yields

$$I = \int^x -\frac{S_0\delta(\xi)}{DA}\frac{[\exp(r_1\xi)\exp(r_2x) - \exp(r_2\xi)\exp(r_1x)]}{(r_2 - r_1)\exp(r_1\xi)\exp(r_2\xi)}d\xi$$
$$= \left[\frac{S_0}{DA(r_1 - r_2)}\right][\exp(r_2x) - \exp(r_1x)]$$

Thus

$$\bar{C}(x) = \left[\frac{S_0}{DA(r_1 - r_2)}\right][\exp(r_2x) - \exp(r_1x)] + A_1\exp(r_1x) + A_2\exp(r_2x)$$

At large $x, \bar{C}(x)$ must be bounded. This is achieved by letting

$$A_1 = \frac{S_0}{DA(r_1 - r_2)} \text{ and } A_2 = 0$$

Thus,

$$\bar{C}(x,s) = \frac{S_0}{DA(r_1 - r_2)}\exp(r_2x)$$
$$= \frac{S_0}{DA\sqrt{\frac{u^2}{D^2} + \frac{4(s+k)}{D}}}\exp\left[\left(\frac{u}{2D} - \frac{1}{2D}\sqrt{u^2 + 4D}\right)x\right]$$
$$= \frac{S_0}{2A\sqrt{D}}\exp\left(\frac{ux}{2D}\right)\left[\frac{\exp\left[-\frac{x}{\sqrt{D}}\sqrt{\frac{u^2}{4D} + (s+k)}\right]}{\sqrt{\frac{u^2}{4D} + (s+k)}}\right]$$

Taking the inverse transform of $\bar{C}(x,s)$ with respect to s yields

$$C(x,t) = \mathcal{L}_s^{-1}\left[\bar{C}(x,s)\right] = \mathcal{L}_s^{-1}\left[\frac{S_0}{2A\sqrt{D}}\exp\left(\frac{ux}{2D}\right)\frac{\exp\left[-\frac{x}{\sqrt{D}}\sqrt{\frac{u^2}{4D}+(s+k)}\right]}{\sqrt{\frac{u^2}{4D}+(s+k)}}\right]$$

$$= \frac{S_0}{2A\sqrt{D}}\exp\left(\frac{ux}{2D}\right)\mathcal{L}_s^{-1}\left[\frac{\exp\left[-\frac{x}{\sqrt{D}}\sqrt{\frac{u^2}{4D}+(s+k)}\right]}{\sqrt{\frac{u^2}{4D}+(s+k)}}\right]$$

Rearranging to put into a form consistent with tables of Laplace transforms, we have

$$\bar{C}(x,s) = \frac{S_0}{2A\sqrt{D}}\exp\left(\frac{ux}{2D}\right)\mathcal{L}_s^{-1}\left[\frac{\exp\left[-\frac{x}{\sqrt{D}}\sqrt{s-\left(-\frac{u^2}{4D}-k\right)}\right]}{\sqrt{s-\left(-\frac{u^2}{4D}-k\right)}}\right]$$

From tables of Laplace transforms (Appendix A), $\mathcal{L}^{-1}[F(s-a)] = \exp(at)\mathcal{L}^{-1}[F(s)]$. Thus,

$$C(x,t) = \frac{S_0}{2A\sqrt{D}}\exp\left(\frac{ux}{2D}\right)\exp\left[\left(-\frac{u^2}{4D}-k\right)t\right]\mathcal{L}_s^{-1}\left[\frac{\exp\left[-x\sqrt{s}/\sqrt{D}\right]}{\sqrt{s}}\right]$$

From tables of Laplace transforms (Appendix A),

$$\mathcal{L}_s^{-1}\left[\frac{\exp\left[-b\sqrt{s}\right]}{\sqrt{s}}\right] = \frac{1}{\sqrt{\pi t}}\exp\left(-\frac{b^2}{4t}\right)$$

Here, $b = x/\sqrt{D}$. Thus,

$$C(x,t) = \frac{S_0}{2A\sqrt{D}}\exp\left(\frac{ux}{2D}\right)\exp\left[\left(-\frac{u^2}{4D}-k\right)t\right]\frac{\exp\left(-x^2/4Dt\right)}{\sqrt{\pi t}}$$

Simplifying yields

$$C(x,t) = \frac{S_0}{A\sqrt{4\pi Dt}}\exp\left[\frac{-(x-ut)^2}{4Dt}\right]\exp(-kt)$$

► **Example 4.2**

Five kilograms of contaminant is released to the environmental stream described in Example 4.1. Transport of the contaminant is modeled using the one-dimensional advection–dispersion model. The dispersion coefficient is $10\,m^2/s$, and the other parameters are the same as in Example 4.1.

(a) Neglecting degradation, at what time will the maximum concentration occur at the receptor located 500 m from the release?

(b) What is the standard deviation of plume concentration when the peak reaches the receptor?

(c) What is the maximum concentration at the receptor?

Solution

(a) The time of the maximum concentration is given by Eq. 4.13. Substituting values, we obtain

$$t^* = \frac{-D + \sqrt{D^2 + u^2 x^2}}{u^2} = \frac{-10\,\mathrm{m^2/s} + \sqrt{(10)^2 + \left[(0.25)^2\,\mathrm{m^2/s^2}\right]\left[(500)^2\,\mathrm{m^2}\right]}}{(0.25)^2\,\mathrm{m^2/s^2}}$$
$$= 1846\,\mathrm{s}$$

(b) The standard deviation of plume concentration is given by Eq. 4.12. Substituting values gives us

$$\sigma = \sqrt{2D\frac{x}{u}} = \sqrt{(2)(10\,\mathrm{m^2/s})(500\,\mathrm{m}/0.25\,\mathrm{m/s})}$$
$$= 200\,\mathrm{m}$$

(c) From part (a), the maximum concentration occurs at $t = 1846$ s. Substituting values into Eq. 4.11 yields

$$C(x,t) = \frac{S_0}{A\sqrt{4\pi Dt}}\exp\left[\frac{-(x-ut)^2}{4Dt}\right]\exp(0)$$
$$= \frac{5\,\mathrm{kg}}{100\,\mathrm{m^2}\sqrt{(4\pi)(10\,\mathrm{m^2/s})(1846\,\mathrm{s})}}\exp\left[-\frac{[(500\,\mathrm{m} - 0.25\,\mathrm{m/s})(1846\,\mathrm{s})]^2}{(4)(10\,\mathrm{m^2/s})(1846\,\mathrm{s})}\right]$$
$$= 323\,\mathrm{mg/m^3}$$

Concentration profiles for a semi-infinite step release with no degradation during transport (Eq. 4.7 with $k = 0$) are shown in Figure 4.6*a* (concentration vs. distance at a given time) and Figure 4.6*b* (concentration vs. time at a given distance). The effects of dispersion can be seen by comparing the profile in Figure 4.6*a* to that in Figure 4.3*a*, in which dispersion was neglected. Dispersion causes the leading edge of the profile to be less abrupt, but it does not affect the concentration behind the front.[2] Dispersion effects are more clearly illustrated in Figure 4.6*b*, where the profiles are shown for Peclet numbers of 10, 100, and 1000. Comparing these to the purely advective profiles in Figure 4.3*b*, it is seen that the effects of dispersion become more pronounced as Pe decreases from 1000 to 10.

[2] This is true only for one-dimensional problems. Dispersion in the y and z directions cause the concentration along $x = 0$ to decline behind the front as well.

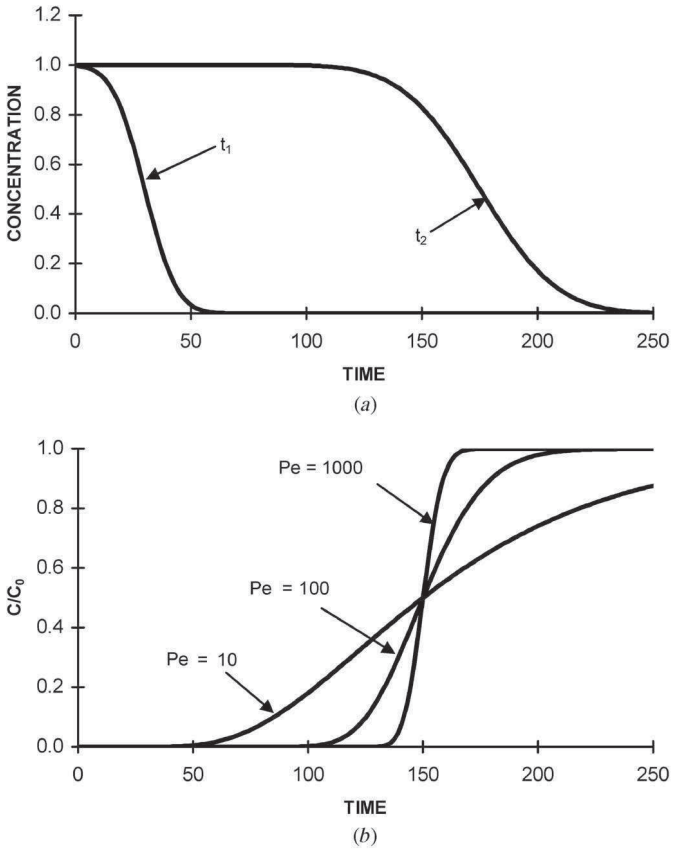

Figure 4.6 Concentration profiles for one-dimensional advection–dispersion of a point, semi-infinite step source without degradation ($k = 0$): (*a*) concentration vs. distance at t_1 and t_2 where $t_1 < t_2$; (b) concentration vs. time at given x for Pe = 10, 100, and 1000.

4.3 THREE-DIMENSIONAL CONTAMINANT TRANSPORT

There are many important environmental transport problems for which one-dimensional approximations are not appropriate. For example, a **two-dimensional approximation** might be necessary for a wide, shallow river or a confined aquifer; and **three-dimensional approximations** are usually needed in the atmosphere, large lakes and oceans, and unconfined aquifers. In the three-dimensional approximation, fluid flow and dispersion are uniform and constant in time and space and the x-axis is oriented to be parallel to the direction of advective flow. Under these simplifications, Eq. 2.22 becomes

$$\frac{\partial C(\mathbf{r},t)}{\partial t} = D_x \frac{\partial^2 C(\mathbf{r},t)}{\partial x^2} + D_y \frac{\partial^2 C(\mathbf{r},t)}{\partial y^2} + D_z \frac{\partial^2 C(\mathbf{r},t)}{\partial z^2} - u \frac{\partial C(\mathbf{r},t)}{\partial x} + g(\mathbf{r},t) - d(\mathbf{r},t) \tag{4.14}$$

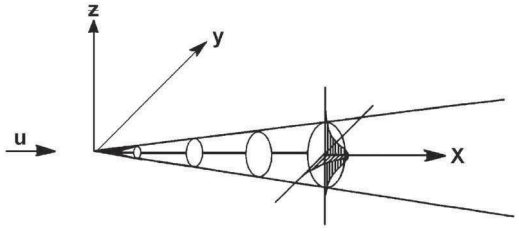

Figure 4.7 Three-dimensional advection–dispersion.

where D_x is **longitudinal dispersion coefficien**t, D_y is **transverse (horizontal) dispersion coefficient** in the y direction, and D_z is **transverse (vertical) dispersion coefficient** in the z direction. The mathematical form of the solution to Eq. 4.14 depends on the geometry of the medium and the spatial and temporal dependence of the release. For a point, instantaneous release of amount S_0 into a medium where boundary effects are negligible, the solution is

$$C(x,y,z,t) = S_0 \frac{\exp\left[-(x-ut)^2/4D_x t\right]}{\sqrt{4\pi D_x t}} \frac{\exp\left(-y^2/4D_y t\right)}{\sqrt{4\pi D_y t}} \frac{\exp\left(-z^2/4D_z t\right)}{\sqrt{4\pi D_z t}} \quad (4.15)$$

Examination of Eq. 4.15 reveals that at any given time, the contaminant concentration profile is Gaussian in the x, y, and z directions, with standard deviations of $\sqrt{2D_x t}$, $\sqrt{2D_y t}$ and $\sqrt{2D_z t}$, respectively. Thus, with increasing time (i.e., as advection carries the contaminant away from the release point), the contaminant distribution becomes broader. This is illustrated in Figure 4.7 for the y and z directions. Spreading in the x direction, which for simplicity is not shown in Figure 4.7, is identical to that found for the one-dimensional solution (Figure 4.4*a*). Each factor in the denominator of Eq. 4.15 has units of length and can be interpreted as a spreading distance. This spreading distance is $\sqrt{2\pi}$ times the standard deviation of contaminant concentration, which is $\sqrt{2Dt}$.

In this section the contaminant transport equation has been applied to problems that are generic in the sense that the transport medium is not specified. In subsequent chapters the equation is applied specifically to atmospheric, surface water, groundwater, and food chain pathways. A common feature of the resulting models is that they each contain one or more empirical transport parameters. A critical part of transport modeling is specifying appropriate values for these empirical parameters.

4.4 ADVANCED SOLUTION METHODS

4.4.1 Numerical Techniques

Often, environmental transport models are too complex to be solved analytically to yield closed-form solutions such as those in Table 4.1. In such cases, numerical techniques can be used to solve Eq. 4.1 for a particular problem. Common techniques for performing such a numerical analysis are **finite-element models** or **finite-**

difference models (Faust and Mercer 1980), in which the transport medium is divided into grids and difference equations based on Eq. 4.1 are written for each node in the grid. The resulting system of coupled simultaneous algebraic equations is solved for concentration at each node. Such models can simulate highly complex systems when properly applied; however, they often require detailed site characterization data to be effective and in some cases require a great deal of computing power.

4.4.2 Superposition Integral[3]

In the sections above, transport is developed from a differential approach (i.e., all of the solutions area obtained by solving a differential equation for contaminant transport). An equivalent alternative is to develop solutions from an integral approach. The **superposition integral** can be developed rigorously from Duhamel's principle (Hildebrand 1976). Alternatively, it can be developed from a physical basis. Depicted in Figure 4.8 is the simultaneous release of the same contaminant at three different locations in a stream. Release S_1 occurs at location x_1, S_2 occurs at x_2, and S_3 occurs at x_3. The problem is to find $C(t)$ for a receptor located at downstream location x_p. Using Eq. 4.11 for a point and instantaneous release (with $k = 0$), the concentration at x_p due to the release at x_1 is

$$\frac{S_1}{A\sqrt{4\pi Dt}}\exp\left\{\frac{-\left[(x_p - x_1) - ut\right]^2}{4Dt}\right\}$$

Similarly, the concentration at x_p due to releases at x_2 and x_3 are

$$\frac{S_2}{A\sqrt{4\pi Dt}}\exp\left\{\frac{-\left[(x_p - x_2) - ut\right]^2}{4Dt}\right\} \quad \text{and} \quad \frac{S_3}{A\sqrt{4\pi Dt}}\exp\left\{\frac{-\left[(x_p - x_3) - ut\right]^2}{4Dt}\right\}$$

respectively. The total concentration at x_p is the superposition of the contributions from the three releases[4]:

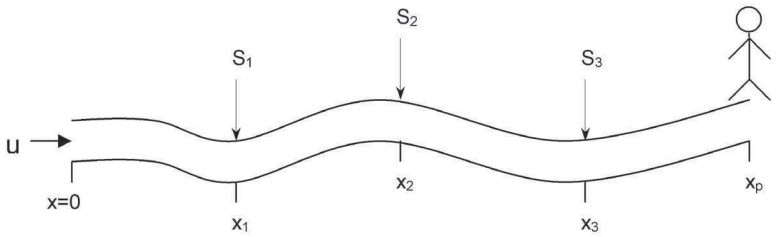

Figure 4.8 One-dimensional environmental transport problem with three simultaneous point, and instantaneous releases.

[3] Integral approaches are common in mathematics, physics, and engineering and are not limited to contaminant transport problems. They are known by various names, including Green's function, point kernel integration, and Duhamel's superposition integral.

[4] There is an implied assumption here that the release from x_1 does not affect the transport of the releases from x_2 or x_3, the release from x_2 does not affect the transport of the releases from x_1 or x_3, and so on. This is a very reasonable assumption because contaminant concentrations are too low, typically in the parts per million range or less, to affect advection and dispersion processes.

$$C(x_p,t) = \frac{S_1}{A\sqrt{4\pi Dt}}\exp\left\{-\frac{[(x_p - x_1) - ut]^2}{4Dt}\right\} + \frac{S_2}{A\sqrt{4\pi Dt}}\exp\left\{-\frac{[(x_p - x_2) - ut]^2}{4Dt}\right\} + \frac{S_3}{A\sqrt{4\pi Dt}}\exp\left\{-\frac{[(x_p - x_3) - ut]^2}{4Dt}\right\} \tag{4.16}$$

Each term in Eq. 4.16 is the product of the mass released, S_i, and the response for an instantaneous release at x_i. This can be generalized for any number, N, of point sources by summing the contributions from each:

$$C(x_p,t) = \sum_{i=1}^{N} \frac{S_i}{A\sqrt{4\pi Dt}}\exp\left\{-\frac{[(x_p - x_i) - ut]^2}{4Dt}\right\} \tag{4.17}$$

Equation 4.17 can be generalized for a release that is continuous in space [i.e., $S(x)$] by changing the summation to an integral:

$$C(x_p,t) = \int_{-\infty}^{\infty} S(x)\frac{\exp\left\{-[(x_p - x_1) - ut]^2/4Dt\right\}}{A\sqrt{4\pi Dt}}dx$$

or equivalently,

$$C(x,t) = \int_{-\infty}^{\infty} S(x')G(x - x', t)dx' \tag{4.18}$$

where $G(x - x')$ is the **spatial point kernel**,

$$C(x - x', t) = \frac{\exp\left\{-[(x - x') - ut]^2/4Dt\right\}}{A\sqrt{4\pi Dt}} \tag{4.19}$$

and $S(x')$ is the mass emitted per unit length. Physically, the spatial point kernel is the concentration at location x due to a point, instantaneous release of one unit mass at location x', and $S(x')dx'$ is the mass released in dx' about location x'.

Similarly, the concentration profile at some time t due to a time-dependent point release at $x = 0$ is

$$C(x,t) = \int_{-\infty}^{t} \dot{S}(t')G(x, t - t')dt' \tag{4.20}$$

where $G(x,t - t')$ is the **temporal point kernel**,

$$C(x, t - t') = \frac{\exp\left\{-[x - u(t - t')]^2/4D(t - t')\right\}}{A\sqrt{4\pi D(t - t')}} \tag{4.21}$$

and $\dot{S}(t')$ is the emission rate. Physically, the temporal point kernel is the concentration at time t due to a point, instantaneous release of one unit mass at time t', and

$S(t')\,dt'$ is the mass released in dt' about time t'. The upper limit of the time integral is t because releases at future times do not contribute to the concentration.

For a general spatial and temporal release,

$$C(x,t) = \int_{t'=-\infty}^{t} \int_{x'=-\infty}^{\infty} \dot{S}(x',t') G(x-x', t-t')\, dx' dt' \tag{4.22}$$

where $G(x - x',t - t')$ is the **temporal/spatial point kernel**

$$G(x-x',t-t') = \frac{\exp\left(-\dfrac{[(x-x')-u(t-t')]^2}{4D(t-t')}\right)}{A\sqrt{4\pi D(t-t')}} \tag{4.23}$$

and $\dot{S}(x',t')$ contains the spatial and temporal dependence of the emission rate. Physically, the point kernel is the concentration at (x,t) due to a point, instantaneous release of one mass unit at (x',t'), and $\dot{S}(x',t')\,dx'\,dt'$ is the total mass released in $dx'\,dt'$ about (x',t'). The technique can be extended to three dimensions by replacing $(x - x')$ in Eqs. 4.22 and 4.23 by $(\mathbf{r} - \mathbf{r}')$ and including factors (from Eq. 4.15) in Eq. 4.23 to account for dispersion in the y and z directions. Also, removal processes that can be approximated as first order can be taken into account by including the factor $\exp[-k(x - x')/u]$ in the point kernel.

The advantage of this approach is that $C(x,t)$ or, more generally, $C(\mathbf{r},t)$, can be determined by inserting the emission rate into Eq. 4.20 or its three-dimensional equivalent, Eq. 4.23. The disadvantage is that the integrals cannot generally be solved analytically. However, with a robust numerical integration tool, it is possible to perform the integrations for any emission rate.

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PROBLEMS

4.1 Consider one-dimensional advection (no dispersion) of a point, instantaneous release.

(a) Based on a physical understanding of advective transport, do the following:

(i) Sketch a plot of $C(x)$ vs. x at t_1 and t_2, where $t_2 > t_1$.

(ii) Sketch a plot of $C(t)$ vs. t at x_1 and x_2, where $x_2 > x_1$.

(b) Use the Laplace transform technique to show that Eq. 4.2 is the solution for this problem. Show that the mathematical solution agrees with the plots from part (a).

4.2 Consider a one-dimensional advection (no dispersion) problem with first-order degradation (rate constant k) and a finite step emission rate $\dot{S}_0$ from $t = 0$ to $t = T$.

(a) Find $C(x,t)$ (Eq. 4.4) by Laplace transforms.

(b) Express $C(x,t)$ in a form such that you can explain the physical significance of each factor or term.

(i) Sketch a plot of $C(x)$ vs. x at t_1 and t_2, where $t_2 > t_1$.

(ii) Sketch a plot of $C(t)$ vs. t at x_1 and x_2, where $x_2 > x_1$.

(iii) Give a brief physical explanation of each plot.

4.3 **(a)** Use the Laplace transform technique to obtain $C(x,t)$ for one-dimensional advection (no dispersion) of an exponentially declining emission rate [i.e., $\dot{S}(t) = \dot{S}_0 e^{-\lambda t} h(t - 0)\,\delta(x)$]. There is no degradation during transport.

(b) Sketch C vs. x at t_1 and t_2 where $t_2 > t_1$ and C vs. t at x_1 and x_2 where $x_2 > x_1$.

4.4 Consider one-dimensional advection (no dispersion) of a linearly decreasing emission rate as shown in Figure 4.9.

(a) Based on a physical understanding of advective transport, do the following:

(i) Sketch a plot of $C(x)$ vs. x at t_1 and t_2, where $t_2 > t_1$.

(ii) Sketch a plot of $C(t)$ vs. t at x_1 and x_2, where $x_2 > x_1$.

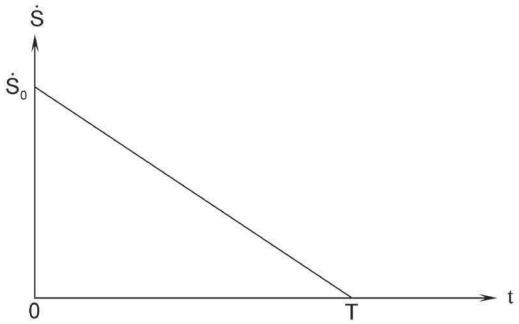

Figure 4.9 Emission rate for Problem 4.4.

(b) Find $C(x,t)$ by Laplace transforms. (*Hint:* You will need to use step functions in your equation for the emission rate.) Show that the mathematical solution agrees with the plots from part (a).

4.5 Consider one-dimensional advection (no dispersion) and first-order degradation of an instantaneous emission S_0 which is distributed uniformly between $x = 0$ and $x = L$.

(a) Find $C(x,t)$ by Laplace transforms.

(b) Express $C(x,t)$ in a form such that you can explain the physical significance of each factor or term.

(i) Sketch a plot of $C(x)$ vs. x at t_1 and t_2, where $t_2 > t_1$.

(ii) Sketch a plot of $C(t)$ vs. t at x_1 and x_2, where $x_2 > x_1$.

(iii) Give a brief physical explanation of each plot.

4.6 One-dimensional advection and first-order degradation of a constant emission rate at $x = 0$ can be formulated in two ways:

$$(1) \quad 0 = -u\frac{dC}{dx} - kC$$

$$C_0 = C(x=0) = \frac{\dot{S}_0}{Q}$$

$$(2) \quad 0 = -u\frac{dC}{dx} - kC + \frac{\dot{S}_0}{A}\delta(x)$$

$$C_0 = C(x=0) = 0$$

Solve formulations (1) and (2) by Laplace transforms and show that they are equivalent.

4.7 Sketch C vs. x at $t^* > t_2$ for one-dimensional advection (no dispersion) of the emission rate in Figure 4.10.

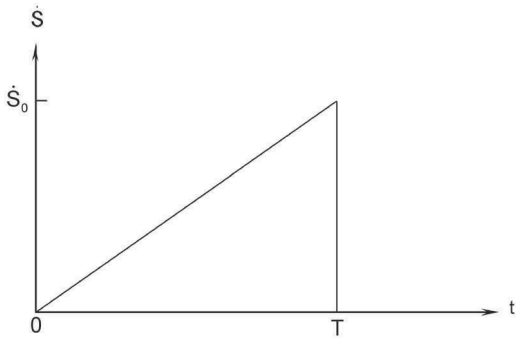

Figure 4.10 Emission rate for Problem 4.7.

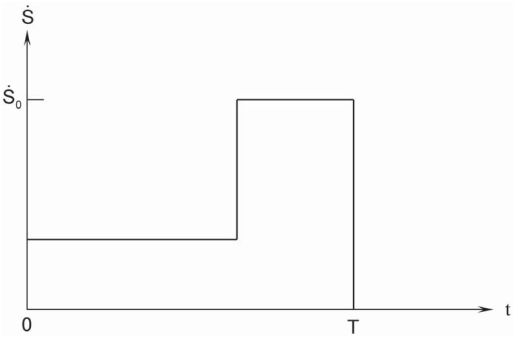

Figure 4.11 Emission rate for Problem 4.8.

4.8 Sketch C vs. x at $t = t_1$ (where $t_1 > T$) for one-dimensional advection (no dispersion) of the emission rate in Figure 4.11.

4.9 Consider the solution of the one-dimensional advection–dispersion problem for a point, semi-infinite step release (Eq. 4.7 in Table 4.1). For no degradation, show the following:

(a) $C(0,t) = \dot{S}_0/Q$.

(b) $\lim_{x\to\infty} C(x,t) = \dot{S}_0/Q$.

(c) Equation 4.7 is often approximated as $C_{\text{approx}}(x,t) \approx (\dot{S}_0/2Q)\text{erfc}[(x - ut)/\sqrt{4Dt}\,]$. This approximation is generally a good one, especially in groundwater applications, except close to the source for small values of t.

(i) Show $\lim_{t\to\infty} C_{\text{approx}}(x,t) = \dot{S}_0/2Q$.

(ii) Show $\lim_{x\to 0} C_{\text{approx}}(x,t) = \dot{S}_0/Q$.

4.10 Develop a tool (Excel Procedure, Fortran, Pascal, C, etc.) for calculating the error function, erf(z), using the equation below (Eq. 7.1.26 from Abramowitz and Stegun 1964). Print a table of z and erf(z) for z varying between −8 and +8 in increments of 0.5 and z varying between 0 and 0.1 in increments of 0.01.

$$\text{erf}\, z = 1 - \left(a_1 t + a_2 t^2 + a_3 t^3 + a_4 t^4 + a_5 t^5\right) e^{-z^2} \qquad t = \frac{1}{1 + pz}$$

$$p = 0.3275911 \qquad a_1 = 0.254829592$$
$$a_2 = -0.284496736 \qquad a_3 = 1.421413741$$
$$a_4 = -1.453152027 \qquad a_5 = 1.061405429$$

Valid only for $z > 0$. Use erf($-z$) = −erf(z) for $z < 0$.

5 Surface Water Transport

5.1 INTRODUCTION

Contaminants in surface water can arise from a variety of sources and give rise to a variety of exposure pathways. Potential sources can include direct discharges from industrial facilities, runoff from contaminated surface soils, seeps from contaminated aquifers, deposition from the atmosphere, and spills and leaks from ships. The principal exposure pathway of interest is usually ingestion of drinking water. However, other potential pathways include incidental ingestion or skin absorption while swimming, inhalation of volatilized contaminants, consumption of food crops irrigated with contaminated surface water, and consumption of fish or shellfish.

Depending on the pathway, the contaminant, and the characteristics of the water body, suspended sediment may affect transport processes. Consequently, it is important to distinguish between contaminants in the aqueous and solid phases. Generally, aqueous-phase contaminants are in dissolved form and concentrations are bounded by solubility limits (Table 5.1). Aqueous-phase contaminants are readily available for assimilation by plants, animals, and humans. Solid-phase contaminants are adsorbed to or otherwise associated with the solid phase and are generally less available; however, particulate matter can sometimes be an important vector for contaminant transport. To account for both phases, it is convenient to subdivide surface water into three physical compartments: water, suspended sediment, and bottom sediment. It is sometimes necessary to couple these compartments with biotic compartments in food chain pathways.

The compartments and processes that may be important in contaminant transport are illustrated in Figure 5.1. Dissolved contaminants can be removed from the water compartment through sorption to suspended sediment and bottom sediment and ingestion or uptake by biota. Contaminants associated with suspended sediment can be ingested by animals and microorganisms, can be taken up by plants, can undergo settling to the bottom, and can dissolve or desorb into the water compartment. Contaminants in biota can be released to the other three compartments by excretion, respiration, and decay of dead organisms. Contaminants associated with bottom sediment are removed to animals by scavenging and to plants by root uptake, to the suspended compartment by resuspension, and to the water compartment by desorption.

This is a simplified conceptualization of the processes that may play a role in surface water transport because it neglects intracompartmental processes. For

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

TABLE 5.1 Water Solubility Limits for Selected Contaminants

Contaminant	Solubility Limit (mg/L)
Aldrin	0.017
Benzene	1,790
Carbon tetrachloride	793
Chlordane	0.056
Chloroform	7,950
Chromium(VI)[a]	1.69×10^6
DDD	0.09
DDE	0.04
DDT	0.0055
Dieldrin	0.25
Ethylene dibromide	3,910
Heptachlor	0.018
Heptachlor epoxide	0.2
Hexachlrorocyclohexane-γ (lindane)	8
Hydrogen sulfide	5,132
Mercury	0.06
Methylene chloride	13,000
Polychlorinated biphenyls	0.277
Tetrachloroethylene (PCE)	206
Trichloroethylene (TCE)	1,280
Vinyl chloride	8,800

Source: RAIS 2005.

[a] Chromium(VI) as particulates or acid mists.

example, aqueous-phase reactions such as hydrolysis, oxidation–reduction, or complexation could yield multiple chemical species of an aqueous-phase contaminant in the water compartment; and the suspended sediment compartment encompasses a spectrum of particle origins, sizes, and morphologies ranging from colloidal precipitates to sand particles. In addition, the water compartment is sometimes divided into two separate components, one for the overlying water and a separate one for pore water in the sediment. For some bodies of water, especially lakes and reservoirs in temperate climates, seasonal thermal stratification of the water column may have a profound effect on transport and degradation processes. None of these refinements is considered here.

Depending on the contaminant and the characteristics of the water body, it may be possible to neglect one or two of the compartments and many, if not all, of the transfer processes. For a nonreacting, nonsorbing highly soluble contaminant in a fast-flowing river or stream, attention may be restricted to the aqueous phase of the contaminant and the water compartment. For a contaminant that is strongly sorbed to particulate matter in a lake, ingestion of bottom feeding fish and benthic organisms may be the most important exposure route; consequently all four compartments and multiple transfer processes might need to be considered.

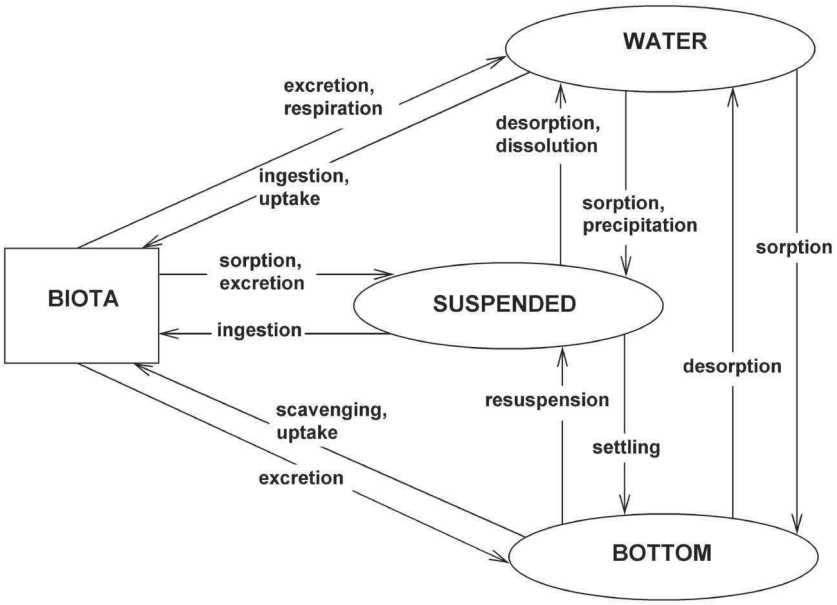

Figure 5.1 Surface water compartments, potential coupling with biotic food chain compartments, and possible mechanisms for contaminant transport among the compartments.

5.2 TYPES OF SURFACE WATER BODIES

Since advection–dispersion processes are dependent on the geometry of the receiving body, it is convenient to divide surface water bodies into the following categories: rivers and streams, reservoirs on a river, lakes, estuaries, and oceans. The general characteristics and relative importance of dissolved-phase transport and solid (sediment)-phase transport can differ greatly for the various systems.

5.2.1 Rivers and Streams

Flow in rivers and streams can usually be approximated by open-channel flow, and the residence time of the aqueous phase is on the order of days or weeks. Although the models describing the spatial and temporal dependence of contaminant concentrations can be complex near the point of discharge, the turbulent flow associated with rivers and streams typically causes the contaminant to become rapidly mixed throughout the depth and breadth of the channel. Dispersion is generally dominated by that in the longitudinal direction, and far-field transport (i.e., far from the point of discharge) can usually be adequately approximated by the one-dimensional contaminant transport equation.

For highly soluble contaminants, the importance of the solid phase depends on the suspended solids concentration and the affinity of the contaminant for suspended particles. The residence time of the solid phase varies because the majority of solid-phase contaminants are associated with small particles. During periods of low flow, these particles settle to the bottom. Depending on the topography of the streambed, sediment may be immobilized in traps or subject to resuspension during

periods of high flow, such as those that occur during the spring. If sediment is resuspended in water with relatively low contaminant concentrations, a fraction of the contaminant may dissolve or desorb back into the water compartment. In addition, seasonal flooding can cause contamination of the surface soils at the banks of the river. This can be important in situations where regular flooding occurs and the riverbanks are under cultivation for food or used for pasturing livestock.

■ Seasonal Flooding

The effect of seasonal flooding is illustrated in Figure 5.2. In late March (Figure 5.2*a*) heavy rains have caused a stream to rise in its banks (upper left part of the photograph) and to flood an adjacent low-lying area (middle of the photograph). By late May (Figure 5.2*b*) the stream has receded and is barely visible.

(*a*)

(*b*)

Figure 5.2 Seasonal flooding of a small stream: (*a*) photograph taken in late March during a period of high flow; (*b*) photograph taken in late May during a period of low flow. (Photo by N.A. Eisenberg.)

5.2.2 Lakes

Small lakes can often be treated as homogeneous compartments. Large lakes have rapid initial dispersion in large-scale turbulence and wind-driven currents. Ultimately, dispersion is limited by the dimensions of the lake. Since flow rates through lakes are small compared to lake volumes, residence times are long, typically years to tens of years. Consequently, virtually all particulate matter is trapped and settles to the lake bottom. Bottom sediments that do not migrate can become an effective sink for sorbed contaminants. However, as illustrated in Figure 5.1, contaminants can be mobilized from bottom sediments through a variety of mechanisms. They can enter the food chain through organisms living in the sediments, they can reenter the water column when sediment becomes resuspended by aquatic animals or the action of water movement, and they can desorb from either bottom sediment or suspended sediment when water chemistry changes or if aqueous-phase concentrations drop due to a reduction in the emission rate. They can also become exposed and available when lake levels drop.

5.2.3 Reservoirs on Rivers

A river–reservoir system, formed by one or more dams, is a hybrid between a river and a lake. Thus, the description above for rivers applies to riverine sections and that for lakes applies to reservoir sections. For modeling purposes reservoirs can sometimes be treated as a series of homogeneous compartments. The dividing line between compartments can be arbitrary or can correspond to a natural geographic feature such as a junction with a major tributary or a human-made feature such as a causeway. The residence time for aqueous-phase contaminants is longer than in rivers, but removal by settling of the solid phase may be significant. Solid-phase contaminants are more likely to be resuspended in a river–reservoir system than in a lake.

5.2.4 Estuaries

In estuaries, advection and dispersion are dominated and enhanced by tidal flows, drag, and volume mixing. Aqueous-phase concentration at a given location can exhibit increases and decreases corresponding to tides. Sediment has net downstream movement in the upper portions of an estuary, but lower portions serve as sediment traps with a large amount of the sediment deposited. The aqueous chemistry is altered by increased salinity. For metals, sorption in brackish water is usually less than that in fresh water. Also, some cations may react with chloride ions to form precipitates that settle to the bottom and lead to high contaminant concentrations in sediments. Because there is no drinking water pathway, dissolved-phase contaminants are generally available only through the food chain.

5.2.5 Oceans

In oceans, coastal turbulence causes rapid dispersion, and advection can be affected by winds and ocean currents. On the continental shelf, residence times for aqueous-

phase contaminants are on the order of hundreds of days. Sediment is a minor concern because sediment loadings offshore are usually low, and sorption of contaminants, especially metals, is generally low in salt water. Contaminants are generally available only through food chain pathways because of the lack of a drinking water pathway.

5.3 SORPTION

When an aqueous-phase contaminant and a solid phase come into contact (either through the addition of the solid phase to a contaminated solution or addition of a contaminant to a solution containing solids), some of the contaminant may become associated with the solid phase due to a variety of mechanisms collectively known as **sorption**. The principal mechanisms responsible for sorption are ion exchange, complexation, hydrolysis, oxidation–reduction, precipitation, and colloid formation. Depending on the contaminant, the sorption mechanism, and the suspended solids concentration, sorption may represent a significant process for removal of a contaminant from the aqueous phase. Sorption can be an important process in contaminant uptake by benthic and aquatic organisms.

5.3.1 Distribution Coefficient

The partitioning of a contaminant between the aqueous and solid phases is illustrated in Figure 5.3, where the aqueous-phase contaminant concentration is denoted by C_l [M(c)/L^3(l)] and the solid-phase contaminant concentration is denoted by C_s [M(c)/M(s)]. In developing mathematical descriptions of the sorption process, it is convenient to consider the addition of an uncontaminated solid phase to a contaminated aqueous phase. The kinetic behavior of C_l and C_s for this situation is the same as that illustrated in Figure 2.7, with the aqueous phase being represented by compartment A and the solid phase being represented

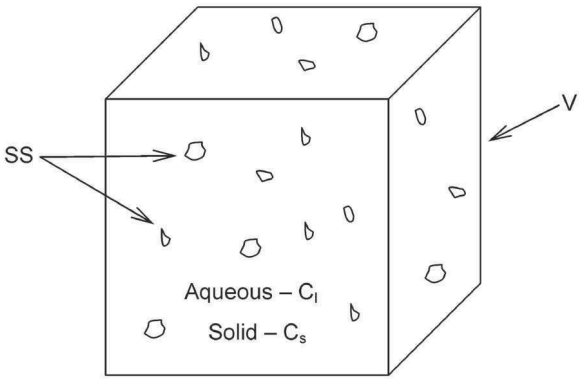

Figure 5.3 Partitioning of a contaminant between the aqueous and solid phases.

by compartment B. At $t = 0$ the contaminant begins to sorb to the solid phase, resulting in an increase in C_s and a decrease in C_l. Eventually, the concentrations reach steady state. When the contaminant becomes bound to the solid phase and consequently does not desorb when the aqueous-phase concentration is reduced, sorption is considered to be **irreversible**. In many environmental settings, however, the sorption process is one of dynamic exchange of contaminant between the solid and aqueous phases and is thus **reversible**, and C_s responds to changes in C_l, and vice versa. For reversible sorption, steady state represents equilibrium between contaminant sorption and desorption. The relationship between the equilibrium concentrations is expressed through a sorption **isotherm**, which is $C_{s,e}$ vs. $C_{l,e}$ at a given temperature. The sorption of contaminants to soils and sediments is usually approximated by one of three isotherms; Langmuir, Freundlich, or linear. The Langmuir isotherm is based on the concept of a finite number of sorption sites. Mathematically, it is given by

$$C_{s,e} = C_{s,\text{sat}} \frac{\alpha C_{l,e}}{1 + \alpha C_{l,e}} \tag{5.1}$$

where $C_{s,\text{sat}}$ is the maximum (i.e., saturated) solid-phase concentration [M(c)/M(s)] and α is a sorption constant [L^3/M(c)]. The Langmuir isotherm finds applications involving relatively high aqueous-phase concentrations, such as in the use of adsorbents to remove contaminants from waste streams. In typical surface waters or groundwaters, where contaminant concentrations are too low to saturate the available sites on the solid phase, the Freundlich and linear isotherms are more commonly used to predict sorption. The Freundlich isotherm is

$$C_{s,e} = K_F C_{l,e}^n \tag{5.2}$$

where K_F is the Freundlich sorption constant [$(L^3)^n/(M(c))^{n-1}$/M(s)] and n is a constant for a given contaminant/solid combination. When $n = 1$, the Freundlich isotherm reduces to the linear isotherm,

$$C_{s,e} = K_D C_{l,e} \tag{5.3}$$

where K_D is the **distribution coefficient** [L^3/M(s)]. The Langmuir isotherm also reduces to the linear isotherm at low aqueous-phase concentrations.

When the time required to reach equilibrium is short compared to the time scale of interest, sorption can be approximated as occurring instantaneously, and the equilibrium concentrations can be replaced by extant concentrations. When combined with linear sorption, this yields

$$C_s = K_D C_l \tag{5.4}$$

Equation 5.4 is a reasonable approximation in many environmental applications because aqueous-phase contaminant concentrations are typically very low [on the

TABLE 5.2 Default Distribution Coefficients for Inorganic Contaminants[a,b]

Contaminant	K_D [L/kg(s)]	Contaminant	K_D [L/kg(s)]
Aflatoxin B1	n/a	Lead	900
Arsenic	29	Mercury	52
Cadmium	75	Plutonium	4,500
Cesium	1,000	Radium	450
Chromium salts	850	Selenium	5
Chromium(VI)	19	Strontium	35
Cobalt	45	Thorium	50,000
Iodine	60	Uranium	450

Source: RAIS 2005.

[a] n/a, not available.

[b] kg(s) refers to mass of solid.

order of mg(c)/L[1] or less]. Where sorption is generally linear, there are usually an abundance of sorption sites, and equilibration times are relatively short compared to transport times in the surface water body.

As noted previously the principal mechanisms responsible for sorption are ion exchange, complexation, hydrolysis, oxidation–reduction, and colloid formation. Consequently, distribution coefficients depend largely on variables that affect these processes, such as the chemical form of the contaminant, the physical and chemical characteristics of the solid phase (mineralogy and specific surface area), and the chemical characteristics of the aqueous phase (principally, pH and ionic strength). Because of the differing mechanisms and variables, distribution coefficients for metals and other inorganic contaminants vary considerably; thus, for a given set of conditions, they cannot be reliably predicted and laboratory measurements must be relied upon. Table 5.2 provides default estimates of distribution coefficients for selected inorganic contaminants.

▶ Example 5.1

The aqueous-phase concentration of mercury in a lake is 10 μg(c)/L. What is the solid-phase concentration of mercury?

Solution The equation relating the aqueous- and solid-phase concentrations is $C_s = K_D C_l$. From Table 5.2, the value of K_D for mercury is 52 L/kg(s). Substitution yields

$$\begin{aligned} C_s &= [52\,\text{L/kg(s)}][10\,\mu\text{g(c)/L}][10^{-3}\,\text{mg(c)}/\mu\text{g(c)}] \\ &= 0.52\,\text{mg(c)/kg(s)} \end{aligned}$$

---

[1] mg(c) refers to mass of contaminant.

TABLE 5.3 Default Distribution Coefficients for Organic Contaminants[a]

Contaminant	K_{oc}	K_D [L/kg(s)]	
		$f_{oc} = 0.001$	$f_{oc} = 0.01$
Aldrin	10,600	11	110
Benzene	166	0.17	1.7
Carbon tetrachloride	49	0.049	0.49
Chlordane	86,700	87	870
Chloroform	35	0.035	0.35
DDD	153,000	150	1,500
DDE	153,000	150	1,500
DDT	220,000	220	2,200
Dieldrin	10,600	11	110
Ethylene dibromide	44	0.044	0.44
Heptachlor	52,400	52	520
Heptachlor epoxide	5,260	5.3	53
Hexachlrorocyclohexane-γ (lindane)	3,380	3.4	34
Methyl mercury	n/a	n/a	n/a
Methylene chloride	24	0.024	0.24
Polychlorinated biphenyls	44,800	45	450
Tetrachloroethylene (PCE)	107	0.11	1.1
Trichloroethylene (TCE)	68	0.068	0.68
Vinyl chloride	24	0.024	0.24

Source: RAIS 2005.

[a] n/a, not available.

Hydrophilic organic contaminants (e.g., certain organic solvents such as alcohols and ethyl acetate) generally do not sorb readily to solids and thus have low distribution coefficients. However, hydrophobic organics such as PCBs, PCHs, dioxins, and many pesticides have high distribution coefficients. Organic contaminants can also sorb to other organic constituents, such as humic acids, that are present in most soils. In the absence of data, the distribution coefficient for sorption of neutral organic compounds to organic carbon in the solid phase can be estimated from the organic carbon–water partition coefficient[2] of the contaminant, K_{oc}, and the fraction of organic carbon in the solid phase, f_{oc}, as follows:

$$K_D = f_{oc} K_{oc} \tag{5.5}$$

Presented in Table 5.3 are default distribution coefficients for selected organic contaminants for soil organic carbon fractions of 0.001 and 0.01.

[2] The organic carbon partition coefficient K_{oc} is the ratio of contaminant concentration in the organic phase [M(c)/M(organic carbon)] to contaminant concentration in water [M(c)/L^3]. It can be obtained from the octanol–water partition coefficient, K_{ow}, which is the ratio of contaminant concentration in octanol to contaminant concentration in water, through empirical relationships (Lyman et al. 1990; Schwarzenbach et al. 1993).

► **Example 5.2**

Estimate K_D for trichloroethylene (TCE) for a soil organic carbon fraction of 2.5%.

Solution From Table 5.3, the K_{oc} for TCE is 68 L/kg(s). From Eq. 5.5, K_D is

$$K_D = f_{oc}K_{oc} = (0.025)[68\,\text{L/kg(s)}] = 1.7\,\text{L/kg(s)}$$

5.3.2 Fraction Sorbed

It is useful to estimate the fraction of contaminant associated with the two phases. The fraction of contaminant associated with the solid phase, or the **fraction sorbed**, f_s, can be approximated with the aid of Figure 5.3, which shows a volume of solution, V, containing a solid phase at a suspended solids concentration SS [M(s)/L^3]. The fraction sorbed is the amount of contaminant associated with the solid phase divided by the amount associated with both the solid and aqueous phases:

$$f_s = \frac{M_s}{M_s + M_l} \tag{5.6}$$

where M_s and M_l are the contaminant masses associated with the solid and aqueous phases, respectively. These masses are related to the solid- and aqueous-phase concentrations by[3]

$$M_s = C_s \cdot \text{SS} \cdot V \tag{5.7}$$

$$M_l \approx C_l V \tag{5.8}$$

The solid-phase concentration in Eq. 5.7 can be expressed in terms of the aqueous-phase concentration through the distribution coefficient (i.e., $C_s = K_D C_l$), yielding

$$M_s = K_D C_l \cdot \text{SS} \cdot V \tag{5.9}$$

Substituting Eqs. 5.9 and 5.8 into Eq. 5.6 yields

[3] Equation 5.8 is an approximation that is valid when the volume occupied by the suspended particles is small compared to the total volume (i.e., the approximation becomes less accurate as suspended solids concentration increases). The exact expression is

$$f_s = \frac{K_D \cdot \text{SS}}{K_D \cdot \text{SS} + (1 - \text{SS}/\rho_P)}$$

where ρ_P is the particle density. At a suspended solids concentration of 10,000 mg/L, the approximation is within 1% of an exact calculation.

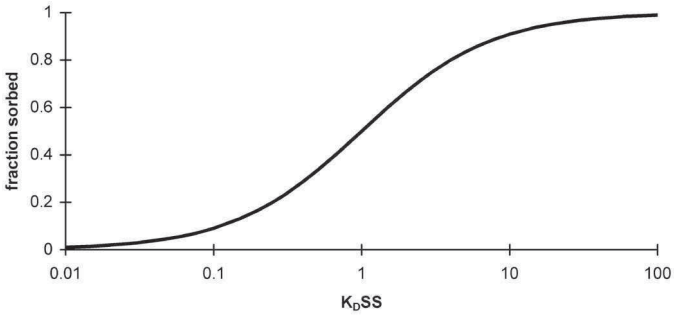

Figure 5.4 Fraction sorbed as a function of the product of the distribution coefficient and the suspended sediment concentration.

$$f_s = \frac{K_D \cdot \mathrm{SS}}{1 + K_D \cdot \mathrm{SS}} \tag{5.10}$$

The corresponding aqueous-phase fraction is $1 - f_s$.

Figure 5.4 is a plot of the fraction sorbed as a function of the $K_D \cdot \mathrm{SS}$ product. This plot can be used to determine the degree to which sorption affects the aqueous-phase concentration. For $K_D \cdot \mathrm{SS} < 0.1$, the fraction sorbed is less than 10%. This means that sorption has a small impact on aqueous-phase concentration, reducing it by less than 10%. In clear water, suspended solids concentrations are typically less than 10^{-5} kg(s)/L [10 mg(s)/L] and sorption is therefore significant only for $K_D > 10^4$ L/kg(s). Although suspended solids concentrations can reach 10^{-2} kg(s)/L [10,000 mg(s)/L] in streams subject to heavy erosion, they rarely exceed 10^{-3} kg(s)/L [1000 mg(s)/L], in which case sorption would be significant for $K_D > 10^2$ L/kg(s).

▶ Example 5.3

Consider a contaminant with $K_D = 5 \times 10^3$ L/kg(s) in surface water in which the suspended solids concentration is 50 mg(s)/L. What fractions of the contaminant are in the solid and aqueous phases?

Solution Partitioning between the solid and aqueous phases depends on $K_D \cdot \mathrm{SS}$:

$$\begin{aligned} K_D \cdot \mathrm{SS} &= [5 \times 10^3 \,\mathrm{L/kg(s)}][50 \,\mathrm{mg(s)/L}][10^{-6} \,\mathrm{kg(s)/mg(s)}] \\ &= 0.25 \end{aligned}$$

Substituting into Eq. 5.10 for the sorbed fraction, we have

$$\begin{aligned} f_s &= \frac{K_D \cdot \mathrm{SS}}{1 + K_D \cdot \mathrm{SS}} = \frac{0.25}{1 + 0.25} \\ &= 0.2 \end{aligned}$$

The aqueous-phase fraction is $1 - f_s = 1 - 0.2 = 0.8$.

5.3.3 Inclusion of Sorption in Transport Models

For situations where either sorption has a significant impact on aqueous-phase concentrations or the solid phase is important in a food chain transport pathway, it is necessary to include sorption in the transport model. A very simplistic way to approximate sorption is first to develop a model for total concentration in the absence of sorption, where total concentration, C_T, refers to the total mass of contaminant (aqueous phase plus solid phase) per unit volume. Then, the aqueous- and solid-phase concentrations can be estimated using the fraction sorbed:

$$C_l = (1 - f_s) C_T \tag{5.11}$$

$$C_s = \frac{f_s C_T}{\mathrm{SS}} \tag{5.12}$$

If greater accuracy is needed, the loss of a sorbed contaminant by settling can be included in the contaminant transport equation as a first-order removal process. Presented in Figure 5.5 is a conceptual model for removal of a sorbed contaminant by settling. The rate of mass lost by settling per unit volume of water, V, is

$$d_s = \frac{\text{flux to bottom} \times \text{area}}{\text{volume}} = \frac{j_s A}{V} \tag{5.13}$$

Substituting for the settling flux, $j_s = v_s C_s \cdot \mathrm{SS} = v_s f_s C_T$ and noting that $Z = V/A$, the loss rate density becomes

$$d_s = \frac{v_s f_s}{Z} C_T \tag{5.14}$$

Equation 5.14 can be written as

$$d_s = \alpha_s C_T \tag{5.15}$$

where $\alpha_s = v_s f_s / Z$ is a first-order rate constant for removal by settling.

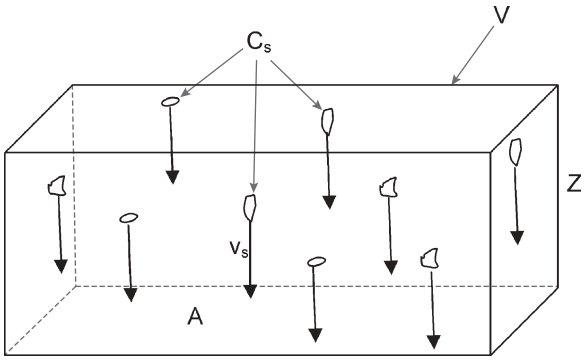

Figure 5.5 Contaminant removal from the water column due to settling of suspended sediment containing sorbed contaminant.

5.4 TRANSPORT MODELING

Developing transport models for all five surface water systems is beyond the scope of this book. For more detailed discussion of modeling the fate and transport of toxic contaminants in surface waters, the reader is referred to other books (Fischer et al. 1979; Logan 1999; Hemond and Fechner-Levy 2000). The discussion here is confined to simple approximations for lakes and rivers. For well-mixed lakes, ponds, and reservoirs, the approximation is a homogeneous compartment model that includes sorption of contaminants to suspended sediments and settling of the sediments. For rivers and streams, the approximation is a one-dimensional open-channel model that includes advection, dispersion, and first-order degradation.

5.4.1 Lakes

Presented in Figure 5.6 is a conceptual model of a lake which consists of two compartments, one for the water column and one for the bottom sediment. The emission rate into the lake from an external source such as the atmosphere or a discharge is $\dot{S}_0$, the area of the lake is A, and the volume of the lake is V. The flow rate of water through the lake is Q. Water flows into the lake with a contaminant concentration of $C_{T,\text{in}}$. Because the lake is well mixed, the contaminant concentration in the water flowing out is the same as that in the lake, which is C_T.

Processes included in the model are contaminant partitioning between the aqueous and solid phases and first-order removal processes such as degradation, dilution, and settling. The conceptual model for the water column is similar to the advective compartment depicted in Figure 2.11, except that multiple first-order processes and a nonadvective source ($\dot{S}_0$) are included here. The mathematical model for the conceptual model of Figure 5.6 is a generalized version of Eqs. 2.23 and 2.24. The differential equation can be written as

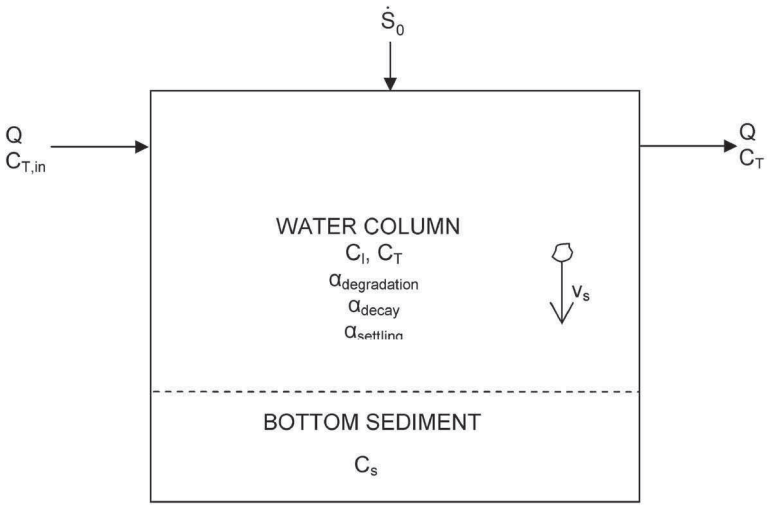

Figure 5.6 Two-compartment model of a lake. Contaminant sorbed to suspended sediment is transported from the water column compartment to the bottom sediment compartment at a mean settling velocity, v_s.

$$\frac{dC_T}{dt} = \frac{\dot{S}_0 + C_{T,\text{in}}Q}{V} - \sum_i \alpha_i C_T \tag{5.16}$$

where C_T is total contaminant concentration (i.e., it includes the contaminant in both the solid and aqueous phases), and α_i represents first-order rate constants for contaminant flow out of the lake ($\alpha_{\text{flow}} = Q/V$) and for destruction processes such as degradation ($\alpha_{\text{degradation}} = k$) (see Appendix B), radiological decay ($\alpha_{\text{decay}} = \ln 2/t_{1/2}$), settling ($\alpha_{\text{settling}} = v_s f_s/Z$), and so on. Equation 5.16 is in the form of the constant-source first-order removal model, and the solution for $C_T(\text{O})$is

$$C_T = \frac{\dot{S}_0 + C_{T,\text{in}}Q}{V\sum_i \alpha_i}\left[1 - \exp\left(-\sum_i \alpha_i t\right)\right] \tag{5.17}$$

The aqueous-phase concentration in the water column is obtained from Eq. 5.11, and the solid-phase concentration in the water column and in the bottom sediment is obtained from Eq. 5.12.

During the summer months, many lakes undergo thermal stratification in which there is a well-mixed upper layer (the epilimnion) and a stable pool of cool water at the bottom (the hypolimnion). The aqueous chemistry of the epilimnion and hypolimnion are distinctly different and from a modeling perspective can be treated as separate homogeneous compartments during the part of the year that the lake remains stratified. However, when the surface water cools in the autumn and whole-lake mixing occurs, the two-compartment model is no longer appropriate. Thus, at some appropriate time it would be necessary to model the lake as a single compartment with initial conditions based on homogeneous mixing of the epilimnion and hypolimnion waters. Lakes in colder climates are subject to stratification again in the winter, resulting in another change in the conceptual model.

▶ Example 5.4

Consider the discharge of DDT into a small, stagnant pond. The pond is 50 m long by 30 m wide and is 1.5 m deep. The suspended solids concentration in the pond is 500 mg(s)/L, the distribution coefficient for sorption of DDT to lake sediments is 2200 L/kg(s), and the settling velocity of the suspended solids is 5×10^{-6} m/s. Determine the first-order rate constant for settling.

Solution The fraction sorbed depends on the product of K_D and SS:

$$\begin{aligned} K_D \cdot \text{SS} &= (500\,\text{mg/L})(2200\,\text{L/kg})[10^{-6}\,\text{kg(s)/mg(s)}] \\ &= 1.1 \end{aligned}$$

and

$$\begin{aligned} f_s &= \frac{K_D \cdot \text{SS}}{1 + K_D \cdot \text{SS}} = \frac{1.1}{1 + 1.1} = \frac{1.1}{2.1} \\ &= 0.524 \end{aligned}$$

The first-order rate constant for settling is

$$k = \frac{v_s f_s}{Z} = \frac{(5 \times 10^{-6}\ \text{m/s})(0.524)}{1.5\,\text{m}}$$
$$= 1.75 \times 10^{-6}\ \text{s}^{-1}$$

▶ **Example 5.5**

Consider a pond of the same dimensions as that in Example 5.4 that is fed by an uncontaminated creek having a volumetric flow rate of $0.02\,\text{m}^3/\text{s}$. A leaking tank adjacent to the pond drips DDT into the pond at a rate of 2 kg/d. In addition to sorption and settling, DDT undergoes photodegradation in the pond with a first-order rate constant of $3 \times 10^{-6}\,\text{s}^{-1}$. Find the steady-state aqueous-phase concentration in the pond.

Solution Letting $C_{T,\text{in}} = 0$ and $t \to \infty$ in Eq. 5.17 yields

$$C_T = \frac{\dot{S}_0}{V \sum \alpha_i} = \frac{\dot{S}_0}{V(\alpha_{\text{dilution}} + \alpha_{\text{degradation}} + \alpha_{\text{settling}})}$$
$$= \frac{[2\,\text{kg(c)/d}]\{(1.16 \times 10^{-5}\,\text{d/s})(10^{-3}\,\text{m}^3/\text{L})[10^6\,\text{mg(c)/kg(c)}]\}}{(50 \times 30 \times 1.5\,\text{m}^3)\left(\frac{0.02\,\text{m}^3/\text{s}}{50 \times 30 \times 1.5\,\text{m}^3} + 3 \times 10^{-6}\,\text{s}^{-1} + 1.75 \times 10^{-6}\,\text{s}^{-1}\right)}$$
$$= 0.754\,\text{mg(c)/L}$$

The aqueous-phase concentration is

$$C_l = (1 - f_s)C_T = (1 - 0.524)[0.754\,\text{mg(c)/L}]$$
$$= 0.359\,\text{mg(c)/L}$$

5.4.2 Rivers and Streams

The discussion here is confined to one-dimensional open-channel approximations which are appropriate for narrow rivers and streams or for wide rivers at locations that are sufficiently far downstream that the concentrations in the cross-flow directions are approximately uniform. The conceptual model is that depicted in Figure 4.2, and the contaminant transport equation is the same as Eq. 4.1.

5.4.2.1 One-Dimensional Advection Advective approximations are appropriate when longitudinal dispersion can be ignored. This occurs when the emission rate varies slowly with time or when the elapsed time is large enough for transients to have traveled past the receptor. Mathematically, this means that $\dot{S}(t) \approx \text{constant} = \dot{S}_0$ and the concentration is given by Eq. 4.5. Generalizing to allow multiple first-order removal processes, Eq. 4.5 takes on the form

$$C(x) = \frac{\dot{S}_0}{Q} e^{-\Sigma \alpha_i (x/u)} \tag{5.18}$$

The factor $\dot{S}_0/Q$ is the concentration at the release point, and the exponential factor represents first-order removal between the release point and the receptor. The first-order rate constants are the same as those described above for the lake model with the exception of α_{flow}. Equation 5.18 is not time dependent; thus, at any given receptor location the concentration does not vary with time.

5.4.2.2 One-Dimensional Advection and Dispersion The one-dimensional advection–dispersion solutions are Eqs. 4.6 to 4.8. To apply these equations, it is necessary to specify a value for the dispersion coefficient. Dispersion in an open channel is due primarily to turbulence and nonuniform velocity distributions. This is illustrated in Figure 5.7. Here, spreading of an instantaneous release across a cross section of the channel at $t = 0$ is illustrated for uniform and nonuniform velocity distributions. If the velocity profile across the channel were uniform, as in Figure 5.7*a*, spreading of the contaminant at $t = t_1$ and $t = t_2$ would be due solely to turbulent diffusion. However, velocities in channels are not uniform; instead, they vary both

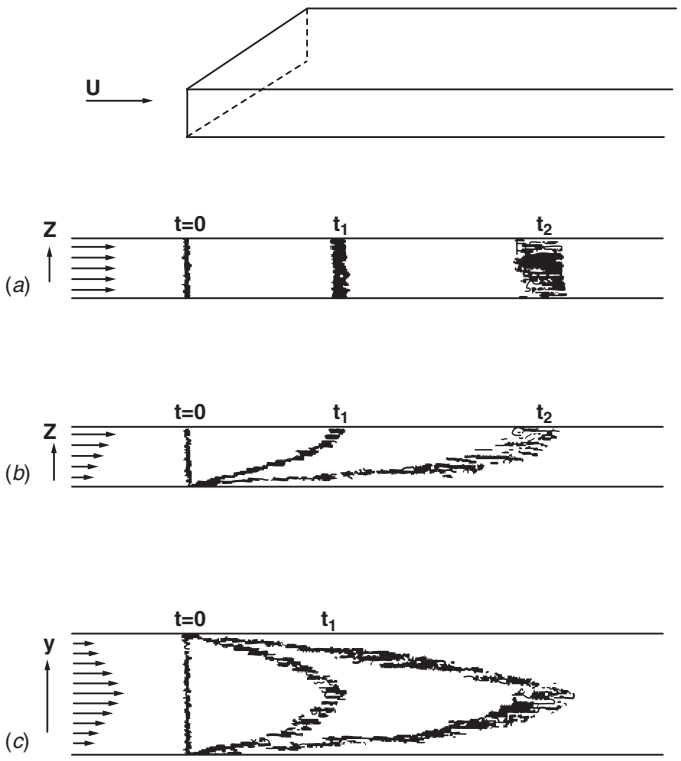

Figure 5.7 Dispersion due to turbulence and nonuniform velocity distributions in an open channel with (*a*) a uniform velocity distribution, (*b*) a nonuniform velocity distribution with depth, and (*c*) a nonuniform velocity distribution across the width. (By permission of Kevin J. Farley, Manhattan College, Riverdale, NY.)

vertically and horizontally. The effect of a nonuniform profile in the vertical direction is illustrated in Figure 5.7*b*. The surface spreading of the contaminant is the same as in Figure 5.7*a*; however, below the surface the water velocity decreases with depth. Thus, contaminant molecules near the bottom of the channel travel only a short distance. Superposed onto this is the effect of a nonuniform velocity profile across the channel, illustrated in Figure 5.7*c*. Turbulent mixing modifies the effect of nonuniform velocity profiles by causing slow-moving parcels of water near the boundaries to mix with the faster-moving ones in the middle of the channel. The combined effect of nonuniform velocity profiles and turbulent mixing is to cause initially sharp concentration profiles to spread and disperse.

Schnoor (1996) provides a summary of measured longitudinal dispersion coefficients for over 30 rivers and streams. Some of those data (from McQuivey and Keefer 1974) are presented in Table 5.4. The dispersion coefficients vary considerably and thus the most reliable values are obtained from measurement. However, this is not always possible, and in the absence of measurements, it is necessary to rely on empirical expressions to estimate the dispersion coefficient. One such expression is provided by Fischer et al. (1979):

$$E_x\left(\mathrm{m}^2/s\right) = 0.011\frac{u^2W^2}{u^*H} \tag{5.19}$$

where u is the mean water velocity, H the channel height (m), W the channel width (m), and

$$u^* = \sqrt{gHs} \tag{5.20}$$

where u^* is the shear velocity (m/s), s the channel slope (m/m), and g the acceleration due to gravity.

▶ Example 5.6

Consider a large, slow-moving river that is 10 m wide and 2 m deep, flowing down a 0.08% slope with a velocity of 0.5 m/s. Determine the longitudinal dispersion coefficient.

Solution The shear velocity is calculated from Eq. 5.20:

$$\begin{aligned} u^* &= \sqrt{gHs} = \sqrt{\left(9.8\,\mathrm{m/s^2}\right)(2\,\mathrm{m})(0.0008)} \\ &= 0.125\,\mathrm{m/s} \end{aligned}$$

The longitudinal dispersion coefficient is calculated from Eq. 5.19,

$$\begin{aligned} E_x &= 0.011\frac{u^2W^2}{u^*H} = (0.011)\frac{(0.5\,\mathrm{m/s})^2\,(10\,\mathrm{m})^2}{(0.125\,\mathrm{m/s})(2\,\mathrm{m})} \\ &= 1.1\,\mathrm{m^2/s} \end{aligned}$$

TABLE 5.4 Measured Dispersion Coefficients

Location	Slope (m/m)	Width (m)	Flow Rate (m^3/s)	Velocity (m/s)	Dispersion Coefficient (m^2/s)
Monacacy River, MD	0.0006	35	2.4	0.11	4.7
		37	5.2	0.21	14
		48	18	0.38	37
Antietam Creek, MD	0.0001	16	2.0	0.20	9.3
		20	4.3	0.27	16
		24	8.8	0.42	26
Missouri River, NE/IA	0.0002	180	380	0.91	460
		200	900	1.24	840
		97	920	1.48	890
Clinch River, TN	0.0006	47	9.1	0.21	14
		53	50	0.44	47
		59	84	0.65	56
Bayou Anacoco, LA	0.0005	20	2.4	0.21	14
		26	8.1	0.34	33
		37	13	0.40	40
Nooksack River, WA	0.0098	64	32	0.68	35
		86	300	1.31	150
Wind and Bighorn Rivers, WY	0.0013	67	57	0.89	42
		69	230	1.56	160
Elkhorn River, NE	0.00073	33	4.2	0.34	9.3
	0.00053	51	9.8	0.41	21
John Day River, OR	0.00355	25	14	1.02	14
	0.00135	34	68	0.82	65
Comite River, LA	0.00078	13	1.0	0.23	7.0
		16	2.4	0.35	14
Amite River, LA	0.00061	37	8.5	0.24	23
		42	14	0.36	30
Sabine River, LA	0.00015	100	120	0.57	320
		130	380	0.65	670
Yadkin River, NC	0.00044	70	70	0.44	110
		72	210	0.76	260
Muddy Creek, NC	0.00083	13	3.9	0.30	14
		20	10.5	0.38	33
Sabine River, TX	0.00018	35	7.3	0.18	40
White River, IN	0.00036	67	13	0.30	30
Chattahoochee River, GA	0.00052	66	29	0.34	33
Susquehanna River, PA	0.00032	200	105	0.33	93

Source: McQuivy and Keefer 1974.

Releases from an accidental discharge such as a spill or a pulse failure of a water purification system can be modeled as an instantaneous release. For the far field, this can be approximated as an instantaneous release uniformly distributed across the river. For no degradition the solution is the same as Eq. 4.6 with D replaced by E_x and with $k = 0$:

$$C(x,t) = \frac{S_0}{WH\sqrt{4\pi E_x t}} \exp\left[-\frac{(x-ut)^2}{4E_x t}\right] \tag{5.21}$$

The shapes of concentration vs. time and concentration vs. distance plots are the same as those in Figure 4.4. At a given location, the time at which the maximum concentration occurs (Eq. 4.13) becomes

$$t^* = \frac{-E_x + \sqrt{E_x^2 + u^2 x^2}}{u^2} \tag{5.22}$$

The maximum concentration can be calculated by inserting t^* into Eq. 5.21. However, in many rivers and streams, advection dominates (i.e., the Peclet number is large) and peak arrival times can be approximated by $t^* \approx x/u$. The maximum concentration can then be estimated by

$$C_{\max} = \frac{S_0}{WH\sqrt{4\pi E_x (x/u)}} \tag{5.23}$$

► **Example 5.7**

Consider the instantaneous release of 10 kg of PAHs into the river in Example 5.6. For a sampling location 1 km downstream from the release, find:

(a) The time at which the concentration reaches a maximum.
(b) The maximum concentration.

Solution

(a) The time at which the concentration reaches a maximum is calculated from Eq. 5.22:

$$t^* = \frac{-E_x + \sqrt{E_x^2 + u^2 x^2}}{u^2} = \frac{-1.1\,\text{m}^2/\text{s} + \sqrt{(1.1\,\text{m}^2/\text{s})^2 + (0.5\,\text{m/s})^2\,(1000\,\text{m})^2}}{(0.5\,\text{m/s})^2}$$
$$= 1996\,\text{s} = 33.26\,\text{min}$$

It is interesting to compare the exact calculation above, which includes the combined effects of advection and dispersion, with an estimate based on advection only:

$$t^* \approx \frac{x}{u} = \frac{1000\,\text{m}}{0.5\,\text{m/s}} = 2000\,\text{s}$$
$$= 33.33\,\text{min}$$

Thus, including dispersion in the calculation does not have a significant affect (~0.2%) on the travel time for this situation.

(b) An exact theoretical calculation can be made by inserting $t = 1996\,\text{s}$ into Eq. 5.21. Alternatively, an approximate calculation can be made using Eq. 5.23:

$$C_{\max} = \frac{S_0}{WH\sqrt{4\pi E_x (x/u)}} = \frac{[10\,\text{kg(c)}]\{[10^6\,\text{mg(c)/kg(c)}](10^{-3}\,\text{m}^3/\text{L})\}}{(10\,\text{m})(2\,\text{m})\sqrt{4\pi(1.1\,\text{m}^2/\text{s})(1000\,\text{m}/0.5\,\text{m/s})}}$$
$$= 3.01\,\text{mg(c)/L}$$

Similar calculations can be carried out for the case of a steady-state release such as that from a continuous discharge. In this case, the steady-state concentration is constant throughout the river and is simply

$$C = \frac{\dot{S}_0}{uWH} = \frac{\dot{S}_0}{Q} \tag{5.24}$$

► **Example 5.8**

EPA's maximum contaminant level (MCL) for PAHs is 0.2 μg/L. How much PAH can be discharged on a continuous basis into the river in Example 5.6 without exceeding this criterion?

Solution The concentration is given by Eq. 5.18 with $\alpha_i = 0$. Rearranging and substituting gives

$$\dot{S}_0 = CQ = [0.2\,\mu\text{g(c)/L}](0.5\,\text{m/s})(10\,\text{m})(2\,\text{m})(10^3\,\text{L/m}^3)$$
$$= 2000\,\mu\text{g/s}$$

or approximately 173 g/d.

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PROBLEMS

5.1 Consider the derailment of a train with a tank car containing 1000 kg of chloroacetylnitrocarbylester-rhodamine (CANCER). The contents leak from the car at a rate of 100 kg/h into an adjacent river which has a cross-sectional area of 500 m^2 and a flow rate of 1000 m^3/s. A municipal water intake is located 10 km downstream from the spill.

(a) Estimate the concentration of the contaminant (in μg/L) at the water intake for conservative transport of the contaminant. (Neglect dispersion.)

(b) If the contaminant undergoes first-order degradation in the river and the rate constant is $5 \times 10^{-4} s^{-1}$, estimate the concentration at the water intake.

5.2 Chromium from a steel mill is discharged continuously to a river that has a flow rate of 13 m^3/s and a cross-sectional area of 30 m^2. The MCL for chromium is 0.1 mg/L. Using a one-dimensional advective approximation, estimate the maximum permissible emission rate of chromium from the steel mill.

5.3 Cesium-137 is released in liquid effluent from a nuclear power plant to a slow-moving river which has a depth of 0.75 m, a width of 5 m, and flow velocity of 0.2 m/s. The total concentration of ^{137}Cs immediately below the release point is 0.5 Bq/L. The suspended sediments have a concentration of 100 mg/L and a settling velocity of 8×10^{-5} m/s. The distribution coefficient for ^{137}Cs with the suspended sediment is 1500 L/kg. Using the one-dimensional advective approximation and neglecting resuspension of settled sediment, what is the ^{137}Cs concentration 10 km downstream from the release?

5.4 A tank rupture at an industrial facility results in the discharge of 400 kg of methylene chloride into a river. The river is 10 m wide and 2 m deep with a 0.02% slope and a flow rate of 10 m^3/s. The intake of a local water supply is located 500 m downstream from the plant.

(a) What is the maximum concentration at the water intake?

(b) At what time following the release will the maximum concentration occur at the water intake?

(c) If methylene chloride degradation in the stream can be approximated as a first-order process with a degradation rate constant of $10^{-3}\,s^{-1}$, what impact will this have on the maximum concentration?

5.5 A nuclear facility releases 210 TBq ($1\,T = 10^{12}$) of tritium (^3H) to a river. The river has a flow rate of $250\,m^3/s$, a depth of 3 m, a width of 150 m, and an elevation change of 30 m over a distance of 100 km.

(a) Based on a point, instantaneous release, calculate and plot the tritium concentration (in Bq/m^3) as a function of time at a highway bridge located 37 km downstream and a water treatment plant located 181 km downstream.

(b) The EPA drinking water standard for tritium, based on an annual average concentration, is $7.4 \times 10^5\,Bq/m^3$. Does the concentration at either the bridge or the water treatment plant exceed this limit? If so, for what period of time?

5.6 A pipe break in a plastics manufacturing plant results in the sudden release of 200 kg of vinyl chloride to a river. The river has a flow rate of $120\,m^3/s$, a velocity of 0.6 m/s, and a slope of 0.00015. A water intake is located 10 km downstream from the plant.

(a) When will the concentration be a maximum at the water intake?

(b) What will be the maximum concentration at the water intake?

5.7 The aqueous-phase concentration of a contaminant in a reservoir is 40 μg/L and the suspended solids concentration is 80 mg/L. The distribution coefficient for sorption of the contaminant to the suspended sediment is 4500 L/kg.

(a) What is the solid-phase contaminant concentration?

(b) What fraction of the contaminant is associated with the solid phase?

5.8 Distribution coefficients are measured in the laboratory by adding the solid phase of interest to an aqueous solution containing the contaminant and allowing the contaminant to equilibrate between the aqueous and solid phases. When it is difficult to measure the solid-phase concentration, the calculation of K_D is based on the aqueous-phase concentration before the addition of the solid phase and the aqueous-phase concentration at equilibrium.

(a) Derive an expression for calculating K_D in terms of initial aqueous-phase concentration C_0, equilibrium aqueous-phase concentration C_e, and suspended solids concentration SS.

(b) Calculate K_D for an experiment in which the initial aqueous-phase concentration is 5 mg/L, the equilibrium aqueous-phase concentration is 1.7 mg/L, and the suspended solids concentration is 5000 mg/L.

5.9 The suspended sediment concentration in the river in Problem 5.6 is 50 mg/L and the organic carbon fraction is 0.03. How much of the vinyl chloride is associated with the solid phase?

5.10 A research laboratory discharges 0.5 kg of benzene per day into a drainage system that discharges directly into a waste pond nearby. The pond is approximately circular, with a radius of 30 m and a depth of 2 m. In the pond, benzene degrades with a 16-day half-life.

(a) Neglecting volatilization, what is the steady-state concentration of benzene in the pond?

(b) If the suspended solids concentration in the pond is 50 mg/L and the distribution coefficient for benzene sorption is 2 L/kg, what is the equilibrium concentration on settling particles?

5.11 Consider the continuous release of chromium to a small lake in the liquid effluent from an electroplating shop. The effluent flow rate is 400 L/yr and the chromium concentration in the effluent is 5 g/L. The lake is 4 m deep, and has a volume of $5 \times 10^5 m^3$, and the flow rate of water through the lake is $5 \times 10^3 m^3$/yr. The suspended solids concentration in the lake is 500 mg/L, the settling velocity of the solids is 0.06 m/yr, and the distribution coefficient for chromium is 400 L/kg.

(a) What fraction of chromium is sorbed to the solid phase?

(b) What is the steady-state concentration of chromium in the aqueous phase?

(c) What is the chromium concentration in the sediment?

5.12 A contaminant from a settling basin located adjacent to a well-mixed reservoir leaches from the basin and enters the reservoir at a rate of 2 kg/yr. The reservoir has a volume of 5×10^8 L and the flow rate through the reservoir is 3×10^9 L/yr. The average suspended sediment concentration is 10 mg/L.

(a) Neglecting sorption, what is the dissolved-phase contaminant concentration (in mg/L) in the reservoir?

(b) The contaminant has a distribution coefficient of 500 L/kg. What is the concentration (in mg/kg) of the contaminant in suspended sediment?

(c) Does the neglect of sorption in part (a) yield a significant error in the dissolved-phase concentration? (Provide a quantitative basis for your answer.)

6 Groundwater Transport

6.1 INTRODUCTION

Groundwater represents an important pathway for human exposure to environmental contaminants. Major sources of groundwater contamination are underground storage tanks, landfills, surface spills, agricultural activity, surface impoundments, and septic tank drain fields. Contaminated groundwater can migrate to wells or outcrop into streams and lakes. Although the principal route of human exposure is ingestion of water, food chain and inhalation pathways are also possible.

The subsurface contaminant transport problem basically consists of two components. One is determining the speed and direction of water flow, and the other is determining how the contaminant migrates given the groundwater velocity field and geochemical conditions. Especially important for contaminant migration is the extent to which sorption to subsurface formations retards contaminant movement with respect to the water.

Earth consists of three successive layers: the dense, metallic inner core, a mantle of dense rocky material, and a lighter outer rock crust. The atmosphere (gases and water vapor) and the hydrosphere (oceans, lakes, and rivers) overlie the crust.[1] For the purposes of environmental risk assessment, groundwater is considered to flow in two parts of the crust: the underlying bedrock and the overlying regolith. **Rock** is defined as (Skinner and Porter 1987) "any naturally formed, firm, and coherent aggregate mass of solid matter that constitutes part of a planet." The **regolith** (literally, blanket rock) is defined (Skinner and Porter 1987) as "the blanket of loose noncemented rock particles that commonly overlie bedrock." The regolith is formed by physicochemical processes that weather rock, such as wind and water erosion. Although the regolith generally overlies the bedrock, outcrops of bedrock are not unusual. **Sediment** is regolith that has been transported from the location of its formation by water, wind, landslide, or other processes acting on Earth's surface. Sediment may be unconsolidated gravel or soils such as sand, clay, silt, or mixtures of these. Rocks are generally classified as sedimentary, igneous, and metamorphic, based on how they were formed.

Groundwater flows either in **pores**, microscopic spaces between solid subsurface material, or **fractures**, macroscopic void spaces in the subsurface. Some subsurface

[1] Some parts of Earth's crust are covered by snow and ice, solid forms of water.

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formations have few, if any, fractures. However, a variety of geologic, geochemical, and biological processes can produce fractures in both rocks and the regolith. These fractures can have a profound effect on groundwater flow and contaminant transport. The importance of fractures for groundwater flow and transport depends on the characteristics of both the fractures and the subsurface formation in which they occur. For example, fractures in a largely impermeable rock, such as granite, are likely to provide a significant path for the movement of groundwater and contaminants dissolved in it. To the contrary, fractures in sandy soil are likely to have a less significant effect on groundwater flow and contaminant transport, so that fractures may be treated by incrementally increasing the permeability of the medium. The identification and characterization of fractures and the modeling of groundwater flow through fractured media can be extremely complex and are beyond the scope of this book.

The regolith and rocks can be either saturated or unsaturated. In saturated media, all of the interstitial spaces are filled with water and the driving force for fluid transport is the hydraulic pressure gradient. In unsaturated media, the interstitial spaces contain both water and air. Water flow occurs as a result of both the hydraulic pressure gradient and capillary action. Airflow occurs primarily as a result of diffusion and the atmospheric pressure gradient.

A generic subsurface formation is depicted in Figure 6.1. The **vadose zone** (also known as the unsaturated zone) extends from the land surface to the water table. The **saturated zone** is the region below the water table. Subsurface media are further characterized according to their ability to transmit water under normal pressure gradients. An **aquifer** is a saturated region that can transmit significant quantities of water, and an **aquitard** is a poorly permeable region that impedes groundwater movement. An aquifer that lies between two aquitards is referred to as a **confined aquifer**, and one whose upper boundary is the water table is referred to as an **unconfined aquifer**.

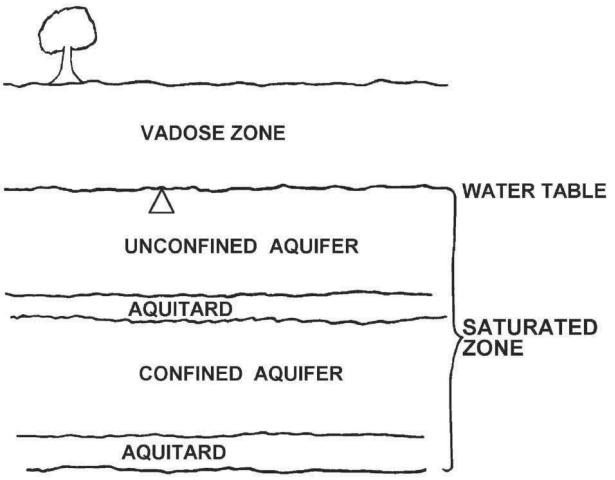

Figure 6.1 Characterization of subsurface formations.

6.2 SUBSURFACE CHARACTERIZATION

For the purposes of flow and contaminant transport modeling, the physical characteristics of subsurface media are quantified by two parameters: porosity and bulk soil density. They are defined for a representative volume, V_T, of the medium as illustrated in Figure 6.2. **Porosity**, n, is the ratio of the interstitial volume to the total volume:

$$n = \frac{V_i}{V_T} \tag{6.1}$$

In some saturated media it is important to distinguish between porosity and **effective porosity**, n_e, which is the ratio of **mobile water volume**, V_{mw}, to total volume:

$$n_e = \frac{V_{\text{mw}}}{V_T} \tag{6.2}$$

The mobile water volume can be less than the interstitial volume due to water that is trapped and immobilized in dead end pores. A related quantity is **moisture content**, which is the ratio of liquid volume to total volume:

$$\theta = \frac{V_l}{V_T} \tag{6.3}$$

In the saturated zone, moisture content is equal to porosity; in the vadose zone, moisture content is less than porosity because of the presence of air.

Bulk soil density is the mass of dry solid material per unit volume,

$$\rho_B = \frac{m_s}{V_T} \tag{6.4}$$

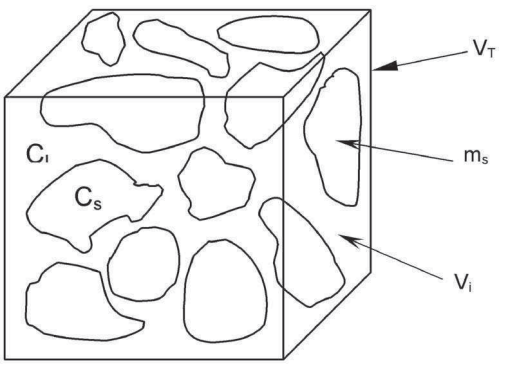

Figure 6.2 Parameters commonly used to characterize the physical properties of subsurface media.

where m_s is the dry solid mass. It can easily be shown that the bulk soil density and the density of the individual particles, ρ_P, in the medium are related by

$$\rho_B = \rho_P(1-n) \tag{6.5}$$

6.3 SATURATED FLOW IN POROUS MEDIA

6.3.1 Groundwater Speed and Direction

As noted in Section 6.1, the first part of the subsurface contaminant transport problem is determination of the speed and direction of water flow. The basic tool for accomplishing this is an empirical expression developed by a French engineer named Henry Darcy in the mid-nineteenth century. In laboratory experiments, he found that the flow rate of water through a porous medium was proportional to the discharge area and the hydraulic head gradient. He expressed these findings through an empirical equation called **Darcy's law**, which in one dimension is

$$\frac{Q}{A_T} = q = -k_H \frac{dH}{dx} \tag{6.6}$$

where Q is the volumetric flow rate [L^3/T], k_H the **hydraulic conductivity** [L/T], H the **hydraulic head** [L], A_T the total cross-sectional area of the medium [L^2], and q the specific discharge [L/T]. Hydraulic head at a given location is a measure of the energy available to drive groundwater flow. Thus, the flow of groundwater is similar to the flow of electricity and the flow of heat in solids, in that the flow rate is proportional to an energy gradient, which is a measurable physical quantity. Whereas electric current is proportional to the voltage gradient and the heat flow in a solid is proportional to the temperature gradient, the flow of groundwater is proportional to the **hydraulic head gradient**. The hydraulic head, representing the total mechanical energy of the groundwater, is the sum of the elevation, pressure, and kinetic energy:

$$H = z + \frac{p}{\rho g} + \frac{v^2}{2g}$$

where z is the elevation above an arbitrary datum, p the gauge pressure of the groundwater, ρ the density of the groundwater, g the acceleration due to gravity, and v the groundwater velocity. For a great many problems of contaminant migration in the subsurface, the kinetic energy term (the last term) is very small because groundwater velocities are small. Similarly, gauge pressure differences are frequently small for this set of problems. Consequently, the hydraulic head gradient is approximately equal to the elevation gradient. That is, for many contaminant transport problems, gravity is the driving force for groundwater flow, and the difference in hydraulic head between two points may be approximated by the difference in elevation.

Darcy's law was developed in the context of water supply, where the objective was to predict flow rate from a subsurface formation. In the context of contaminant transport the need is for an effective advective velocity for use in obtaining solu-

tions to the contaminant transport equation. For surface water problems the advective velocity is a physically measurable quantity. For groundwater problems such an advective velocity cannot be measured, because groundwater flow consists of a microscopic scale of parcels[2] of water molecules flowing around, between, and through the particles of soil or rock. Since water parcels can follow different three-dimensional paths between two points, a distribution of travel times and speeds results. However, an effective speed along the macroscopic path of groundwater travel has been found to be useful for predicting contaminant transport. This effective speed u, known as the **mean linear velocity** or seepage velocity, is defined as

$$u = \frac{1}{n_e}\frac{Q}{A_T} = \frac{q}{n_e} = -\frac{k_H}{n_e}\frac{dH}{dx} \tag{6.7}$$

where all variables are as defined previously. A simplified, heuristic understanding of the physical meaning of the mean linear velocity may be obtained by considering a macroscopic volume V_T of an aquifer as depicted in Figure 6.3. The mean linear velocity u is the mean distance traveled L, divided by the mean residence time of the groundwater in the volume τ:

$$u = \frac{L}{\tau} \tag{6.8a}$$

Consider the imaginary plane surfaces perpendicular to the macroscopic direction of groundwater flow as representative of the mean forward linear motion of the fluid. These imaginary plane surfaces are separated by the average distance L, and the mean residence time is the volume of mobile water between the surfaces divided by the flow rate: $\tau = V_{mw}/Q$. Substituting into Eq. 6.8*a* and noting that $V_{mw} = n_e V_T$ and $V_T = A_T L$, an approximation for the mean linear velocity is obtained:

$$u = \frac{1}{n_e}\frac{Q}{A_T} \tag{6.8b}$$

which is the same as the definition in Eq. 6.7. Note that because $n_e \le 1$, $u \ge q$.

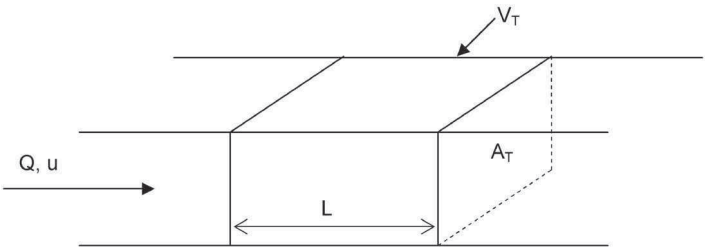

Figure 6.3 Groundwater flow through a macroscopic volume V_T. Mean linear velocity is the residence time in V_T divided by the travel distance L.

[2] For the purposes of this discussion, a parcel of water is a hypothetical assemblage of water molecules that travel together. The assemblage is small compared to the soil particles and the interstitial space between soil particles.

▶ Example 6.1

A waste burial ground at a nuclear facility has been used for the disposal of trash contaminated with TCE, mercury, and ^{137}Cs. Due to the proximity of the burial ground to the site boundary, there is a concern that these contaminants may migrate off-site and threaten residential drinking water wells. The burial ground is located in an area of relatively homogeneous sandy clay soils. A first step in addressing the problem is to estimate the travel time of the groundwater to the site boundary, which is located 500 meters from the burial ground. The hydraulic conductivity of the subsurface soils at the site is 3×10^{-3} cm/s, the porosity is 0.33, and the average hydraulic gradient is –0.01 m/m. Determine the time required for groundwater to travel from the burial ground to the site boundary.

Solution The mean linear velocity of the groundwater is

$$u = -\frac{k_H}{n_e}\frac{dH}{dx} = -\frac{3\times10^{-3}}{0.33}(-0.01) = 9.1\times10^{-5}\ \text{cm/s}$$
$$= 29\ \text{m/yr}$$

The mean groundwater travel time to the site boundary is thus

$$t = \frac{x}{u} = \frac{500\,\text{m}}{29\,\text{m/yr}}$$
$$= 17\ yr$$

The three-dimensional form of Eq. 6.8*b* is

$$\mathbf{v} = -\frac{k_H}{n_e}\nabla H \tag{6.9}$$

Solutions to this equation provide the flow field, $\mathbf{v}(x,y,z)$, for the region of interest. Equation 6.9 is applicable to steady flow. For time-varying flows, additional terms must be added to account for the storage of water by the subsurface medium. A treatment of transient groundwater flow is beyond the scope of this book.

For steady flow, conservation of mass requires that $\nabla \cdot \mathbf{v} = 0$. Thus,

$$\nabla \cdot \left[\frac{k_H}{n_e}\nabla H\right] = 0 \tag{6.10}$$

For an isotropic homogeneous medium, this reduces to Laplace's equation for the head:

$$\nabla^2 H = 0 \tag{6.11}$$

The hydraulic head field, $H(x,y,z)$, for a given region is determined by a combination of (1) field measurements of hydraulic head, (2) pump tests and other field

experiments, (3) laboratory measurements of field samples, and (4) assumptions about boundary conditions. Each of these can be supplemented by calculations using a variety of analytical techniques.

6.3.2 Porosity and Hydraulic Conductivity

The values of parameters such as hydraulic conductivity and effective porosity are strongly dependent on the type of material comprising the subsurface. Presented in Tables 6.1 and 6.2 are results of an analysis of measurements of porosity and hydraulic conductivity for various aquifer materials. Since porosity and hydraulic conductivity both show considerable variability, they usually are determined on a site-specific basis. Porosity can be measured by weighing a sample of the aquifer material fully saturated and after drying. Hydraulic conductivity can be measured in the field through pumping tests or in the laboratory through permeameter tests. In the absence of data from these measurements, an estimate of hydraulic conductivity can be made from the grain size through empirical relationships such as the Kozeny–Carmen equation (Bear 1972; Freeze and Cherry 1979):

$$k_H = \frac{1}{180}\left(\frac{\rho_w g}{\mu_w}\right)\frac{n^3}{(1-n)^2}d_m^2 \tag{6.12}$$

TABLE 6.1 Representative Values of the Effective Porosity of Aquifer Materials

Aquifer Material	*N*	Range	Arithmetic Mean
Sedimentary materials			
Sandstone			
Fine	47	0.02–0.40	0.21
Medium	10	0.12–0.41	0.27
Siltstone	13	0.01–0.33	0.12
Sand			
Fine	287	0.01–0.46	0.33
Medium	297	0.16–0.46	0.32
Coarse	143	0.18–0.43	0.30
Gravel			
Fine	33	0.13–0.40	0.28
Medium	13	0.17–0.44	0.24
Coarse	9	0.13–0.25	0.21
Silt	299	0.01–0.39	0.20
Clay	27	0.01–0.18	0.06
Limestone	32	~0–0.36	0.14
Wind-laid materials			
Loess	5	0.14–0.22	0.18
Eolian sand	14	0.32–0.47	0.38
Tuff	90	0.02–0.47	0.21
Metamorphic rocks			
Schist	11	0.22–0.33	0.26

Source: McWhorter and Sunada 1977.

TABLE 6.2 Representative Values of the Hydraulic Conductivity of Aquifer Materials

Aquifer Material	*N*	Range (cm/s)	Arithmetic Mean (cm/s)
Igneous rocks			
Weathered granite	7	$(3.3–52) \times 10^{-4}$	1.6×10^{-3}
Weathered gabbro	4	$(0.5–3.8) \times 10^{-4}$	1.9×10^{-4}
Basalt	93	$(0.2–4250) \times 10^{-8}$	9.4×10^{-6}
Sedimentary materials			
Sandstone (fine)	20	$(0.5–2270) \times 10^{-6}$	3.3×10^{-4}
Siltstone	8	$(0.1–142) \times 10^{-8}$	1.9×10^{-7}
Sand			
Fine	159	$(0.2–189) \times 10^{-4}$	2.9×10^{-3}
Medium	255	$(0.9–567) \times 10^{-4}$	1.4×10^{-2}
Coarse	158	$(0.3–6610) \times 10^{-4}$	5.2×10^{-2}
Gravel	40	$(0.3–31.2) \times 10^{-1}$	4.0×10^{-1}
Silt	39	$(0.09–7090) \times 10^{-7}$	2.8×10^{-5}
Clay	19	$(0.1–47) \times 10^{-8}$	9×10^{-8}
Metamorphic rocks			
Schist	17	$(0.002–1130) \times 10^{-6}$	1.9×10^{-4}

Source: McWhorter and Sunada 1977.

where d_m is a representative grain size, n the porosity, ρ_w the density of water, μ_w the viscosity of water, and g is acceleration due to gravity. The equation is valid for any consistent set of units. At 20°C, Eq. 6.12 becomes

$$k_H = 543 \frac{n^3}{(1-n)^2} d_m^2 \tag{6.13}$$

where k_H is in cm/s and d_m is in centimeters.

6.3.3 Dispersion

Dispersion in the subsurface is due to the combined effects of molecular diffusion and advective heterogeneities. **Advective heterogeneities** can occur at both the pore level and the aquifer level. At the pore level (Figure 6.4), advective heterogeneity refers to (1) variability in the size of the pore channels resulting in differing peak velocities in the pores, (2) variability in the length of flow paths that water parcels follow, and (3) nonuniform velocity distributions across the pore channels (Fetter 1999). Contaminant spreading due to these variations in water velocities and flow paths is usually referred to in the groundwater literature as **mechanical dispersion** (Bear 1972). At the aquifer level, variability in hydraulic conductivity and porosity due to heterogeneities of the porous medium causes additional variability of groundwater velocity and a corresponding increase in dispersion, although at a larger spatial scale. Many subsurface media are highly heterogeneous, containing regions where water flow is high and regions where it is low. For example, a sand medium may contain clay lenses. Water flow through a clay lens is slower than

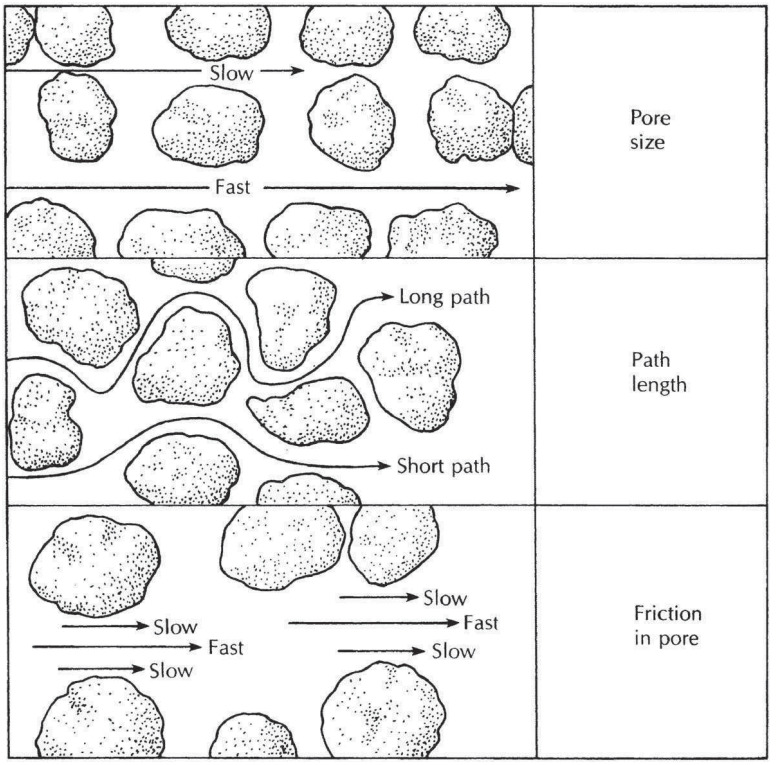

Figure 6.4 Pore-level dispersion processes. (From Fetter 2001; reprinted by permission of Prentice Hall, Inc.)

it is in the surrounding sand, resulting in enhanced spreading of contaminants dissolved in the water.

Mechanical dispersion coefficients are taken to be proportional to the mean linear velocity, and the proportionality constant is the **dispersivity**, α. The effects of molecular diffusion and mechanical dispersion are combined linearly to yield the following expression for hydrodynamic dispersion:

$$D_L = D + \alpha_L u \tag{6.14}$$

where D_L is the longitudinal dispersion coefficient (i.e., the dispersion coefficient parallel to the direction of flow [L^2/T]), D the effective molecular diffusion coefficient for porous media, which is between 0.1 and 1 times the molecular diffusion coefficient in water only, and α_L the longitudinal dispersivity [L].

Estimating dispersivity is difficult, and the most reliable estimates come from field tests performed at the site and at the spatial scale of interest. Field test methods include single-well injection and withdrawal tests, double-well tests, and tracer tests. In the absence of field data, laboratory tests can be conducted on soil samples collected from the site. Dispersivity can be inferred from particle size

analysis using an empirical expression similar to Eq. 6.13 or from column breakthrough curves for a conservative tracer and analytical transport equations such as those in Section 6.5. However, values of dispersivity based on laboratory tests are smaller than those measured in the field. This scale effect arises because the field tests account for large-scale advective heterogeneities that do not appear in laboratory-scale samples. Lacking field or laboratory data, it is often necessary to use generic empirical relationships. Gelhar et al. (1992) have collected and reviewed published estimates of dispersivity from field studies at 59 different sites representing a wide range of aquifer materials and test methods. The results of this analysis are shown in Figure 6.5, where longitudinal dispersivity is shown as a function of distance from the source. Although there is considerable scatter in the data, longitudinal dispersivity increases with the length scale, ranging from approximately 0.01 m at 1 m to 100 m at 100 km. Also, there is no clear distinction between values for porous and fractured media. Xu and Eckstein (1995) subsequently performed a least-squares fit to the data, weighted by reliability of the measurement, and they provide the following relationship between longitudinal dispersivity and distance from the source:

$$\alpha_L = 0.83(\log_{10} x)^{2.414} \tag{6.15}$$

where the distance along the flow direction x and the longitudinal dispersivity are both in meters. Although Eq. 6.15 may be used to estimate dispersivity, the uncertainties inherent in the relationship should be recognized.

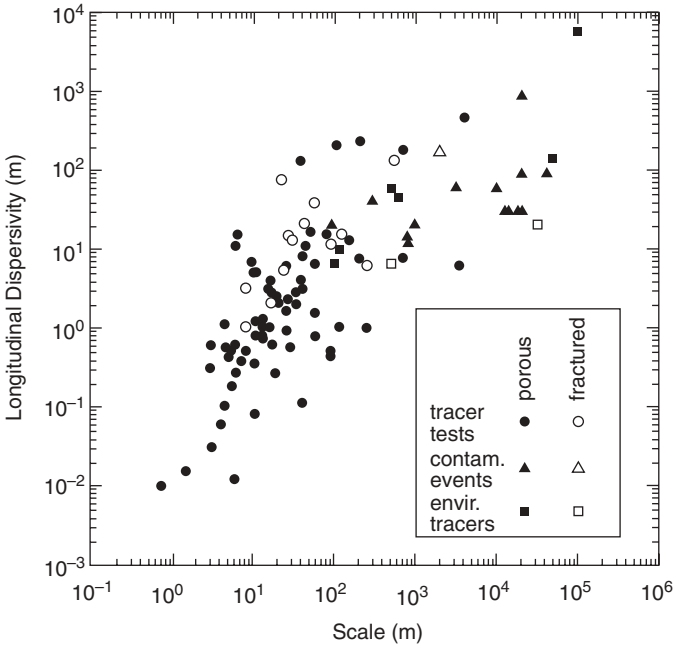

Figure 6.5 Field-scale dispersivity data. (From Gelhar et al. 1992.)

► **Example 6.2**

The molecular diffusion coefficient for TCE is 4.5×10^{-6} cm^2/s. Determine the longitudinal dispersion coefficient (D_L) for the problem in Example 6.1. Which process is most significant: mechanical dispersion or molecular diffusion?

Solution The dispersivity α_L for a length scale of 500 m (corresponding to the distance to the site boundary) is calculated from Eq. 6.15:

$$\begin{aligned}\alpha_L &= 0.83(\log_{10}x)^{2.414} = 0.83(\log_{10}500)^{2.414} \\ &= 9.1\,\text{m}\end{aligned}$$

The longitudinal dispersion coefficient is calculated from Eq. 6.14:

$$\begin{aligned}D_L &= D + \alpha_L u \\ &= (4.5 \times 10^{-6}\,\text{cm}^2/\text{s})\ (10^{-4}\,\text{m}^2/\text{cm}^2 + 9.1\,\text{m})\ (9.1 \times 10^{-5}\,\text{cm/s})\ (10^{-2}\,\text{m/cm}) \\ &= 4.5 \times 10^{-10}\,\text{m}^2/\text{s} + 8.3 \times 10^{-6}\,\text{m}^2/\text{s} \\ &= 8.3 \times 10^{-6}\,\text{m}^2/\text{s}\end{aligned}$$

The mechanical dispersion coefficient is a factor approximately 20,000 greater than the molecular diffusion coefficient and thus dominates.

6.4 SORPTION

Sorption was introduced in Chapter 2 and amplified in Chapter 5 in the context of contaminants partitioning between the aqueous and solid phases of a surface water. Such partitioning can also occur between the aqueous and solid phases in the subsurface environment. The treatment of sorption in surface water and in groundwater has many similarities, but it also has some important differences. One similarity is calculation of the fraction of contaminant in the sorbed and aqueous phases. With the aid of Figure 6.2, an approach analogous to that in Section 5.3.2 can be followed to yield the following for the sorbed fraction in the subsurface:

$$f_s = \frac{(\rho_B / n)K_D}{1 + (\rho_B / n)K_D} \tag{6.16}$$

The relative importance of sorption in surface water and groundwater transport is illustrated in Table 6.3, where sorbed fractions are given for solid/liquid mass ratios ranging from 10^{-6} to 100. In surface waters, mass ratios range from 10^{-6}, which corresponds to a suspended solids concentration of 1 mg(s)/L and is representative of a clear lake, to 10^{-2}, which corresponds to a suspended solids concentration of almost 10,000 mg(s)/L and is representative of runoff from heavily eroding soil. In the subsurface, the mass ratio ranges from a minimum of about 1, which corresponds to a moisture content of 0.71 and represents an upper bound on the porosity of clays, to greater than 100, which corresponds to a moisture content of less than 0.03 and is representative of crystalline rock such as granite. The table includes

TABLE 6.3 Fraction Sorbed as a Function of the Liquid Volume Ratio and Distribution Coefficient[a]

Solid/Liquid Mass Ratio, m_s/m_l [kg(s)/kg(w)]	Natural System Equivalents Volume Fraction Water, V_w/V_T [L(w)/L(T), L(w)/kg(T)]	Suspended Solids Concentration, SS [mg(s)/L(T)]	Representative Natural System	Fraction Sorbed	
				$K_D = 10$ L(w)/kg(s)	$K_D = 0.1$ L(w)/kg(s)
10^{-6}	1	1	Clear lake or stream	10^{-5}	10^{-7}
10^{-4}	1	100	Turbid lake or stream	10^{-3}	10^{-5}
10^{-2}	0.996	9,900	Runoff from heavily eroding field	0.09	0.001
1	0.71	714,000	Peat bog	0.88	0.09
10	0.2	2×10^6	Saturated limestone	0.95	0.50
100	0.024	2.44×10^6	Saturated granite	0.96	0.91

[a] $\rho_B = 2500\,\text{kg/m}^3$.

calculations for distribution coefficients of 10 and 0.1 L/kg(s). For solid/liquid ratios greater than 1, sorption causes a significant fraction of the contaminant to partition to the solid phase. For example, for a saturated zone moisture content of 0.2, the sorbed fraction is 95% for K_D = 10 L/kg(s), and it is 50% for K_D = 0.1 L/kg(s). This is in sharp contrast to surface waters (i.e., suspended solids concentrations of less than 10,000 mg/L), where the sorbed fractions are very low. The implication is that sorption must be considered in groundwater transport modeling unless the distribution coefficient is very low. The physical reason is the extremely large surface area per unit volume of water available for sorption in the subsurface.

6.5 SUBSURFACE CONTAMINANT TRANSPORT MODELING

6.5.1 Equilibrium Model of Subsurface Contaminant Transport

As a consequence of the importance of sorption in groundwater transport, the advection–dispersion equation (Eq. 4.1) is typically modified to explicitly incorporate the effect of sorption on aqueous-phase concentrations. In the **linear equilibrium model** presented here, sorption is considered to occur instantaneously (which, in practical terms, means that the time scale to reach equilibrium is small compared to advection, dispersion, generation, or destruction time scales), and the solid-phase concentration is related linearly to the aqueous-phase concentration as in Eq. 5.4. This yields an equilibrium one-dimensional equation for subsurface contaminant transport:

$$\frac{\partial C_l(x,t)}{\partial t} = \frac{D}{R}\frac{\partial^2 C_l(x,t)}{\partial x^2} - \frac{u}{R}\frac{\partial C_l(x,t)}{\partial x} + \frac{g_T(x,t)}{nR} - kC_l(x,t) \tag{6.17}$$

where $C_l(x,t)$ is the aqueous-phase contaminant concentration, $g_T(x,t)$ the total (aqueous and solid phase) contaminant emission rate per unit volume to the subsurface, and R the **retardation factor**:

$$R = 1 + \frac{\rho_B K_D}{n} \tag{6.18}$$

The retardation factor is a measure of the importance of sorption. If the contaminant does not sorb (i.e., if $K_D = 0$), $R = 1$. As K_D increases, R increases.

Physically, $1/R$ is the fraction of contaminant in the aqueous phase. The degradation term in Eq. 6.17 applies to the situation in which degradation occurs at the same rate in the aqueous and solid phases. For chemical reactions or biological processes that occur only in the aqueous phase, the degradation term is kC_l/R. The subscript T is included with the generation term in Eq. 6.17 to emphasize that it is the total amount of contaminant that is released per unit of subsurface volume. The factor $1/R$ in the generation term accounts for the fraction that partitions into the aqueous phase, and n accounts for the fraction of the total volume occupied by the aqueous phase.

■ Derivation of the Subsurface Contaminant Transport Equation

As indicated earlier, sorption causes a significant fraction of the contaminant to partition to the solid phase. For this reason the partitioning of the contaminant between the aqueous and solid phases must be explicitly included in the governing equation for groundwater transport of contaminants. The simplest model of subsurface transport is the linear equilibrium model. In this model, the sorption process is approximated as instantaneous, linear (i.e., $C_s = K_D C_l$), and reversible.

The contaminant transport equation (Eq. 4.1) applies to the total (i.e., aqueous phase plus solid phase) contaminant concentration. With first-order removal, Eq. 4.1 becomes

$$\frac{\partial C_T(x,t)}{\partial t} = D\frac{\partial^2 C_T(x,t)}{\partial x^2} - u\frac{\partial C_T(x,t)}{\partial x} + g_T(x,t) - kC_T(x,t)$$

The relationship between the total contaminant concentration and the aqueous- and solid-phase concentrations is facilitated through Figure 6.2. The total concentration is

$$C_T = \frac{V_l}{V_T}C_l + \frac{m_s}{V_T}C_s = nC_l + \rho_B C_s$$

Substituting C_T into the contaminant transport equation and dropping (x,t) for simplicity yields

$$\frac{\partial}{\partial t}(nC_l + \rho_B C_s) = D\frac{\partial^2}{\partial x^2}(nC_l + \rho_B C_s) - u\frac{\partial}{\partial x}(nC_l + \rho_B C_s) + g_T - k(nC_l + \rho_B C_s)$$

Since the solid-phase contaminant is not mobile, C_s can be eliminated from the advection and dispersion terms. (Contaminant diffusion into the soil grains is being neglected because the diffusion process is generally slow compared to dispersion in the liquid.) Also, the distinction between mobile and nonmobile water in the soil matrix is not being considered in this formulation.

$$\frac{\partial}{\partial t}(nC_l + \rho_B C_s) = Dn\frac{\partial^2 C_l}{\partial x^2} - un\frac{\partial C_l}{\partial x} + g_T - k(nC_l + \rho_B C_s)$$

Dividing by n and substituting $C_s = K_D C_l$ gives us

$$\frac{\partial}{\partial t}\left(C_l + \frac{\rho_B K_D C_l}{n}\right) = D\frac{\partial^2 C_l}{\partial x^2} - u\frac{\partial C_l}{\partial x} + \frac{g_T}{n} - k\left(C_l + \frac{\rho_B K_D C_l}{n}\right)$$

Rearranging yields

$$\left(1 + \frac{\rho_B K_D}{n}\right)\frac{\partial C_l}{\partial t} = D\frac{\partial^2 C_l}{\partial x^2} - u\frac{\partial C_l}{\partial x} + \frac{g_T}{n} - \left(1 + \frac{\rho_B K_D}{n}\right)kC_l$$

Dividing by $R = 1 + \rho_B K_D/n$ gives

$$\frac{\partial C_l}{\partial t} = \frac{D}{R}\frac{\partial^2 C_l}{\partial x^2} - \frac{u}{R}\frac{\partial C_l}{\partial x} + \frac{g_T}{nR} - kC_l$$

This is the form of the subsurface contaminant transport equation for instantaneous equilibrium linear sorption.

► **Example 6.3**

The bulk soil density for the aquifer material in Example 6.1 is 1600 kg(s)/m^3 and the organic carbon fraction is 0.02. What is the retardation factor for TCE?

Solution The distribution coefficient can be calculated from Eq. 5.5:

$$K_D = f_{oc} K_{oc}$$

where $K_{oc} = 68$ L/kg(s) (from Table 5.2). Substitution gives

$$K_D = (0.02)\ [68\,\text{L/kg(s)}] = 1.36\,\text{L/kg(s)}$$

The retardation factor is calculated from Eq. 6.18:

$$R = 1 + \frac{\rho_B K_D}{n} = 1 + \frac{[1600\,\text{kg(s)/m}^3][1.36\,\text{L/kg(s)}](10^{-3}\ \text{m}^3/\text{L})}{0.33}$$
$$= 7.6$$

Comparing the dispersive and advective transport terms in Eq. 6.17 to those in Eq. 4.1, it is seen that the forms are the same except that D is replaced by D/R and u is replaced by u/R. Since $R > 1$ for a sorbing contaminant, the net effect of sorption is to make dispersive and advective transport of the contaminant to be slower than that of the water by the factor $1/R$. Thus, **mean linear contaminant velocity**, u_c, is defined as

$$u_c = \frac{u}{R} \tag{6.19}$$

The effect of sorption on contaminant velocity is illustrated in Table 6.4, where the ratio of contaminant velocity to water velocity is given for various values of distribution coefficient for a given porosity and bulk soil density. Sorption is seen to have a dramatic effect on the advection of a contaminant. For example, at a distribution coefficient of 10 L/kg(s), which is at the low end of the range for metals in a subsurface medium containing clay, the contaminant migrates only 1/50 as fast as the water. The reason is that at any instant in time only 1/50 of the contaminant is associated with the aqueous phase and is mobile; the remainder is associated with the solid phase and is immobile.

TABLE 6.4 Effect of Sorption on Contaminant Velocity in the Saturated Zone[a]

K_D [L/kg(s)]	R	u_c/u
0	1	1
0.1	1.5	0.65
1	6.3	0.16
10	54	0.02
100	5330	0.002

[a] $n = 0.2$, $\rho_B = 1500\,\text{kg(s)/m}^3$.

► **Example 6.4**

How long does it take for TCE to travel from the burial ground to the site boundary for the problem in Example 6.1?

Solution The speed of TCE is calculated from Eq. 6.19:

$$u_c = \frac{u}{R}$$

From Example 6.1, $u = 9.1 \times 10^{-7}\,\text{m/s}$, and from Example 6.3, $R = 7.6$. Substitution gives

$$u_c = \frac{u}{R} = \frac{9.1\times10^{-7}\,\text{m/s}}{7.6} = 1.2\times10^{-7}\,\text{m/s} = 3.8\,\text{m/yr}$$

The contaminant travel time to the site boundary is

$$t = \frac{500\,\text{m}}{3.8\,\text{m/yr}} = 132\,\text{yr}$$

This is 7.6 times longer than the travel time for the groundwater, which was 17 years.

6.5.2 Saturated-Zone Transport Solutions

6.5.2.1 One-Dimensional Solutions Solving Eq. 6.17 can be complex. As discussed in Chapter 2, there are two general approaches for solving the contaminant transport equation: numerical and analytical. Numerical solutions typically involve the use of finite-element or finite-difference techniques. Analytical solutions provide fundamental insights into the transport behavior of contaminants. Analyti-

cal solutions to Eq. 6.17 for a wide variety of initial conditions and boundary conditions are given by van Genuchten and Alves (1982). The two one-dimensional problems considered here were solved for no sorption ($R = 1$) in the general discussion of advective–dispersive transport in Chapter 4. In both cases, releases occur at $x = 0$. In the first, the release is instantaneous in time; in the second it is semi-infinite in time. Solutions can be obtained either by solving Eq. 6.17 with the appropriate initial conditions or by letting $D \rightarrow D/R \approx \alpha_L u/R$ and $u \rightarrow u/R$ in Eqs. 4.6 and 4.7. When applying the latter approach to Eq. 4.6, S_0 must be replaced by S_0/nR to account for the fraction of S_0 that is in the aqueous phase ($1/R$) and the fraction of the volume that is occupied by liquid (n). It must be remembered that these solutions are for aqueous-phase contaminants in homogeneous and isotropic media, with no sources or sinks of groundwater (e.g., wells or recharge areas).

The solution for the instantaneous release for a constant dispersivity is

$$C_l(x,t) = \frac{S_0}{nA\sqrt{4\pi\alpha_L uRt}} \exp\left[-\frac{(Rx-ut)^2}{4\alpha_L uRt}\right] \exp\left[-k\frac{xR}{u}\right] \tag{6.20}$$

Presented in Figure 6.6 is concentration as a function of down-gradient distance at a given time after the release for retardation factors of 1 and 5. Longitudinal dispersivity is a constant value of 1 m in Figure 6.6, regardless of the distance from the source. The contaminant that has a retardation factor of 5 travels one-fifth the distance of a contaminant that has a retardation factor of 1, and its mass in the aqueous phase, which is the area under the concentration vs. distance curve, is one-fifth as large.

The solution for the semi-infinite step release for a constant dispersivity is

$$C_l(x,t) = \frac{C_{l0}}{2}\left[\operatorname{erfc}\left(\frac{Rx-ut}{\sqrt{4\alpha_L uRt}}\right) + \exp\left(\frac{x}{\alpha_L}\right)\operatorname{erfc}\left(\frac{Rx+ut}{\sqrt{4\alpha_L uRt}}\right)\right]\exp\left(-k\frac{Rx}{u}\right) \tag{6.21}$$

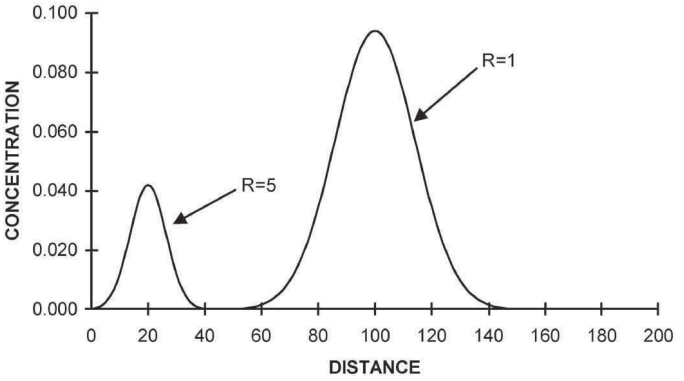

Figure 6.6 Aqueous-phase concentration vs. distance for an instantaneous release with $R = 1$ and $R = 5$. Values of other parameters are $S_0 = 1$, $n = 0.3$, $A = 1\,\text{m}^2$, $u = 10\,\text{m/yr}$, $t = 10\,\text{yr}$, and $\alpha_L = 1\,\text{m}$.

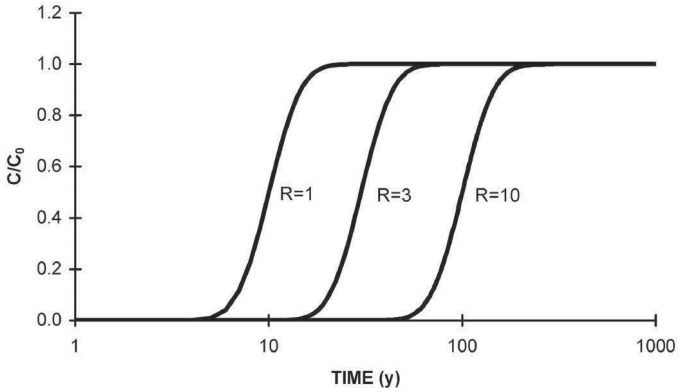

Figure 6.7 C/C_0 as a function of time for a semi-infinite step release with $R = 1$, 3, and 10. Values of other parameters are $x = 100\,\text{m}$, $u = 10\,\text{m/yr}$, and $\alpha_L = 4.42\,\text{m}$ (from Eq. 6.15).

where C_{l0} is aqueous phase concentration at $x = 0$. In most groundwater transport problems, the Peclet number (Pe = ux/D) is large and the second term in brackets in Eq. 6.21 can be neglected, yielding

$$C_l(x,t) \approx \frac{C_{l0}}{2}\text{erfc}\left(\frac{Rx-ut}{\sqrt{4\alpha_L uRt}}\right)\exp\left(-k\frac{Rx}{u}\right) \tag{6.22}$$

Calculations based on Eq. 6.22 are given in Figure 6.7, where C_l/C_{l0} is plotted against time at a given location for $R = 1$, 3, and 10. As R increases, the time required for the contaminant to reach a given location increases. Physically, this occurs because the fraction of contaminant in the aqueous phase decreases with increasing R, and the contaminant must travel more slowly to maintain equilibrium with the solid-phase concentration.

6.5.2.2 Multidimensional Solutions One-dimensional solutions such as those presented in Section 6.5.2.1 are useful in illustrating the temporal and spatial behavior of a plume along its centerline, but they overestimate concentrations because transverse dispersion is neglected. This can be illustrated for the situation in Figure 6.8, which is the conceptual model once used by the EPA to evaluate hazardous waste delisting petitions. Depicted is a rectangular area of uniform contaminant concentration C_0 in the saturated zone as might occur beneath a hazardous waste burial site. The contaminated area has width W and depth H, and it is perpendicular to the direction of groundwater flow. The top of the contaminated region coincides with the top of the saturated zone, and groundwater flow is in the x direction. The steady-state expression for $C_l(x,y,z)/C_{l0}$ is (Domenico and Palciauskas 1982)

$$\frac{C_l(x,y,z)}{C_{l0}} = \frac{1}{4}\left[\text{erf}\left(\frac{y+W/2}{2\sqrt{\alpha_H x}}\right)-\text{erf}\left(\frac{y-W/2}{2\sqrt{\alpha_H x}}\right)\right]\left[\text{erf}\left(\frac{z+H}{2\sqrt{\alpha_V x}}\right)-\text{erf}\left(\frac{z-H}{2\sqrt{\alpha_V x}}\right)\right] \tag{6.23}$$

The centerline concentration (i.e., along the x-axis) is

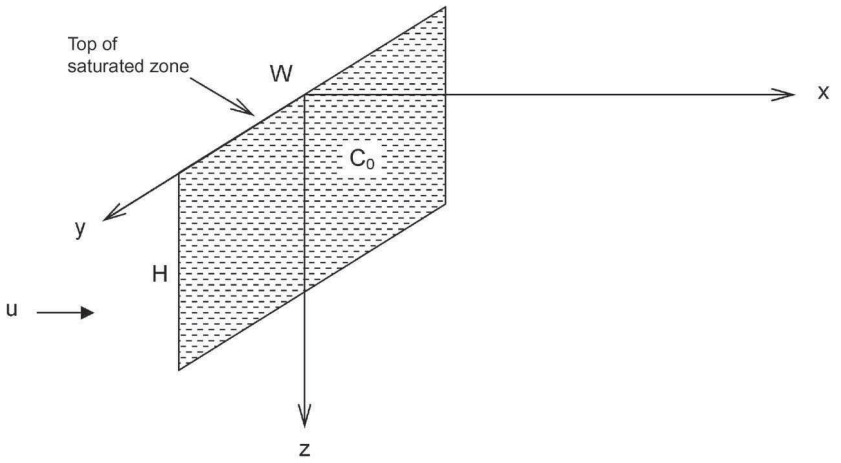

Figure 6.8 Conceptual model for Eq. 6.23.

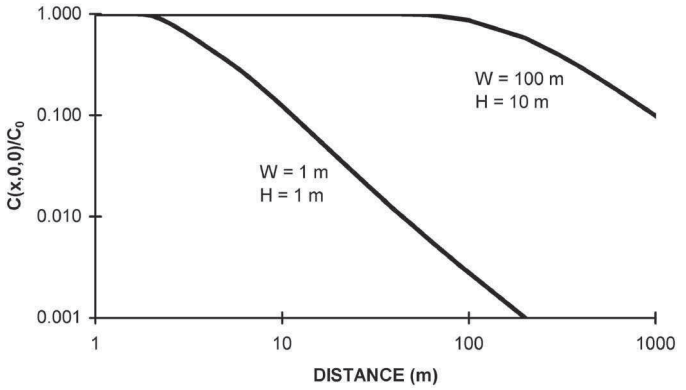

Figure 6.9 Effect of transverse dispersion on centerline concentration for small (1 m × 1 m) and large (100 m × 10 m) contaminated zones.

$$\frac{C_l(x,0,0)}{C_{l0}} = \text{erf}\left(\frac{W}{4\sqrt{\alpha_H x}}\right)\text{erf}\left(\frac{H}{2\sqrt{\alpha_V x}}\right) \tag{6.24}$$

where α_H and α_V are transverse dispersivities in the horizontal and vertical directions, respectively. In the absence of site-specific data, the following are recommended (ASTM 1995):

$$\alpha_H \approx 0.33\alpha_L \tag{6.25}$$

$$\alpha_V \approx 0.05\alpha_L \tag{6.26}$$

Presented in Figure 6.9 is $C_l(x,0,0)/C_{l0}$ as a function of distance for a small (1 m width × 1 m height) and a large (100 m width × 10 m height) contaminated zone. For the small contaminated zone, transverse dispersion begins to have an impact on

the centerline concentration within 2 m of the source, whereas for the large contaminated zone, the impact occurs first over 50 m from the source. Referring to Eq. 6.24, the error function is unity for values of the argument greater than about 2. Thus, transverse dispersion is important for $W <\approx 8\sqrt{\alpha_H x}$ or $H <\approx 4\sqrt{\alpha_V x}$.

Multidimensional groundwater transport problems are typically solved by numerical methods. However, analytical approximations are available (Codell et al. 1982; Wexler 1992) which are useful to illustrate plume behavior and to conduct scoping studies. A relatively simple method for formulating a three-dimensional time-dependent approximation (Domenico and Robbins 1985; Domenico 1987) is illustrated in Example 6.5. The approach is to multiply a one-dimensional time-dependent solution such as Eq. 6.20 (instantaneous release) or Eq. 6.22 (semi-infinite step release) by a steady-state solution such as Eq. 6.23 (plane perpendicular to flow direction) corresponding to the source geometry in the y and z directions.

► Example 6.5

An orphaned waste site for pesticides is discovered in a rural area. A test well installed to sample water leaching from the waste into the unsaturated zone beneath the site reveals the presence of lindane. A preliminary analysis suggests that the principal exposure pathway is ingestion of groundwater from a well located 100 m down-gradient from the site. An analysis is to be performed to evaluate the risk posed by the site.

In the conceptual model of the site, rainwater infiltrates the waste, leaches lindane from the waste at a constant rate, and travels vertically through the unsaturated zone to the top of the saturated zone. When the percolating water enters the saturated zone, it displaces the clean groundwater (i.e., no dilution), yet moves at the same velocity as the clean groundwater. This results in a uniformly contaminated area of width W and height H at the down-gradient end of the region below the waste site as depicted in Figure 6.10.

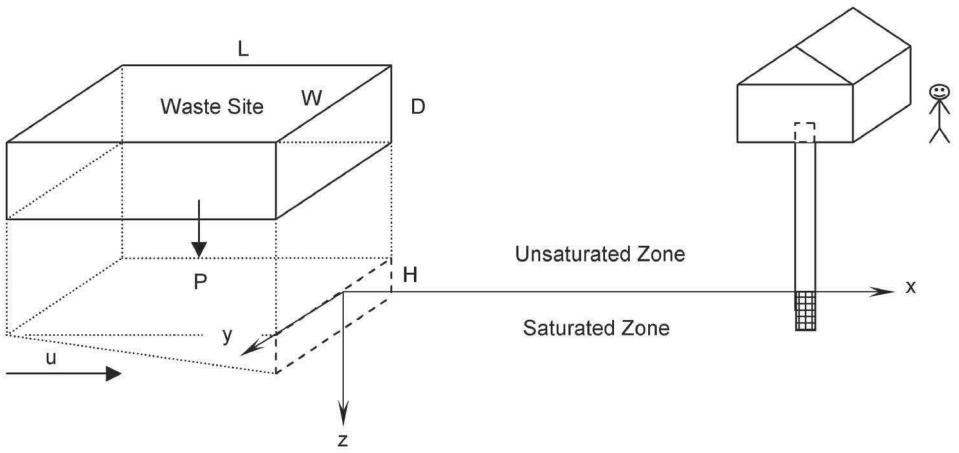

Figure 6.10 Conceptual model for Example 6.5.

The concentration of lindane in the percolating water beneath the site is 40 μg/L. The waste site is 10 m long, 30 m wide, and 2 m deep. The groundwater velocity is 15 m/yr, the percolation rate (see Section 6.6.1) of water through the unsaturated zone is 40 cm/yr, and $R = 30$.

(a) What is the height, H, of the contaminated area in the saturated zone?
(b) If the aquifer beneath the site has been contaminated for 20 years, use a one-dimensional model to determine when the lindane concentration is expected to exceed the EPA's maximum contaminant level (MCL) for lindane, which is 2 μg/L.
(c) Repeat part (b) with a multidimensional model.

Solution

(a) The depth of contamination (H) at the downstream end of the waste site can be calculated by equating the volumetric flow rate of water out of the waste site to that through the contaminated area:

$$PLW = nuWH$$

Solving for H and substituting values gives us

$$H = \frac{PL}{nu} = \frac{(0.4\,\text{m/yr})(10\,\text{m})}{(0.3)(15\,\text{m/yr})} = 0.89\,m$$

(b) Neglecting degradation, the one-dimensional time-dependent model for a semi-infinite source is Eq. 6.22:

$$C_l(x,t) \approx \frac{C_{l0}}{2}\,\text{erfc}\left(\frac{Rx - ut}{\sqrt{4\alpha_L uRt}}\right)$$

where C_0 = 40 μg/L, R = 30, u = 15 m/yr, and $\alpha_L = 0.83(\log_{10}x)^{2.414} = 0.83(\log_{10}100)^{2.414} = 4.42$ m.

(c) Applying the approach of Domenico (1987), a three-dimensional time-dependent model can be formed by the product of Eqs. 6.22 and 6.24:

$$\frac{C_l(x,0,0,t)}{C_{l0}} \approx \frac{1}{2}\,\text{erfc}\left(\frac{Rx - ut}{\sqrt{4\alpha_L uRt}}\right)\text{erf}\left(\frac{W}{\sqrt{4\alpha_H x}}\right)\text{erf}\left(\frac{H}{2\sqrt{\alpha_V x}}\right)\exp\left(-k\frac{Rx}{u}\right)$$

where $W = 30$ m, $H = 0.89$ m, $\alpha_H \approx 0.33\alpha_L = 1.46$ m, $\alpha_V \approx 0.05\alpha_L = 0.22$ m, and $k = 0$. The one- and three-dimensional estimates are plotted in Figure 6.11. Based on the one-dimensional model, the lindane concentration at the well is predicted to reach the MCL 90 years after the initial contamination of the aquifer. Based on the three-dimensional model, the time to reach the MCL is predicted to be about 140 years.

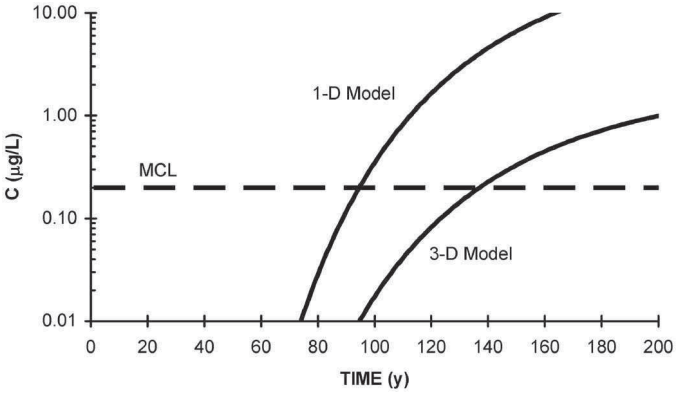

Figure 6.11 Comparison of one- and three-dimensional predictions in Example 6.5.

6.6 OTHER CONSIDERATIONS IN GROUNDWATER TRANSPORT

6.6.1 Vadose Zone Transport

The modeling of water flow and contaminant transport in the vadose zone is more complex and difficult than that in the saturated zone because of the presence of both air and water in the pore spaces. Estimates of contaminant travel time vertically downward in the unsaturated zone can be made using the conceptual model depicted in Figure 6.12. The **mean percolation rate** P is the mean flow rate per unit area into the vadose zone. It is obtained from a water balance on the top soil layer, or root zone. A simple **water balance** involves estimating water inputs and losses to the root zone, averaged over a period of time, typically a year. Precipitation, in the form of rain or snowmelt, provides water input to the root zone. Losses can occur by runoff (overland flow) to surface water bodies and percolation into the vadose zone and ultimately to the saturated zone. Losses can also occur by water in the soil being evaporated to the atmosphere or being absorbed by plant roots and subsequently entering the atmosphere by **transpiration.** This simple water balance is based on a quasi-steady-flow approximation. If the flow is time varying, changes in moisture content within the soil layer can also affect the amount of water available for percolation.

For a constant-tension head and moisture content, mean linear velocity through the vadose zone, v, can be estimated by

$$v = \frac{P}{\theta} \tag{6.27}$$

The contaminant velocity thus becomes

$$v_c = \frac{P}{R_V \theta} \tag{6.28}$$

where v_c is the contaminant velocity and R_V is the retardation factor for the vadose zone sediments.

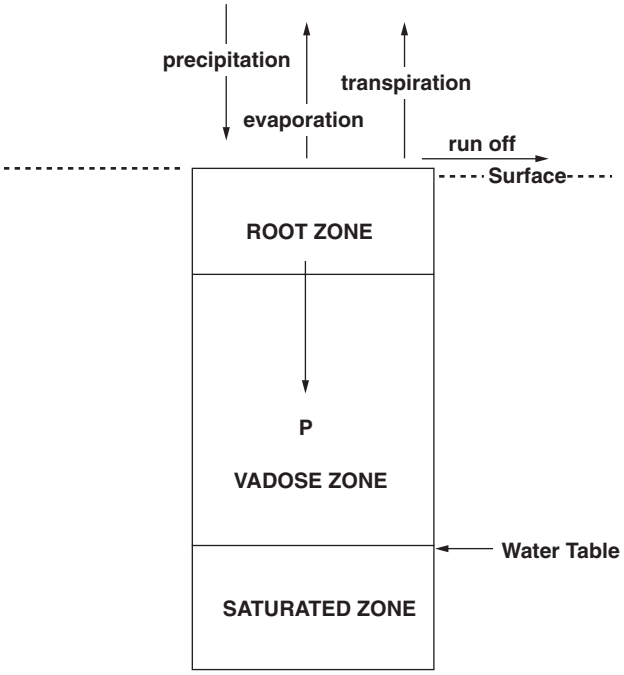

Figure 6.12 Compartmental model of water flow and contaminant transport in the unsaturated zone.

▶ Example 6.6

The distance from the bottom of the waste site in Example 6.5 to the water table is 5 m. If the average moisture content is 0.18 and the lindane retardation factor in the unsaturated zone is 12, how long does it take lindane to migrate from the waste site to the saturated zone?

Solution The mean linear velocity in the vadose zone is estimated from Eq. 6.28:

$$v_c = \frac{P}{R_u \theta}$$

Substitution yields

$$v_c = \frac{0.4\,\text{m/yr}}{(12)(0.18)} = 0.185\,\text{m/yr}$$

The travel time to the saturated zone is

$$t = \frac{5\,\text{m}}{0.185\,\text{m/yr}} = 27\,\text{yr}$$

6.6.2 Colloidal Transport

Colloids are very small particles (smaller than 1 μm in diameter) which because of their size can be suspended in and migrate with groundwater. Colloids can be soil particles, macromolecules, suspensions of insoluble liquids, or bacteria. They are a complicating feature in groundwater transport because they represent a mobile solid phase which undergoes transport at a much higher velocity than that predicted by Eq. 6.19. In some situations the effect of colloids on transport predictions can be estimated by assigning a separate K_D (or, equivalently, a separate R) to the fraction of the contaminant associated with colloids. However, the colloidal fraction cannot be reliably predicted and must be inferred from field or laboratory data.

6.6.3 Transformations

Depending on the type of contaminant present, transformation processes can significantly affect the concentration of a contaminant at a location down-gradient from a source. These processes are nonconservative and can result in either an increase or, more commonly, a decrease in contaminant concentration. The transformations can be divided into two categories: biotic and abiotic. **Biotic transformations** are those that are produced by biological processes of microscopic flora (typically, bacteria) in the subsurface. **Abiotic transformations** are those that are due to physical processes such as radioactive decay or chemical reactions between the contaminant and surrounding media. Although the basic processes underlying the transformations are often fairly well understood, rigorous application of the relevant models is often difficult, and transformations are often approximated as first-order processes. When modeling transformations, it must be remembered that transformation products are generated. Some transformations yield products that are more toxic than the parent contaminant, and their impact on risk must also be taken into account.

Biological transformations are typically the result of subsurface microbes which utilize organic contaminants as an energy source. These can occur either under aerobic conditions or if an electron acceptor such as nitrate is present, under anaerobic conditions. Although the modeling of biodegradation can be highly complex, certain simplifying assumptions can be made to approximate the process as first order. These assumptions are that the concentration of the compound is very low and that the amount of microbial activity and oxygen are relatively constant.

■ Reductive Dechlorination of PCE

Tetrachloroethylene (C_2Cl_4), or PCE, is widely used for dry cleaning of fabrics and for degreasing of metal objects, and it is toxic to humans. Through a series of anaerobic biotic transformations called reductive dechlorination, chlorine atoms are sequentially replaced by hydrogen, and PCE is ultimately transformed to ethene (C_2H_4). This series of transformations is shown in Figure 6.13. Two of the transformation products, trichloroethylene (TCE) and vinyl chloride (VC), are also toxic.

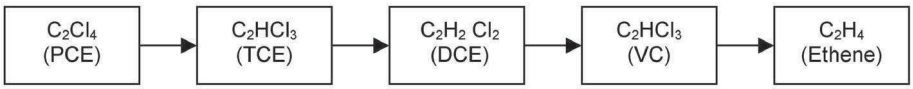

Figure 6.13 Biotic transformations of PCE and its degradation products.

Thus, risk assessments of tetrachloroethylene in environments where reductive dechlorination is possible must consider risk due to TCE and VC as well.

Common abiotic transformations include radioactive decay (for radioactive contaminants), hydrolysis, dehydrohalogenation, oxidation, and reduction. Hydrolysis is a chemical process in which water reacts with a contaminant molecule, the contaminant and the water molecules are split, and the fragments combine. For example, a halogenated hydrocarbon contaminant and water can react to yield a hydrocarbon group and a halogen ion from the contaminant and a hydroxyl ion and an H^+ ion from the water. The hydroxyl ion can combine with the hydrocarbon group, and the H^+ ion can form an acid with the halogen ion. In general, hydrolysis rates for slightly halogenated hydrocarbons are greater than those for highly halogenated hydrocarbons. However, the converse is true for dehydrohalogenation, in which a halogen atom and a hydrogen atom are removed from a hydrocarbon compound, leaving a double bond between the carbon atoms formerly linked by a single bond. This type of process proceeds rapidly for highly halogenated compounds and slows as the degree of halogenation decreases. Oxidation and reduction can cause changes in the speciation of the contaminant, which can strongly affect its transport properties.

6.6.4 Non-Aqueous-Phase Liquids

Non-aqueous-phase liquids pose a particular modeling problem. Up to this point, the discussion has been focused on contaminants in an aqueous phase. However, some important contaminants are relatively immiscible in water. They are known as **non-aqueous-phase liquids** (NAPLs). The density of these compounds can either be greater or less than that of water. Non-aqueous-phase liquids which are lighter than water, such as gasoline, are known as **light non-aqueous-phase liquids** (LNAPLs), and those that are denser than water, such as trichloroethylene, are referred to as **dense non-aqueous-phase liquids** (DNAPLs).

A LNAPL spill results in a pool of liquid that floats on top of the saturated zone and represents a constant input of dissolved contaminant to the groundwater (Figure 6.14). A DNAPL spill sinks through the saturated zone, pooling on top of low-permeability formations such as clay or rock (Figure 6.15). Since a residual saturation of NAPLs can be retained in the soil pores as a discontinuous mass, it can constitute a continuing and variable source of dissolved-phase contamination. Since some of the liquid dissolves, the modeling of the dissolved phase may be handled by the techniques discussed above. Quantitative modeling of the movement of NAPLs is quite complex. However, the theory is quite well developed, as the problem has been of interest to petroleum geologists for many years (Fetter 1999).

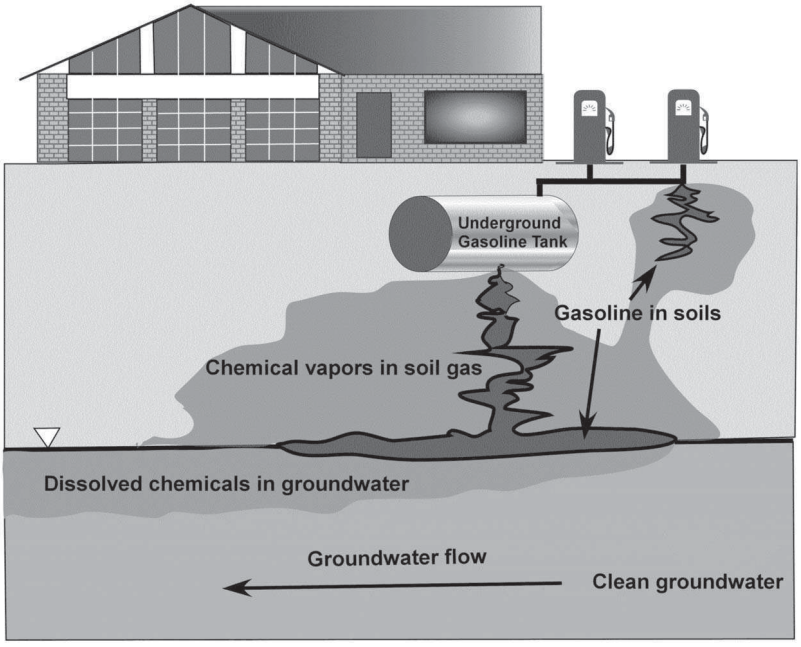

Figure 6.14 LNAPL spill. (By permission of R. W. Falta, Jr., Clemson University, Clemson, SC.)

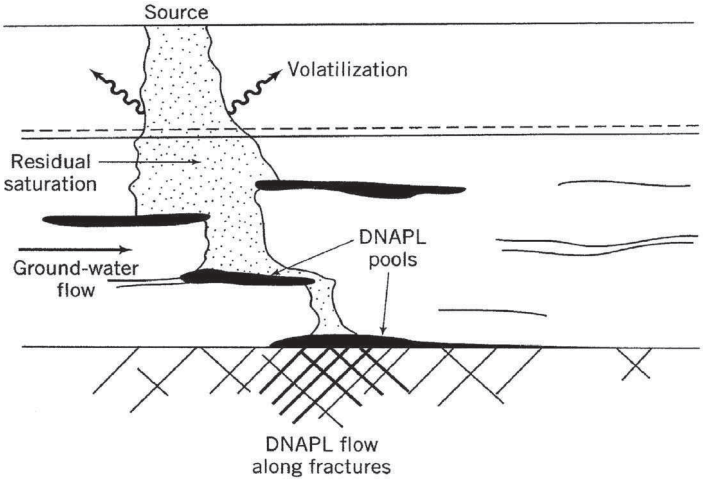

Figure 6.15 DNAPL spill. (From Domenico and Schwartz 1998; reprinted by permission of John Wiley & Sons, Inc.)

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PROBLEMS

6.1 The hydraulic pressure gradient along the direction of groundwater flow is −0.008 m/m, the porosity is 0.25, and the hydraulic conductivity is 0.003 cm/s. Find the mean linear groundwater velocity.

6.2 The hydraulic head field in a region is described by $H(x,y,z) = 3x^2 - xy^2 + \ln z$.

(a) Find the direction of groundwater flow.

(b) If a contaminant percolating downward from the unsaturated zone reaches the water table at (2,3,5), in what direction does it begin to migrate?

6.3 Using the definitions of porosity and bulk soil density, show that $\rho_B = \rho_P(1 - n)$.

6.4 Estimate the hydraulic conductivity in an aquifer in which the median grain size is 0.08 mm and the porosity is 0.32.

6.5 Using an approach analogous to that used in Chapter 5 for surface water transport, derive Eq. 6.16 for fraction sorbed in a subsurface medium.

6.6 Show the following relationships utilized in Table 6.3:

(a) $\text{SS} = \dfrac{1}{1/\rho_B + m_l / \rho_w m_s}$.

(b) $\theta = \dfrac{1}{1 + (1/\rho_p)(m_s / m_l)}$.

(c) For $m_s/m_l = 10^{-2}$, verify the remaining values in the table.

6.7 Nickel from a metal plating operation is found in nearby groundwater. Find the fraction sorbed if the distribution coefficient for nickel with the sediments is 35 L/kg(s), the porosity is 0.3, and the bulk soil density is 1750 kg(s)/m^3.

6.8 Show that the fraction of contaminant in the aqueous phase is $1/R$. (*Hint:* Derive f_l in terms of K_D, n, and ρ_B.)

6.9 The distribution coefficients for the remaining two contaminants in Example 6.1 are 1000 L/kg(s) for ^{137}Cs and 52 L/kg(s) for mercury.

(a) Find the retardation factor for each contaminant.

(b) Find the travel time to the site boundary for each contaminant.

(c) Find the fraction of each contaminant in the aqueous phase.

6.10 Consider a one-dimensional advective approximation (neglect dispersion) for the transport of ethylene dibromide in groundwater. The release of ethylene dibromide to the groundwater can be approximated as a semi-infinite step with an emission rate of 30 kg/yr. The aquifer is composed of sandy soil with a hydraulic conductivity of 10^{-5} m/s, an effective porosity of 0.4, a bulk soil density of 1600 kg/m^3, an organic fraction of 0.015, and a hydraulic gradient of −0.01 m/m. The cross-sectional area of the contaminated region in the aquifer is 20 m^2. From Table 5.2, the K_{oc} for ethylene dibromide is 44 L/kg(s).

(a) How long will it take the ethylene dibromide to reach a well located 50 m from the source?

(b) Estimate the concentration of ethylene dibromide (in mg/L) at the well.

(c) If the ethylene dibromide undergoes degradation with an effective half-life of 100 days, calculate the concentration of ethylene dibromide (in mg/L) at the well.

6.11 Two hundred kilograms of a contaminant is released to an aquifer from a waste disposal trench. The distribution coefficient for the contaminant is 10 L/kg(s), the bulk soil density is 1500 kg/m^3, and the porosity is 0.3.

(a) If the average linear groundwater velocity is 20 m/yr, what is the contaminant velocity?

(b) What fraction of the contaminant is associated with the aqueous phase?

6.12 A low-level radioactive waste facility contains a large inventory of ^{60}Co ($t_{1/2} = 5.2$ yr, $K_D = 45$ L/kg). The average hydraulic gradient between the waste site and the site boundary, which is 500 m away, is −0.01 m/m. The average hydraulic conductivity is 3×10^{-3} cm/s, the porosity is 0.3, and the bulk soil density is 1600 kg/m^3.

(a) How long does it take ^{60}Co to reach the site boundary?

(b) If the concentration of ^{60}Co below the waste site is 100 Bq/L, what will the concentration be when it reaches the site boundary? (Neglect dilution.)

6.13 The concentrations of carbon tetrachloride in wells 20 and 50 m downgradient from a hazardous waste site are 200 and 80 µg/L, respectively. Using a one-dimensional advective transport approximation (neglect dispersion), estimate the degradation half-life of carbon tetrachloride in the subsurface. The mean linear groundwater velocity is 15 m/yr and the carbon tetrachloride retardation factor is 1.003.

6.14 A three-dimensional mathematical model for contaminant transport in the saturated zone due to a semi-infinite step source can be formed by the product of the one-dimensional longitudinal solution (Eq. 6.22) and the two-dimensional transverse solution (Eq. 6.23).

(a) Using the software tool of your choice (Excel, C++, Fortran, Visual Basic, etc.), implement this approximation. Variables to be input into the computer solution are groundwater velocity, retardation factor, porosity, first-order reaction rate, depth of contaminated region at $x = 0$, width of contaminated region at $x = 0$, and well or outcrop location (x,y,z).

(b) Given the following parameters:

$$\begin{aligned} k &= 0.01\ \text{yr}^{-1} & W &= 20\ \text{m} \\ R &= 3 & H &= 5\ \text{m} \\ u &= 20\ \text{m/yr} & n_e &= 0.3 \end{aligned}$$

(i) Determine $C(50,5,2)/C_0$ at a time of 7 years. Check with a hand calculation.

(ii) Compute $C(50,0,0)/C_0$ as a function of time for $R = 1, 2$ and 10. Use the following increments (years):

$$\begin{aligned} 0 < t \le 10 \quad & \Delta t = 1 \\ 10 < t \le 100 \quad & \Delta t = 10 \\ 100 < t \le 1000 \quad & \Delta t = 100 \end{aligned}$$

Plot these results on the same graph (log-log). Explain the behavior that you observe.

7 Atmospheric Transport

7.1 INTRODUCTION

Contaminants released to the atmosphere can have impacts over a wide range of spatial scales. At the global level, there is a strong link between the release of chlorinated ßuorocarbon compounds and deterioration of the ozone layer, and there is mounting evidence linking releases of carbon dioxide to global climate change. At the regional level, releases of sulfur dioxide and oxides of nitrogen in one locale contribute to acid precipitation and its concomitant ecological impacts in another location. Examples are releases in the United Kingdom affecting Scandinavia, those in the central United States affecting areas in the southeast and northeast, and releases from the United States affecting Canada. Although the global and regional atmospheric contamination issues are important, they are beyond the scope of this book, which is concerned with localized effects (i.e., those within a few tens of kilometers of the source).

A typical risk assessment problem involving localized atmospheric transport is illustrated conceptually in Figure 7.1. A contaminant is emitted from a source, either routinely or accidentally, at an emission rate $\dot{S}(t)$. The objective is to predict the contaminant concentration at one or more receptor locations. For a deÞned emission, the concentration depends primarily on wind direction, wind speed, release height, and turbulent diffusion. However, removal or transformation mechanisms can be important under certain meteorological conditions (such as a precipitation event) or for selected contaminants (such as hydrogen sulÞde, photochemical oxidants, or radionuclides with short half-lives). Simple atmospheric transport models that take into account the various physical and chemical processes of importance are presented in this chapter.

7.2 ATMOSPHERIC DISPERSION

As noted above, the focus here is on localized effects close to the release point (up to about 50 km). Localized transport occurs in the part of the atmosphere adjacent to the surface of the Earth known as the planetary boundary layer, which can vary in thickness between approximately 100 and 3000 m. The dispersion of contaminants in the planetary boundary layer depends on the stability of the atmosphere. Atmospheric stability depends largely on the vertical temperature gradient, or

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

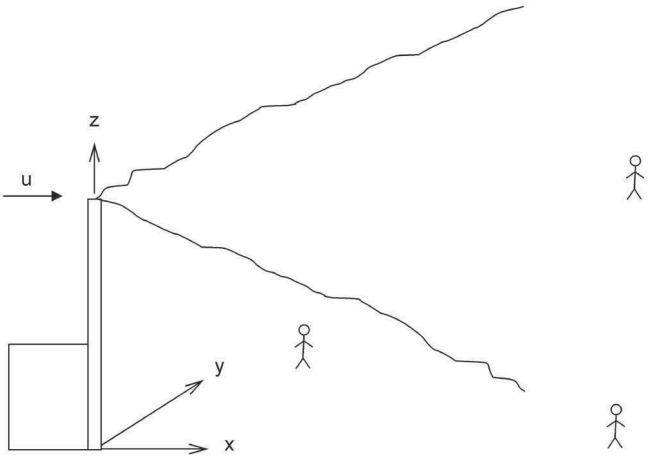

Figure 7.1 Typical local atmospheric transport scenario.

lapse rate. The temperature gradient, in turn, depends primarily on the decrease in pressure with height above the surface but can also be affected by cloud cover, solar radiation, and winds.

The vertical temperature gradient that results solely from the decrease in pressure with height above the surface is the **dry adiabatic lapse rate** and is approximately –1¡C per 100 m (–5.4¡F per 1000 ft). The dry adiabatic lapse rate is applicable as long as water vapor does not condense to liquid water. This phase transition releases heat to the air, which decreases the lapse rate to a value lower than the dry adiabatic lapse rate. Generally, as the air cools with altitude, the relative humidity increases. At an altitude where the relative humidity has a value around 100%, water begins to condense from the air and the lapse rate is less than the dry adiabatic lapse rate.

Under dry adiabatic conditions, the atmosphere is considered to be neutral with respect to contaminant dispersion (i.e., vertical motions are neither enhanced nor suppressed). However, if the actual temperature gradient is not as steep as the adiabatic lapse rate or if the temperature increases with elevation, vertical motions are suppressed. This behavior can be understood by considering the relative temperatures of a displaced parcel of air and the surrounding air as illustrated in Figure 7.2. For purposes of explanation, the parcel of air can be considered to be contained in a virtual sac which does not permit heat transfer between the parcel and its surroundings. If the parcel is displaced upward from its initial position, its temperature decreases according to the dry adiabatic lapse rate. The temperature of the surrounding air, on the other hand, increases and thus is greater than that of the air parcel. Since both the surrounding air and the air parcel are at the same pressure, the ideal gas law requires that the ratio of densities be inversely proportional to the ratio of temperatures. Consequently, the density of the air parcel is greater than that of the surrounding air, and the parcel experiences a net force (the difference between the buoyancy force upward and the gravitational force downward) that pushes it down toward its original position. Similarly, a parcel that is displaced

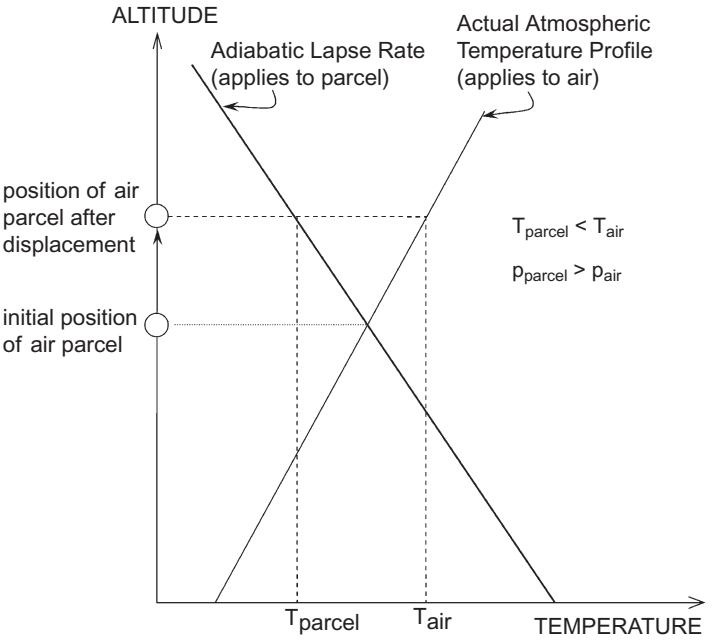

Figure 7.2 Suppression of vertical motion when the temperature proÞle in the atmosphere increases with altitude.

downward has a higher temperature than the surrounding air and a corresponding lower density. It experiences a net force that pushes it upward toward its original position. Thus, under these conditions, the atmosphere is inherently stable and dispersion is low. Conversely, if the temperature decreases more rapidly than the adiabatic lapse rate, vertical motions are enhanced, the atmosphere is inherently unstable, and dispersion is high.

Near EarthÕs surface the actual temperature proÞle can differ signiÞcantly from the dry adiabatic proÞle due to surface heating from solar radiation, surface cooling due to radiation emission, the movement of air masses (i.e., cold fronts and warm fronts), the localized effects of wind and topography, and stationary high- or low-pressure systems. The effect of actual temperature proÞles on dispersion is illustrated in Figure 7.3. If the actual proÞle follows the dry adiabatic proÞle, the atmosphere is neutral with respect to vertical motions; and a contaminant plume forms a nearly symmetrical cone (Figure 7.3*a*). Under stable conditions, there is relatively little dispersion vertically. When this is accompanied by signiÞcant spreading in the direction transverse to wind ßow, the plume forms a thin fan (Figure 7.3*b*). Under unstable conditions, the enhanced vertical motion of the air causes the plume to loop (Figure 7.3*c*). An atmospheric layer in which air temperature increases with altitude is known as an **inversion.** Figure 7.3*b* shows an inversion from ground level to the highest altitude shown; Figure 7.3*d* shows an inversion layer at a medium height. Inversions can result in very high contaminant concentrations at ground level. For example, in Figure 7.3*d*, the inversion layer at medium height effectively traps contaminants released at lower altitudes, while the unstable

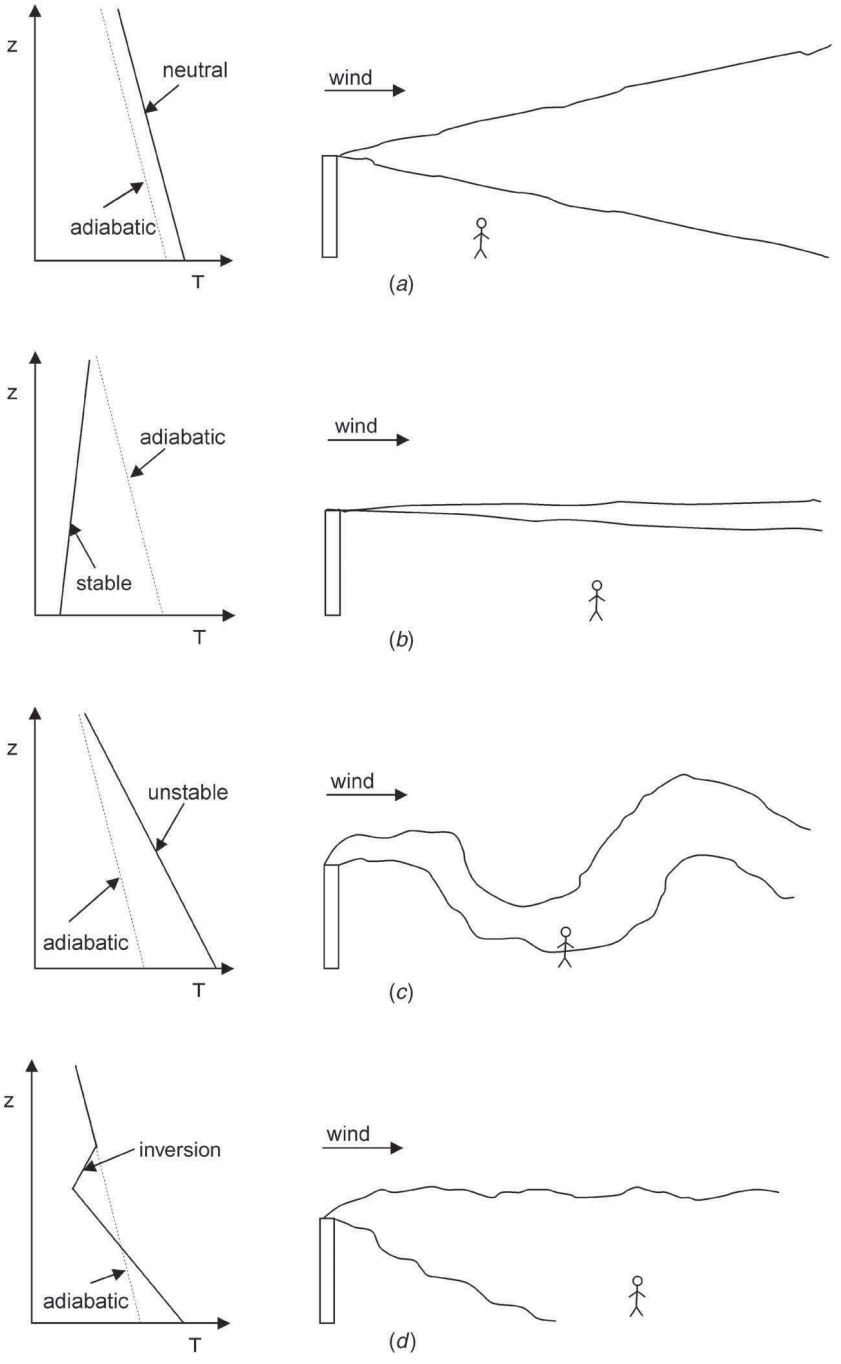

Figure 7.3 Representative actual temperature proÞles: (*a*) neutralÐfanning; (*b*) stableÐ fanning; (*c*) unstableÐlooping; (*d*) inversionÐfumigation.

(superadiabatic) temperature proPle below the inversion layer provides for efPcient mixing below the inversion. This condition is referred to as **fumigation**. Quantifying the effects of inversions on contaminant transport is an advanced topic that is not addressed fully in this book.

Nearly neutral conditions can occur on an overcast day with light winds. Unstable conditions can occur on clear days, especially in the summer, when solar heating causes the temperature of EarthÕs surface to rise. This causes the air near EarthÕs surface to rise and yield a temperature gradient that exceeds the adiabatic lapse rate. One common cause of stable conditions and inversions is radiation cooling. On a clear night, the EarthÕs surface cools by radiating energy into space. This results in cooling of air near EarthÕs surface to a lower temperature than that of air higher up. Thus, the temperature of the atmosphere in the early morning can actually increase with height. Stable conditions can also result from subsidence of air in a high-pressure system. Air descends in the center of a high-pressure system. As it falls, it is heated by compression according to the adiabatic lapse rate and can exceed the temperature of the air near the surface, which is determined by conditions on the ground. Since subsidence inversions are caused by high-pressure systems, they can occur in both the summer and winter. Further, unlike radiation inversions, which are short-lived, subsidence inversions can persist as long as the high-pressure system is in place.

For purposes of dispersion modeling, it is customary in the United States to characterize atmospheric stability in terms of the **Pasquill–Gifford stability classification system**. As developed by Pasquill (1961), atmospheric stability is inferred from surface wind speed and insolation or nighttime cloud cover through six discrete stability classes, shown in Table 7.1. This system was later modiPed by the Nuclear Regulatory Commission to include a stability class G, which would correspond to wind speeds of less than 2 m/s at night in Table 7.1. Also, the criteria for selection of the appropriate class were based on either the vertical temperature gradient or the standard deviation of wind direction as described in Table 7.2.

TABLE 7.1 Pasquill Stability Classification System[a]

Surface Wind Speed (m/s)	Insolation (Day)			Night	
	Strong	Moderate	Slight	Thinly Overcast or >4/8 Low Cloud	<3/8 Cloud
<2	A	AÐB	B	Ñ	Ñ
2Ð3	AÐB	B	C	E	F
3Ð5	B	BÐC	D	D	E
5Ð6	C	CÐD	D	D	D
>6	C	D	D	D	D

Source: Pasquill 1961.

[a] ÒStrong insolation corresponds to sunny midday in midsummer in England; slight insolation, to similar conditions in midwinter. Night refers to the period from 1 h before sunset to 1 h before sunrise. The neutral category D should also be used, regardless of wind speed, for overcast conditions during day or night and for any sky conditions during the hour preceding or following night as dePned above.Ó

TABLE 7.2 Pasquill–Gifford Stability Classification Systems as Implemented by the Nuclear Regulatory Commission

Stability Class	Description	σ_θ (deg)	Temperature Gradient (¡C/100 m)
A	Extremely unstable	25.0	$\frac{\Delta T}{\Delta z} \le -1.9$
B	Moderately unstable	20.0	$-1.9 < \frac{\Delta T}{\Delta z} \le -1.7$
C	Slightly unstable	15.0	$-1.7 < \frac{\Delta T}{\Delta z} \le -1.5$
D	Neutral	10.0	$-1.5 < \frac{\Delta T}{\Delta z} \le -0.5$
E	Slightly stable	5.0	$-0.5 < \frac{\Delta T}{\Delta z} \le 1.5$
F	Moderately stable	2.5	$1.5 < \frac{\Delta T}{\Delta z} \le 4.0$
G	Extremely stable	1.7	$\frac{\Delta T}{\Delta z} < 4.0$

Source: NRC 1972.

► **Example 7.1**

A paper manufacturing plant located in a ßat region is equipped with a 25-m stack. At this plant the average wind speed is 2.5 m/s. During the day, when insolation is Òmoderate,Ó what is the Pasquill stability category?

Solution Table 7.1 indicates that the stability class is B, moderately unstable.

7.3 ATMOSPHERIC TRANSPORT MODELS

Presented in this section are four models for estimating contaminant concentrations in the atmosphere. Two of the models apply to point sources with constant emission rate (Gaussian plume and sector averaged), one applies to a point, instantaneous emission (Gaussian puff model), and one applies to a linear source with constant emission rate. The theoretical basis for each of these is the contaminant transport equation (Eq. 4.14). In the atmosphere, turbulent diffusion is quantiÞed by the **eddy diffusivity** (Kao 1984), K [L^2/T], which is generally a tensor quantity. By choosing the x-axis parallel to the direction of the mean ßow, so the D_i in Eq. 4.14 may be replaced by K_{ii}, and neglecting the off-diagonal terms

$$\frac{\partial C}{\partial t} = K_{xx}\frac{\partial^2 C}{\partial x^2} + K_{yy}\frac{\partial^2 C}{\partial y^2} + K_{zz}\frac{\partial^2 C}{\partial z^2} - u\frac{\partial C}{\partial x} + g - d \tag{7.1}$$

7.3.1 Constant Emission Rate: Gaussian Plume Model

For modeling purposes, many atmospheric transport problems can be approximated by the simplified problem depicted in Figure 7.1. Consistent with the conventions discussed in Chapter 4, the x-axis is aligned with the wind direction and the z-axis is aligned with the vertical direction. The origin of the x and y axes is at the source and the origin of the z-axis is at ground level. The release occurs at $z = h$, $x = y = 0$. When applied to a constant rate, elevated release without generation or removal, the solution to Eq. 7.1 is

$$C(x,y,z) = \frac{\dot{S}_0}{u} \frac{\exp\left[-y^2/4K_{yy}(x/u)\right]}{\sqrt{4\pi K_{yy}(x/u)}} \frac{\exp\left[-(z-h)^2/4K_{zz}(x/u)\right]}{\sqrt{4\pi K_{zz}(x/u)}} \tag{7.2}$$

In its present form Eq. 7.2 is not useful because of the lack of suitable theoretical or empirical equations for eddy diffusivity. The operational form of Eq. 7.2 is known as the **Gaussian plume model**. This is obtained by substituting $\sigma_x = \sqrt{2K_{xx}(x/u)}$ and $\sigma_y = \sqrt{2K_{yy}(x/u)}$ (from Eq. 4.12) and assuming complete reflection of contaminant at the ground. The resulting expression for concentration at any point downwind of the release point is

$$C(x,y,z) = \frac{\dot{S}_0}{2\pi u \sigma_y \sigma_z} \exp\left(-\frac{y^2}{2\sigma_y^2}\right) \left\{ \exp\left[-\frac{(z-h)^2}{2\sigma_z^2}\right] + \exp\left[-\frac{(z+h)^2}{2\sigma_z^2}\right] \right\} \tag{7.3}$$

where σ_y and σ_y are the dispersion parameters (also called dispersion coefficients) in the cross-wind (y) and vertical (z) directions. They have units of [L] and both are functions of downwind distance x.

The expression for the ground-level concentration ($z = 0$) is

$$C(x,y,0) = \frac{\dot{S}_0}{\pi u \sigma_y \sigma_z} \left[\exp\left(-\frac{y^2}{2\sigma_y^2}\right) \exp\left(-\frac{h^2}{2\sigma_z^2}\right) \right] \tag{7.4}$$

The highest ground-level concentrations are found along the centerline of the plume. The centerline concentration is calculated by letting $y = 0$ in Eq. 7.4, yielding

$$C(x,0,0) = \frac{\dot{S}_0}{\pi u \sigma_y \sigma_z} \exp\left(-\frac{h^2}{2\sigma_z^2}\right) \tag{7.5}$$

Equations 7.3, 7.4, and 7.5 require values of the **dispersion parameters**, σ_y and σ_z, which are standard deviations of the Gaussian distribution of contaminant concentration in the y and z directions, respectively. A number of schemes are available for specifying σ_y and σ_z as a function of downwind distance. The Briggs curves are used in this book because they represent a "consistent estimation scheme for σ_y and σ_z over flat terrain from distances <1 km to 10 km" (Barr and Clements 1984, p. 519). Plots of σ_y and σ_z are displayed in Figure 7.4*a* and *b*, respectively, and algebraic expressions are given in Table 7.3. Although strictly applicable only for downwind distances up to about 10 km, they are commonly

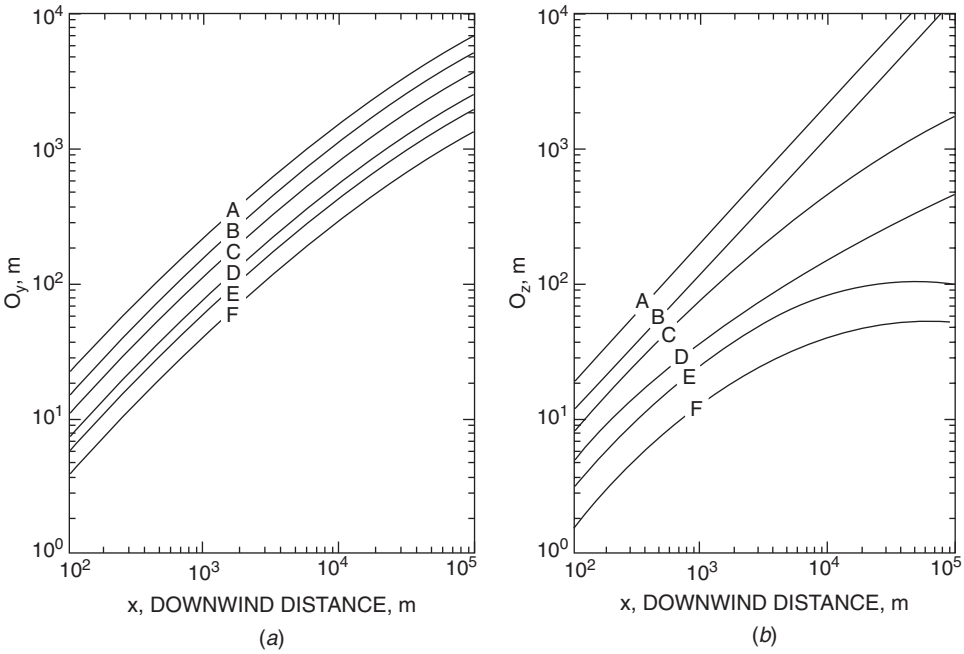

Figure 7.4 Briggs curves for estimating σ_y and σ_z. (From Barr and Clements 1984.)

TABLE 7.3 Equations Recommended by Briggs for σ_y and σ_z as a Function of Downwind Distance *x*

Stability Class	σ_y (m)	σ_z (m)
A	$\dfrac{0.22x}{\sqrt{1+0.0001x}}$	$0.20x$
B	$\dfrac{0.16x}{\sqrt{1+0.0001x}}$	$0.12x$
C	$\dfrac{0.11x}{\sqrt{1+0.0001x}}$	$\dfrac{0.08x}{\sqrt{1+0.0002x}}$
D	$\dfrac{0.08x}{\sqrt{1+0.0001x}}$	$\dfrac{0.06x}{\sqrt{1+0.0015x}}$
E	$\dfrac{0.06x}{\sqrt{1+0.0001x}}$	$\dfrac{0.03x}{1+0.0003x}$
F	$\dfrac{0.04x}{\sqrt{1+0.0001x}}$	$\dfrac{0.016x}{1+0.0003x}$

Source: Barr and Clements 1984.

used for distances up to 50 km. The Briggs dispersion parameters are used for EPA's National Emissions Standards for Hazardous Air Pollutants (NESHAP) analyses, but different systems are also in common use. The Nuclear Regulatory Commission, for example, uses the Pasquill–Gifford–Turner system (Figure 7.5). The Briggs and Pasquill–Gifford–Turner dispersion parameters are generally within 10 to 20% of one another under B, C, and D stability. However, values of σ_z under A, E, and F stability can differ substantially, especially at downwind distances greater than 1000 m.

▶ Example 7.2

A community is located 250 m downwind from the facility in Example 7.1. Calculate the dispersion parameters using the equations in Table 7.3.

Solution From Example 7.1, the atmospheric stability is class B. From Table 7.3, the equations for the dispersion parameters for stability class B are as follows, with 250 m substituted in for x:

$$\sigma_y = \frac{0.16x}{\sqrt{1+0.0001x}} = \frac{(0.16)(250)}{\sqrt{1+(0.0001)(250)}} = \frac{40}{1.01}$$
$$= 39.6\,\text{m}$$

$$\sigma_z = 0.12x = (0.12)(250)$$
$$= 30\,\text{m}$$

Alternatively, the parameters could be estimated from the graphs in Figure 7.4.

To illustrate the effect of release height on the ground level, centerline concentration, it is convenient to normalize the concentration as $C(x,0,0)u/\dot{S}_0[\text{L}^{-2}]$. Presented in Figure 7.6 is normalized concentration as a function of downwind distance for release heights ranging from 0 to 200 m. For a ground-level release (i.e., for $h = 0$), the concentration is at its highest close to the source and decreases with distance. In contrast, for an elevated release the concentration is low close to the source, rises to a maximum, and then decreases. With increasing release height, the maximum concentration decreases, and the distance at which the maximum occurs increases. If σ_y is proportional to σ_z, which is approximately true under neutral and stable conditions, the location of the maximum concentration occurs at the distance where $2\sigma_z^2 = h^2$. The maximum concentration is

$$C_{\max} = \frac{2\dot{S}_0\sigma_z}{\pi h^2 e u \sigma_y} \tag{7.6}$$

Finally, the centerline concentration for a ground-level release is obtained by letting $h = 0$ in Eq. 7.5:

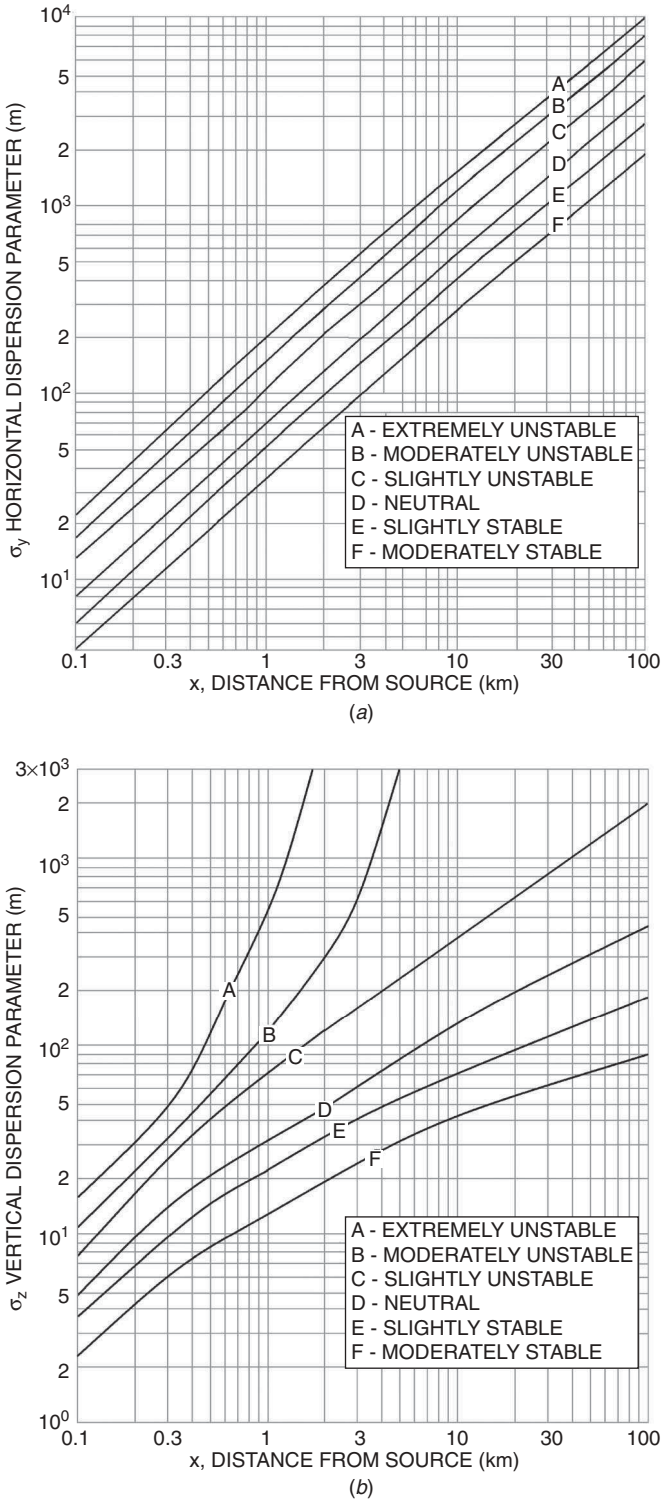

Figure 7.5 PasquillÐGiffordÐTurner curves for estimating σ_y and σ_z. (From Barr and Clements 1984.)

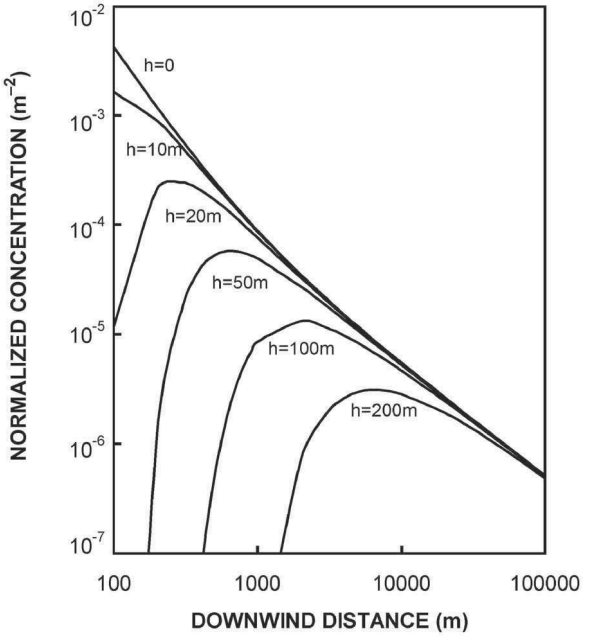

Figure 7.6 Ground-level centerline concentration as a function of downwind distance for various release heights.

$$C(x,0,0)|_{h=0} = \frac{\dot{S}_0}{\pi u \sigma_y \sigma_z} \tag{7.7}$$

The Gaussian plume model applies to gases and to particulate matter smaller than about 10 μm and is based on constant wind speed and stability conditions between the source and the receptor. Also, as discussed later in this chapter, it is also valid for time periods on the order of tens of minutes. When applied to relatively flat terrain, it can yield predictions that are within a factor of 2 of measurements.

▶ Example 7.3

The paper mill in Example 7.1 continuously releases hydrogen sulfide, H_2S, from the stack at a rate of 50 mg/s. The wind is blowing in the direction of a nearby community that is located 250 m downwind from the release (i.e., $y = 0$) at 2.5 m/s. Use the Gaussian plume model to calculate the concentration of H_2S in the community.

Solution Substitute into the equation for centerline concentration (Eq. 7.5):

$$\begin{aligned} C(250\,\text{m},0,0) &= \frac{\dot{S}_0}{\pi u \sigma_y \sigma_z} \exp\left(-\frac{h^2}{2\sigma_z^2}\right) = \frac{50\,\text{mg/s}}{\pi(25\,\text{mg/s})(39.6\,\text{m})(30\,\text{m})} \exp\left[-\frac{25^2}{(2)(30^2)}\right] \\ &= (5.36\,\mu\text{g/m}^3)\exp(-0.347) \\ &= 3.97\,\mu\text{g/m}^3 \end{aligned}$$

7.3.2 Long-Term Averages

The typical atmospheric transport problem in a risk assessment application requires calculation of the concentration at a Þxed receptor location (or locations) resulting from either an accidental release of short duration (on the order of hours) or a routine release of long duration (on the order of years). However, when applied to a Þxed receptor over an extended period of time, the Gaussian plume model with Briggs or PasquillÐGiffordÐTurner dispersion parameters is valid only for time periods on the order of tens of minutes. This is because (1) the dispersion parameters are based on observations made over this time span, (2) wind directions tend to ßuctuate, causing the position of a Þxed receptor relative to the centerline of a plume to ßuctuate as well, and (3) even in the absence of ßuctuations in wind direction, plumes meander due to large-scale eddies. Consequently, the Gaussian plume model overestimates concentrations for releases longer than about an hour. Two averaging techniques are presented here for longer time periods. Both of these require hourly measurements of wind direction, wind speed, and temperature gradient (or stability class).

7.3.2.1 Summation of Gaussian Plumes In the **summation of Gaussian plumes method**, the Gaussian plume model is used to calculate the concentration for each hourly period; and these hourly concentrations at each location are averaged over the entire exposure period. The average concentration is given by

$$\bar{C} = \frac{\sum_{n=1}^{N} C_{g,n}}{N} \tag{7.8}$$

where $C_{g,n}$ is the Gaussian plume concentration for the nth set of hourly meteorological measurements and N is the total number of measurements. In prospective analyses performed to support permitting or licensing applications, background meteorological data are collected for at least a year (i.e., $N = 8760$, corresponding to 365 days of hourly measurements) and preferably longer. If Eq. 7.8 is applied to each node on a grid surrounding the release point, a map of concentration isopleths, such as illustrated in Figure 7.7, can be produced.

7.3.2.2 Sector-Averaged Approximation The **sector-averaged approximation** (NRC 1977), which is not as computationally intensive as the summation of Gaussian plumes, is used by the Nuclear Regulatory Commission in the licensing of nuclear power plants. The region surrounding the release is divided into 16 sectors, each centered on one of the 16 compass points (N, NNE, NE, ENE, etc.). The sector is that region enclosed by two radii separated by the angle θ from the point of release and the arc between the radii (Figure 7.8). Based on observations for each hourly period, a single sector is identiÞed as the primary wind direction and the concentration (i.e., the sector-averaged concentration) is taken to be constant across the sector arc at a given downwind distance x.

The sector-averaged concentration is obtained by using the principle of conservation of mass and the predictions of the Gaussian plume model. The contaminant ßux leaving the sector at x is $j_{SA} = uHC_{SA}x\theta$, where j_{SA} is the sector-averaged ßux, H an arbitrary height, and C_{SA} the (constant) sector-averaged concentration at x. The ßux for a Gaussian plume is

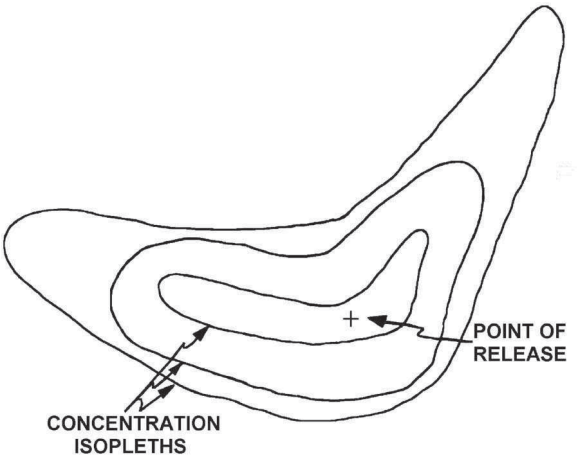

Figure 7.7 Concentration isopleths in the vicinity of a release point.

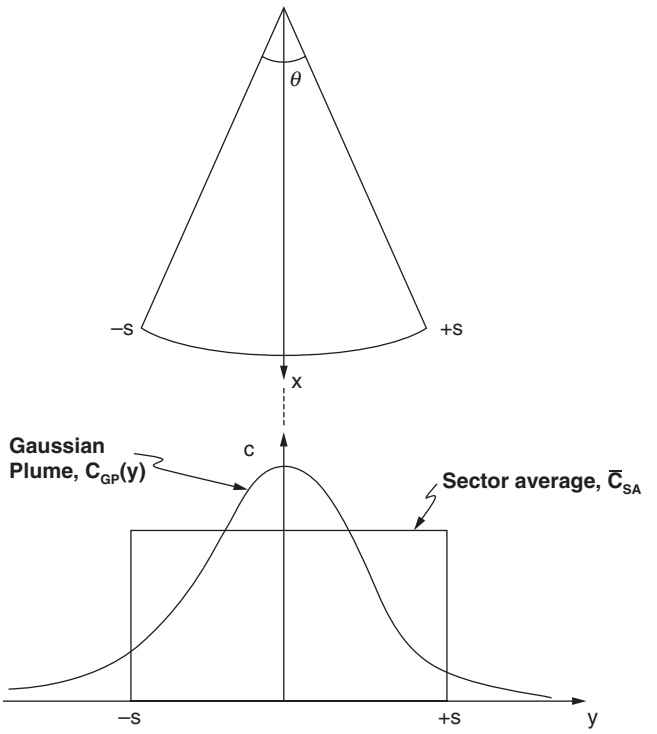

Figure 7.8 Sector-averaged approximation.

$$j_{GP} = uH\int_{-\infty}^{+\infty} C_{GP}dy$$

where C_{GP} is concentration based on the Gaussian plume approximation, conservation of mass requires these ßuxes to be equal, so

$$C_{SA} = \frac{1}{x\theta}\int_{-\infty}^{\infty} C_{GP}(y)dy \tag{7.9}$$

Substituting for C_{GP}, Eq. 7.9 becomes

$$C_{SA} = \frac{1}{x\theta}\int_{-\infty}^{\infty} \frac{\dot{S}_0}{\pi u\sigma_y\sigma_z}\exp\left(-\frac{y^2}{2\sigma_y^2}\right)\exp\left(-\frac{h^2}{2\sigma_z^2}\right)dy \tag{7.10}$$

However, because the wind blows into the given sector only part of the time, a factor, f, is multiplied by the result to account for the fraction of time during the averaging period that winds blow into the sector. This yields

$$C_{SA} = \sqrt{\frac{2}{\pi}}\frac{f\dot{S}_0}{u\sigma_z x\theta}\exp\left(-\frac{h^2}{2\sigma_z^2}\right) \tag{7.11}$$

If the 16 compass points, (i.e., E, WSW, NE, etc.) are used to tabulate hourly meteorological observations, it is convenient to deÞne a sector as a 360¡/16 = 22.5¡ slice of the region surrounding the release point. Substituting $\theta = 22.5¡/180¡\,\pi$ into Eq. 7.11 and simplifying yields

$$C_{SA} = \sqrt{\frac{2}{\pi}}\frac{8}{\pi}\frac{f\dot{S}_0}{u\sigma_z x}\exp\left(-\frac{h^2}{2\sigma_z^2}\right) \tag{7.12}$$

However, $\sqrt{2/\pi}(8/\pi) = 2.032$, so this result may be approximated by

$$C_{SA} = \frac{2f\dot{S}_0}{u\sigma_z x}e^{-h^2/2\sigma_z^2} \tag{7.13}$$

When used in conjunction with hourly meteorological data, the sector-averaged concentration is calculated by

$$\bar{C}_{SA} = \frac{2\dot{S}_0}{x}\sum_{j=A}^{F}\sum_{i=1}^{I}\frac{f_{i,j}}{u_i\sigma_{z,j}}e^{-h^2/2\sigma_{z,j}^2} \tag{7.14}$$

where I is the number of wind speed groups and $f_{i,j}$ is the fraction of time that winds blow into the given sector under stability j and with speed u_i. Hourly meteorological observations for a year or several years are processed to yield tabulated values of $f_{i,j}$, which comprise the joint frequency distribution of wind direction, wind speed, and stability class.

▶ Example 7.4

Yearly meteorological data for winds blowing from the SSW sector into the NNE sector of the plant in Example 7.1 are given in Table 7.4. Each entry is the number of hourly observations (out of 8533) in which winds were blowing into the NNE sector with speeds in a given range (represented by the midpoint of the range) and under a given stability class. The sector-averaged concentration is determined as follows. The first step is to determine the source-normalized sector-averaged concentration, $\chi_{SA}/\dot{S}_0$, using Eq. 7.14. The contribution to the total annual average from each stability class is obtained by performing the sum over wind speeds for a given value of j:

$$\frac{2}{x}\sum_{i=1}^{I}\frac{f_{i,j}}{u_i\sigma_{z,j}}\exp\left(-\frac{h^2}{2\sigma_{z,j}^2}\right)$$

The results are shown in Table 7.5. The total annual average concentration is the sum of the contributions from each stability class, which is 1.07×10^{-6} s/m^3. Multiplying this by the emission rate of 50 mg/s, the yearly sector-averaged concentration is 0.054 μg/m^3.

TABLE 7.4 Joint Frequency Distribution of Wind Speed and Atmospheric Stability for Example 7.4[a]

	Average Wind Speed (m/s)					
Stability Class	1	2.25	4	6.25	8.75	10
A	0	2	3	7	2	2
B	0	1	5	11	8	1
C	1	5	19	22	15	7
D	1	23	54	58	71	17
E	2	14	28	86	48	6
F	3	22	79	104	23	0

[a] Each entry is the number of hourly observations.

TABLE 7.5 Contribution to the Source Normalized Concentration for Each Stability Class

Stability Class	σ_z (m)	Contribution to $C/\dot{S}_0$
A	50	0.5×10^{-7}
B	30	1.17×10^{-7}
C	16	2.92×10^{-7}
D	13	6.05×10^{-7}
E	7	0.10×10^{-7}
F	3.7	$<10^{-14}$
Total (annual average)		1.07×10^{-6} s/m^3

Although the Gaussian plume model is often used to a distance of 50 km from the source, the uncertainty is quite large beyond about 10 km. Also, the model is best applied to smooth terrain. An alternative is the variable trajectory model, which takes into account spatial and temporal variations of airßow and should provide more accurate estimates of atmospheric concentrations. In such a model, individual ÒpuffsÓ are tracked over time intervals on the order of 30 minutes. However, it requires detailed data on wind direction in the vicinity of a site that are normally not available.

7.3.3 Instantaneous Emission: Gaussian Puff Model

Releases such as those that might result from an explosive accident which occur over a time period that is very short compared to the travel time from the source to the receptor can be approximated by the **Gaussian puff model.** This model is obtained by solving Eq. 7.1 for an instantaneous point source, and the solution can be obtained by replacing D in Eq. 4.14 by K, letting $\sigma_I = \sqrt{2Kt}$, and multiplying by a factor of 2 to account for total reßection of contaminant at the ground. This yields

$$C(x,y,0,t) = \frac{S_0}{\sqrt{2}\pi^{3/2}\sigma_{xI}\sigma_{yI}\sigma_{zI}} \exp\left[-\frac{(x-ut)^2}{2\sigma_{xI}^2}\right] \exp\left(-\frac{y^2}{2\sigma_{yI}^2}\right) \exp\left(-\frac{h^2}{2\sigma_{zI}^2}\right) \tag{7.15}$$

where σ_{xI}, σ_{yI}, and σ_{zI}, are dispersion parameters for an instantaneous release. Equation 7.15 gives the concentration at any given time. For purposes of risk assessment, it is necessary to integrate Eq. 7.15 over the entire exposure period to obtain the **time-integrated concentration**, $\overline{Ct}$. In Chapter 9 it is used in the calculation of contaminant dose for situations where concentration varies with time. Integrating Eq. 7.15 (Gifford 1968) yields

$$\overline{Ct}(x,y,0) = \frac{S_0}{\pi u \sigma_{yI}\sigma_{zI}} \exp\left(-\frac{y^2}{2\sigma_{yI}^2}\right) \exp\left(-\frac{h^2}{2\sigma_{zI}^2}\right) \tag{7.16}$$

It is interesting that Eq. 7.16 is very similar to the Gaussian plume model, Eq. 7.4. However, the dispersion parameters presented in Section 7.3.1 for the Gaussian plume model do not apply to an instantaneous release and are not appropriate for Eqs. 7.15 and 7.16. Islitzer and Slade (1968) suggest the values presented in Table 7.6. Since the dispersion parameters in Table 7.6 are based on sparse data, there is considerably more uncertainty in calculations based on the Gaussian puff model than those based on the Gaussian plume model.

7.3.4 Infinite Line Source

A problem that could be encountered in risk analysis is that of contaminants released from a linear source such as automobiles on a highway. For a constant emission rate, if the angle between the wind direction and the line is greater than 45¡, the concentration at some distance x downwind from the line can be estimated by (Turner 1994)

TABLE 7.6 Dispersion Parameters for Instantaneous Releases

Dispersion Parameter	Stability	Equation (x in meters)
σ_{yI} (m)	Unstable	$0.14x^{0.92}$
	Neutral	$0.06x^{0.92}$
	Very stable	$0.02x^{0.89}$
σ_{zI} (m)	Unstable	$0.53x^{0.73}$
	Neutral	$0.15x^{0.70}$
	Very stable	$0.05x^{0.61}$

Source: Islitzer and Slade 1968.

$$C(x,y,0) = \frac{2\tilde{\dot{S}}_0}{\sqrt{2\pi}\sigma_z u} e^{-h^2/2\sigma_{zI}^2} \tag{7.17}$$

where $\tilde{\dot{S}}_0$ is the emission rate per unit length [M/(LT)].

7.4 OTHER CONSIDERATIONS

The plume models presented above incorporate the principal factorsÑwind direction, wind speed, atmospheric stability, and release heightÑthat typically dominate contaminant concentrations in air. However, other factors can be important in certain situations. Although many of these other factors are complex and difÞcult to model accurately, simple expressions are available for making estimates of their impact on atmospheric concentrations. These simple expressions are given below.

7.4.1 Effective Release Height and Plume Rise

Because ground-level concentration is proportional to $\exp(-h^2)$, it is particularly sensitive to the height of a release. However, the physical height of a release is often not the appropriate height to use in calculations. For example, in Figure 7.9 buoyancy and momentum of the exhaust gases (termed **plume rise**) cause the effective height of the release to differ from the physical height of the stack. **Effective release height**, h_e, is given by

$$h_e = h_s + h_{\text{pr}} \tag{7.18}$$

where h_s is physical release height and h_{pr} is plume rise. Although not shown in Figure 7.9, building wake can also affect effective release height.

A number of empirical equations of varying levels of complexity are available for predicting plume rise (Briggs 1969; Turner 1994). Presented here are relatively simple expressions that treat the effects of momentum and buoyancy separately (Turner 1994). In practice, both are calculated and the larger of the two is used in Eq. 7.18. Plume rise due to momentum can be estimated by

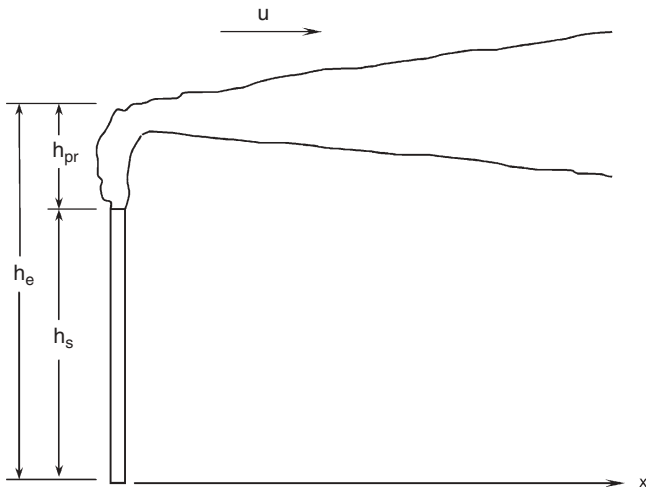

Figure 7.9 Effect of plume rise and receptor location on effective release height.

$$h_{\text{pr}} = \frac{3D_s v_s}{u} \tag{7.19}$$

where D_s is stack diameter [L] and v_s is plume exit velocity [L/T]. More accurate estimates require additional parameters, such as efßuent and ambient temperature and temperature gradient. The calculation of buoyant rise is based on the buoyancy ßux,

$$F = \frac{g v_s D_s^2 \Delta T}{4 T_s} \tag{7.20}$$

where F is buoyancy ßux [L^4/T^3], g is acceleration due to gravity [L/T^2], ΔT is the difference between stack gas and ambient temperature (¡K), and T_s is stack gas temperature (¡K). Plume rise is then calculated from

$$h_f = \frac{1.6 F^{1/3} x_F^{2/3}}{u} \tag{7.21}$$

For unstable and neutral conditions, the parameter x_F is given by

$$x_F = \begin{cases} 49F^{5/8} & F < 55 \\ 119F^{2/5} & F \geq 55 \end{cases} \tag{7.22}$$

where x_F is in meters and F is in m^4/s^3. If the receptor is not at ground level, the general Gaussian plume solution (Eq. 7.3) must be used with the receptor height z.

7.4.2 Building Wake

Building wake refers to the flow patterns and turbulence that develop as air passes around and over a building. This is important because it can increase near-field dispersion. The preferred methods for predicting the effects on contaminant concentrations are field measurements or wind tunnel simulations. However, these approaches are rarely practical, and in their absence, empirical adjustments to the dispersion parameters are made. Gifford (1968) gives

$$\sigma_y^* = \left(\sigma_y^2 + \frac{cA}{\pi} \right)^{1/2} \tag{7.23}$$

$$\sigma_z^* = \left(\sigma_z^2 + \frac{cA}{\pi} \right)^{1/2} \tag{7.24}$$

where σ_y^* and σ_z^* are modified dispersion parameters, A is the cross-sectional area of the building normal to the wind direction, and c is a constant between 0.5 and 0.67. Since the dispersion parameters increase with downwind distance, the adjustment term cA/π decreases in importance with distance from the release. Building wake can also affect release height. A release that is less than the height of the building is captured in the wake and is effectively released at ground level. As the release height increases above the building height, the effect of building wake is difficult to predict. Brenk et al. (1983) recommend the following: For a release greater than 2.5 times the building height, the release is unaffected by building wake; for a release below the height of the building, a ground-level release is assumed; for a release between the building height and 2.5 times the building height, a ground-level release is assumed for part of the time, and an elevated release is assumed for the remainder of the time.

7.4.3 Release with Inversion Aloft

As noted in Section 7.2 and illustrated in Figure 7.3*d*, an inversion aloft (i.e., above the effective release height) can result in high contaminant concentrations for receptors at ground level. At a sufficient downwind distance, it can be assumed that the concentration in the region between the ground and the inversion layer is uniform (Turner 1994). The concentration is then given by

$$C(x,y,0) = \frac{\dot{S}_0}{\sqrt{2\pi}\sigma_y u L} \exp\left(-\frac{y^2}{2\sigma_{yI}^2} \right) \tag{7.25}$$

where L is the mixing height. This equation is applicable for $x \geq 2X_L$, where X_L is the downwind distance at which the stable layer begins to limit upward dispersion of the plume. It is determined from

$$\sigma_z(X_L) = 0.47(L - h_e) \tag{7.26}$$

■ Mixing Height

A series of temperature proÞles during the day after a clear night is shown in Figure 7.10. During the night, radiation cooling causes the temperature of the air at EarthÕs surface to be cooler than that higher up. During the day, solar heating of EarthÕs surface results in an unstable well-mixed layer of air adjacent to the ground. As the day progresses, the thickness of this mixed layerÑthe **mixing height**Ñincreases. For purposes of contaminant transport modeling, the mixing height is the elevation at which the dry adiabatic proÞle (based on the maximum ground-level temperature in the afternoon) intersects the actual morning temperature proÞle.

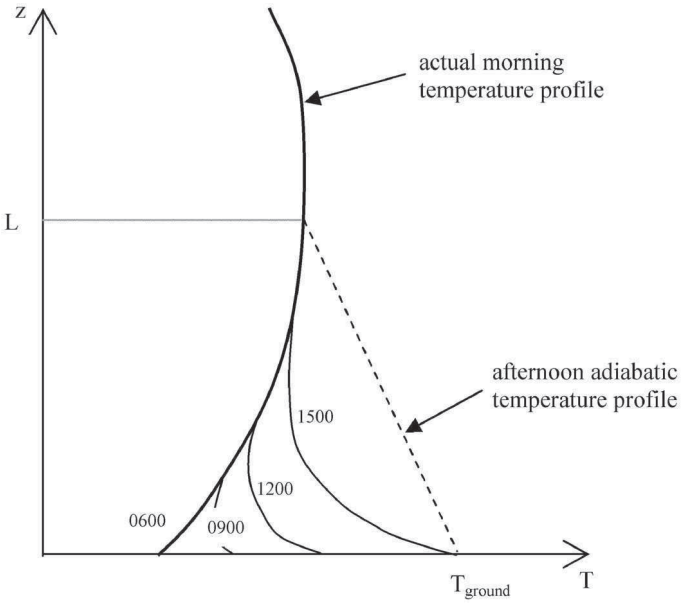

Figure 7.10 Mixing height.

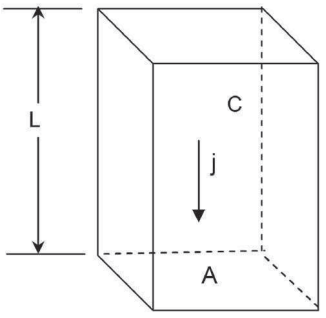

Figure 7.11 Conceptual model for approximating dry deposition and precipitation scavenging as Þrst-order processes.

If the mixing height is not known, it can be approximated by requiring the total mass of contaminant in the mixing volume (LA in Figure 7.11) at a given location to be equal to the total mass of contaminant in the Gaussian plume. For a ground-level release,

$$C_{gl}LA = \int_0^\infty C_{gl}e^{-z^2/2\sigma_z^2}dzA$$

where C_{gl} is ground-level concentration. Dividing both sides by $C_{gl}A$ and integrating gives us

$$L = \int_0^\infty e^{-z^2/2\sigma_z^2}dz = \sqrt{\frac{\pi}{2}}\sigma_z$$

For an elevated release, Slinn (1984) gives the following:

$$L \approx \sqrt{\frac{\pi}{2}}e^{-h^2/2\sigma_z^2}\sigma_z$$

which is valid provided $L >> h_e$.

7.4.4 Nonconservative Processes

The concentration of atmospheric contaminants can be affected significantly by nonconservative processes. Some contaminants are generated in the atmosphere through chemical reactions. For example, photochemical smog and ozone are produced from atmospheric reactions in the presence of sunlight, and hydrocarbon vapors present in the atmosphere increase the concentrations of these species. Removal mechanisms that can be important in the atmosphere are dry deposition, precipitation scavenging, degradation, and radioactive decay. **Dry deposition** refers either to gravitational settling of particulate matter or the attachment of gaseous or particulate contaminants to surfaces. **Precipitation scavenging** (also called **wet deposition**) refers to the incorporation of atmospheric contaminants into precipitation which is subsequently deposited onto the ground, vegetation, or surface water. None of these nonconservative processes is included in the Gaussian plume model.

Contaminant generation is far too complex for consideration here, as are rigorous treatments of removal mechanisms. However, by treating the removal mechanisms as first-order processes, their effect on contaminant concentration can be approximated by the following:

$$C = C^*e^{-k(x/u)} \tag{7.27}$$

where C is contaminant concentration with removal processes taken into consideration, C^* is concentration based on one of the models presented above, and k is a first-order rate constant for the removal mechanism of interest. For degradation, the rate constant is the relevant chemical reaction rate constant, and for radioactive decay it is the radioactive decay constant. Rate constants for dry deposition and precipitation scavenging can be developed through the conceptual model presented in Figure 7.11. The volume defined by the area A and the mixing height L is a

homogeneous compartment in which the concentration is the same as that at ground level. The removal rate to the ground due either to dry deposition or precipitation scavenging is jA, where j is the contaminant flux to the ground. For dry deposition, $j = v_{d,D}C$ where $v_{d,D}$ is dry deposition velocity. Deposition velocity is discussed further in Chapter 8. Precipitation scavenging can be estimated in either of two ways. The first is analogous to dry deposition, i.e., $j = v_{d,W}C$, where $v_{d,W}$ is wet deposition velocity. The other is $j = \dot{R}\omega_v C$, where $\dot{R}$ is the rainfall rate [L_{water}/T] and ω_v is the volumetric washout factor [L^3_{water}/L^3_{air}]. Volumetric washout factor, ω_v, is a partition coefficient for contaminant partitioning between the air and precipitation, or $\omega_v = \frac{C_w}{C_a}$, where C_w is the concentration in precipitation and C_a is the concentration in the surrounding. The volumetric washout factor is equal to the inverse of the dimensionless Henry's law constant. Values of volumetric washout factors and dimensionless Henry's law constants for selected contaminants are given in Table 7.7.

TABLE 7.7 Volumetric Washout Factors and Henry's Law Constants for Selected Contaminants

Contaminant	ω_v (Peterson 1983) (m^3_{air}/m^3_{water})	H (RAIS 2005) (m^3_{water}/m^3_{air})
Aldrin		1.8×10^{-3}
Benzene	4.4	2.3×10^{-1}
Carbon tetrachloride	0.88	1.1
Chlordane		2.0×10^{-3}
Chloroform	6.7	1.5×10^{-1}
Cl_2		1.0
DDD		2.7×10^{-4}
DDE		1.7×10^{-3}
DDT		3.4×10^{-4}
Dieldrin		4.1×10^{-4}
Ethylene dibromide		2.7×10^{-3}
Heptachlor		1.2×10^{-2}
Heptachlor epoxide		8.6×10^{-4}
Hexachlorocyclohexane (lindane)		2.1×10^{-4}
Hydrogen sulfide		3.6×10^{-1}
Iodine		
Elemental	$0.2–2.5 \times 10^6$	
Methyl (alkyl)	$0.3–4.1 \times 10^3$	
Particulate	$2.5–12.4 \times 10^5$	
Mercury (elemental)		1.0
Methylene chloride		1.3×10^{-1}
PAH (benz[a]pyrene)		1.9×10^{-5}
PCB (Aroclor 1260)		1.4×10^{-2}
SO_2	$0.5–8.8 \times 10^5$	
Tetrachloroethylene (PCE)		7.2×10^{-1}
Trichloroethylene (TCE)		4.0×10^{-1}
Vinyl chloride		1.1
Water vapor		
10¡C	9×10^4	
20¡C	5×10^4	

The loss rate per unit volume (i.e., the destruction term in the contaminant transport equation) is

$$d = \frac{jA}{LA} = \frac{j}{L} \tag{7.28}$$

Thus, for dry deposition, the loss-rate density is

$$d_d = \frac{v_d}{L} C \tag{7.29}$$

and for precipitation scavenging, the loss-rate density is

$$d_{\text{ps}} = \frac{\dot{R}\omega_v}{L} C \tag{7.30}$$

Thus, the rate constants are

$$k_d = \frac{v_d}{\text{L}} \tag{7.31}$$

$$k_{\text{ps}} = \frac{\dot{R}\omega_v}{\text{L}} \tag{7.32}$$

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PROBLEMS

7.1 Draw a graph analogous to Figure 7.2 showing the relative temperatures of a parcel of air and the surrounding ambient air when the temperature of the surrounding air decreases more rapidly than the adiabatic lapse rate. Use the graph to explain why vertical motions are enhanced in this situation.

7.2 Using the temperature data that follow, determine the stability class as a function of height (25, 75, 125, 225, and 275 m) based on the NRC criteria given in Table 7.2.

Elevation (m)	Temperature (¡C)	Elevation (m)	Temperature (¡C)
0	20	200	17.2
50	19.1	250	17.2
100	18.2	300	17.7
150	17.7		

7.3 The emission rate of a contaminant to the atmosphere under normal operating conditions of an incinerator is 8 g/s. The effective release height is 50 m and the wind speed is 2 m/s. Under slightly unstable conditions, determine the following:

(a) Maximum short-term concentration at a distance of 500 m from the release.

(b) Distance at which the centerline concentration is a maximum and the concentration at that location.

(c) Concentration at a point 3 km downwind and 200 m off the downwind axis.

7.4 Consider the conditions in Example 7.3. The odor threshold for H_2S is 0.1 mg/m^3. What stack height is required to reduce the maximum centerline H_2S concentration to the odor threshold?

7.5 Consider conditions in which $\sigma_y = \alpha\sigma_z$, where α is a constant.

(a) Using the Gaussian plume model, show that the maximum centerline concentration occurs at the distance x^* such that $2\sigma_z^2 = h^2$. (Do not use the curve Þts in Table 7.3 in this derivation.)

(b) Using the graphs in Figure 7.4, determine x^* for a 60-m stack under B&E stabilities.

7.6 A power plant burning coal emits 120 g of selenium per day. A residential neighborhood is located 400 m northeast of the 50-m stacks of the power plant. The wind blows into the northeast sector approximately 15% of the time. For a wind speed of 1.5 m/s and D stability, what is the sector-averaged concentration of selenium at the neighborhood?

7.7 The emission rate of PCE to the atmosphere from a large dry cleaning facility is 1500 kg per year. A residence is located 1000 m ENE from the facility. The table that follows contains approximate meteorological data for the ENE direction.

Fraction of Year	Wind Speed (m/s)	Stability
0.04	1.5	D
0.08	3	B

Neglecting building wake effects, estimate the average atmospheric concentration at the residence. The effective release height is at ground level.

7.8 The meteorological data in Table 7.8 are for the NE sector (i.e., winds blowing from NE to SW) in the vicinity of a coal-Þred power plant. Given

TABLE 7.8 Joint Frequency Distribution of Wind Speed and Atmospheric Stability for Problem 7.8[a]

Stability Class	Average Wind Speed (m/s)				
	1	3	5	7	9
A	59	125	21	2	0
B	11	65	55	3	0
C	19	136	161	34	1
D	14	189	225	46	7
E	8	84	181	10	0
F	3	14	62	7	0

[a] Each entry is the number of hourly observations for winds blowing into the SW sector out of a total of 16,627 observations.

are the number of hourly observations (out of a total of 16,627) in which winds were blowing into the SW sector with speeds in a given class (represented by the average) and under the given stability condition. The effective release height is 30 m. Determine the long-term source-normalized sector-averaged concentration (i.e., $C_{SA}/\dot{S}_0$) at a distance of 500 m from the source. (Do the sum over wind speeds Þrst and put the results in a table that shows the contribution for each stability class.)

7.9 An explosion in a chemical processing plant results in the release of 200 kg of a toxic volatile organic compound into the atmosphere over a short period of time. Calculate the time-integrated concentration at the plant boundary, which is located 200 m from the site of the explosion. The wind speed is 3 m/s, the temperature gradient is –1.3¡C per 100 m, and the effective release height is 25 m.

7.10 A highway has a trafÞc ßow of 15,000 vehicles per hour emitting an average of 2 g/mile of NO_x. If the wind speed is 1.5 m/s, calculate the NO_x concentration 100 m downwind from the highway under moderately unstable conditions.

7.11 Repeat Problem 7.3(c) for an inversion layer at 75 m.

7.12 A plume is emitted from a 1-m-diameter stack at a temperature of 325¡C and a velocity of 30 m/s. The ambient temperature is 20¡C and the wind speed is 2 m/s.

(a) What is the plume rise due solely to buoyancy?

(b) What is the plume rise due solely to momentum?

7.13 Write a subroutine or function (using Visual Basic in Excel) that is capable of computing ground-level contaminant concentrations using the Gaussian plume model with Briggs equations (Table 7.3) for dispersion parameters. The arguments passed to the subroutine or function should include the emission rate, effective release height, downwind distance, cross-wind distance, wind speed, and stability class. Check the subroutine with a manual calculation using x = 500 m, y = 25 m, u = 2 m/s, h = 60 m, C stability, and $\dot{S}_0$ = 0.2 kg/s.

7.14 **(a)** Using the subroutine or function from Problem 7.13, compute the source-normalized concentration, $C/\dot{S}_0$, as a function of downwind distance (100 m – 10,000 m), for the following: $h = 60$ m, $u = 2$ m/s, B&E stabilities, and $y = 0$.

(b) Repeat part (a) for a ground-level release.

(c) Make a log–log plot of source normalized concentration, $C/\dot{S}_0$ (no more then six decades) vs. distance. Discuss the implications of the results in parts (a) and (b) with respect to release height and stability. Put all of the plots on a single graph.

(d) From the graphs in part (c), determine the location at which the maximum ground-level concentration occurs. Compare to values determined in Problem 7.5(b).

7.15 Consider a Chernobyl-like accident at a nuclear power plant, i.e., the release of a large amount of radioactivity to the atmosphere. This problem focuses on ^{138}Xe ($t_{½} = 0.29$ h), which is responsible for a significant fraction of the dose to an exposed population during the first few hours of the accident. The inventory of ^{138}Xe is 1.2×10^{18} Bq (32×10^6 Ci), all of which is assumed to be released over a period of 30 minutes; the release height is 30 m; the wind speed is 1.5 m/s; and the atmospheric stability is neutral. Use the Gaussian plume model to estimate concentration vs. time at a distance of 2000 m for a 2-hour period following the release. (*Hint*: Approximate the release as a finite step.)

8 Food Chain Transport

8.1 INTRODUCTION

Consumption of contaminated food products such as produce, meat, milk, fish, and shellfish may be significant pathways of human exposures to contaminants. These pathways may be relatively simple, such as ingestion of fish contaminated as a result of a contaminated surface water body, or they may involve many compartments, such as air/water → soil → plants → animals → milk. The food chain pathways and food chain compartments addressed here are indicated in boldface type in Figure 8.1.

The principal source of food on Earth is photosynthesis in plants. This food is consumed by herbivores, which in turn are consumed by carnivores, which in turn are consumed by other carnivores. Such a path of food consumption is a **food chain** (Figure 8.2), and each link in the chain is a **trophic level**. The plants at the lowest trophic level are called **primary producers**, the herbivores at the next level are **primary consumers**, the carnivores at the next level are **secondary consumers**, and carnivores at higher levels are **tertiary consumers**. Although not important in the context of human health risk assessment, the bacteria responsible for decay of organisms following death comprise a trophic level known as **decomposers**, which completes the chain.

Food chain transport involves determination of contaminant concentrations at various points in the food chain. In human health risk assessments, the final endpoint is typically determined by the type of food raised and consumed in the contaminated region. Although physical processes usually control the initial release and dispersion of a contaminant, biological processes become important in the food chain. In addition, ecological risk assessments may have as an endpoint the level of contamination of a species of interest, such as protected plants or animals.

Figure 8.3 is a representation of a biological organism as a homogeneous, nonadvective environmental compartment, as discussed in Section 2.4.3.1. Contaminant may enter the organism from contaminated environmental media in which the organism lives; for example, a fish may take up contaminants from the water in which it lives. Contaminant may also enter the organism from ingestion of contaminated food; for example, a bird of prey may consume contaminated fish. Contaminant may be eliminated by the organism in feces, urine, and expired air. In addition, the amount of contaminant within the organism may be decreased by a variety of internal metabolic processes that degrade or transform the contaminant. The term

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

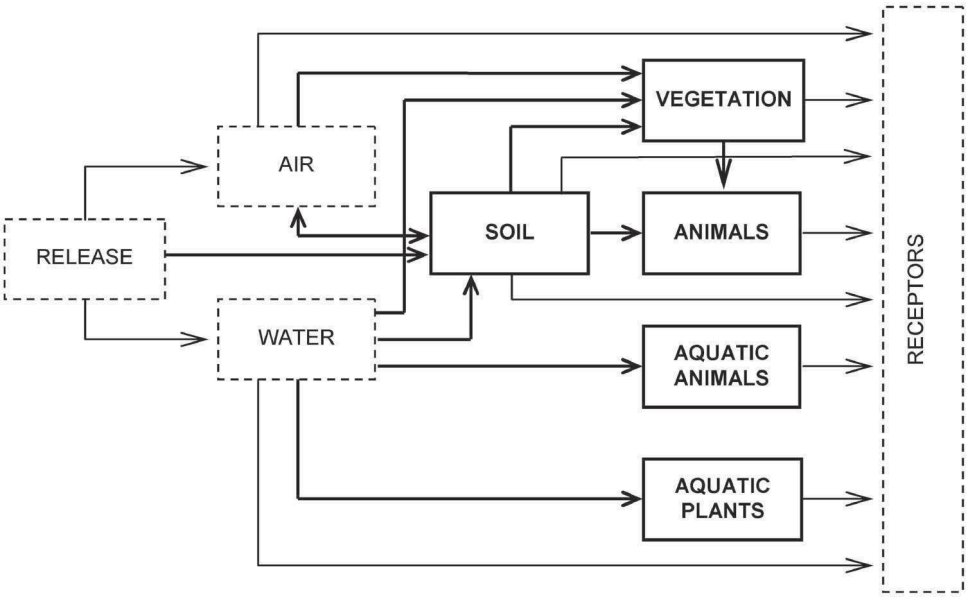

Figure 8.1 Principal food chain compartments and transport pathways. Pathways and compartments in boldface type are addressed in this chapter.

Figure 8.2 Simple food chain.

bioaccumulation refers to the general process by which a contaminant concentrates in an organism or in certain tissues in the organism due to exposure to contaminated media and/or ingestion of contaminated food (EPA 1991). Another term, **bioconcentration** (EPA 1991), refers to the process by which a contaminant concentrates in an organism due to exposure to contaminated media. Initially, this term was used to describe processes in aquatic animals, so the process moved the contaminant from water through gills or epithelial tissue into animals such as fish.

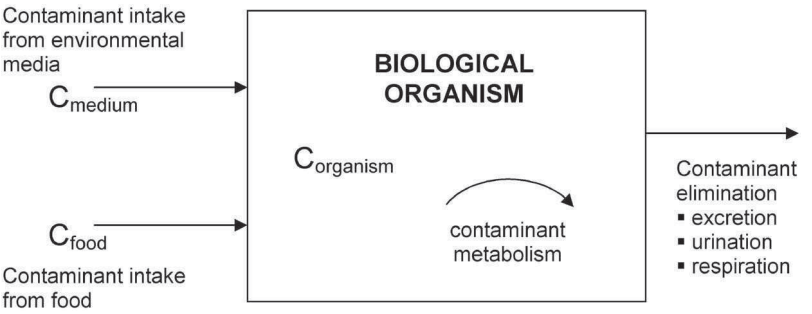

Figure 8.3 Biological organism modeled as a nonadvective compartment to illustrate bioaccumulation, dilution ($C_{\text{orgamism}}/C_{\text{food}} < 1$), bioconcentration ($C_{\text{orgamism}}/C_{\text{medium}} > 1$), and biomagnification ($C_{\text{orgamism}}/C_{\text{food}} > 1$).

The term *bioconcentration* has been extended to terrestrial organisms and implies the increase of a contaminant in the organism to a concentration higher than that in the environmental medium which provided the contaminant; in the context of Figure 8.3, $C_{\text{organism}} > C_{\text{medium}}$. Bioaccumulation at successive trophic levels in a food chain can lead to contaminant concentrations in secondary and tertiary consumers being orders of magnitude higher than those in producers. This is referred to as **biomagnification** (EPA 1991). This potential for concentration of contaminants by living organisms contrasts sharply to the atmospheric, surface water, and groundwater compartments considered so far, in which contaminant concentrations usually decline due to dilution, degradation, and dispersion.

A classic example of biomagnification is DDT transport through aquatic ecosystems, where concentrations in the tertiary consumers (ducks, ospreys, hawks, etc.) at the top of the food chain were as much as four orders of magnitude higher than in the lake sediment where the primary producers draw their nutrients. Contaminants that are lipid soluble, for example, may build up in fatty tissues of an organism and attain concentrations higher than those in the food that is consumed. Lipid solubility is quantified through the octanol–water partition coefficient, K_{ow}, introduced in Chapter 5. Values of K_{ow} greater than about 4 or 5 (Suter 1993) indicate a significant bioaccumulation potential. This occurs because the contaminants accumulate successively from sediment to benthic organisms, from benthic organisms to fish, and from fish to birds.

Contaminant transport through food chain pathways can involve a variety of complex physical, chemical, and biological processes. Even though the basic mechanisms for some of these processes are known, the systems are so complex and depend on so many variables that integrated theoretical models either do not exist or require data that are not readily available. Consequently, food chain transport models used in risk assessments are largely empirical, and they rely on the base of contaminant and species-specific laboratory and field data that exist.

For modeling purposes, food chain transport data are almost universally expressed in terms of one of the nonadvective homogeneous compartmental models introduced in Chapter 2: either (1) the instantaneous partitioning model or (2) the constant-source first-order removal model. Presented in this chapter are the models that are commonly used to predict contaminant concentrations in the soil, vegetation, and animal compartments in Figure 8.1.

8.2 CONCENTRATION IN SOIL

8.2.1 Conceptual Model

Since a major pathway for food contamination involves uptake from contaminated soil by plants, determination of soil concentrations is often a first step in a food chain transport assessment. The major deposition and removal processes are illustrated in Figure 8.4. Deposition pathways include dry or wet deposition from the atmosphere, direct deposition from a leak or spill, and irrigation with contaminated water. Removal can occur by degradation, decay, resuspension to the atmosphere, runoff from the surface, and leaching to deeper soil. For modeling purposes, contaminants are considered to be deposited on the soil surface and to become mixed uniformly with the underlying soil. The level of contamination on the surface is expressed as the **areal concentration** (or areal contamination density), $\chi[M(c)/L^2]$, which is contaminant mass per unit area. Conversion of areal concentration to concentration in the underlying soil is accomplished by considering the contaminant to become uniformly mixed to the **depth of the root zone**, which is typically taken to be 0.15 m in the absence of site-specific information. With reference to Figure 8.4, the concentration in the soil is the total amount of contaminant on the surface, which is χA, divided by the mass of soil, which is $\rho_B A Z_R$. This yields

$$C_s = \frac{\chi}{\rho_B Z_R} \tag{8.1}$$

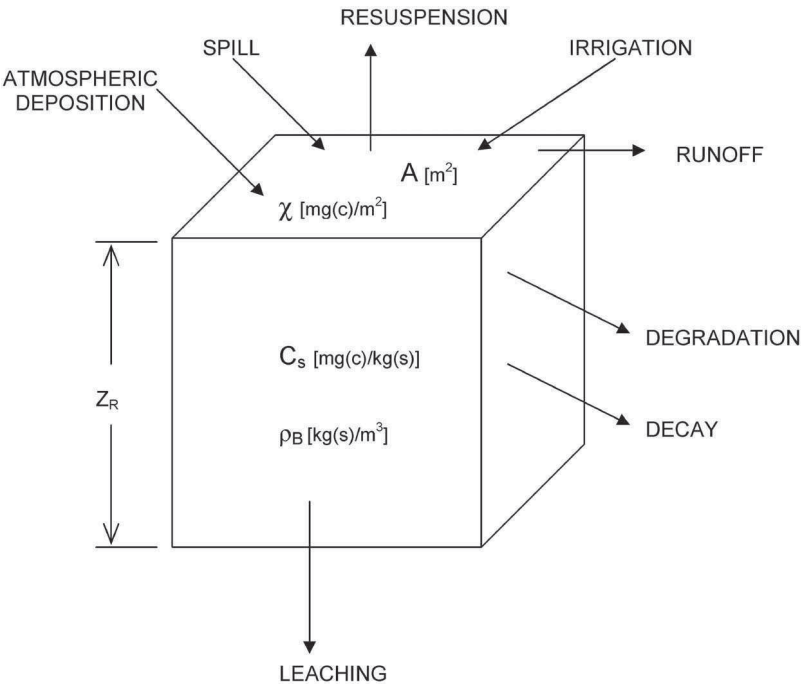

Figure 8.4 Contaminant deposition and removal processes for soil.

where C_S is soil concentration [M(c)/M(s)], ρ_B is bulk soil density [M(s)/L^3], and Z_R is the depth of the root zone [L].

▶ **Example 8.1**

Levels of ^{137}Cs in soil as a result of the Chernobyl accident are often given in terms of areal concentration. In the region of highest contamination, areal concentrations of ^{137}Cs can exceed 1.5 TBq/km^2. If the contamination is well mixed throughout the root zone, what is the equivalent soil concentration? The bulk soil density is 1700 kg(s)/m^3 and the depth of the root zone is 20 cm.

Solution This is a straightforward application of Eq. 8.1:

$$C_s = \frac{\chi}{\rho_B Z_R} = \frac{(1.5\times10^{12}\ \text{Bq/km}^2)(10^{-6}\ \text{km}^2/\text{m}^2)}{[1700\,\text{kg(s)/m}^3](0.2\,\text{m})} = 4.4\times10^3\ \text{Bq/kg(s)}$$

Predictions of soil concentration as a result of the processes mentioned above are made using the constant-source (i.e., constant deposition) first-order removal model. When applied to soil, Eq. 2.24 takes the form

$$\frac{dC_s}{dt} = \frac{j_d}{\rho_B Z_R} - k_s C_s \tag{8.2}$$

where j_d is deposition flux to the surface [M(c)/L^{2}T] and k_s is a first-order rate constant for removal from soil. Depending on the contaminant and the soil, the rate constant may actually be the sum of rate constants for degradation, decay, leaching, resuspension, and so on. The solution to Eq. 8.2 is

$$C_s(t) = \frac{j_d}{k_s \rho_B Z_R}[1-\exp(-k_s t_{s,e})] \tag{8.3}$$

where $t_{s,e}$ is the soil exposure time. Equation 8.3 is typically used for long-term exposures such as those due to routine atmospheric releases from a facility. The limiting (i.e., steady-state) concentration is

$$C_s = \frac{j_d}{k_s \rho_B Z_R} \tag{8.4}$$

This is the concentration resulting from a continuous long-term release. For exposures resulting from short-term accidental releases, Eq. 8.3 reduces to

$$C_s = \frac{j_d}{\rho_B Z_R} t_{s,e} \tag{8.5}$$

Applying Eq. 8.4 or Eq. 8.5[1] to a specific scenario requires determination of the deposition flux and removal-rate constants.

8.2.2 Atmospheric Deposition

Dry and wet surface deposition fluxes are calculated from

$$j_d = (1 - f_v) v_d C_a \tag{8.6}$$

where C_a is the contaminant concentration in air, f_v the fraction of the contaminant intercepted by vegetation (taken as 0.25 in the absence of site-specific data), and v_d the deposition velocity.

The **deposition velocity** is an empirical quantity that varies widely depending on the physical and chemical form of the contaminant, the type of surface, and meteorological conditions, especially precipitation rate. Dry deposition velocities for contaminants typically range between 10^{-3} and 10 cm/s, although they can be as high as 100 cm/s for particles (Sehmel 1984). For screening purposes, IAEA (2001) recommends 1 cm/s as a conservative value to account for both wet and dry deposition.

► Example 8.2

Selenium is a trace constituent in coal that is released in the gaseous effluent from coal-fired power plants. The average atmospheric concentration of selenium at a certain location downwind from a power plant is 5×10^{-6} mg(c)/m^3, and the deposition velocity is 0.2 m/s. What is the selenium flux to the ground at this location?

Solution The deposition flux is calculated from Eq. 8.6,

$$\begin{aligned} j_d &= (1 - f_v) v_d C_a \\ &= (1 - 0.25)(0.2\,\text{m/s})[5 \times 10^{-6}\,\text{mg(c)/m}^3](3600\,\text{s/h})(24\,\text{h/d})(365\,\text{d/yr}) \\ &= 24\,\text{mg(c)/(m}^2 \cdot \text{yr)} \end{aligned}$$

8.2.3 Irrigation Deposition

Deposition by irrigation can be calculated from

$$j_d = \dot{I} C_w \tag{8.7}$$

where $\dot{I}$ is the irrigation rate [L/T] and C_w is the contaminant concentration in the irrigation water. Irrigation rates vary depending on the water needs of the crop and can range from 0.23 to 1.4 m/yr (Peterson 1983). The default agricultural irrigation

[1] Equation 8.5 is obtained from the first two terms of the Taylor's series expansion of Eq. 8.3. That is, if $k_s t_{s,e} << 1$, then $\exp(-k_s t_{s,e}) \approx 1 - k_s t_{s,e}$ and

$$C_s(t) = \frac{j_d}{k_s \rho_B Z_R}(1 - e^{-k_s t_{s,e}}) \approx \frac{j_d}{k_s \rho_B Z_R}(1 - 1 + k_s t) = \frac{j_d}{\rho_B Z_R} t_{s,e}$$

rate used by the Nuclear Regulatory Commission is 0.76 m/yr (NRC 1992). Deposition due to a spill can be considered a special case of deposition by irrigation, in which the deposition occurs for a very short period of time (i.e., the time over which the spill occurs).

▶ **Example 8.3**

A crop of lettuce is irrigated from a well at a rate of 0.25 m/yr. The water in the well is contaminated with TCE at a concentration of 300 mg/L. What is the flux of TCE to soil?

Solution The irrigation flux is calculated from Eq. 8.7:

$$\begin{aligned} j_d &= \dot{I} C_w = 0.25\,\text{m/yr}(300\,\text{mg/L})(1000\,\text{L/m}^3) \\ &= 7.5 \times 10^4\,\text{mg/(m}^2 \cdot \text{yr)} \end{aligned}$$

8.2.4 Atmospheric Resuspension

Surface soils can be resuspended into the atmosphere through the action of wind and mechanical disturbance. Resuspension is quantified through the **resuspension factor**, κ [L^{-1}], which is defined by

$$\kappa = \frac{k_{s,r}}{v_d} \tag{8.8}$$

where $k_{s,r}$ is the resuspension rate constant. Physically, the resuspension factor is the fraction of contaminant resuspended per meter in the direction of the wind. It is affected by many factors, including the type of soil, the atmospheric conditions, and the level of mechanical disturbance (e.g., plowing, vehicular traffic, gardening); and it ranges from 10^{-5} to $10^{-10}\,\text{m}^{-1}$, with typical values being on the order of $10^{-8}\,\text{m}^{-1}$ (Sehmel 1984; NRC 1992).

▶ **Example 8.4**

In Example 8.2, the deposition flux of selenium from the atmosphere as a result of a coal-fired power plant was calculated to be $24\,\text{mg(c)/m}^2 \cdot \text{yr}$. The resuspension factor for selenium is $10^{-7}\,\text{m}^{-1}$, the bulk soil density is $1600\,\text{kg(s)/m}^3$, and the depth of the root zone is 0.15 m. Plot the soil concentration as a function of time.

Solution The soil concentration is calculated from Eq. 8.3:

$$C_s(t) = \frac{j_d}{k_s \rho_B Z_R}[1 - \exp(-k_s t_{s,e})]$$

Everything in the equation is known except for the resuspension rate constant. It is obtained by rearranging Eq. 8.8:

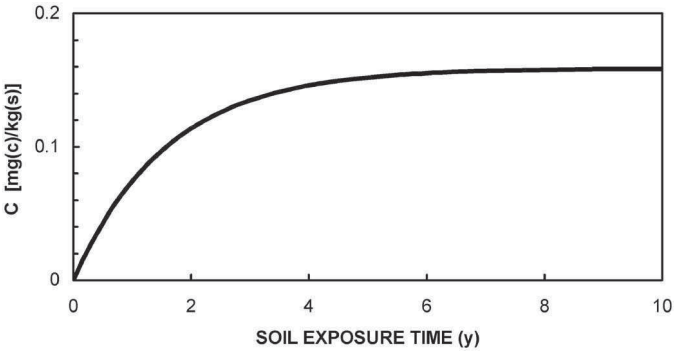

Figure 8.5 Contaminant concentration in soil vs. time in Example 8.4.

$$k_{s,r} = \kappa \cdot v_d = (0.2\,\text{m/s})(10^{-7}\,\text{m}^{-1})(3600\,\text{s/h})(24\,\text{h/d})(365\,\text{d/yr})$$
$$= 0.63\,\text{yr}^{-1}$$

Substituting the numerical values into Eq. 8.3 yields

$$C_s(t) = \frac{j_d}{k_s \rho_B Z_R}[1 - \exp(-k_s t_{s,e})]$$
$$= \frac{24\,\text{mg(c)/m}^2 \cdot \text{yr}}{(0.63\,\text{yr}^{-1})(1600\,\text{kg/m}^3)(0.15\,\text{m})}\{1 - \exp[(-0.63\,\text{yr}^{-1})t_{s,e}]\}$$

The time dependence of contaminant concentration in soil is plotted in Figure 8.5. After about 5 years the concentration is very close to the steady-state level, which is calculated by taking the limit as time goes to infinity:

$$C_s(t \to \infty) = \frac{j_d}{k_s \rho_B Z_R} = \frac{24\,\text{mg(c)/m}^2 \cdot \text{yr}}{(0.63\,\text{yr}^{-1})(1600\,\text{kg(s)/m}^3)(0.15\,\text{m})}$$
$$= 0.16\,\text{mg(c)/kg(s)}$$

8.3 CONCENTRATION IN VEGETATION

Vegetation can become contaminated as a result of uptake from contaminated soils through the roots and direct deposition or absorption from the air through foliage or flowers. Removal processes (Figure 8.6) include those related to the plant, such as transpiration, wind removal, and wash-off by rain; and those related to the contaminant, such as degradation and decay. For modeling purposes, these are approximated as independent first-order processes. They are incorporated into a single vegetative removal rate constant, k_v, which is the sum of rate constants for the individual processes, $k_v = \Sigma_i k_{v,i}$. In the absence of data, a default value of $0.05\,\text{d}^{-1}$, which corresponds to a half-time of 14 days, is recommended (IAEA 2001).

The concentration of a contaminant in vegetation could be obtained from a mass balance that considers the various processes adding or removing contaminant mass from the vegetation. In a simplified approach, the total concentration is calculated as the linear combination of the contributions from deposition and uptake:

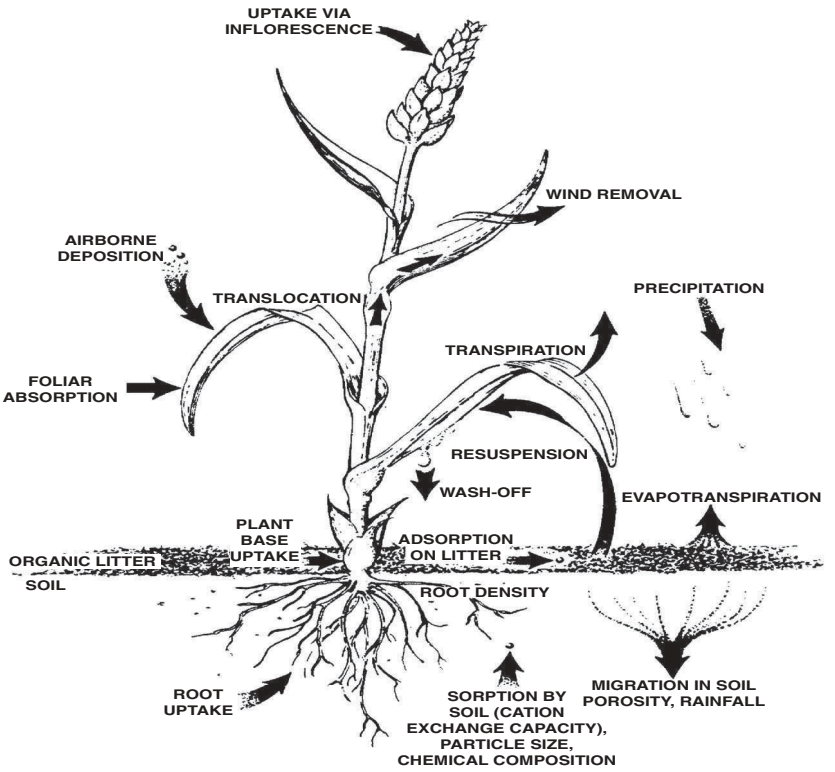

Figure 8.6 Processes affecting contaminant concentration in food crops. (From Peterson 1983.)

$$C_v = C_v^d + C_v^u \tag{8.9}$$

where C_v is the contaminant concentration in vegetation [M(c)/M(v,wet)], C_v^d the contribution to the total due to deposition on foliage, and C_v^u the contribution to the total due to uptake from the soil. Direct deposition is modeled through the constant-source first-order removal model, which takes on the form

$$\frac{dC_v^d}{dt} = v_d C_a f_v \frac{T_v}{Y} - k_v C_v^d \tag{8.10}$$

where T_v is the **translocation factor** and Y is the **vegetative yield** [M(v,wet)/L^2]. Physically, $v_d C_a$ is the areal deposition rate (i.e., mass of contaminant deposited per unit area). This is multiplied by the fraction of the deposited contaminant that is intercepted by the vegetation (f_v) and the fraction of the deposited contaminant that is transferred to the edible portion of the plant (T_v). This yields an areal uptake rate into the vegetation due to foliar deposition. Conversion to a mass concentration in the vegetation is achieved through the vegetative yield, which is the mass of crop harvested per area planted. Translocation factor is taken to be 1 for produce such as leafy vegetables and 0.1 for most other crops.

The solution to Eq. 8.10 is

$$C_v^d = \frac{f_v v_d C_a T_v}{Y k_v}\left[1 - \exp\left(-k_v t_{v,e}\right)\right] \tag{8.11}$$

where $t_{v,e}$ is the exposure time for vegetation. For annual crops, the maximum exposure time is the length of the growing season, about 90 days. For a short-term exposure, Eq. 8.11 reduces to

$$C_v^d = \frac{f_v v_d C_a T_v}{Y} t_{v,e} \tag{8.12}$$

► **Example 8.5**

Returning to the example of selenium released from the power plant, a dairy farm is located at the site boundary, where the long-term average selenium concentration in air is 5×10^{-6} mg(c)/m^3. Grass is allowed to grow in a pasture at the farm for 30 days before being opened to grazing. The interception fraction is 0.25, the deposition velocity is 0.2 m/s, the translocation factor is 0.1, and the yield of pasture grass is 1.5 kg(v,wet)/m^2. What is the concentration of selenium in pasture grass at the end of 30 days?

Solution Using Eq. 8.11, the concentration of selenium in pasture grass due to foliar deposition is

$$\begin{aligned} C_v^d(t) &= \frac{f_v v_d C_a T_v}{Y k_v}\left[1 - \exp\left(-k_v t_{v,e}\right)\right] \\ &= \frac{(0.25)(0.2\,\text{m/s})\left[5 \times 10^{-6}\,\text{mg(c)/m}^3\right](0.1)(3600\,\text{s/h})(24\,\text{h/d})}{\left[1.5\,\text{kg(v,wet)/m}^2\right]\left(0.05\,\text{d}^{-1}\right)} \\ &\quad \left\{1 - \exp\left[\left(-0.05\,\text{d}^{-1}\right)(30\,\text{d})\right]\right\} = 0.022\,\text{mg(c)/kg(v,wet)} \end{aligned}$$

Contaminant uptake from soil is a highly complex biophysical process which is affected by factors such as the physicochemical form of the contaminant, the plant species, the extent of translocation within the plant, the characteristics of the soil, the levels of nutrients and trace elements already present in the soil, and the spatial distribution of the contaminant in the soil (Peterson 1983). A model for predicting uptake as a function of all of these variables is generally not available. The alternative usually employed is an instantaneous partitioning model in which contaminant concentrations in the soil and in the plant are taken to be instantaneously in equilibrium. The results of experimental studies are used to determine the value of the partitioning coefficient. The form of the model is

$$C_v^u = B_v C_{s,\text{ext}} \tag{8.13}$$

where B_v is the **bioaccumulation factor** [M(s)/M(v,wet)] (also known as the concentration ratio) and $C_{s,\text{ext}}$ is the **extractable soil concentration**. The extractable

soil concentration is the portion of the contaminant that is biologically available for uptake by the plant. For freshly contaminated soils, most of the contaminant is generally available for uptake; hence the extractable concentration differs little from the total concentration. However, for naturally occurring contaminants and for soils that have been contaminated for a number of years, some portion of the contaminant may not be readily available to the plant; hence the extractable concentration can be significantly smaller than the total concentration. For some contaminants and some plants, separate bioaccumulation factors are available for roots, leaves, and fruit. Although a rigorous model would account for all of the factors given above, bioaccumulation factors are typically given only as a function of the contaminant and the plant. Vegetative bioaccumulation factors for selected contaminants are given in Table 8.1.

► **Example 8.6**

The contribution of foliar deposition to selenium in pasture grass was considered in Example 8.5. What is the contribution due to uptake from soil, and what is the total selenium concentration in pasture grass? Assume that all of the selenium is extractable.

Solution The concentration due to uptake from soil is calculated from Eq. 8.13:

$$C_v^u = B_v C_{s,\text{ext}}$$

From Table 8.1, the soil–plant bioaccumulation factor for selenium is 0.1. From Example 8.4, the concentration of selenium in soil depends on the period of time that the soil is exposed to the selenium in air. Since the life of a power plant is typically 40 years or more, the steady-state concentration of 0.16 mg(c)/kg(s) is used for this calculation. Thus,

$$\begin{aligned} C_v^u = B_v C_{s,\text{ext}} &= [0.1\,\text{kg(s)/kg(v,wet)}][0.16\,\text{mg(c)/kg(s)}] \\ &= 0.016\,\text{mg(c)kg(v,wet)} \end{aligned}$$

The total concentration of selenium in pasture grass is thus

$$\begin{aligned} C_v = C_v^d + C_v^u &= 0.022 + 0.016 \\ &= 0.038\,\text{mg(c)/kg (v,wet)} \end{aligned}$$

In this example, both foliar deposition and uptake from the soil are significant contributors to selenium in the plant.

Because animal (Section 8.4) and human (Chapter 9) uptake rates are usually based on the wet weight of food consumed, the concentration in vegetation is expressed in terms of wet weight as well. However, parameters such as vegetative yield and bioaccumulation factor are sometimes given in terms of dry weight and must be converted to a wet weight basis by the ratio of fresh (i.e., wet) weight to

TABLE 8.1 Food Chain Transport Parameters

Contaminant	B_v [kg(s)/kg(v,wet)]	B_{fish} [L/kg(fish)]	F_{milk} [d/L(milk)]	F_{meat} [d/kg(meat)]
Aldrein/dieldrin	1.4×10^{-1}	2.0×10^{4}	7.9×10^{-6}	2.5×10^{-5}
Arsenic	1.0×10^{-2}	3.2	6.0×10^{-5}	2.0×10^{-3}
Benzene	4.7×10^{-1}	8.7	9.9×10^{-7}	3.1×10^{-6}
Cadmium	1.4×10^{-1}	3.2	1.0×10^{-3}	4.0×10^{-4}
Carbon tetrachloride	1.8×10^{-1}	3.0×10^{1}	5.0×10^{-6}	1.5×10^{-5}
^{137}Cs	1.4×10^{-2}	2.0×10^{3}	7.9×10^{-3}	5.0×10^{-2}
^{137}Cesium/^{137m}Ba	1.7×10^{-2}	2.0×10^{3}	8.4×10^{-3}	5.0×10^{-2}
Chlordane	5.0×10^{-3}	1.2×10^{4}	2.5×10^{-3}	7.9×10^{-3}
Chlorine	1.8×10^{1}	3.2	1.7×10^{-2}	2.0×10^{-2}
Chloroform	5.3×10^{-1}	6.6	7.9×10^{-7}	2.6×10^{-6}
Chromium (+6)	1.0×10^{-4}	2.0×10^{2}	1.0×10^{-5}	9.0×10^{-3}
Chromium (+3)	1.0×10^{-4}	2.0×10^{2}	1.0×10^{-5}	9.0×10^{-3}
Cobalt	2.3×10^{-2}	3.2	7.0×10^{-5}	1.0×10^{-4}
DDD	3.3×10^{-3}	8.6×10^{3}	5.0×10^{-3}	1.6×10^{-2}
DDE	3.8×10^{-3}	2.1×10^{4}	4.0×10^{-3}	1.3×10^{-2}
DDT	1.6×10^{-3}	4.2×10^{4}	1.8×10^{-2}	5.7×10^{-2}
Ethylene dibromide	4.1×10^{-1}	9.2	1.3×10^{-6}	4.0×10^{-6}
Heptachlor	2.5×10^{-2}	9.9×10^{3}	1.6×10^{-4}	5.0×10^{-4}
Heptachlor epoxide	5.7×10^{-3}	1.4×10^{3}	2.0×10^{-3}	6.3×10^{-3}
Hexachlrorocyclohexane-γ (lindane)	5.5×10^{-2}	3.1×10^{2}	4.0×10^{-5}	1.3×10^{-4}
^{3}H (as HTO)		1.0	1.5×10^{-2}	
Hydrogen sulfide	2.2	3.2	7.0×10^{-8}	2.2×10^{-7}
^{131}I (aerosol or vapor)	2.0×10^{-3}	4.0×10^{1}	1.0×10^{-2}	4.0×10^{-2}
Lead	7.6×10^{-4}	3.2	3.0×10^{-4}	4.0×10^{-4}
Mercury (+2)	3.0×10^{-1}	3.2	4.7×10^{-4}	1.0×10^{-2}
Methyl mercury		1.0×10^{2}		
Methylene chloride	1.4	1.8	1.6×10^{-7}	5.0×10^{-7}
^{239}Pu	4.9×10^{-6}	3.0×10^{1}	1.1×10^{-6}	1.0×10^{-5}
Polychlorinated biphenyls	2.5×10^{-3}	5.8×10^{4}	7.9×10^{-3}	2.5×10^{-2}
^{226}Ra	9.3×10^{-3}	5.0×10^{1}	1.3×10^{-3}	9.0×10^{-4}
^{226}Ra + decay products	1.9×10^{-2}	4.6×10^{2}	2.8×10^{-3}	1.2×10^{-2}
Selenium	1.0×10^{-1}	3.2	1.0×10^{-2}	1.0×10^{-1}
^{90}Sr	2.1×10^{-1}	6.0×10^{1}	2.8×10^{-3}	8.0×10^{-3}
^{90}Sr/^{90}Y	2.1×10^{-1}	9.0×10^{1}	2.8×10^{-3}	9.0×10^{-3}
Tetrachloroethylene (PCE)	2.4×10^{-1}	8.3×10^{1}	3.1×10^{-6}	1.0×10^{-5}
Trichloroethylene (TCE)	3.1×10^{-1}	15	2.0×10^{-6}	6.3×10^{-6}
Uranium salts	6.3×10^{-4}	1.0×10^{1}	4.0×10^{-4}	3.0×10^{-4}
^{235}U	6.3×10^{-4}	1.0×10^{1}	4.0×10^{-4}	3.0×10^{-4}
^{235}U + decay products	7.7×10^{-4}	1.1×10^{2}	4.1×10^{-4}	4.0×10^{-4}
^{238}U	6.3×10^{-4}	1.0×10^{1}	4.0×10^{-4}	3.0×10^{-4}
^{238}U + decay products	2.0×10^{-3}	1.3×10^{2}	4.2×10^{-4}	4.0×10^{-4}
Vinyl chloride	1.2	3.5	2.0×10^{-7}	6.3×10^{-7}

Source: RAIS 2005.

dry weight. This ratio ranges from close to 1 for grains to 20 for leafy produce such as lettuce. Nominal values for food crop yields range from 4 kg(v,wet)/m^2 for vegetables to 2 kg(v,wet)/m^2 for fruits and leafy vegetables to 1 kg(v,wet)/m^2 for grains (NRC 1992).

8.4 CONCENTRATION IN ANIMALS

Animals such as fish, other aquatic organisms, and terrestrial grazing animals are food sources, and they are important pathways of human exposure. The transfer of contaminants from water and vegetation to animals is another very complex set of processes that generally are not rigorously modeled given the current state of knowledge. As a result, relatively simple partitioning and uptake/removal models are applied to these problems.

Aquatic organisms such as fish may be exposed to contaminants in the water column, while bottom feeding fish and benthic organisms (animals that live in the sediments at the bottom of a surface water body) may be exposed to contaminants from the sediments and the water. Contaminant uptake in fish and other aquatic animals through exposure to aqueous-phase contaminants is approximated through an instantaneous partitioning model:

$$C_f = B_f C_w \tag{8.14}$$

where C_f is the concentration in fish [M(c)/M(f,wet)], C_w the concentration in the aqueous phase [M(c)/L^3], and B_f the bioaccumulation factor for fish [L^3/M(f,wet)]. Here again, the concentration in fish is expressed in terms of wet weight to be consistent with exposure calculations, and it is important that bioaccumulation factors be expressed in terms of wet weight as well.

Grazing animals take up contaminants through grazing, eating stored feed, or drinking water. Regardless of the source, accumulation of the contaminant is modeled using the constant-source first-order removal model. It is typically applied to either cows or goats, and it is used to determine contaminant concentration in both milk and meat. For milk, the differential equation takes the form

$$\frac{dC_{\text{milk}}}{dt} = \frac{C_v \dot{U}_v f_{\text{milk}}}{V_{\text{milk}}} - kC_{\text{milk}} \tag{8.15}$$

where C_{milk} is the concentration in milk [M(c)/L^3(milk)], $\dot{U}_v$ the uptake rate of vegetation [M(v,wet)/T], f_{milk} the fraction of contaminant transferred to milk, V_{milk} the milk volume [L^3], and k a first-order removal-rate constant. The solution is

$$C_{\text{milk}}(t) = C_v \dot{U}_v \left\{ \frac{f_{\text{milk}}}{kV_{\text{milk}}} [1 - \exp(-kt)] \right\} \tag{8.16}$$

The term in braces in Eq. 8.16 is defined as the **milk transfer factor**, F_{milk} [T/L^3(milk)], which physically is the fraction of daily intake of contaminant that appears in the milk per unit milk volume. Thus, the calculation of contaminant concentration in milk simplifies to

TABLE 8.2 Animal Feed and Water Intake Rates [kg(w)/d]

Intake Media	Beef Cattle	Dairy Cattle
Fresh forage	27	36
Stored hay	14	29
Stored grain	3	2
Water	50	60

Source: NRC 1992.

$$C_{\text{milk}} = \dot{U}_v C_v F_{\text{milk}} \tag{8.17}$$

An analogous approach is used for the calculation of contaminant concentration in the meat of grazing animals, yielding

$$C_{\text{meat}} = \dot{U}_v C_v F_{\text{meat}} \tag{8.18}$$

where C_{meat} is contaminant concentration in meat [M(c)/M(meat)] and F_{meat} is the **meat transfer factor** [T/M(meat)]. Values of F_{milk} and F_{meat} for cows are included in Table 8.1. Transfer factors for other foods, such as goat milk, goat meat, pork, poultry, and eggs, are available for some contaminants (IAEA 1994).

The uptake rate of vegetation is an important factor in determining the total contaminant uptake of the animal. Total uptake rates for different animals and animal products are shown in Table 8.2. Since these are total uptake rates, they must be reduced proportionally if animals are grazed or fed on uncontaminated crops for part of their diet.

► **Example 8.7**

A final link in the food chain transport of selenium is cow's milk. What is the selenium concentration in cow's milk?

Solution The concentration in cow's milk is calculated from Eq. 8.17:

$$C_{\text{milk}} = \dot{U} C_v F_{\text{milk}}$$

From Table 8.2 the vegetative uptake rate for dairy cows is 36 kg(w)/d, from Table 8.1 the milk transfer factor for selenium is 1.0×10^{-2} d/L(milk), and from Example 8.6 the selenium concentration in vegetation is 0.038 mg(c)/kg(v,wet). Thus,

$$\begin{aligned} C_{\text{milk}} = \dot{U} C_v F_{\text{milk}} &= [36\,\text{kg(v,wet)/d}][0.038\,\text{mg(c)/kg(v,wet)}][1.0 \times 10^{-2}\,\text{d/L(milk)}] \\ &= 0.014\,\text{mg(c)/ L(milk)} \end{aligned}$$

A final consideration is the time required for food products to reach consumers. This time, known as a holdup time, can range from 20 days for beef to 14 days for fruits and vegetables to 1 day for leafy vegetables and dairy products (NRC 1992). For crops exposed to long-term releases of contaminants that degrade quickly, such

as short-lived radionuclides, holdup times may significantly reduce the amount of contaminant taken up by humans. In such cases, the appropriate degradation correction should be applied.

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PROBLEMS

8.1 The areal concentration of TCE at an industrial site is $50\,g/m^2$. What is the concentration in soil if the depth of the root zone is 25 cm and the bulk soil density is $1800\,kg/m^3$?

8.2 The arsenic concentration in an aquifer used for irrigating crops in an agricultural region is $100\,\mu g/L$. Consider a field for which the irrigation rate is 0.8 m/yr, the bulk soil density if $1600\,kg/m^3$, and the depth of the root zone is 0.15 m.

(a) Neglecting removal, calculate the arsenic concentration in the soil after 3 months of irrigation.

(b) If the removal-rate constant for leaching of arsenic from the soil is $5 \times 10^{-3}\,yr^{-1}$, what is the steady-state concentration of arsenic in soil, and how long does it take for the concentration to be within 1% of the steady-state value?

8.3 If the concentration of extractable lead in soil is 350 mg/kg, what is the concentration in vegetation?

8.4 If the concentration of chromium (+3) in a stream below an electroplating facility is $50\,\mu g/L$, what is the concentration in fish?

8.5 During the late 1950s and early 1960s, atmospheric tests of nuclear weapons resulted in ^{90}Sr contamination of milk. For a ^{90}Sr concentration in pasture grass of 5 Bq/kg(v,wet), calculate the concentration in milk.

8.6 Due to releases from a plutonium fabrication facility, the long-term average atmospheric concentration of ^{239}Pu is 0.07 Bq/m^3 at the site boundary.

(a) Calculate the steady-state concentration of ^{239}Pu in soil. The deposition velocity is 0.15 m/s, the resuspension factor is 10^{-6} m^{-1}, the depth of the root zone is 0.15 m, the plant interception fraction is 0.25, and the bulk soil density is 1500 kg/m^3.

(b) Calculate the steady-state concentration of ^{239}Pu in grains used for cattle feed. The translocation factor is 1, the wet yield is 1 kg(v,wet)/m^2, and the removal rate constant from vegetation is 0.05 d^{-1}. Obtain the soil–plant bioaccumulation factor from Table 8.1.

(c) Calculate the concentration of ^{239}Pu in the beef resulting from the contamination. Obtain the intake rate for stored grain from Table 8.2 and the meat transfer factor from Table 8.1.

8.7 An accident at a nuclear reactor results in the release of ^{131}I ($t_{1/2}$ = 8 d) to the atmosphere. The contaminant passes through the grass–cow–milk food chain. A dairy farm is located in the path of the plume, and pasture grass becomes contaminated. Measurements of atmospheric concentrations of the contaminant are made at the farm during the 1-hour period while the plume passes by. The average airborne concentration during this period is 80 Bq/m^3, the deposition velocity is 10^{-2} m/s, the vegetative yield for grass is 0.75 kg(wet)/m^2, the translocation factor is 1, and the fraction intercepted by vegetation is 1. Obtain the fresh forage intake rate for cows from Table 8.2 and the milk transfer factor from Table 8.1. Uptake of contaminant by the grass from soil may be neglected.

(a) If cows must be kept out of the pasture and use stored food when the contaminant concentration exceeds 4 Bq/L in milk, will it be necessary for the farmer to keep the cows out of the pasture?

(b) If so, for how long?

8.8 A fire in an industrial facility causes the release of a contaminant over a period of 30 minutes. Winds carry the contaminant plume past a farm located 2 km downwind from the facility. The average ground-level concentration at the farm is 0.5 mg/m^3 during the 30-minute period when the plume is present.

(a) What is the contaminant concentration in the soil as a result of this release? The deposition velocity is 0.2 m/s, the depth of the root zone is 0.15 m, and the bulk soil density is 1600 kg/m^3.

(b) Suppose that the farmer plows the farm and plants wheat, which is estimated to have a bioaccumulation factor of 0.05 kg(soil)/kg(v,wet) for the contaminant. What is the concentration of the contaminant in the fresh (not dried) crop?

9 Exposure Assessment

9.1 INTRODUCTION

Exposure is the contact of humans with contaminants. For environmental risk assessment, contaminants are present in environmental media (air, water, soil, sediment) or in food (plant products, such as vegetables, fruits, nuts, and grains; and animal products, such as meat, eggs, and milk). Chapters 4 to 8 describe the analytical methods for determining the concentrations of contaminants in environmental media and food. The avenues through which the contaminants move from environmental media and food into the body of an exposed human are **exposure routes** (EPA 1992). For environmental contaminants, typical exposure routes are (1) inhalation of contaminated air; (2) ingestion of contaminated water, food, or soil; and (3) dermal contact with a contaminated medium such as water or soil. In the special case of radiological contaminants, exposure is also possible without direct contact with the contaminant because radiological contaminants emit radiation that can expose persons located some distance away. The effect of exposure to environmental contaminants depends on a variety of factors, including the contaminant concentration in the media or food, the exposure route, the rate at which the exposure occurs, the duration and, if appropriate, the frequency of the exposure, and the chemical and/or physical form of the contaminant. **Exposure assessment** is the process of identifying exposed individuals or populations and quantifying the various factors determining the exposure. The quantitative result of an exposure assessment is usually a contaminant dose, although contaminant concentration (sometimes with exposure duration) is used in some situations. The dose (or concentration) is subsequently combined with a dose–response (or concentration–response) relationship to estimate risk.

Receptors are individuals or populations subject to exposure to the contaminant. Although many risk assessments provide the distribution of risks among the entire exposed population, risk estimates are often calculated for individuals in the population. An example of an individual receptor within a population is a person who receives a "reasonable maximum exposure", defined by the EPA (1989) for some regulatory purposes as the upper 95th percentile of dose. Other individual

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receptors might be specific persons at specific locations in the vicinity of the contaminant release. Receptors can also include critical subgroups such as the very young, the very old, home gardeners, indigenous populations, or fishermen. Sometimes, the receptor is a hypothetical person whose risk in some fashion bounds the population distribution of risk. For example, for some regulatory assessments, the Nuclear Regulatory Commission (NRC 1996) requires that risk be determined for a hypothetical maximum-exposed person.

Another term frequently used in environmental risk assessments is **exposure pathway**, which denotes the particular route followed by a contaminant from its source to a particular receptor (EPA 1992). For example, the set of examples in Chapter 8 trace selenium released from a coal-fired power plant through dispersion in air, deposition on soil and plant surfaces, uptake by pasture grass from roots and foliage, and consumption by dairy cows leading to a concentration in milk intended for human consumption. This entire passage from the point of release to the human consuming the milk constitutes an exposure pathway.

Traditionally, exposure duration has been characterized qualitatively as acute, subchronic, or chronic. **Acute exposure** refers to exposure that occurs over a short period of time, on the order of days or less. A **subchronic exposure** is one that is protracted, either continuous or intermittent, but the duration is not a significant fraction of an entire lifetime. The duration of a subchronic human exposure can range from several months to a few years. A **chronic exposure** is exposure that occurs on a continuing, long-term basis. The EPA (2002a) defines a subchronic exposure as less than 10% of total lifetime and chronic as greater than 10%. Thus, an exposure duration of 7 years (corresponding to an average human life span of 70 years) or less is considered to be subchronic, and more than 7 years is considered to be chronic. Recently, a recommendation (EPA 2002a) has been made to modify this formalism by categorizing exposures as acute, short term, long term, and chronic, with respective durations of less than 1 day, 1 to 30 days, 30 days to 10% of species lifetime, and greater than 10% of species lifetime. For the laboratory animals typically used in laboratory toxicity testing, the dividing line between long term and chronic is taken to be 90 days.

In addition to the qualitative definition of exposure, the term is sometimes used quantitatively. For example, the term cumulative exposure is sometimes defined as the time integral of concentration over some time interval t_1 to t_2 [i.e., $\int_{t_1}^{t_2} C(t)dt$]. This time integral is useful in the calculation of doses when the concentration varies over time and when the health effects are proportional to the total intake rather than the rate at which the contaminant is taken into the body.

9.2 DOSE

Dose is the metric that is used to quantify exposure. There are two different traditions in which the specific quantitative definitions of the dose metrics used in risk assessment have developed. Toxicologists typically quantify a chemical dose as a mass of contaminant per unit body mass (i.e., milligrams of contaminant per kilogram of body weight). Health physicists have defined two quantities, equivalent dose and effective dose, for expressing radiological dose. Both quantities have the

same special unit, the sievert (Sv). Regardless of whether it refers to a chemical or radiological exposure, **dose rate** ($\dot{D}$) is dose per unit time, and **total dose** (D_T) is the time integral of dose rate over the entire exposure period:

$$D_T = \int_0^{t_e} \dot{D}(t)\,dt \tag{9.1}$$

where t_e is the exposure time.

9.2.1 Chemical Dose

For acute exposures, chemical dose is expressed as the total mass of contaminant taken into the body per unit body weight. This quantity is also termed the **potential dose** (by EPA) or the **administered dose** (in the context of animal studies). As discussed in Chapter 11, this is a surrogate for the biologically active concentration in the target organ (termed **internal dose** or **effective dose**[1]) that is the ultimate basis for the toxic effects of the contaminant. Pharmacokinetic models, which are also discussed in Chapter 11, can be used to estimate an effective dose from an administered dose. However, for many contaminants, dose–response relationships are expressed in terms of potential (or administered) dose. Consequently, most of the doses calculated in this book are potential doses.

The calculation of dose due to acute exposures to chemical contaminants is a direct application of Eq. 9.1. Chronic and subchronic exposures, however, are typically expressed as an average dose rate where the averaging time may differ from the exposure duration:

$$\tilde{\dot{D}} = \frac{\int_0^{t_e} \dot{D}(t)\,dt}{t_{\text{avg}}} = \frac{D_T}{t_{\text{avg}}} \tag{9.2}$$

where $\tilde{\dot{D}}$ is average dose rate, t_e the exposure duration, and t_{avg} the averaging time. If $t_{\text{avg}} = t_e$, $\tilde{\dot{D}}$ is a true time average; if $t_{\text{avg}} \neq t_e$, $\tilde{\dot{D}}$ is the average dose rate prorated over the averaging time. The latter arises because most chemical dose–response relationships for humans are inferred from animal tests. Thus, in estimating risks, human dose rates are averaged over a period of time corresponding to the conditions of the laboratory animal tests. For example, most animal carcinogenicity studies are performed over the animal's lifespan, which is about 2 years for rodents. Consequently, chronic human exposures are expressed as dose rate averaged over the human lifespan. Traditionally, a human lifespan of 70 years has been used in risk assessment, but a recent recommendation (EPA 1997) is 75 years.

[1] Because chemical and radiological contaminants have traditionally been studied and regulated by different groups, the terminology for describing exposure was developed independently. As a consequence, some terms have two meanings; one for chemical contaminants and one for radiological contaminants. The dose terms with two meanings in this text are effective dose and internal dose, and in Chapter 11, absorbed dose. For this reason, the descriptor *chemical* or *radiation* is sometimes included in the term either for emphasis or to avoid confusion.

▶ Example 9.1

An individual drinks water from a contaminated well for 30 years. The resulting dose rate changes because the concentration of the contaminant in the well decreases with time. If the dose rate is given by $\dot{D}(t) = 0.05\ \exp(-t/7000\,\text{d})\text{mg(c)/kg·d}$, where t has units of days, find the following:

(a) The total dose.
(b) The dose rate averaged over the exposure period (i.e., average daily dose).
(c) The dose rate averaged over an averaging time of 70 years (i.e., lifetime average daily dose).

Solution

(a) Total dose is calculated by integrating dose rate over the exposure period, or

$$\begin{aligned} D_T &= \int_0^{t_e} \dot{D}(t)\,dt = \int_0^{30\,\text{yr}\times 365\,\text{d/yr}} 0.05[\text{mg(c)/kg·d}]\exp(-t/7000\,\text{d})\,dt \\ &= 350[\text{mg(c)/kg·d}]\left[-\exp\left(-\frac{t}{7000\,\text{d}}\right)\right]\Bigg|_0^{10{,}950\,\text{d}} \\ &= 277\,\text{mg(c)/kg} \end{aligned}$$

(b) The average dose rate is calculated from Eq. 9.2. Here, $t_{\text{avg}} = t_e = 30\,\text{yr}$, and from part (a), the numerator (total dose) is 277 mg(c)/d. Thus,

$$\tilde{\dot{D}} = \frac{277\,\text{mg(c)/kg}}{(30\,\text{yr})(365\,\text{d/yr})} = 0.025\,\text{mg(c)/(kg·d)}$$

(c) This is the same as part (b) except that $t_{\text{avg}} = 70\,\text{yr}$. Thus,

$$\tilde{\dot{D}} = \frac{277\,\text{mg(c)/kg}}{(70\,\text{yr})(365\,\text{d/yr})} = 0.011\,\text{mg(c)/(kg·d)}$$

In risk assessment practice; "dose" is both a qualitative term that refers to a generic measure of exposure to a contaminant as in "dose–response relationship" and a quantitative definition such as mass of contaminant per unit body mass, as in "lethal dose to 50% of those exposed." Various quantitative definitions are used in toxicology and in regulatory practice (EPA 1992). EPA's **average daily dose** is the average dose rate defined in Eq. 9.2 for $t_{\text{avg}} = t_e$. For simple, continuous exposure scenarios, the integration indicated in Eq. 9.2 is straightforward; for exposures with intermittent or periodic exposures, factors must be introduced into the integral to account for those time periods when the dose rate is zero. For example, for a person exposed in the workplace only, the exposure time is the number of working hours (2000 h/yr) and the averaging time is the total number of hours (8760 h/yr). The **lifetime average daily dose** is the average dose rate for t_{avg} equal to the lifespan (70 to 75 years) rather than the actual exposure time.

Estimating chemical effective dose requires modeling the distribution and transformation of the contaminant in the body through the use of a pharmacokinetic model that tracks the absorption, distribution, metabolism, and elimination of chemicals in humans and animals. However, for a majority of contaminants, data are not yet available to construct pharmacokinetic models properly. For some contaminants, it is possible to calculate chemical absorbed dose. However, for the majority of contaminants, dose–response relationships are expressed in terms of administered dose. Consequently, most of the doses calculated in this book are administered doses which, for simplicity, are designated here as D.

9.2.2 Radiological Dose

Biological damage due to radiation exposure depends primarily on the amount of energy deposited per unit mass of tissue and the type of radiation. Four primary types of nuclear radiation are encountered in risk assessments: alpha, beta, gamma, and neutron radiation. Environmental contaminants emit primarily the first three types of radiation. Alpha radiation consists of energetic helium nuclei (two protons and two neutrons) and is highly damaging, although it has a limited ability to penetrate matter and only poses a significant risk if α-emitting radioactive contaminants are taken into the body. Beta radiation consists of energetic electrons and can pose a risk due to doses to the skin or by β-emitting radioactive contaminants taken into the body. Gamma radiation is highly penetrating and thus poses a risk from radioactivity located either inside or outside the body. Neutron radiation does not typically arise from environmental contamination, since most radiation from environmental contaminants is a result of radioactive decay processes that produce only alpha, beta, or gamma radiation. However, large neutron exposure can arise from manufactured neutron sources or nuclear criticality accidents.

The basic dosimetric quantities are radiation **absorbed dose**, D_R; **equivalent dose**, H; and **effective dose**, E (ICRP 1991). Absorbed dose rate, equivalent dose rate, and effective dose rate are denoted, respectively, by $\dot{D}_R$, $\dot{H}$, and $\dot{E}$. Radiation absorbed dose is the amount of energy absorbed per unit mass of tissue from the radiation. It has units of J/kg, which is given the special name gray (Gy). The combined effect of absorbed energy and type of radiation on biological damage is taken into account by the equivalent dose,

$$H = \omega_R D_R \tag{9.3}$$

where ω_R is radiation weighting factor. The radiation weighting factor is 1 for gamma rays and electrons, 5 for protons, 20 for alpha particles, and ranges from 5 to 20 for neutrons depending on their energy. Radiation equivalent dose, which has units of sieverts (Sv), is used to estimate the adverse systemic effects of contaminants classified in Chapter 10 as deterministic. Deterministic effects are of concern only for equivalent doses in excess of 0.1 Sv. Radiation effective dose, which also has units of sieverts, is used to estimate the risk of cancer and hereditary effects, which are classified in Chapter 10 as stochastic. Radiation effective dose accounts for the different sensitivities of different body tissues to cancer and hereditary effects and is essentially a risk-weighted equivalent dose. It is given by

$$E = \sum_T \omega_T H_T \tag{9.4}$$

where ω_T is the tissue weighting factor. Tissue weighting factors to account for cancer risk are 0.12 for the colon, stomach, lungs, and red marrow; 0.05 for the urinary bladder, liver, esophagus, and thyroid; and 0.01 for the bone surface and skin. The weighting factor to account for risk of inherited effects is 0.2 for the gonads. Equivalent dose and effective dose are measures that are independent of the specific radionuclide or type of radiation. This permits the consequence of exposure to multiple radionuclides to be combined by summation of the individual equivalent doses or effective doses.

9.3 CONTAMINANT INTAKE

A contaminant can enter the body and exert its toxic effect through inhalation, ingestion, or absorption through the skin. As mentioned above, external sources of radioactive contaminants can also be harmful. This section is concerned with quantification of contaminant intake through inhalation, ingestion, and skin absorption. Throughout the discussion, default and typical values are given. However, a complete exposure assessment requires a more accurate estimation of the uptake rates for the population actually exposed, along with the uncertainty and variability in these estimates. Default values are useful primarily for screening and should not be taken as representative of the exposed population without further verification. Detailed guidance on exposure assessment and exposure factors is provided in EPA documents (EPA 1992, 1997, 2004).

Contaminant **intake rate** is the mass or activity of contaminant entering the body per unit time,

$$\dot{I} = \mathrm{CR} \cdot C \tag{9.5}$$

where $\dot{I}$ is the rate at which the contaminant enters the body [M/T for chemicals or activity/T for radionuclides] and CR is the **contact rate** with the contaminated medium [L^3/T for inhalation, L^3/T for ingestion of water, M/T for ingestion of food, L^3/T for skin absorption from water, and M(s)/T for skin absorption from soil]. For ingestion and inhalation, the contact rate is sometimes called the **uptake rate**.

9.3.1 Inhalation

Airborne contaminants subject to inhalation may be present in either particulate or gaseous form. The contact rate is the air inhalation rate, which is also known as the pulmonary ventilation rate. The inhalation rate is a function of age, weight, gender, activity level, and physical condition, although it is rare for all of these factors to be taken into account. Presented in Table 9.1 are values recommended by the EPA (EPA 1997) for inhalation and other exposure routes. Values range from less than 0.2 m^3/h (4.5 m^3/d) for infants to 3.2 m^3/h for adults engaged in short-term heavy activity. Default values are 15 m^3/d for adult males and 11 m^3/d for adult females.

TABLE 9.1 Exposure Factors

Exposure Factor	Value	Normalized Value[a]
Adult body weight	71.8 kg	
Life expectancy	75 yr	
Inhalation rate		
Children (<1 yr)	4.5 m^3/d (average)	
Children (1–12 yr)	8.7 m^3/d	
Adult females	11.3 m^3/d (average)	
Adult males	15.2 m^3/d (average)	
Drinking water uptake	1.5 L/d (average)	21 mL/(kg·d)
	2.4 L/d (90th percentile)	34 mL/(kg·d)
Total fruit uptake	240 g/d (average)	3.4 g/(kg·d)
	870 g/d (95th percentile)	12.4 g/(kg·d)
Total vegetable uptake	300 g/d (average)	4.3 g/(kg·d)
	700 g/d (95th percentile)	10 g/(kg·d)
Total meat uptake	150 g/d (average)	2.1 g/(kg·d)
	360 g/d (95th percentile)	5.1 g/(kg·d)
Total dairy uptake	560 g/d (average)	8.0 g/(kg·d)
	2100 g/d (95th percentile)	29.7 g/(kg·d)
Grain uptake	290 g/d (average)	4.1 g/(kg·d)
	760 g/d (95th percentile)	10.8 g/(kg·d)
Breast milk uptake	—	742 mL/(kg·d) (average)
	—	1033 mL/(kg·d) (upper %tile)
Fish uptake	General population	
	20.1 g/d (total fish average)	
	14.1 g/d (marine average)	
	6.0 (freshwater/estuarine average)	
	63 g/d (total fish 95th percentile)	
	Recreational marine	
	2–7 g/d (finfish only)	
	Recreational freshwater	
	8 g/d (average)	
	Native American subsistence	
	70 g/d (average)	
	170 g/d (95th percentile)	
Soil uptake		
Children	100 mg/d (average)	
	400 mg/d (upper percentile)	
Pica child	10 g/d	
Adults	50 mg/d (average)	
Showering time	10 min/d (average)	
	35 min/d (95th percentile)	
Bathing time	20 min/event (median)	
	45 min/event (90th percentile)	
Swimming	1 event/month	
	60 min/event (median)	
	180 min/event (90th percentile)	

TABLE 9.1 *Continued*

Exposure Factor	Value	Normalized Value[a]
Time indoors		
Children (3–11)	19 h/d (weekdays)	
	17 h/d (weekends)	
Adults (12 and older)	21 h/d	
	16.4 h/d (residential)	
Time outdoors		
Children (3–11)	5 h/d (weekdays)	
	7 h/d (weekends)	
Adults (12 and older)	1.5 h/d	
	2 h/d (residential)	
Time inside vehicle	1 h 20 min/d	
Occupational tenure	6.6 yr	
Population mobility	9 yr (average)	
	30 yr (95th percentile)	

Source: EPA 1997.

[a] Based on a body weight of 70 kg.

9.3.2 Ingestion

Intake through ingestion is conceptually similar to that by inhalation in that the ingestion rate is simply the amount of the contaminated medium (water, food, or soil) taken into the body per unit time. For contaminated water, intake occurs due to ingestion of drinking water and incidental ingestion of water while swimming. The actual amounts vary depending on the individual level of physical activity and the ambient temperature and relative humidity. Sources include water direct from the tap and beverages made with tap water, such as concentrated juices and coffee or tea. EPA has historically used a default value of 2 L/d for adults and 1 L/d for children. The current recommendations (EPA 1997) range from 0.3 L/d (infant mean) to 2.4 L/d (lactating female: 95th percentile). A value of 1.4 L/d is the recommended mean for adults.

For food, the uptake rate is a function of the type of food and the age and gender of the person. Ingestion of various foodstuffs is typically determined from dietary surveys. Major food groups used in environmental risk assessments that may be subject to contamination are fruits, vegetables, dairy products, meat, and fish. Since a portion of a person's total diet may not be produced locally, the fraction of the total diet that is affected by contamination must be taken into account. The contaminated food is usually that produced (or caught) by the receptor for personal consumption, although situations may exist (such as the Chernobyl accident) in which contaminated food is distributed commercially. Typically, the percentage of locally produced food is highest for persons living in rural areas and lowest for those residing in cities, with suburban residents lying between the two. In addition, people engaging in hunting and fishing are likely to consume higher fractions of locally produced game and fish and may have higher-than-average consumption rates. Default uptake rates for each of the food categories are given in Table 9.1.

The final ingestion pathway of concern involves contaminated soils. Under normal circumstances, this is due either to ingestion of airborne particulate matter

that becomes deposited in the respiratory system or to incidental ingestion of soil from homegrown food products or of dust from handling food or cigarettes with unwashed hands. Normally, young children (less than 7 years old) can be expected to ingest, on average, between 100 and 200 mg of soil per day, although ingestion rates of up to 1000 mg/day are possible for normal ingestion by children (EPA 1997). However, a phenomenon known as pica may cause soil ingestion rates to be abnormally high for some subpopulations, particularly children from lower-socioeconomic backgrounds. Pica can be of significant concern if it leads to an abnormal level of direct ingestion of contaminated soil. Unfortunately, it is very difficult to quantify accurately the amount of soil that pica children ingest, as it can vary widely.

9.3.3 Dermal Absorption

Although the skin is an effective barrier to the entry of contaminants, it is possible for small amounts to penetrate and enter the body. The two approaches for estimating contaminant absorption through the skin recommended by the EPA for Superfund sites (EPA 1989) are adopted here. One applies to contact with contaminated water and the other to contact with contaminated soil. For both scenarios, the contact rate does not have an easily described, intuitive physical meaning as it did for ingestion and inhalation, where it was the volume of water or mass of food taken into the body every hour or every day. For dermal contact, the medium does not enter the body and the contact rate incorporates either water or soil contact with the skin and contaminant penetration of the skin. It has units of water volume or soil mass per unit time which when multiplied by concentration yields the rate of contaminant entry into the body. Unlike the intake calculations for ingestion and inhalation, which ultimately yield estimates of potential chemical dose, dermal absorption calculations ultimately yield estimates of applied chemical dose.

The equation for contact rate due to absorption from water is

$$\mathrm{CR} = k_p A_{\mathrm{skin}} \tau_d \tag{9.6}$$

where k_p is dermal permeability constant [L/T], A_{skin} the area of skin exposed [L^2], and τ_d the daily exposure duration [T/T]. The physical interpretation of the permeability constant is contaminant penetration rate per unit area of skin per unit concentration, and the expanded units are[(M(c)/T)/L^2]/[M(c)/L^3]. Values of the permeability constants for selected contaminants are given in Table 9.2. The typical scenarios for dermal absorption due to water contact are showering, bathing, and swimming where the total body is exposed. EPA recommends conservative, health-protective default values for the various parameters (EPA 2004). Default skin areas are 18,000 cm^2 for adults and 6600 cm^2 for children. Default values for exposure duration for showering, bathing, and swimming are given in Table 9.1.

For contaminated soil, contact rate is calculated by

$$\mathrm{CR} = \alpha_F f_{\mathrm{abs}} v_e A_{\mathrm{skin}} \tag{9.7}$$

where α_F is the soil-to-skin adherence factor [M(s)/L^2], f_{abs} the dermal absorption fraction, and v_e the exposure frequency [event/T]. The soil-to-skin adherence factor

TABLE 9.2 Dermal Permeability Constants for Water

Contaminant	k_p (cm/h)
Aldrein/dieldrin	1.4×10^{-3}
Arsenic	[a]1.0×10^{-3}
Benzene	1.5×10^{-2}
Cadmium	[a]1.0×10^{-3}
Carbon tetrachloride	1.6×10^{-2}
Cesium	[a]1.0×10^{-3}
Chlordane	3.4×10^{-2}
Chloroform	6.8×10^{-3}
Chromium (+6)	2×10^{-3}
Chromium (+3)	1×10^{-3}
Cobalt	4×10^{-4}
DDD	1.8×10^{-1}
DDE	1.6×10^{-1}
DDT	2.7×10^{-1}
Hexachlrorocyclohexanes (lindane)	1.1×10^{-2}
Iodine	[a]1.0×10^{-3}
Lead	1×10^{-4}
Mercury (+2)	1×10^{-3}
Mercury vapor	0.24
Methyl mercury	1×10^{-3}
Methylene chloride	3.5×10^{-3}
Plutonium	[a]1.0×10^{-3}
Polychlorinated biphenyls	7.5×10^{-1}
Radium	[a]1.0×10^{-3}
Strontium	[a]1.0×10^{-3}
Tetrachloroethylene (PCE)	3.3×10^{-2}
Trichloroethylene (TCE)	1.2×10^{-2}
Uranium	[a]1.0×10^{-3}
Vinyl chloride	5.6×10^{-3}

Source: EPA 2004.

[a] The default value for inorganics is 1×10^{-3}.

is the mass of soil that can adhere to a unit area. It is weighted by the fraction of body surface area that is exposed and thus depends on the type of activity that is responsible for contact with soil. Values vary widely and include geometric mean values of 0.01 mg/cm^2 for children indoors and adult grounds keepers, 0.1 mg/cm^2 for adults playing soccer, 0.3 mg/cm^2 for archaeologists, and over 20 mg/cm^2 for children playing in mud. Health-protective default values recommended by EPA (EPA 2004) are 0.07 mg/cm^2 for adults and 0.2 mg/cm^2 for children, which represent conservative values for typical individuals experiencing a variety of activities. The value for adults is the median value for gardening, a high-end activity, and the value for children is the 95th percentile for children playing at a day care center, a central tendency activity. Dermal absorption fraction is the fraction of contaminant absorbed through the skin. Values for selected contaminants are given in Table 9.3.

TABLE 9.3 Dermal Absorption Fraction for Soil

Contaminant	f_{abs}
Arsenic	0.03
Cadmium	0.0001
Chlordane	0.04
2,4-Dichlorophenoxyacetic acid	0.05
DDT	0.03
TCDD and other dioxins	0.03
If soil organic content > 10%	0.001
Benzo[*a*]pyrene and other PAHs	0.13
Arolcors 1254/1242 and other PCBs	0.14
Pentachlorophenol	0.25
Semivolatile organic compounds	0.1

Source: EPA 2004.

9.4 DOSE CALCULATIONS

9.4.1 Chemical Dose Calculations

The generic equation for total dose due to inhalation, ingestion, or skin penetration is

$$D_T = \int_0^{t_e} \frac{\mathrm{CR}(t)C(t)}{\mathrm{BW}(t)}\,dt \tag{9.8a}$$

where CR t is the contact rate: that is, volume of air per day [L^3/T], volume of water per day [L^3/T], or mass of food/soil per day [M/T]; and BW t is the body weight [M]. The parallel expression for average daily dose is

$$\tilde{\tilde{D}} = \frac{\int_0^{t_e} [\mathrm{CR}(t)C(t)/\mathrm{BW}(t)]/dt}{t_{\mathrm{avg}}} \tag{9.8b}$$

If the uptake rate of the contaminated medium, contaminant concentration, and body weight are constant over time, Eq. 9.8 reduces to

$$D_T = \frac{\mathrm{CR}\cdot Ct_e}{\mathrm{BW}} \tag{9.9a}$$

and

$$\tilde{\tilde{D}} = \frac{\mathrm{CR}\cdot Ct_e}{\mathrm{BW}(t)_{\mathrm{avg}}} \tag{9.9b}$$

For intermittent exposures, it is useful to modify Eq. 9.9 to explicitly take into account the frequency of contact on a yearly basis:

$$D_T = \frac{\mathrm{CR} \cdot C v_y t_e}{\mathrm{BW}} \tag{9.10a}$$

and

$$\tilde{\dot{D}} = \frac{\mathrm{CR} \cdot C v_y t_e}{\mathrm{BW}(t)_{\mathrm{avg}}} \tag{9.10b}$$

where v_y is the exposure frequency [T/T]. Although Eq. 9.10 applies to both continuous and intermittent exposures, it reduces to Eq. 9.9 for continuous exposures because $v_y = 365\,\mathrm{d/yr}$ is just a unit conversion factor.

▶ Example 9.2

A remediation worker is confined to an enclosed space containing high levels of airborne cadmium. The concentration is $20\,\mathrm{mg/m^3}$, and the worker is engaged in heavy lifting and moving of equipment prior to removal of waste drums. Removal of the equipment will take approximately 2 hours. What is the total dose to the worker?

Solution Acute exposures are quantified by the total dose, D_{T}, given by Eq. 9.1:

$$\begin{aligned} D_T &= \int_0^{t_e} \dot{D}(t)\,dt \\ &= \int_0^{t_e} \frac{\mathrm{CR}(t)C(t)}{\mathrm{BW}(t)}\,dt \end{aligned}$$

Since uptake rate and concentration are constant, this reduces to

$$D_T = \frac{\mathrm{CR} \cdot C t_e}{\mathrm{BW}}$$

For heavy activity, $\mathrm{CR} = 4.8\,\mathrm{m^3/h}$. Thus,

$$\begin{aligned} D_T &= \frac{(4.8\,\mathrm{m^3/h})(20\,\mathrm{mg/m^3})(2\,\mathrm{h})}{70\,\mathrm{kg}} \\ &= 2.74\,\mathrm{mg/kg} \end{aligned}$$

▶ Example 9.3

The air concentration of PCE in a dry cleaning establishment is $10\,\mathrm{mg/m^3}$. Calculate the average daily dose received by a worker exposed 8 hours a day, 5 days a week, and 50 weeks per year for 30 years.

Solution Since the uptake rate and concentration are constant, the average daily dose can be calculated by Eq. 9.10*a* with $\mathrm{CR} = 0.8\,\mathrm{m^3/h}$, $\mathrm{BW} = 70\,\mathrm{kg}$, and $v_y = (8\,\mathrm{h/d})(5\,\mathrm{d/wk})(50\,\mathrm{wk/yr}) = 2000\,\mathrm{h/yr}$:

$$\tilde{D} = \frac{\mathrm{CR}\cdot Cv_y t_e}{\mathrm{BW}\cdot t_{\mathrm{avg}}} = \frac{(0.8\,\mathrm{m^3/h})(10\,\mathrm{mg/m^3})(2000\,\mathrm{h/yr})(30\,\mathrm{yr})}{(70\,\mathrm{kg})(70\,\mathrm{yr})(365\,\mathrm{d/yr})}$$
$$= 0.268\,\mathrm{mg/(kg\cdot d)}$$

▶ **Example 9.4**

A suburban family living adjacent to a hazardous waste site uses well water to irrigate a vegetable garden that supplies the family with 20% of its vegetables over the course of the year. Due to leaching from the hazardous waste site, levels of mercury in the vegetables are, on average, 1.3×10^{-2} mg/g. The average rate of consumption of vegetables by adults in the family is 201 g/d. What is the lifetime average daily dose due to 25 years of exposure?

Solution Since this represents a continuous exposure (i.e., daily consumption of vegetables), the dose can be obtained from Eq. 9.9*b*, but the fraction of the food that is contaminated must be considered:

$$\tilde{D} = \frac{\mathrm{CR}\cdot Ct_e}{\mathrm{BW}\cdot t_{\mathrm{avg}}} = \frac{(0.2)(201\,\mathrm{g/d})(1.3\times10^{-2}\,\mathrm{mg/g})(25\,\mathrm{yr})}{(70\,\mathrm{kg})(70\,\mathrm{yr})}$$
$$= 2.67\times10^{-3}\,\mathrm{mg/(kg\cdot d)}$$

9.4.2 Radiological Dose Calculations

The terms internal and external when used in the context of radiological dose calculations refer to the location of the radioactivity. A dose due to radioactivity taken into the body as a result of inhalation, ingestion, or skin penetration is referred to as an **internal dose**. Similarly, a dose due to radioactivity located outside the body is referred to as an **external dose**.

9.4.2.1 Internal Dose The calculation of radiation internal dose is quite complex because it involves the calculation of both radionuclide transport in the body using a pharmacokinetic model and calculation of the absorbed dose to a particular organ due to radionuclides in that organ and in other body tissues. The task is simplified by the use of effective dose factors, which have been derived and made widely available for all radionuclides of technological interest (EPA 2002b). The dose factors provide the integrated dose (over a 50-year period of time) resulting from the instantaneous intake of 1 Bq of radioactivity. The dose factors take into account radionuclide transport in and removal from the body and the energy deposited in individual body organs for the 50-year period of integration. Since an increase in cancer risk is the principal concern associated with radioactivity taken into the body in environmental settings, the relevant dosimetric quantity is the effective dose, calculated as

$$E = I\cdot \mathrm{DF}_{E,\mathrm{int}} \tag{9.11}$$

where I [activity] is the total amount of radioactivity taken into the body during the exposure and $\mathrm{DF}_{E,\mathrm{int}}$ [dose/activity] is the effective dose factor. Presented in

Table 9.4 are effective dose factors for selected radionuclides. In terms of contact rate and contaminant concentration, the effective dose can be calculated from

$$E = \left[\int_0^{t_e} \mathrm{CR}(t)C(t)\,dt\right]\mathrm{DF}_{E,\mathrm{int}} \tag{9.12}$$

For a constant contact rate and contaminant concentration, Eq. 9.12 reduces to

$$E = \mathrm{CR}\cdot Ct_e\cdot \mathrm{DF}_{E,\mathrm{int}} \tag{9.13}$$

Equations 9.11 to 9.13 can be used for either acute or chronic exposures. When applied to chronic exposures, it is important to recognize that the dose factor applies to a 50-year period following exposure and may yield an inflated risk for the elderly; for example, when applied to a person exposed at age 20, the dose factor yields the dose received between ages 20 and 70; when applied to a person exposed at age 80, the dose factor yields the dose received between ages 80 and 130.

► **Example 9.5**

A facility that processes uranium routinely emits uranium into the atmosphere through process vents. The long-term average airborne concentrations of ^{235}U and ^{238}U at the fence line of the facility are 2×10^{-4} and 9×10^{-6} Bq/m^3, respectively. What is the effective dose from inhalation to a hypothetical person who spends 30 years at the fence line? The uranium is in a form that is removed slowly from the lungs.

Solution The effective dose can be calculated from Eq. 9.13. From Table 9.4, the inhalation dose factors (S lung clearance rate) for ^{235}U and ^{238}U (and their short-lived decay products) are 8.47×10^{-6} and 1.74×10^{-5} Sv/Bq, respectively. Letting the inhalation rate be 15.2 m^3/d, the effective dose for ^{235}U is

$$\begin{aligned} E &= \mathrm{CR}\cdot Ct_e\cdot \mathrm{DF}_{E,\mathrm{inh}} \\ &= (15.2\,\mathrm{m^3/d})(2\times 10^{-4}\,\mathrm{Bq/m^3})(365\,\mathrm{d/yr})(30\,\mathrm{yr})(8.47\times 10^{-6}\,\mathrm{Sv/Bq}) \\ &= 2.82\times 10^{-4}\,\mathrm{Sv} \end{aligned}$$

The effective dose for ^{238}U is

$$\begin{aligned} E &= \mathrm{CR}\cdot Ct_e\cdot \mathrm{DF}_{E,\mathrm{inh}} \\ &= (15.2\,\mathrm{m^3/d})(9\times 10^{-6}\,\mathrm{Bq/m^3})(365\,\mathrm{d/yr})(30\,\mathrm{yr})(1.74\times 10^{-5}\,\mathrm{Sv/Bq}) \\ &= 2.61\times 10^{-5}\,\mathrm{Sv} \end{aligned}$$

The total effective dose is the sum of the contributions from ^{235}U and ^{238}U:

$$\begin{aligned} E &= 2.82\times 10^{-4}\,\mathrm{Sv} + 2.61\times 10^{-5}\,\mathrm{Sv} \\ &= 3.08\times 10^{-4}\,\mathrm{Sv} \end{aligned}$$

9.4.2.2 External Dose External dose calculations must take into account radiation transport from the source to the receptor and subsequent interactions and energy deposition in body organs. Here again, the results of these calculations are available in terms of dose factors. Effective dose factors for radioactivity in the air and on the ground are given in Table 9.4. Unlike the dose factors for internal sources, which yield the dose for a 50-year period following intake, the external dose factors yield the instantaneous dose rate:

$$\dot{E}(t) = C(t) \cdot \mathrm{DF}_{E,\mathrm{ext}} \tag{9.14}$$

where $\mathrm{DF}_{E,\mathrm{ext}}$ is the external effective dose factor [(dose/T)/(activity/L^2) for radioactivity on the ground and (dose/T)/(activity/L^3) for submersion in air]. The total effective dose is obtained by multiplying the external effective dose factor by the time integral of concentration:

$$E_T = \mathrm{DF}_{E,\mathrm{ext}} \int_{t_1}^{t_2} C(t)\,dt \tag{9.15}$$

In assessing risks associated with some nuclear activities, specifically the impacts of accidental releases of radioactivity such as occurred at Chernobyl, the deterministic (i.e., noncarcinogenic) effects of acute external exposures could be important. Rigorously, this requires an estimate of equivalent dose, H, rather than effective dose, E. However, an initial estimate of deterministic risk can be made by letting $H \approx E$. If the results suggest deterministic effects (it was previously noted that deterministic effects become important for an acute equivalent dose in excess of 0.1 Sv), a more precise determination can be made by a health physicist.

► **Example 9.6**

The surface concentration of ^{137}Cs on the ground in certain regions of the former Soviet Union as a result of the Chernobyl accident is 500 kBq/m^2. What is the radiological dose to someone exposed 10 hours a day for 20 years?

Solution The problem solution can be formulated in either of two ways. In the first, E_T can be obtained from Eq. 9.15:

$$E_T = \mathrm{DF}_{E,\mathrm{ext}} \int_{t_1}^{t_2} C(t)\,dt$$

where $C(t) = C_0 \exp(-kt)$. Alternatively, the total effective dose can be obtained from Eq. 9.1 with D being replaced by E:

$$E_T = \int_{t_0}^{t_e} \dot{E}(t)\,dt$$

where $\dot{E}$ is given by Eq. 9.14:

$$\dot{E}(t) = C(t) \cdot \mathrm{DF}_{E,\mathrm{ext}}$$

and

TABLE 9.4 Radiation Effective Dose Conversion Factors[a]

Radionuclide	Half-Life	Inhalation[b] (Sv/Bq)	Ingestion (Sv/Bq)	Atmospheric Submersion [(Sv/s)/(Bq/m^3)]	Ground Irradiation [(Sv/s)/(Bq/m^2)]
^{3}H (as HTO)	12.2 yr	1.83×10^{-11}	1.92×10^{-11}	n/a	n/a
^{60}Co	5.2 yr	5.27×10^{-9} (F) 1.02×10^{-8} (M) 3.07×10^{-8} (S)	3.42×10^{-9}	1.19×10^{-13}	2.30×10^{-15}
^{85}Kr	10.8 yr	n/a	n/a	2.40×10^{-16}	1.05×10^{-17}
^{88}Kr	2.84 h	n/a	n/a	9.72×10^{-14}	1.72×10^{-15}
^{90}Sr/^{90}Y	28.8 yr/64 h	2.44×10^{-8} (F) 3.70×10^{-8} (M) 1.59×10^{-7} (S)	2.77×10^{-8}	8.91×10^{-16}	1.12×10^{-18}
^{131}I	8.0 d	7.39×10^{-9} (F) 2.44×10^{-9} (M) 1.60×10^{-9} (S) 1.98×10^{-8} (V) 1.54×10^{-8} (O)	2.18×10^{-8}	1.69×10^{-14}	3.64×10^{-16}
^{137}Cs/^{137}Ba	30 yr/2.5 m	4.67×10^{-9} (F) 9.69×10^{-9} (M) 3.92×10^{-8} (S)	1.36×10^{-8}	2.70×10^{-14}	5.81×10^{-16}
^{138}Xe	14.1 min	n/a	n/a	5.48×10^{-14}	1.07×10^{-15}
^{222}Rn/decay products[c]	3.8 d	(3.0×10^{-8})[d]	(3.5×10^{-9})[e]	7.24×10^{-14}	1.44×10^{-15}

^{226}Ra	1,600 yr	3.59×10^{-7} (F) 3.46×10^{-6} (M) 9.51×10^{-6} (S)	1.03×10^{-8}	5.59×10^{-15}	6.11×10^{-18}
^{235}U/^{231}Th[f]	7.0×10^{8} yr/ 26 h	5.21×10^{-7} (F) 3.09×10^{-6} (M) 8.47×10^{-6} (S)	4.7×10^{-8}	6.94×10^{-15}	1.56×10^{-16}
^{238}U/decay products[f,g]	4.5×10^{9} yr	1.06×10^{-6} (F) 3.43×10^{-6} (M) 1.74×10^{-5} (S)	9.74×10^{-8}	5.45×10^{-12}	9.06×10^{-14}
^{239}Pu	24,100 yr	1.19×10^{-4} (F) 5.01×10^{-5} (M) 1.60×10^{-5} (S)	2.51×10^{-7}	1.26×10^{-14}	1.32×10^{-15}

Source: Unless noted otherwise, values are from EPA (2002b).

[a] n/a, not available.

[b] Lung clearance class indicated in parentheses (ICRP 1994). F is fast dissolution (100% dissolved rapidly), M is moderate dissolution (10% dissolved rapidly), and S is slow dissolution (0.1% dissolved rapidly). For ^{131}I, V is vapor and O is the organic form, which is methyl iodide.

[c] Decay products are ^{218}Po ($t_{1/2} = 3.05$ min), ^{214}Pb ($t_{1/2} = 26.8$ min), ^{214}Bi ($t_{1/2} = 19.9$ min), and ^{214}Po ($t_{1/2} = 64\,\mu$s).

[d] Based on a back calculation from the BEIR VI (NAS–NRC 2000) risk estimate of 1.6×10^{-4} $(\text{Bq/m}^3)^{-1}$ for a lifetime exposure in the home. The back calculation used Eq. 9.13 with $R = \rho E$, $\rho = 8 \times 10^{-2}$/Sv, CR = 15.2 m^3/h, and $t_e = 50$ yr.

[e] NAS–NRC 1999.

[f] For ^{238}U and ^{235}U, the values include the contribution to dose from short-lived decay products and thus would be applicable to processed uranium (i.e., uranium that has been separated from ore deposits). These values would not be applicable to undisturbed uranium in the natural environment due to the presence of additional decay products that build up over long periods.

[g] Decay products include ^{234}Th ($t_{1/2} = 24.1$ d), ^{234m}Pa($t_{1/2} = 1.17$ min), and ^{234}U ($t_{1/2} = 2.44 \times 10^5$ yr).

$$E_T = \mathrm{DF}_{E,\mathrm{ext}} \int_{t_1}^{t_2} C(t)\,dt$$

which is identical to Eq. 9.15. Substituting and integrating yields

$$\begin{aligned} E_T &= \mathrm{DF}_{E,\mathrm{ext}} \int_0^{t_e} C_0 \exp(-kt)\,dt \\ &= \frac{\mathrm{DF}_{E,\mathrm{ext}} C_0}{k} [1 - \exp(-kt_e)] \end{aligned}$$

The half-life of ^{137}Cs is 30 years and

$$\begin{aligned} k &= \frac{\ln 2}{t_{1/2}} = \frac{\ln 2}{30\,\mathrm{yr}} \\ &= 2.31 \times 10^{-2}\ \mathrm{yr}^{-1} \end{aligned}$$

From Table 9.4, the value for ground irradiation due to ^{137}Cs is 2.11×10^{-12} [(dose/T)/(activity/L^2)]. Note that the table lists ^{137m}Ba with ^{137}Cs because it is a decay product of ^{137}Cs, which reaches equilibrium within about 10 minutes of the release of pure ^{137}Cs. This means that ^{137m}Ba has the same activity and decays at the same rate as ^{137}Cs. Thus, in virtually all environmental settings, the two are found together. Substituting into the equation for E_T, we have

$$\begin{aligned} E_T = {} & \frac{(500\,\mathrm{kBq/m^2})(10^3\ \mathrm{Bq/kBq})[5.81 \times 10^{-16}(\mathrm{Sv/s})/(\mathrm{Bq/m^2})](3.15 \times 10^7\ \mathrm{s/yr})}{0.0231\,\mathrm{yr}^{-1}} \\ & \{1 - \exp[(-0.0231\,\mathrm{yr}^{-1})(20\,\mathrm{yr})]\} \\ = {} & 0.396\,\mathrm{Sv}\,(1 - 0.630) = 0.147\,\mathrm{Sv} \end{aligned}$$

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PROBLEMS

9.1 A residential development has been constructed close to a landfill that was closed several years ago. The soil in the developed area is contaminated with PCBs at a level of 2.3 mg/kg. Estimate the mean average daily dose due to ingestion for a young child living in the area and playing in the contaminated soil.

9.2 The atmospheric concentration of cadmium in an industrial facility is 25 μg/m^3. Estimate the average daily dose received by a worker exposed 8 hours a day, 5 days a week, and 50 weeks per year for 30 years for an inhalation rate of 1.6 m^3/h.

9.3 Due to leaks from a ruptured process sewer, groundwater is contaminated with TCE at a concentration of 0.3 mg/L. Determine the lifetime average daily dose due to ingestion of contaminated drinking water at 1.5 L/d for 20 years.

9.4 An apartment complex is located adjacent to a lead smelting factory. Due to atmospheric emissions from the factory, soil concentrations of lead at the apartments are 100 mg/kg. Estimate the average daily dose to a pica child living in the complex for a period of 4 years. The child's average body weight over the period of exposure is 30 kg and the child ingests, on average, 1 gram of soil per day.

9.5 Groundwater near an industrial area is contaminated with benzene at a concentration of 1 μg/L. Make a conservative estimate of the lifetime average daily dose rate from dermal absorption through bathing by neglecting benzene volatilization from the water.

9.6 As a result of the Chernobyl accident, the average concentration of cesium-137 in food samples in the Ovruch region of the Ukraine is 800 Bq/kg_{wet}. If this concentration is representative of fruits, vegetables, meat, and dairy products, what is the mean effective dose rate (Sv/yr) due to consumption of contaminated food in the region?

9.7 Aflatoxin is a naturally occurring toxic mold that is found in peanut butter among other products. If the average concentration of aflatoxin in peanut butter is 3 ppb and the average amount of peanut butter in a sandwich is 30 g, what is the lifetime average daily dose to a 70-kg adult who eats one sandwich a day for 20 years?

9.8 What effective dose does a person receive by drinking water containing tritium at a concentration of 40 Bq/L at a rate of 1 L/d for 15 years?

9.9 **(a)** Consider the Chernobyl-like accident described in Problem 7.15 and a person located 2000 m downwind from the release. Determine the external radiation effective dose from ^{138}Xe in the atmosphere for the 2-hour period following the release.

(b) Repeat Problems 7.15 and 9.9(a) for ^{88}Kr ($t_{½} = 2.8$ h), which has an inventory of 4.5×10^{17} Bq (12×10^{6} Ci).

(c) The majority of the external radiation dose in the first few hours following the accident is due to ^{88}Kr and ^{138}Xe. What is the effective dose to the person due to the combination of these two radionuclides?

9.10 In occupational settings, the effective dose cannot exceed 0.02 Sv from the ingestion or inhalation of radioactive material during a one-year period. Find the maximum allowable airborne concentration of ^{239}Pu for an exposure period of 2000 hours (corresponding to 40 hours per week for 50 weeks). Assume fast (F) absorption of ^{239}Pu.

10 Basic Human Toxicology

10.1 INTRODUCTION

Understanding the risk resulting from exposure to a contaminant requires knowledge of both the dose that a person receives as a result of exposure and the potential health effects of the contaminant. In Chapter 9 we focused on approaches for quantifying the exposure of a person to actual or potential releases of contaminants into the environment. The next step of the risk assessment procedure is to characterize the impact of contaminant exposures on human health and make quantitative estimates of health effects. This chapter, which is the first of two dealing with consequence assessment in the context of human health, focuses on the ways in which contaminants can disrupt normal development and functions, including reproduction, in the human body. In Chapter 11 we address the methods by which the human health consequences of exposure to a contaminant are quantified.

Characterization of the potential health effects of exposure to environmental contaminants lies within the field of toxicology, which is the science that deals with "poisons" and their effects. The concept of a "poison" is described by Klaasen and Eaton (1991): "One could define a poison as any agent capable of producing a deleterious response in a biologic system." This is not, however, a useful working definition, for the very simple reason that virtually every known chemical has the potential to produce injury or death if present in sufficient amount. Paracelsus (1493–1541) phrased this well when he noted: "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy." The notion that "the dose makes the poison" is illustrated in Table 10.1, where the dose that is lethal to 50% of exposed rats is given for substances ranging from table sugar to aflatoxin.

The purpose of this chapter is to provide a condensed treatment of some of the basic biological mechanisms that give rise to toxic effects from chemicals and radiation. In many cases toxicological information on contaminants is summarized and compiled by government agencies and others. In other cases (e.g., a new industrial use of a chemical compound), additional toxicological studies might be required to characterize the risk. The information presented in this chapter is intended to assist the risk analyst in understanding the biological bases of potential toxic effects discussed in such reports.

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

TABLE 10.1 Approximate Oral LD_{50} Values in Rats

Chemical	LD_{50} (mg/kg body weight)
Sucrose (table sugar)	29,700
Sodium chloride (common salt)	3,000
Vanillin	1,580
Aspirin	1,000
Copper sulfate	960
Chloroform	908
Caffeine	192
DDT	113
Nicotine	53
Strychnine	16
Sodium cyanide	6
Aflatoxin B1	5

Source: Environ 1988.

10.2 FUNDAMENTALS OF ANATOMY AND PHYSIOLOGY

Scientists arrange the components of the body into a hierarchy of systems. At its most basic, the body consists of a structured arrangement of water and chemical compounds. These chemical compounds include both complex macromolecules and dissolved electrolytes. **Cells**, the fundamental building blocks of a living organism, consist of a closed membrane encapsulating structured assemblages of organic macromolecules, inorganic electrolytes, and water. The human body comprises a system of specialized, interdependent cells that are structurally and functionally related. Cells are assembled into a higher level of organization in which a **tissue** is defined in terms of the types of cells, the way in which the cells are connected, and the function that the assemblage performs. Various tissues, in turn, are grouped together to create **organs**. Organs that work together to carry out major functions of the body, such as digestion or locomotion, comprise **organ systems**. Organ systems are physiological constructs centered around a particular body function, while an organ is an anatomical construct centered around an anatomical feature. One organ may be part of more than one organ system (e.g., the large intestine is part of the digestive system but plays an important role in the immune system). These components carry out their functions by a series of controlled physical and chemical changes. **Anatomy** is the study of the structure of these components. **Physiology** is the study of the function of a component as expressed by its structure and the physical and chemical changes that it undergoes to fulfill its role in the body.

At the heart of toxicology is an understanding of the normal biological functioning that the contaminant disrupts to produce a characteristic effect. **Homeostasis**, a central concept in biology, is defined as the "normal, internal stability in an organism maintained by coordinated responses of the organ systems that automatically compensate for environmental changes" (NLM 2005). A number of biological systems must work together for the body to maintain homeostasis. In the following sections we provide an overview of the normal human physiology and a description

of ways in which contaminants exert their deleterious effects by disrupting various homeostatic mechanisms.

10.2.1 Cellular Anatomy and Physiology

Cells contain a variety of complex organic macromolecules and dissolved inorganic electrolytes. Four types of **macromolecules** are of particular importance in describing physiologic function: nucleic acids, proteins, lipids, and carbohydrates. **Nucleic acids** such as DNA and RNA are formed by chains of nitrogenous bases in a specific sequence and shape. There are two types of these nitrogenous bases: purines (guanine and adenine) and pyrimidines [cytosine and thymine (DNA) or uracil (RNA)]. Purines and pyrimidines are complementary in that guanine is always paired with cytosine, and adenine is always paired with either thymine or uracil. This pairing gives rise to the double helical structure of DNA identified by Watson and Crick (1953). The complementary nature of the two strands of DNA that follow from the principle of base pairing is of central importance in biology, as it ensures that a complete DNA molecule can be accurately reproduced from only one of the strands. These chains of bases serve as "blueprints" from which cellular components are synthesized. Triplets of the four base pairs in DNA, known as *codons*, code for specific amino acids. Although there are 64 possible triplet combinations of the four base pairs, the codons code only for 20 possible amino acids.

Proteins are synthesized from the patterns encoded in DNA using these 20 amino acids. These amino acids are joined in a specific sequence, originating from the DNA, that determines the structure and function of the protein. Proteins are the most abundant organic substances in the body and serve a wide range of functions. Enzymes catalyze biochemical reactions (e.g., digestion of fats, sugars, and proteins, and synthesis of complex molecules). Antibodies are important in allowing the immune system to recognize foreign substances in the body. Protein hormones such as insulin serve to carry messages throughout the body by binding to membrane-embedded proteins. Proteins also are specialized for transporting other chemical substances in systemic circulation (e.g., the iron in hemoglobin is used to transport oxygen from the lungs to the cells, where it is needed for respiration). Fibrous proteins provide structural support to cells (e.g., the collagen that connects tissues and the keratin that provides the toughness of skin cells); and allow them to alter their shape by contracting, conferring the ability for movement (e.g., actin and myosin in muscle cells).

Lipids include a variety of water-insoluble molecules including fats (triglycerides) that serve as energy reserves, phospholipid molecules that serve as a major structural component of cellular membranes, and steroids that function as hormones (e.g., cholesterol, estrogen, and testosterone). **Carbohydrates** are compounds composed of carbon, hydrogen, and oxygen that serve as an energy source for the cell and are also used in supporting cellular structures. They are typically present in animal cells as glycogen and in plant cells as starch or cellulose; however, ribose and dexoxyribose linked through phosphate groups are sugars that form the backbone of RNA and DNA, respectively. Electrolytes such as sodium, potassium, magnesium, calcium, phosphate, sulfate, and bicarbonate are present in solution within bodily fluids. Chemical reactions involving electrolytes allow the cells to maintain the chemical environment necessary for cellular survival and function, to

express the biological responses characteristic of the cell (e.g., maintenance of the voltage difference across nerve cell membranes by potassium and sodium), and to modify protein products to allow them to carry out their function (e.g., phosphorylation of adenosine).

The primary components of the cell are the cytosol, the cytoskeleton, the cell membrane, and various organelles. These components are illustrated in Figure 10.1*a*. The **cytosol** is the fluid comprising the interior of the cell. It consists largely of water, proteins, and electrolytes. Within the cell, rigid fibrous proteins form the cytoskeleton, and interior membranes isolate cellular components to form organelles. The **cytoskeleton** includes both solid structural members and hollow passages that regulate the transport of intracellular compounds within the cell. **Cellular membranes** contain the cellular contents and control the entry and excretion of material from the cell. They are composed of two layers of phospholipid molecules: a hydrophobic (water-insoluble) region and hydrophilic (water-soluble) region. These molecules are oriented such that the external surfaces of the membrane are hydrophilic and the internal surfaces are hydrophobic. This is illustrated in Figure 10.1*b*. The hydrophobic internal region of the membrane impedes the passage of water through the membrane. Such a structure protects the cell against foreign substances by greatly impeding the diffusion of compounds outside the cell through the cellular membrane. However, cellular membranes cannot be completely impermeable. Cells must take in material to survive, and they must excrete waste products. The membrane therefore contains a variety of embedded proteins that assist in transferring materials into and out of the cell and in receiving "chemical signals" from extracellular compounds via receptor–ligand interactions.

■ Ligands and Receptors

A **ligand** is a molecule, ion, or group that can form a chemical complex by binding to another chemical, **a receptor**. In the body, there are ligands in systemic circulation that bind only to very specific receptors. Cellular membranes and interiors contain proteins known as receptors that form complexes only with specific ligands. Formation of a complex by one of these receptor proteins with a ligand causes the cell to express some specific programmed biological response. Other cells typically secrete the extracellular ligands. The secretion, transport, and binding of ligands to receptor proteins therefore constitute an important messaging system for communication between cells. Many biological processes in the body occur through receptor–ligand interactions. Important ligands include hormones (ligands carried in systemic circulation that act between different cell types) and neurotransmitters (ligands that exist in the gaps between neurons to transmit nerve signals from one neuron to another). Exogenous compounds that are able to attach to receptor sites normally used by endogenous ligands can interfere with these messaging systems by triggering or preventing the programmed response and breaking the feedback loops that allow homeostatic control of the body.

Organelles are membrane-enclosed regions of the cytoplasm that allow specific cellular functions to be carried out. A few of the more important organelles include

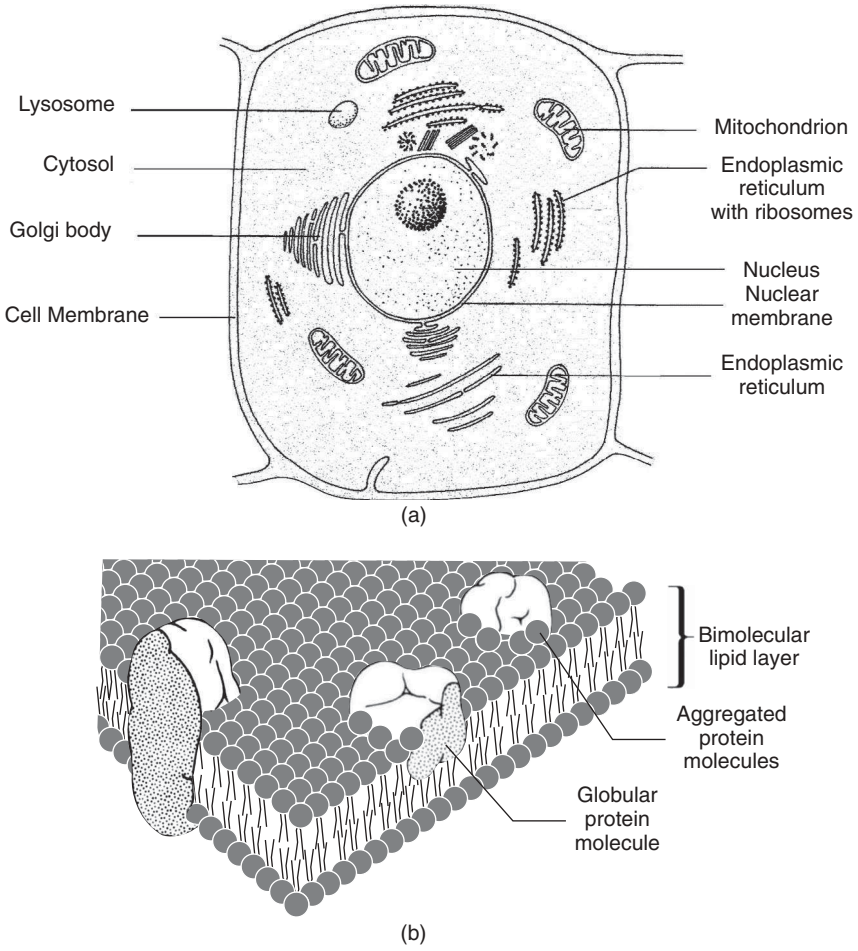

Figure 10.1 Cellular anatomy: (*a*) cell components (from Burke 1980; reprinted by permission of John Wiley & Sons, Inc.); (*b*) cellular membrane (from McClintic 1985; reprinted by permission of John Wiley & Sons, Inc.).

the nucleus, ribosomes, endoplasmic reticulum, Golgi apparatus, and mitochondria. The **nucleus** is the most prominent organelle in the human cell and is a center for cell regulation and reproduction. The nucleus contains strands of nucleic acids [primarily, deoxyribonucleic acid (DNA)] that serve as a "master blueprint" for the function and reproduction of the organism. All normal cells in a human, except for germ cells, contain identical DNA. For normal functioning of succeeding cell generations, the DNA must remain intact and be replicated when the cell divides. The complementarity of base pairs serves as the primary mechanism for ensuring that DNA remains intact by allowing accurate reproduction of DNA strands during cell division and by allowing special enzymes to catalyze DNA repair of damaged sections by reading the base pairs on the opposite strand. Messenger ribonucleic acid (RNA), the working blueprints from which the cell manufactures proteins, are generated from the master blueprint contained in the DNA by the actions of the

enzyme RNA polymerase. The RNA polymerase enzyme reads a specified section of the DNA to generate an RNA molecule, starting at DNA sections known as promoters and stopping when a termination signal is reached in the DNA sequence. This process of generating single-stranded RNA from the double-stranded DNA chain is known as transcription. Following transcription, the RNA may be subject to further processing prior to its use in protein synthesis.

Ribosomes, comprising an RNA subunit and a protein subunit, are centers for protein synthesis within the cell. Ribosomal RNA reacts with messenger RNA to synthesize the particular amino acid sequence coded by the RNA strand in a process that is termed translation. This amino acid sequence, once post-processed, will constitute the protein that is the product of the original DNA sequence read by the RNA polymerase. Free ribosomes (ribosomes that are not attached to any internal membrane) typically synthesize cytosolic proteins. The **endoplasmic reticulum** and the **Golgi apparatus** serve as centers for post-translational processing of proteins destined for incorporation into a cellular membrane or for secretion from the cell. Ribosomes found embedded within the membranes of the rough endoplasmic reticulum synthesize these proteins. The Golgi apparatus is involved in further processing and delivery of the synthesized proteins to their final destination. The **mitochondria** are the power plants of the cell. Oxidation of carbohydrates provides energy for cellular processes. The energy released by cellular respiration is stored in the phosphate–phosphate bonds of adenosine triphosphate (ATP). Hydrolysis of one of the ATP phosphate bonds releases this stored energy and is coupled to biochemical reactions that require the input of energy, such as active transport of biologically required chemicals through the cell membrane or protein synthesis. The resulting adenosine diphosphate (ADP) molecule is then again phosphorylated back to ATP to complete the cycle.

The number of cells in the body is not constant. New cells are constantly being generated to allow the body to grow or to replace cells that are destroyed by normal physiological processes or as a result of injuries. The phase in which active cell division results in a generation of new cells is known as mitosis. Cells that are not actively undergoing cellular division are considered to be in interphase, which can be further divided into three distinct phases. The cell spends most of its life cycle in a gap phase (G1), which is the period of normal cellular activity when cell growth, protein synthesis, and other characteristic cellular activities are carried out. A cell prepares for division by duplicating its DNA in a second phase (S) and then preparing for the mitotic phase in a separate, typically shorter, gap phase (G2). Although all of the cells in a person originate from the division of the single cell formed by the fertilized egg, successive differentiation of cells during cell division eventually results in a tremendous variety of cells in a human body. Differentiation occurs as a result of tightly regulated expression of different genes, mainly during the embryonic development process. The end products of cellular differentiation are the highly specialized cells that make up the variety of human tissues.

10.2.2 Cellular Mechanisms of Toxicity

Most compounds exert their toxic effect through interactions at the cellular level. There are several basic mechanisms through which a chemical compound interferes with normal cellular function (Klaasen and Eaton 1991). These mechanisms include

direct damage to cellular components, interference with normal cellular function, or disruption of communication between cells. Toxic agents can damage cells directly by disrupting cellular energy production or interfering with calcium homeostasis. Interference with ATP production and utilization can result in cellular degradation and death. Chemicals that interfere with transport of oxygen, such as hydrogen sulfide, can also lead to a degradation of cellular energy production. Interference with calcium homeostasis can lead to cell death through disruption of cellular mechanisms regulated by calcium as well as through disruption of the cytoskeleton leading to degradation of cellular structure.

Within the cell, the macromolecules described above must work in concert to allow the cell to survive and carry out its particular function. A contaminant may either damage critical macromolecules directly or may damage them indirectly by forming free radicals. Examples of cellular malfunction induced by direct interference with macromolecules are the binding of carbon monoxide to the iron in hemoglobin, thus impairing the ability of the blood to deliver oxygen to tissues, and ionizing radiation damage to DNA as a result of ionization of an atom in the DNA molecule. Indirect damage accounts for a wide array of toxic effects. For example, in addition to its direct effects, ionizing radiation can also damage DNA indirectly by the formation of free radicals, which subsequently react with and damage the DNA. Chemical contaminants are also suspected of exerting toxic or carcinogenic effects by the generation of highly reactive species. Other forms of DNA damage include strand breaks and cross-links between DNA strands caused by free radical attack on genetic material or by the absorption of energy from ionizing radiation and chromosomal aberrations and rearrangements. Damage to nucleic acids can inhibit or alter the expression of critical gene products, thereby leading to cell death, cellular malfunction, or heritable genetic effects. If the damage to DNA is unrepaired, this damage can lead to mutations in the cell as it divides and replicates its DNA. Although DNA is regularly monitored and repaired by special enzymes, these processes are not error free. In addition, some DNA repair processes themselves can introduce mutations when attempting to repair damaged DNA. Unrepaired genetic damage or inaccurate repair can lead to genetic mutations that may be passed on to cells derived from the mutated cell. Mutations of somatic cells may lead to cancer, and mutations of germ cells may lead to hereditary disorders.

■ Free Radicals

A free radical is a highly reactive chemical species containing an unpaired electron in its outer electron shell. Some free radicals occur naturally in the body; however, additional free radicals may be derived from interactions of normal body compounds with chemical contaminants in the body or with ionizing radiation. **Radiolysis** is the process by which ionizing radiation breaks water, inorganic, or organic molecules into highly reactive species. For example, radiolysis of water can result in the formation of the hydroxyl (OH·) and hydrogen (H·) free radicals. These free radicals can then react with other aqueous species to form other highly reactive species. Although DNA is one of the target macromolecules for these reactive species, other macromolecules can also be damaged by them. For example,

lipids can be damaged by peroxidative attack. Because lipids are a primary component of cellular membranes, such damage can lead to ruptures in the cell membrane and consequent cell death.

► **Example 10.1**

Carbon tetrachloride, widely used as a solvent, has been implicated in liver damage. The primary effect is accumulation of fatty deposits within liver cells followed by cellular necrosis. Various elements of the cells are damaged by carbon tetrachloride. Although the exact mechanisms are unclear, it appears that a reactive metabolite of carbon tetrachloride attacks cellular macromolecules, interfering with cellular function and potentially resulting in cell death. Other halogenated alkanes and alkenes (e.g., PCA, DCA, TCA, TCE) seem to follow similar mechanisms of action, albeit with different levels of response. However, all of these compounds produce reactive metabolites and are thus implicated in varying degrees with carcinogenesis as well.

Finally, chemicals can block the communication functions of the body. Some chemicals interfere with receptor–ligand interactions. These chemicals frequently target the nervous or endocrine systems. Chemical contaminants can act as exogenous ligands that interfere with the chemical messaging systems described previously, thereby disrupting the normal pattern of chemical communication within the body. Other chemicals can disrupt physiological communication systems by interfering with excitable membranes. The effect of DDT on the nervous system (by blocking open a sodium gate on the axonal membrane, thus degrading the ability of the cell to achieve a normal resting state) is a prime example. Interference with calcium metabolism can also lead to disruption of intercellular communication, as calcium frequently serves as a second messenger in the regulation of intracellular functions.

10.2.3 Major Organ Systems

There are 12 major organ systems in the human body. The digestive and urinary systems process food for energy and eliminate waste products; the respiratory system provides the source of oxygen necessary for oxidative energy production; the cardiovascular, lymphatic, and immune systems circulate oxygen, nutrients, and other compounds within the body and provide a communication network to regulate the function of the various specialized cells and to ward off disease; the nervous and endocrine systems coordinate the actions of the various organ systems; the integumentary, muscular, and skeletal systems provide protection, support, and locomotion for the body; and the reproductive system allows the survival of the species by reproduction. In the discussion that follows we provide an overview of these organ systems, with an emphasis on those of particular significance as either a route of exposure, a target of toxic action, or both. Although the toxic action frequently occurs at the point of entry of the toxin (inhalation, ingestion, dermal exposure), organs in other systems may be affected by migration of the toxin within

the body. For example, inhalation of carbon tetrachloride through the respiratory system may have a large impact on the liver, which is part of the digestive system.

All of the organs are composed of variations of four basic tissue types. **Epithelial tissues** are membrane tissues that often serve as selective barriers, allowing the passage of beneficial compounds and blocking the passage of harmful compounds. **Connective tissues** provide structure for the network of cells. **Nerve tissues** are typically specialized for the transmission of information in the form of electrical signals. **Muscular tissues** are characterized by an ability to expand and contract, imparting the potential for movement to individual tissues and the organism as a whole. Most organs contain all four types of tissues; it is the type and arrangement of these tissues that defines how the organ carries out its particular functions.

10.2.3.1 Digestive and Urinary Systems The digestive and urinary systems process food for energy and eliminate waste products. These two systems are of particular significance for environmental toxicology in their role as portals for the entry and exit of environmental contaminants to and from the body. The digestive system is one of the primary routes by which environmental contaminants are taken into the body and transformed chemically, and it is the main route by which insoluble waste materials are excreted. The urinary system plays a key role in the filtration and elimination of soluble toxic substances and is a primary route by which soluble waste products and excess water are eliminated. A simplified representation of the routes through which environmental contaminants can enter and leave the body through the digestive and urinary systems is provided in Figure 10.2, which is the basis for a mathematical model used for estimating the effects of ingested radioactive materials. Use of this model is discussed in more detail in Example 11.1.

The digestive system consists of the alimentary canal and several accessory glands. Food and water enter the body through the mouth and pass through the esophagus into the stomach. Within the stomach, secreted hydrochloric acid and enzymes combine with muscular contractions to reduce the food to a semiliquid, highly acidic (pH 0.9 to 1.5) mass, which is then expelled into the small intestine. The small intestine is in the form of a coiled tube about 7 m in length. It is lined with a layer of epithelial tissue that allows absorption of the nutrients in solution. This is surrounded by a layer of tissue that is highly penetrated by small blood vessels. A layer of connective and muscular tissues supports these layers and moves the food mass through the tube by peristaltic action. Within the small intestine, the food is further metabolized by enzymatic action and absorbed into the bloodstream or the lymphatic system. Bile, produced by the liver, is an alkaline emulsifying agent that raises the pH and breaks down fat particles to aid in their absorption. After passage through the small intestine, the food enters the large intestine, where the water is reabsorbed and waste products are concentrated into a fecal mass. Waste products comprise both indigestible foodstuffs and excess chemical constituents (calcium, magnesium, iron, phosphates) that must be eliminated to maintain the proper chemical balance of the body. This mass is then expelled through the rectal cavity.

Several accessory glands work in conjunction with the alimentary canal to carry out digestion and metabolism. The salivary glands, the liver, the gallbladder, and

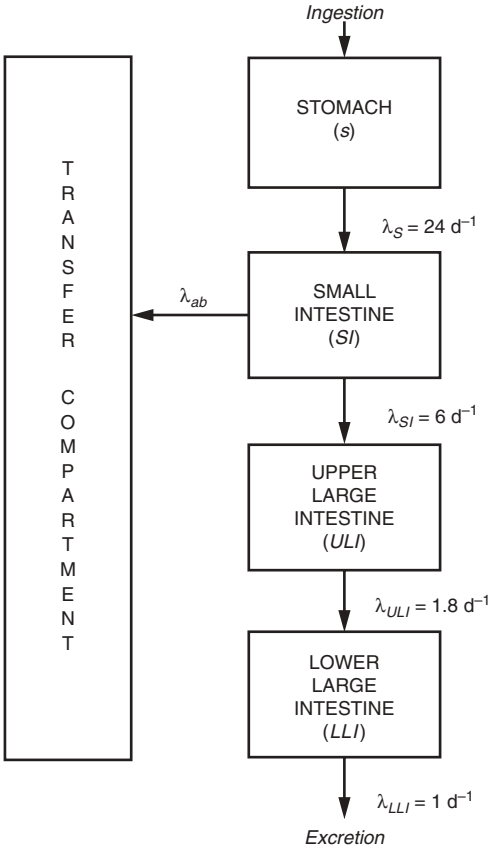

Figure 10.2 ICRP model of the digestive system. (From Till and Meyer 1983.)

the pancreas secrete enzymatic fluids that aid in digestion. Nutrients absorbed from the small intestine are carried by blood to the liver. In the liver, the nutrients are metabolized and released into the circulatory system for delivery to the different parts of the body. The liver serves a variety of functions, the most important of which revolve around its role in digestion and the metabolism of nutrients absorbed from the small intestine. The metabolic action of the liver is a major mechanism for detoxifying substances absorbed along with the nutrients. The liver also plays an important role in the circulatory system through the synthesis of blood proteins, the destruction of old red blood cells, and the storage of iron and vitamins.

Because of the liver's many functions, contaminants can act in a variety of ways to cause liver injury. The principal types of liver damage are necrosis (tissue destruction), cirrhosis (hardening of tissue), and the accumulation of abnormal amounts of fat. A particularly important function of the liver from the standpoint of poisons is the detoxification of compounds by transforming them into a more water-soluble form. Whereas this is an important protection mechanism for some compounds, others that are relatively benign in their administered form can be transformed into more toxic species. This process, known as **metabolic activation**,

is carried out by enzymatic action. For example, the nonmetabolized form of carbon tetrachloride affects primarily the nervous system. However, the critical effect for carbon tetrachloride is typically taken to be liver toxicity, due to metabolites of carbon tetrachloride (ATSDR 2005a). Although the enzymes responsible for metabolic activation are present throughout the body, they are particularly abundant in the liver. Electrophilic intermediate metabolites, produced by metabolic activation of certain compounds, can react with DNA, leading to the initiating step in carcinogenesis.

The kidneys, ureters, urinary bladder, and urethra make up the urinary system, which is the second major route of excretion for waste products. The kidney serves several functions, including the regulation of the water balance and the electrolytic balance of the blood. It carries out this function by the selective filtration of the blood and plasma to remove waste products for elimination from the body. Reabsorption of water, salts, sugar, amino acids, and other essential components such as calcium serves to maintain the water and electrolytic balance of the body. The excess water and waste products are removed from the body through the ureters, bladder, and urethra. The kidney also secretes hormones that regulate red blood cell production, blood pressure, and calcium levels.

Toxic insults to the kidney can result in either decreases in the blood flow, reduction in the removal of wastes, excessive elimination of required chemicals, or an alteration in enzyme production. A variety of contaminants, including metals such as lead, mercury, and uranium; halogenated hydrocarbons such as chloroform, TCE, and PCE; and organic solvents such as ethylene glycol and toluene are known to induce renal (i.e., kidney) dysfunction (Hewitt et al. 1991).

10.2.3.2 Respiratory System The respiratory system is responsible for the intake of oxygen necessary for energy production and the exhalation of waste gases from the body. In the context of toxicology, the respiratory system is of considerable significance because it is the primary route of entry for exposure to airborne contaminants, which may be in a gaseous state or in the form of aerosols (microscopic solid or liquid particles suspended in air). As depicted in Figure 10.3, the respiratory system consists of a series of passages that carry air from the nasal or oral cavities through the trachea into the lungs. Within the lungs, the trachea branch successively into the bronchi, bronchioles, and alveoli. A simplified representation of the routes through which airborne contaminants can enter through the respiratory system and be distributed throughout the body is presented in Figure 10.4. This compartmental model is the basis for a mathematical model used for estimating the effects of inhaled radioactive materials. The use of such models is discussed in more detail in Chapter 11.

The respiratory system is equipped with natural defenses against airborne particulate matter. The nasal cavities are lined with epithelial cells that secrete mucus and support hairs that filter and trap airborne particulates. Particles larger than approximately 10 μm in diameter are generally trapped within the nasal cavity, from where they are either expelled or are swallowed and subsequently eliminated through the alimentary canal. Smaller particles (1 to 10 μm) can be trapped within the tracheobronchial region and subsequently cleared upward into the esophagus, from where they are swallowed and eliminated through the alimentary canal. Respirable particles (less than 1 μm) and gases are inhaled into the alveoli, which

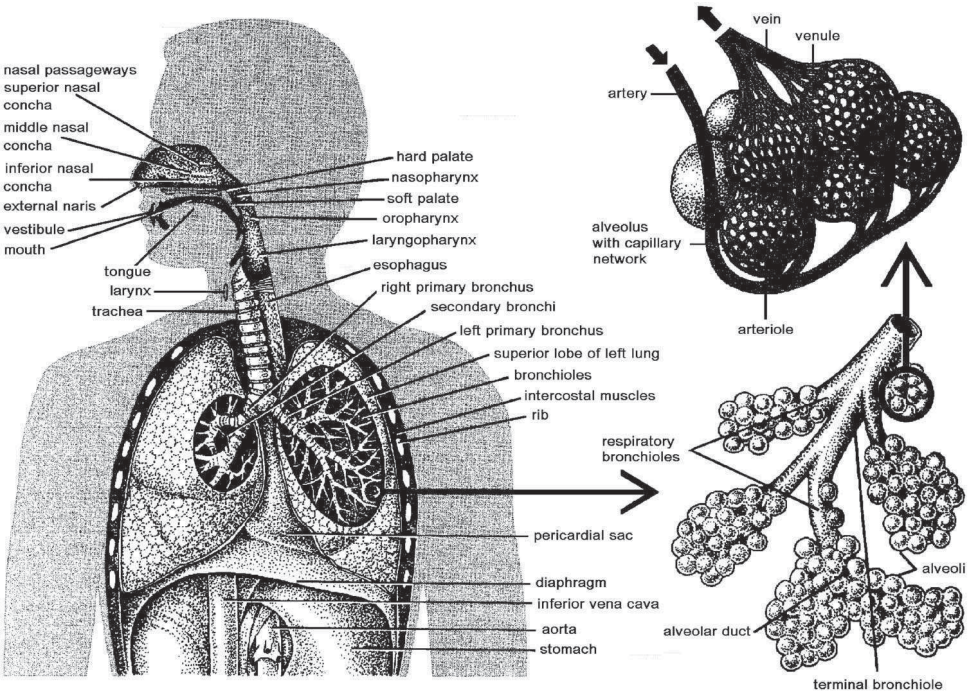

Figure 10.3 Respiratory tract anatomy. (From Burke 1980; reprinted by permission of John Wiley & Sons, Inc.)

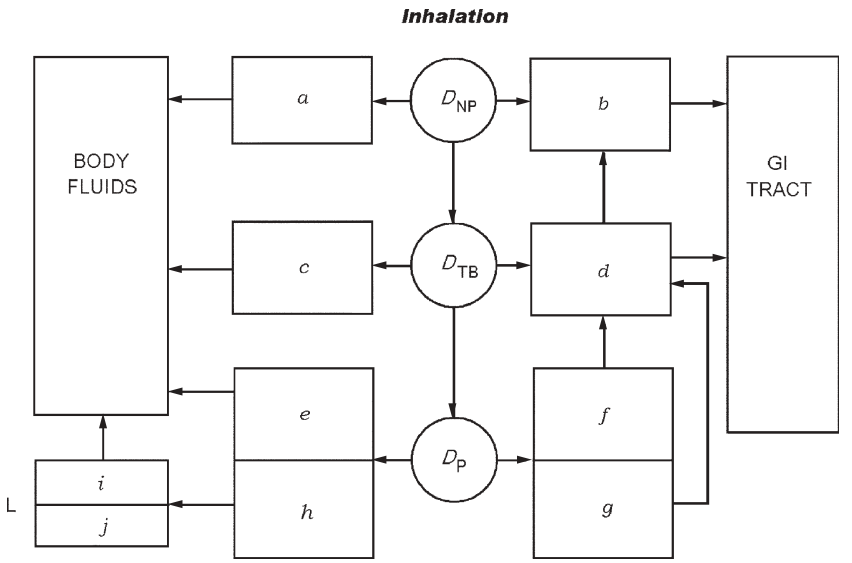

Figure 10.4 ICRP model of the respiratory system. D_{NP}, D_{TB}, and D_P refer to deposition in the nasopharangeal, tracheobronchial, and pulmonary regions, respectively. L refers to the lymphatic tissue. Boxes *a* to *h* represent subcompartments in each of the 3 respiratory regions, and *i* and *j* are subcompartments of lymphatic tissue. (From Till and Meyer 1983.)

are small sacs lined by epithelial tissues. Gas exchange in the alveoli results in the intake of oxygen into the blood and the elimination of waste gases such as carbon dioxide in expired air.

Exposure to airborne contaminants can result in a variety of acute and chronic toxic effects in the respiratory system, including irritation, aggravation of preexisting conditions (e.g., asthma), structural damage leading to chronic diseases (e.g., pulmonary fibrosis and emphysema), and cancer. For example, the effects of inhaling gases such as Cl_2, SO_2, and H_2S which are subject to accidental and routine release to the environment range from coughing to difficulty in breathing to death, depending on the exposure. Ozone aggravates asthma and increases the severity of respiratory infections. Particulate matter, asbestos, arsenic, and nickel are known lung carcinogens.

10.2.3.3 Cardiovascular, Lymphatic, and Immune Systems The cardiovascular and lymphatic systems serve as a means of physical transport of biologically important substances (i.e., oxygen, nutrients, waste products, hormones, disease-fighting cells, etc.) throughout a series of interconnected fluid transfer systems. These substances are circulated within bodily fluids such as blood, lymph, and intracellular fluid. Although both blood and lymphatic fluid circulate throughout the body, the motive force for the two systems is different. Blood is circulated through arteries (blood-supplying tubes), veins (blood collection tubes), and capillaries (the blood distribution network comprising very narrow tubes) via the pumping action of the heart, whereas the lymphatic fluid has no centralized pump to force circulation. Intracellular fluid originates from blood plasma that is forced through the capillary walls by hydrostatic pressure, leaving the larger blood cells within the capillaries. This interstitial fluid is transported from the interstices of body tissues through lymphatic capillaries to lymphatic ducts that are typically at a lower pressure than the interstitial fluid. The lymphatic fluid is then returned from the ducts to the bloodstream, forming plasma.

Blood serves several vital functions in the body in its role as a chemical transport and communication system. Among the most important functions are transport of dissolved substances in the blood plasma; oxygen transfer by hemoglobin in red blood cells; and resistance to disease associated with white blood cells. New blood cells are continuously produced in the marrow of long bones to replace old cells (~120 days), which are destroyed in the liver and spleen. Thus, damage to the bone marrow typically results in clinical effects being observed in the affected person's blood chemistry. The circulatory system also plays an important role in heat regulation by varying the size of capillaries near exterior surfaces of the body, resulting in the ability to control the transfer of heat from the body to outside air. In conjunction with the white blood cells (leukocytes), the lymphatic system provides the body with the ability to recognize and eliminate harmful agents (thereby conferring immunity) by both adapting white blood cells formed in the bone marrow and generation of lymphocytes. Key components of the immune system are the lymphocytes and macrophages, which encapsulate foreign substances for their subsequent destruction or excretion, and antibodies, which are proteins that attach to foreign substances and trigger an immune reaction.

Toxic insults to the blood and immune system can occur in a variety of ways. Some chemicals affect the ability of the hemoglobin in red blood cells to transfer

oxygen. Carbon monoxide, which binds readily to hemoglobin, is well known for reducing oxygen transfer by the blood. A wide array of environmental contaminants has been implicated in interfering with immune system function. Benzene, halogenated hydrocarbons, dioxins, pesticides, and metals are all known to suppress the immune system. Organic compounds such as vinyl chloride, TCE, and PCE, and metals such as gold and mercury can cause an autoimmune response (i.e., an immune response to a person's own tissue). Many contaminants, including organic solvents and metals such as beryllium and mercury, can induce allergic responses.

▶ **Example 10.2**

At one time, polychlorinated biphenyls were widely used as fire retardants and in the electrical industry as an insulating fluid. However, concerns about health effects from PCB exposure have limited their use. Among the concerns posed by PCB exposure are effects on the immune systems of exposed individuals. In one study (Tryphonas et al. 1989), lowered immune system response was observed in rhesus monkeys fed Aroclor 1254 at different levels. This was detected by the observation of lower than expected levels of certain antibodies and lymphocytes following a transfusion of sheep red blood cells (a common laboratory surrogate for pathogenic infection).

10.2.3.4 Nervous and Endocrine Systems The body maintains several communication systems. Two of the most important are the endocrine system, in which chemical messengers known as hormones are circulated through the body; and the nervous system, which uses both a "hard-wired" electrical system and a short-range chemical messaging system.

The endocrine system comprises glands that secrete messenger ligands, known as hormones, into the intercellular fluid. These ligands regulate the behavior of distant cells by binding to receptor proteins embedded in the membranes or interior of the target cell, forming a complex that triggers the target cell to express a characteristic biological response. Major endocrine glands include the thyroid, parathyroid, pituitary, and adrenal glands; the pancreas; and the testes and ovaries. The hormones secreted by the endocrine glands regulate a wide variety of bodily functions related to growth, reproduction, digestion, excretion, calcium homeostasis, and others. These hormones are dissolved within plasma and transported by the circulatory system to target cells. At the target cell, receptor proteins can be found in the membranes, within the cellular body, or within the nucleus. Hormones that are not fat-soluble cannot diffuse through the cellular membrane and therefore interact with receptor proteins embedded within the membrane. These hormones typically induce their effect by causing a momentary change in the concentration of electrolytes inside the cell. These intracellular compounds are known as second messengers. Fat-soluble hormones such as steroids can diffuse through cellular membranes and interact with nuclear proteins. These receptor–ligand complexes typically affect genetic expression, thereby affecting the growth and differentiation of the cell (Darnell et al. 1990).

▶ Example 10.3

Endocrine disruptors are human-made chemicals that mimic endogenous (i.e., natural) hormones and may alter the normal functioning of the endocrine system, which regulates hormonal activity in the body. The postulated mode of action is activating similar responses or blocking the functioning of endogenous hormones by competing for available receptor sites. Endocrine disruption has been hypothesized to be the cause of a variety of health problems in both humans and wildlife, including increased rates of cancer in reproductive and endocrine systems, reproductive system abnormalities, learning and behavioral problems, and immune system deficiencies. Although there is not yet sufficient data to draw conclusions regarding the endocrine disruption hypothesis, concern about endocrine disruption was sufficiently great that in 1996 the U.S. Congress directed the EPA to develop a screening program to identify potential endocrine disruptors (EPA 2006b). Suspected endocrine disruptor compounds include naturally occurring estrogenlike chemicals produced by plants and fungi, pesticides (DDT, methoxychlor, and chlorodecone), dioxins, PCBs, and alkyl phenols (German Environmental Agency 1996).

The nervous system consists of millions of nerve cells known as neurons, which are responsible for receiving and processing sensory information and then controlling muscles to respond to external stimuli. It is divided into two primary components: the central nervous system and the peripheral nervous system. Neurons (Figure 10.5) consist of the cell body or soma, where the nucleus resides; an axonal portion, which extends outward from the soma; and dendrites, which are short-branched structures extending from the soma. The junction between the axon terminus of one neuron and the dendrites of an adjacent neuron is termed the synapse or synaptic junction. Neurons are able to regulate the electrical potential across their cellular membrane as a part of normal cell function. In the nervous system, such membranes are responsible for the transmission of electrical impulses along the cell. Electrical signals are transmitted along the axon by an ionic pump. In its resting state, the neuron has an electrical potential, or voltage difference, of about –70 mV across the cell membrane controlled by the balance between the concentration of sodium external to the cell and of potassium within the cell. Nerve signal transmissions are achieved by selective alteration of the permeability of the membrane, thereby allowing the ions to cross the membrane and temporarily reduce the potential difference across the membrane. This selective permeability is achieved by the action of short-range chemical messengers known as neurotransmitters that open ion channels, allowing the flow of ions through the cell membrane. At the terminus of the axon, neurotransmitters are released by adjacent neurons. This increases the concentration of the neurotransmitter within the synaptic junction. The receptor–ligand complex formed as the result of bonding between a neurotransmitter and a receptor protein in the dendrites of the adjacent neuron provides the signal for the adjacent neuron to fire, thereby transmitting the message across the synaptic junction. Once the cell has reached its excited state, sodium and potassium pumps transport the ions across the cell membrane to

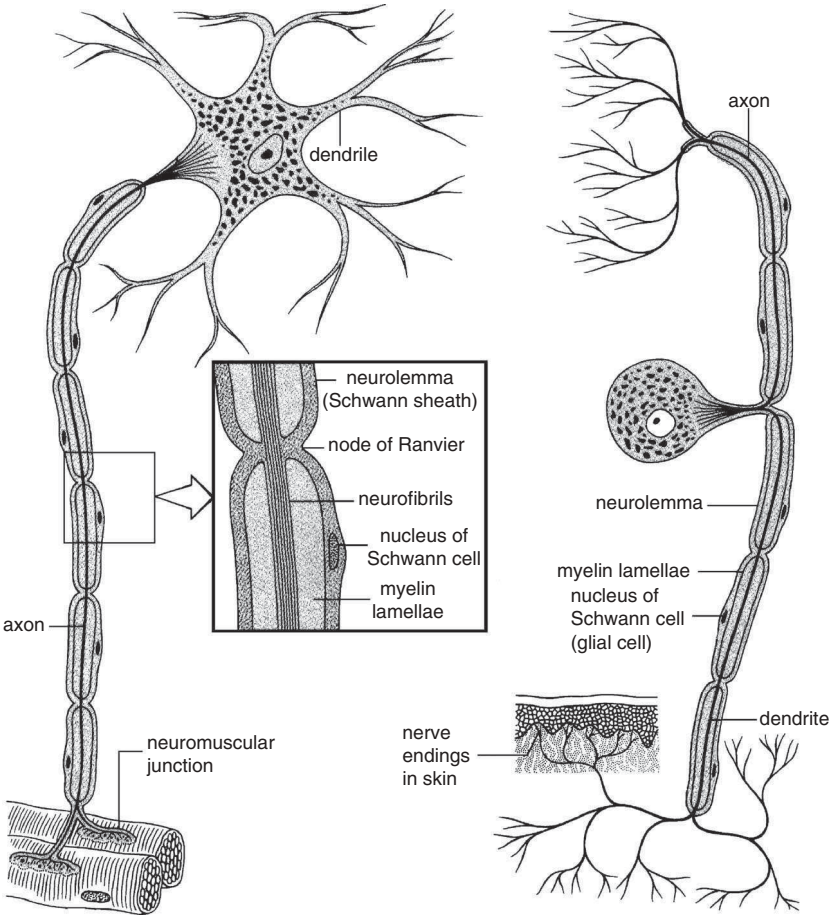

Figure 10.5 Neuronal structure. (From Burke 1980; reprinted by permission of John Wiley & Sons, Inc.)

reestablish the resting potential. The axon is insulated by a cellular membrane (the myelin sheath) that inhibits the flow of ions between the intra- and extracellular fluids, allowing faster signal transmission.

Chemical compounds that mimic neurotransmitters can interfere with the interneuronal transmission of signals. Compounds that interfere with the ability of the neurons to transmit electrical pulses are known as neurotoxins. Along with their other effects, pesticides are typically acute neurotoxins, although the mechanisms of action are different for the different compounds. Organochlorine compounds such as DDT and DDE affect primarily the membrane functions of the axon, leading to the inability to regulate cellular ion levels necessary for the controlled firing of the cells. Organophosphate pesticides, on the other hand, typically act by interfering with neurotransmitter receptor sites, causing the cells to fire repeatedly following a single stimulation.

► **Example 10.4**

Lead is toxic to a number of organs and organ systems, including the nervous system, the blood, and the kidneys. The neurotoxicity of lead has been recognized for millennia (ATSDR 2005b). Human exposure to lead has historically been high, due to the many industrial uses of lead, such as fabrication of corrosion-resistant water pipes, as a paint additive, and as an additive in gasoline. The chemical similarity of lead and calcium allows lead to mimic calcium, thereby disrupting a variety of calcium-mediated effects. Lead neurotoxicity seems to be associated with interference with the normal neurotransmitter functions of the nervous system, due to its ability to mimic calcium chemically as well as its ability to interfere with synaptic receptor–ligand interactions. The peripheral nervous system effects of lead involve the degradation of motor nerve function due to damage to the myelin lamellae surrounding the axons.

10.2.3.5 Integumentary, Skeletal, and Muscular Systems The integumentary, skeletal, and muscular systems serve to protect and support the organs of the body, to provide for voluntary and involuntary motion, and to maintain body temperature. These systems are of importance to environmental risk analysis by virtue of their role in restricting exposure to contaminants and because of the interrelationship between the skeletal system and the creation of blood cells.

The skin is the major organ of the integumentary system. As the primary barrier between the external environment and the body, the skin plays an important role in limiting exposures to environmental contaminants. In cases where the contamination is not inhaled or ingested, the extent to which a contaminant can penetrate or be absorbed through the skin determines the person's exposure. The skin comprises three layers. From the outside in, these are the epidermis, which protects the skin; the dermis, which contains blood vessels, sensory receptors, and various glands; and the hypodermis, which connects the skin to the internal organs and tissues and contains an insulating layer of fat cells. The epidermis consists of a sequence of layers in which cells are generated in an innermost basal layer. As these cells undergo rapid division, younger cells displace older cells that migrate outward over a period of several weeks. As these cells migrate outward, they produce a tough, fibrous protein called keratin. These cells eventually die, producing a tough external layer of dead cells. The protective function of the skin accounts for the limited effect of external exposure to charged particle radiation such as alpha and low-energy beta radiation. External alpha radiation, for example, is stopped within the dead skin layer and therefore yields no significant biological effects. The skin also assists in regulating the heat balance of the body. Constriction and dilation of blood vessels in the dermis and cooling due to evaporation of sweat from glands located in the skin control the rate at which heat is dissipated to the environment. The balance between heat loss from the skin and heat generation in cells (primarily, muscular cells) determines body temperature. In addition to its heat-generating function, the muscular system provides the body with the ability to move and to support the body.

The skeletal system both supports and protects the other organ systems of the body. The skeletal system provides robust protection for vital organs such as the

brain (skull), spinal cord (vertebral column), and internal organs (rib cage and vertebral column). The skeletal system is also the location where blood cells are created and certain important minerals (primarily, calcium and phosphorus, but also magnesium and sodium) are stored. The red bone marrow (principally in the sternum, pelvic bone, vertebrae, and at the ends of the arm and thigh bones) is the site of red and white blood cell formation. A typical long bone has a hard outer layer of compact bone surrounding an inner cavity, with regions of red marrow–filled spongy bone located at either end. This is illustrated in Figure 10.6.

A variety of organ systems affect the stability of the bone. Calcium homeostasis, regulated primarily by the parathyroid hormone and vitamin D, controls the amount of calcium stored in the bone. Loss of homeostatic control can cause excessive release of stored calcium in the bone, resulting in osteoporosis. Because lead is sequestered in the bone, release of calcium from the bone can also result in the release of lead from prior exposures. Toxic effects on the bone can also affect the ability of the red bone marrow to produce red and white blood cells. Ionizing radia-

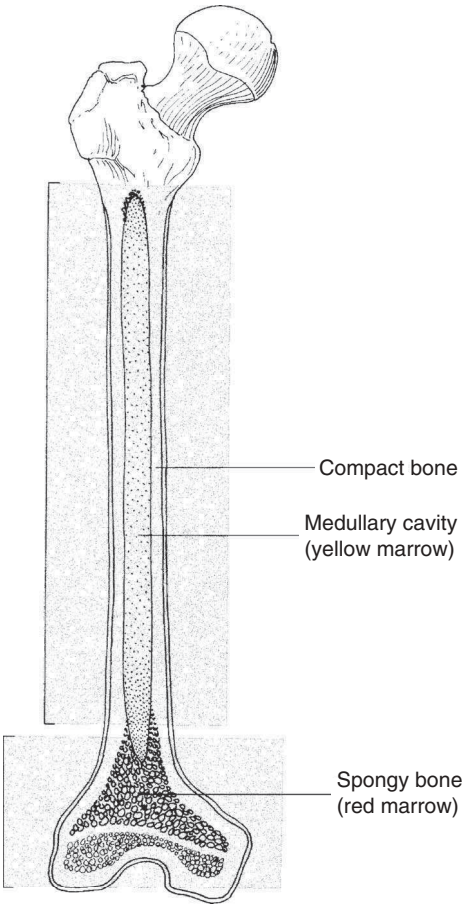

Figure 10.6 Bone structure. (From Burke 1980; reprinted by permission of John Wiley & Sons, Inc.)

tion, benzene, lindane, chlordane, and arsenic have all been shown to depress the production of blood cells at sufficient levels of exposure.

10.2.3.6 Reproductive System The human reproductive system has two primary functions: production of the germ cells that will be joined to produce offspring, and support for the development of a fertilized egg into a viable human infant. Germ cells are produced in the gonads, which are the testes in the male and the ovaries in the female. Male germ cells (spermatozoa) are produced in the testes. Female germ cells (ova) are produced in the ovaries. Each germ cell contains half (23) of the genetic complement of 46 chromosomes. The union of a spermatozoon and an ovum during conception results in a cell (zygote) in which these half-complements are paired to generate a full set of chromosomes.

The zygote undergoes a specific sequence of cell divisions and tissue differentiation developing ultimately into a viable fetus. Following conception, the fertilized cell migrates down the Fallopian tube into the uterus. After approximately 6 days, the cellular mass (blastocyst) becomes attached to the lining of the uterus. At this point, the blastocyst comprises an inner portion and an outer portion that ultimately becomes the placenta, an organ that transfers oxygen and nutrients from the mother to the developing embryo and removes waste products. Part of the blastocyst also develops into the protective amniotic sac enclosing the developing embryo. After implantation, the cells of the inner portion differentiate as they divide, forming the basis of the organ systems of the developing child. The period between the development of initial differentiation and the point at which all of the major organ systems begin to form is known as the embryonic stage (roughly the first trimester). Because of the essentially irreversible nature of cellular differentiation, interference with the normal developmental process during this stage can result in severe developmental abnormalities. Thus, at this point the developing embryo is most sensitive to toxic insults. After about 8 weeks, all major organ systems have begun to form, and the embryo has developed into a fetus. From about 8 to 25 weeks (roughly the second trimester), the organ systems continue to develop, but the developing organism is not yet capable of surviving on its own. During the last trimester, the fetus begins to take over regulation of its own bodily functions, and the likelihood of its survival increases significantly.

Toxic insults to the reproductive system are of concern primarily because of potential adverse effects to offspring rather than adverse impacts on the parent. Adverse impacts on offspring are either hereditary or teratogenic effects, depending on when the exposure occurs. Hereditary effects arise from exposures prior to conception that alter the genetic material of the germ cells in the parent. Teratogenic effects are due to exposure of the developing embryo or fetus.

10.3 MECHANISMS AND EFFECTS OF TOXICITY

It is convenient to divide the clinical effects of contaminants into four categories: systemic, carcinogenic, hereditary, and teratogenic. **Systemic effects** refer to deterministic effects in body organs (i.e., liver, kidneys, brain, etc.) or systems (i.e., immune, respiratory, digestive, etc.). **Carcinogenic effects** refer to the induction of

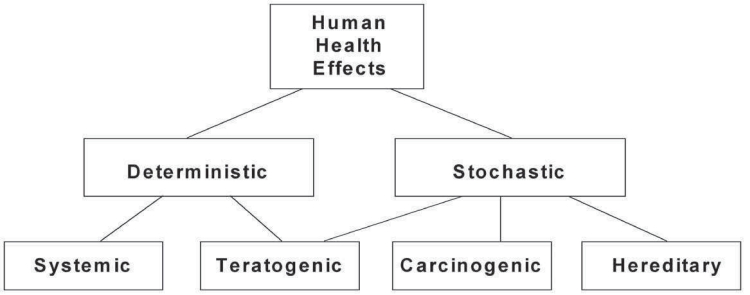

Figure 10.7 Classification of human health effects.

malignant tumors. **Hereditary effects** are adverse effects observed in offspring or in future generations as a result of parental exposure prior to conception. **Teratogenic effects** are developmental abnormalities induced between conception and birth as a result of direct exposure of the developing organism. The relationship between deterministic or stochastic effects (discussed in Chapters 1 and 11) and the clinical taxonomy used in this chapter is illustrated in Figure 10.7. Contaminants may trigger more than one type of effect; for example, ionizing radiation can result in both systemic effects (at high doses and dose rates) and carcinogenic effects. Other classification schemes for clinical effects of toxic substances exist (e.g., EPA 2006a), but this scheme has the advantage of being able to accommodate both chemical and radioactive contaminants.

10.3.1 Systemic Effects

Since different tissues or organs often react differently to a given contaminant, systemic effects are often the result of selective toxicity. The systemic effects of different contaminants were discussed briefly in the preceding discussion of the physiology of the various organ systems. Specific systemic effects associated with selected contaminants were given in Table 3.1. For example, compounds that affect receptor–ligand interactions often express themselves by endocrine or nervous system impacts, and compounds that interfere with excitable membranes typically affect the nervous system. Selective toxicity can also arise from the metabolism of a contaminant, in that certain compounds can be selectively transported or metabolized in the body and thereby concentrate in certain tissues or organs. At an extreme, some compounds are selectively cytotoxic, affecting only certain types of cells. The neurotoxicity of manganese, for example, stems from its toxic effect on certain brain cells.

For radiation, this phenomenon is known as radiosensitivity, in which different organs, tissues, or cells are more or less sensitive to the deleterious impacts of radiation. This phenomenon, observed over a century ago by French scientists, has been stated as the law of Bergonié and Tribondeau (Bergonié and Tribondeau 1906): "The radiosensitivity of a tissue is directly proportional to its reproductive capacity and inversely proportional to its degree of differentiation." Updated by modern observations, four indicators of increased radiosensitivity of cells are iden-

tified as a "rule of thumb", because significant exceptions are extant (DOE 1999): (1) high rate of cell division, (2) high cellular metabolic rate, (3) less differentiated cells, and (4) well-nourished cells. Application of this rule explains the high radiosensitivity of red blood cell precursors, which have all four characteristics, and the low radiosensitivity of muscular and nerve cells, which have few or none. This variable radiosensitivity of different tissues is important in determining the overall consequences from exposure to radiation.

10.3.2 Carcinogenic Effects

In 2005, 22% of deaths in the United States were from cancer. Along with heart attacks and strokes, it is one of the three leading causes of death. Although life-style choices such as the use of tobacco products, alcohol consumption, and diet are thought to be responsible for a majority of cancers, it is also known that cancer can result from exposure to ionizing radiation and to some chemicals in the environment. In fact, cancer is often the primary stochastic effect analyzed in risk assessments. Cancer is of particular concern because it can be induced at doses far below the level required to induce an observable systemic effect (and possibly, at any nonzero dose). Thus, controlling exposures to prevent systemic effects may be ineffective in providing an acceptable level of protection against cancer.

Of the many chemicals present in the environment, only a few have been determined conclusively to cause cancer. Table 3.1 includes several environmental contaminants that are known or probable carcinogens. Cancer is a class of diseases, all characterized by an uncontrolled growth of cells. **Carcinogenesis** is the process whereby a normal somatic cell is modified so that it begins to divide abnormally, leading to an abnormal cellular mass in the body. The cancerous cells consume most of the body's energy and starve healthy cells, they secrete digestive enzymes that destroy healthy cells, and they suppress the immune system, making the person susceptible to secondary infection. Consequently, if the growth of cancerous cells is not checked by some combination of radiation therapy, chemical therapy, and surgery, the person affected is likely to die.

Although the mechanisms for the induction of cancer are not known precisely, there is evidence to suggest a multistage process consisting of several distinct phases: initiation, promotion, and progression. **Initiation** is the mutation of DNA in a single cell by chemicals or radiation (which can be either of natural origin or products of human activities). This mutation proliferates through succeeding cell generations. Except for the presence of the mutated gene, these cells are normal in that they remain differentiated and their growth is regulated. In the **promotion** phase, which is not well understood, the cells begin to grow and divide abnormally, creating a **tumor** (also termed a neoplasm). In some tumors, the cells retain most of the characteristics of their differentiated ancestor cells and are contained by the surrounding tissues. These tumors are termed benign (although the abnormal growth of a benign tumor can sometimes lead to significant health effects). Some tumors enter a third phase known as **progression**. If the internal controls on genetic expression are disrupted, cells can regress or become less differentiated. Whereas mature, differentiated cells are destroyed by the immune system if they are found outside their normal tissue, immature cells are not. They can migrate away from

the site of the primary tumor through the blood and lymphatic circulatory systems and start new tumors in distant tissues and organs. The process is known as **metastasis**, and tumors that have the capability to metastasize are known as **malignant**.

Carcinogens are often divided into two classes: **mutagenic** (genotoxic) **carcinogens**, which can initiate a cancer by transforming the DNA of the target cell, and **epigenetic carcinogens**, which exert their carcinogenic effects without interacting with genetic material. There is considerable evidence for the key role of mutagenicity in cancer, including the monoclonal nature of most cancers and the fact that most carcinogens are capable of damaging DNA. Although all of the factors that cause a chemical to be mutagenic are not clear, it appears that most mutagenic carcinogens are either highly electrophilic compounds or those that have electrophilic metabolites. If these compounds enter the cell, their chemical characteristics allow them to form a covalent bond with DNA. The resulting bonded molecule is known as an adduct. A number of human carcinogens have been identified as having significant reactivity with DNA. These can either be mutagenic in their parent form, or they may require metabolic activation to produce their mutagenic effects. Activation independent mutagens include alkylating agents and certain metals, such as nickel and cadmium. Activation-dependent mutagens include such compounds as arylamine, nitrosamine, and polycyclic aromatic hydrocarbons.

Epigenetic carcinogens do not affect DNA directly but instead act to promote the growth of a tumor or suppress natural defenses against cancer. These include hormone-modifying compounds, cytotoxic compounds, and immunosuppressive agents. Often, these compounds induce DNA synthesis and cellular proliferation. Unlike genotoxic carcinogens, most epigenetic carcinogens require relatively high and prolonged exposure to manifest their carcinogenic effect. Chemically induced promotion is a major mechanism of epigenetic carcinogenicity and is the mechanism of action of such compounds as organochlorine pesticides, polychlorinated/polybrominated biphenyls, and saccharin. Hormone modification by such agents as diethylstilbestrol, estrogens, amitrole, and androgens is another mechanism of action. Chemicals that act through other mechanisms include diethylhexylphthalate, nitrotriacetic acid, cyclosporine A, asbestos, and some plastics.

10.3.3 Teratogenic Effects

Teratogenic effects are those induced between conception and birth. Although a teratogenic effect is defined rigorously as a birth defect (either a malformation or a retardation), embryonic or fetal death can occur from the same mechanisms that cause birth defects. Approximately 3% of live-born infants have major congenital malformations that are recognized in the first year of life (NCBDDD 2005). As is the case with other health effects, it is difficult to distinguish between the incidence of effects caused by a given contaminant exposure and the background incidence. Nonetheless, there is a substantial body of epidemiological data to establish a long list of known teratogens.

Toxic insults in the preembryonic stage typically result in a binary response, in which the pregnancy is either terminated or survives the toxic insult unharmed.

During the embryonic stage, toxic insults may result in developmental abnormalities. This is due to the fact that the cells of the embryo are still undergoing differentiation. Arrested or altered differentiation can result in the elimination or alteration of entire organ systems. After the developing embryo matures into a fetus, cellular growth rather than differentiation predominates, and toxic insults typically result in stunted growth rather than gross malformation of the organ systems.

Teratogenic action predominates during the embryonic stage (first 8 weeks) of development when rapid and complex changes are occurring as a result of differentiation of the embryonic cells into the various organ systems. Selective cytotoxins are of particular concern during embryonic development because loss of a relatively limited number of cells in the developing embryo can have dramatic consequences. There are critical windows of development for the various organs and body systems, and the timing of an exposure plays a critical role in the potential effect. Most organ development occurs between 21 and 56 days after conception. For example, the basis of the nervous system develops between 18 and 21 days, and the lower limbs, between 31 and 32 days. The destruction of a small number of cells by low doses early in a critical period may result in a normal but growth-retarded organ. This is because the surviving cells partly compensate by enhanced cell production. High doses late in the period may yield a malformed or nonfunctional organ because there is no time for cell replacement. However, the effects may be nonspecific if there is a general inhibition of cellular processes in all tissues, resulting in overall growth retardation or death of the embryo. The alteration of specific cells during a very specific time in their differentiation can result in a well-defined anomaly or malformation. For example, thalidomide affects the development of limb bud cells during the sixth or seventh week of pregnancy, yielding rare malformations of the arms.

Presented in Table 10.2 is a partial list of known human teratogens. It must be recognized that human data are very limited, and many toxicologists believe that any contaminant can alter embryonic development if exposure occurs at the appropriate dose and time. Over 900 agents are known to cause teratogenic effects in animals.

▶ Example 10.5

Methyl mercury has been implicated in developmental neurotoxicity, primarily on the basis of animal studies consistent with epidemiological data on a variety of populations (NAS–NRC 2000). Methyl mercury has been shown to interact with critical molecules (DNA and RNA) in the cells of the central nervous system, causing interference with RNA synthesis. As the cells divide, the altered cells are no longer able to reproduce properly, leading to abnormal growth and development of the brain cells. Among its other effects, ionizing radiation can exert teratogenic effects. An increase in mental retardation was observed in Japanese children irradiated in utero at Hiroshima and Nagasaki. Those exposed during the period from 8 to 15 weeks after conception showed the clearest effects, with the effects increasing with increasing dose.

TABLE 10.2 Known Human Teratogens

Androgenic hormones	Trimethadione
Aminopterin	Courmarin anticoagulants
Cyclophosphamide	Valproic acid
Busulfan	Tetracyclines
Thalidomide	13-*cis*-retinoic acid
Mercury (organic)	Lithium
Chlorobiphenyls	Methimazole
Diethylstilbestrol	Radiation
Diphenylhydantoin	

Source: Manson and Wise 1991.

10.3.4 Hereditary Effects

Whereas carcinogenic and teratogenic effects occur as a result of damage to genetic material in somatic cells, hereditary effects occur as a result of damage to genetic material in germ cells in the reproductive system. Hereditary effects are those that are expressed in the immediate or remote descendants of the person exposed. There are two general types of damage: alterations of DNA (gene mutations) and alterations in the structure or number of chromosomes (chromosomal aberrations). **Dominant mutations** can be expressed as a result of inheritance of the mutation from either parent, whereas **recessive mutations** must be present in both parents for the effect to be expressed. Recessive mutations yield few actual effects in the first few generations, but they contribute to the general pool of genetic damage in subsequent generations.

Approximately 30% of all spontaneous embryonic and fetal deaths and 6% of stillbirth and infant deaths are attributed to genetic damage. Among live births, the severity of inherited effects varies over a wide range with approximately one-third to one-half characterized as serious. It is estimated that 2 to 4% of newborns have a serious genetic abnormality, and 10% of people ultimately experience a serious genetic disorder. Dominant mutations can result in hereditary disorders such as polydactyly (extra fingers and toes), achondroplasia (short-limbed dwarfism), Huntington's chorea (progressive involuntary movements and mental deterioration), muscular dystrophy, anemia, and retinoblastoma (an eye cancer). Recessive mutations are known to cause hemophilia (failure of blood clotting), color blindness, phenylketonuria (PKU, a type of mental deficiency), Tay–Sachs disease (blindness and death in the first few years of life), sickle cell anemia, and cystic fibrosis. Although there is no human epidemiological evidence of hereditary effects due to environmental contaminants, there is ample evidence in animal studies for contaminant-induced hereditary effects.

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PROBLEMS

10.1 Define, describe, or explain each of the following:
- **(a)** Toxicology.
- **(b)** Law of Bergonié and Tribondeau.
- **(c)** Carcinogenesis.
- **(d)** Metabolic activation.

10.2 Identify the four types of macromolecules and provide an example of each.

10.3 Describe the process by which information encoded within DNA is used to synthesize proteins.

10.4 List four ways in which a poison can exert its effect at the cellular level.

10.5 Categorize each of the following as either a systemic, teratogenic, carcinogenic, or hereditary health effect.
- **(a)** Leukemia due to exposure to benzene.
- **(b)** Cirrhosis of the liver due to consumption of alcohol.
- **(c)** Thyroid cancer due to consumption of iodine-131 in milk.
- **(d)** Missing arm due to fetal exposure to thalidomide.
- **(e)** Mental retardation due to fetal exposure to lead.

10.6 Explain the difference between somatic and hereditary health effects.

10.7 Explain why the timing of an exposure can be a critical factor in a teratogenic effect.

10.8 Describe two ways in which exposure to a chemical can cause cancer.

11 Dose–Response and Risk Characterization

11.1 INTRODUCTION

Presented in Chapter 10 were qualitative descriptions of the mechanisms through which contaminants can affect normal structures and functions in the body as well as the ultimate effects on human health. The risk assessment process as presented in Chapter 1 requires a quantitative characterization of the human health risk posed by contaminant exposures. This is accomplished through dose–response assessment, which is the process of quantifying the relationship between contaminant dose and the resulting toxicological response.

In the contemporary practice of risk assessment, human health risk is characterized by one of two generic metrics. Selection of the appropriate metric is dictated by the dose–response relationship. Some contaminant effects are only expressed once a toxic threshold is exceeded, and the typical risk management objective is prevention of the effect. This can be accomplished simply by keeping exposures under the threshold. The generic metric used for these types of effects is the **margin of safety**, which is a comparison, usually a ratio, between the estimated dose and the dose at which adverse effects are expected to occur. In contrast, some contaminant effects can be expressed at any dose (i.e., there is a nonzero response at any nonzero dose). The only way to prevent the risk is to prevent or eliminate exposure, which is usually not possible. Thus, the typical risk management objective is to minimize the risk by keeping it below an allowable level. The generic metric used to characterize nonthreshold effects is the **fractional response**, which is the probability of the effect occurring in the exposed population. With respect to the deterministic–stochastic categorization of effects introduced in Chapter 1 and described in more detail below, deterministic effects are frequently characterized by a margin of safety, whereas most stochastic effects are characterized by some type of fractional response metric.

11.2 BIOLOGICAL BASIS OF DOSE–RESPONSE MODELING

Toxic effects are due to alterations of the structure and/or function of molecules in the body that lead to the disruption of normal cellular and physiological function. If cells are damaged, cellular repair mechanisms may be able to repair the

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

cell. If the effects are reversible (as a result of either cellular repair or replacement), permanent physiological damage may be avoided, although symptoms of an adverse effect may be observable while the exposure is ongoing or before the body has repaired itself. If cellular damage is not repaired, it may (1) cause cells to malfunction, die, or not reproduce, or (2) modify the genetic material (DNA) in cells without affecting their ability to reproduce. These two types of cellular damage ultimately yield the fundamentally different types of effects introduced in Chapter 1. **Deterministic effects** (those for which the *severity* is a function of dose) are the result of the first type of damage. As the number of affected cells increases due to increases in contaminant dose, the severity of the effect increases. An example for a chemical contaminant is shown in Table 11.1, where data are shown on the clinical effects of breathing SO_2. It is seen that effects on the respiratory system become more severe as the SO_2 concentration increases. The results are quite different if the genetic material in the cell is modified. If the cell survives and reproduces, the genetic change is passed on to future cell generations, potentially increasing the number of affected cells with each new generation. If the initial damage is in a somatic cell, the ultimate result can be cancer. If it is in a germ cell in a reproductive organ, the ultimate result can be a hereditary effect in descendants. These are referred to as **stochastic effects** (those for which the *probability* is a function of dose). Since the effect is binary (i.e., either it occurs or it does not), the severity is not related to the dose. Examples of stochastic effects include cancer and some teratogenic effects. Contaminants that are known carcinogens in humans are asbestos (lung cancer), benzene (leukemia), cigarette smoke (lung, bladder, laryngeal, and esophogeal cancer), and radiation (leukemia, bone, thyroid, lung, and others).

Many, if not most, tissues and organs can continue to function normally even if a large number of cells are killed or otherwise incapacitated. For these, there exists a toxic threshold below which adverse effects do not occur (Aldridge 1986). Such a threshold could also exist for stochastic effects. Such effects are referred to as **threshold effects**. The **threshold dose** is the dose below which adverse effects are not expected to occur. Equivalently, it is the dose at which the most sensitive member of the population first exhibits an effect. Because deterministic effects occur primarily as a result of cells being killed by the action of the contaminant, they are typically assumed to exhibit a threshold. Effects that can result from the

TABLE 11.1 Deterministic Effects of Inhalation of SO_2

Concentration (ppm)	Duration	Effect
0–0.06	1 wk	None
0.15–0.25	1–4 d	Cardiorespiratory
1–5	1 h	Tightness in chest
5–10	1 h	Choking
10	1 h	Severe respiratory distress

action of a single molecule of a contaminant or radioactive emission are **nonthreshold effects** since any exposure, however small, carries some increased risk. Nonthreshold stochastic effects include cancers that can be initiated by a single interaction with DNA. Also, teratogenic effects that result from the alteration or destruction of a single bud cell in a developing fetus, such as occurred with the drug thalidomide, would not have a threshold.

11.3 ELEMENTS OF QUANTITATIVE DOSE–RESPONSE ANALYSIS

11.3.1 Factors Affecting Toxicity: Pharmacokinetics and Pharmacodynamics

Quantitative dose–response analysis requires a quantitative understanding of how the response is related to the dose. There are three assumptions required to establish a dose–response relationship (Klaassen and Eaton 1991): There is a molecular site within the target tissue with which the contaminant interacts to produce a biological response, the production and degree of the response are related to the concentration of the contaminant at these molecular sites, and the concentration of the contaminant in the target tissues is related to a person's exposure.

The relationship between exposure and the concentration of the contaminant within the target tissues constitutes the discipline of **pharmacokinetics**,[1] which examines the uptake, distribution, transformation, and elimination of a contaminant. **Pharmacodynamics** examines the mechanism of action of the contaminant. Pharmacodynamic parameters include identification of the relevant molecular interaction targets and the quantitative relationship between contaminant concentration at the reactive site and the production and degree of the response.

11.3.1.1 Intake, Uptake, Administered Dose, and Effective Dose For radiological contaminants, **equivalent dose** to an organ is the energy absorbed in the organ per unit organ mass weighted by the effectiveness of the radiation in causing biological damage. **Radiological effective dose** is equivalent dose weighted by the sensitivity of the various organs to radiation-induced cancer. Because the weighting factors used to quantify the effective and equivalent dose are derived on the basis of an observable biological effect, they are therefore by definition an appropriate measure of biological response.

However, the situation is somewhat different for the variety of chemical contaminants where the toxicological knowledge is less complete. The dose obtained from the calculations described in Chapter 9 is more precisely termed the **administered dose** D_{adm}, which reflects the total mass of contaminant entering the body. This dose quantifies the intake of the contaminant. However, this is not necessarily

[1] The term pharmacokinetic refers to the evaluation of the kinetics of therapeutic substances within the field of pharmacology. Pharmacokinetic models are referred to as toxicokinetic models when used to examine the effect of toxic substances. The radiological health community typically uses the term biokinetic model when examining the health effects of internally deposited radionuclides. The term "pharmacokinetic" is used here because it is probably the most widespread term for such models in the risk assessment literature.

the same as the mass of contaminant that actually passes through an exchange boundary such as the lung, intestinal lining, or skin. The amount eventually presented to an exchange boundary after uptake is sometimes termed the **applied dose**, and the amount that actually passes through the boundary is represented by the **absorbed dose**, D_{abs}. The absorbed dose is a measure of the uptake of the contaminant into internal body tissues. A final dose metric represents the biologically active concentration in the target tissue and is given the name **effective** (also *target*, *delivered*, or *tissue*) **dose**, D_{eff}. There are a variety of competing terms for describing these concepts. In assessing human health risks from environmental exposures, the EPA uses the terms "potential dose" rather than "administered dose", "applied dose" to represent the amount presented to an exchange boundary, "internal dose" rather than "absorbed dose", and "biologically effective dose" rather than effective dose. The concepts are similar, as illustrated in Figure 11.1.

The majority of toxicological response information from animal experiments and human epidemiological studies for chemical contaminants is in terms of the gross amount of contaminant taken into the body per unit body mass, or the administered dose. This frequently forces dose–response relationships to be expressed in terms of administered dose, referred to hereafter simply as "dose".

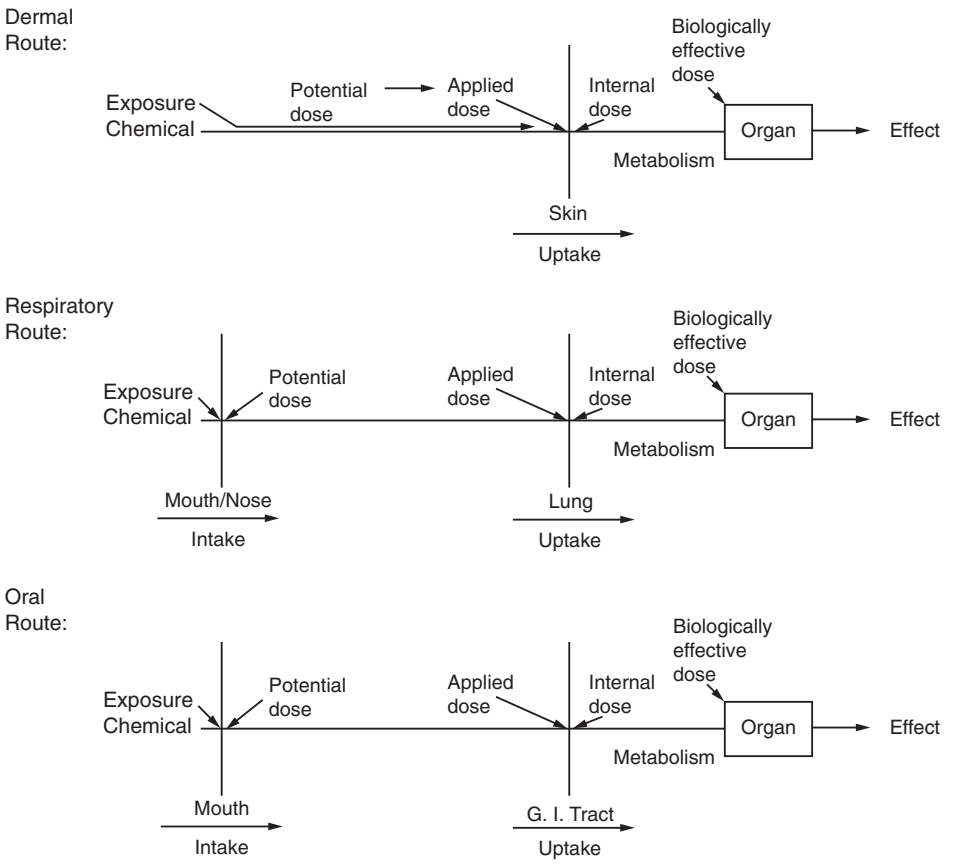

Figure 11.1 Relationship between different dose metrics. (From EPA 1992b.)

11.3.1.2 Pharmacokinetic Models The chemical absorbed dose is a fraction of the chemical administered dose. This fraction can be highly variable, being a function of the individual, the chemical and physical form of the contaminant, the pathway through which the chemical enters the body (e.g., ingestion, inhalation, or dermal exposure), and the person's age and diet. Determination of the chemical effective dose as a function of the chemical absorbed dose involves another layer of complexity. The effective dose is a function of both the total administered dose and the dose rate and the route of exposure, as shown in Figure 11.1. Determination of absorbed and effective doses on the basis of an administered or applied dose can be accomplished with a **pharmacokinetic model** that tracks the absorption, distribution, metabolism, and elimination of chemicals. These models are typically compartmental models of contaminant transport in humans and animals. The compartments can be defined empirically for mathematical convenience, or they can be chosen to simulate actual organ systems characterized by physiological parameters such as organ weight or organ specific metabolic rates. The transport rate from one compartment to another is approximated as either a constant or a linear function of the concentration in the compartments. The result is a set of coupled differential equations that relate changes in contaminant concentration in various compartments due to chemical transfer and metabolic transformation. A graphical representation of a widely used pharmacokinetic model is that used by the EPA to estimate childhood exposure to lead (EPA 1994) is illustrated in Figure 11.2. Similar models are used to examine the effect of ingested or inhaled radioactive materials.

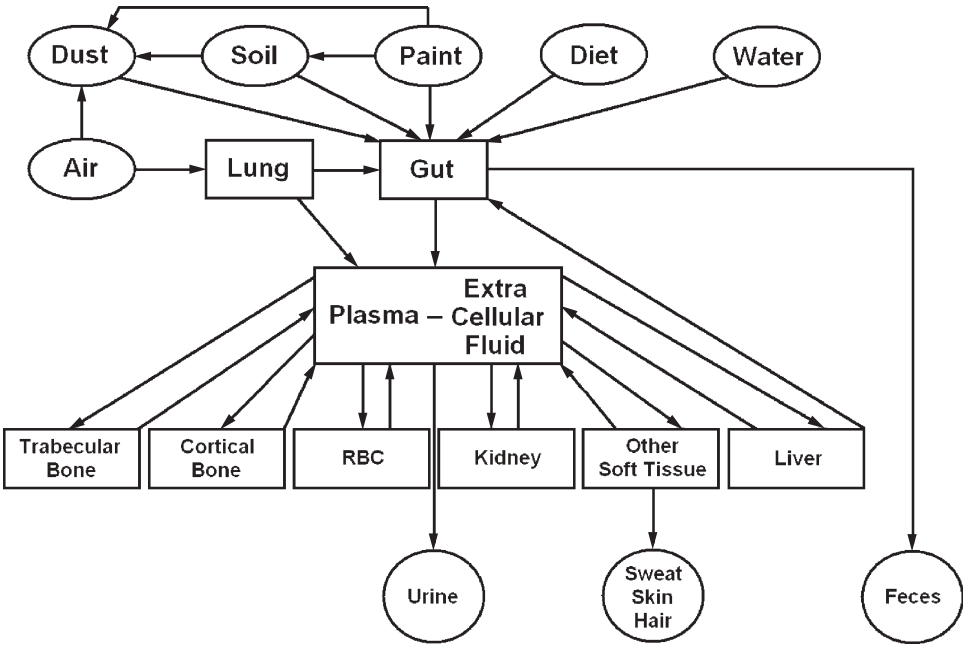

Figure 11.2 Biokinetic model for lead. (From EPA 1994.)

▶ Example 11.1

Dosimetric models to evaluate the effects of radionuclide intake are examples of pharmacokinetic models applied in a radiological health setting. A very simple model is the ICRP model of radionuclide uptake through the gastrointestinal (GI) tract (ICRP 1978) shown previously in Figure 10.2. The model consists of four compartments: the stomach, the small intestine, the upper large intestine, and the lower large intestine. Clearance from one compartment to the next is based on fixed rate constants derived largely from barium studies. Absorption into the blood occurs from the small intestine and is quantified by the fraction absorbed, f_1. The rate constant for absorption from the small intestine is based on f_1. Although the GI tract model is common to all radionuclides, element-specific models are used for different radionuclides to track distribution within the body. The element-specific biokinetic model is coupled to the uptake model to quantify the distribution of the element within the body. An example of such a model is that used to estimate doses resulting from exposure to plutonium (EPA 2002), shown in Figure 11.3.

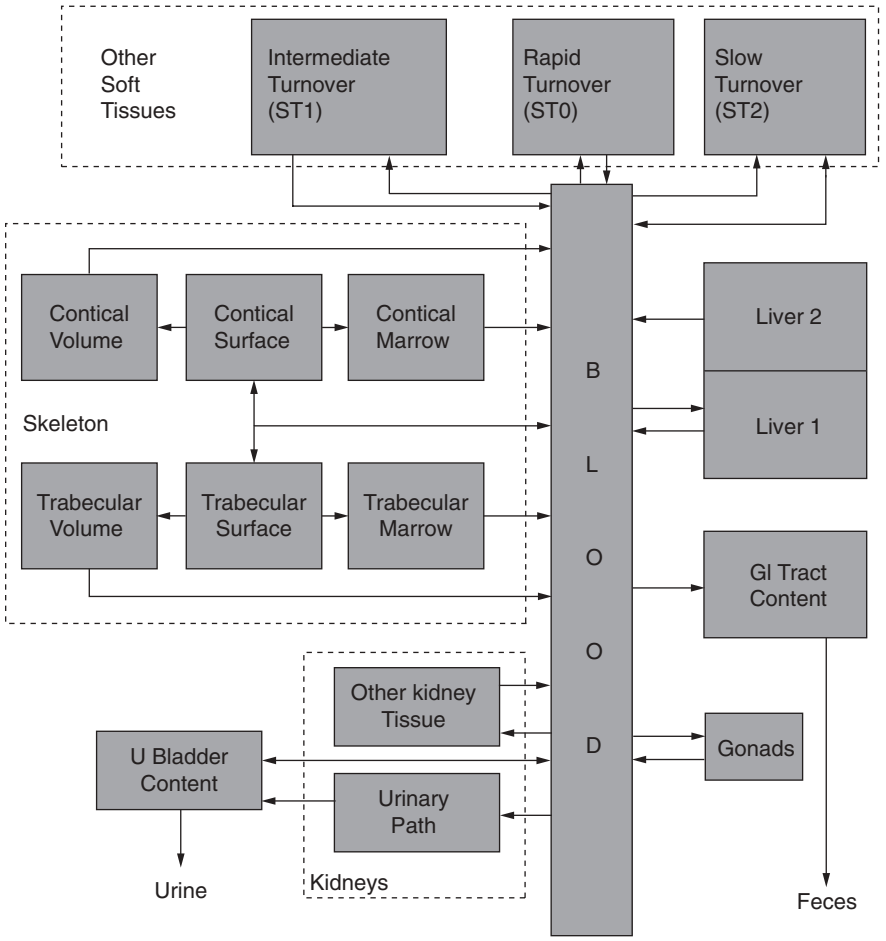

Figure 11.3 ICRP biokinetic model for plutonium. (From ICRP 1993; EPA 2002.)

The GI tract model is highly simplified and was originally developed to estimate doses to workers. However, it has also been used to estimate doses to members of the public, including infants and children. Application to populations other than workers requires changing, for example, estimated masses of the different compartments and modifications of the f_1 factor. Several revised conceptual models have been suggested to make use of new data (Poston et al. 1996; Simko and Noßke 1996). Committee 2 of the ICRP is engaged in the development of an updated model known as the Human Alimentary Tract Model (HATM) that will replace the ICRP 30 GI tract model.

For many contaminants, data are not available to construct a pharmacokinetic model properly. Thus, most environmental risk assessments use the chemical administered or chemical absorbed dose rather than the chemical effective dose. The use of a chemical administered dose to approximate a chemical effective dose increases the importance of understanding the impact of dose rate and route of exposure on responses that are expressed as a function of chemical administered dose.

11.3.1.3 Dose-Rate Dependence of Toxicity An example of the influence of the biokinetic behavior of a contaminant is the dependence of toxicity on the dose rate. For many contaminants, the dose rate (or time over which a given dose is administered) can have a significant effect on the toxicity of the exposure. The administered dose rate for chemical contaminants is simply the rate at which the contaminant is taken into the body. Frequently, a dose received over a short period of time is more toxic than the same dose received over a protracted period. This is often due to differences in the time dependencies of administered dose and effective dose, these differences being due to the ability of the body to eliminate the contaminant or repair toxic insults. If the body can quickly degrade and eliminate the contaminant, or rapidly repair injuries, lowering the dose rate by keeping the total dose constant while extending the period of exposure may lead to reduction or elimination of the toxic response. However, a toxic response may be observed if the natural protective mechanisms of the body are overwhelmed. This is illustrated in Figure 11.4, where contaminant concentration at some target organ is

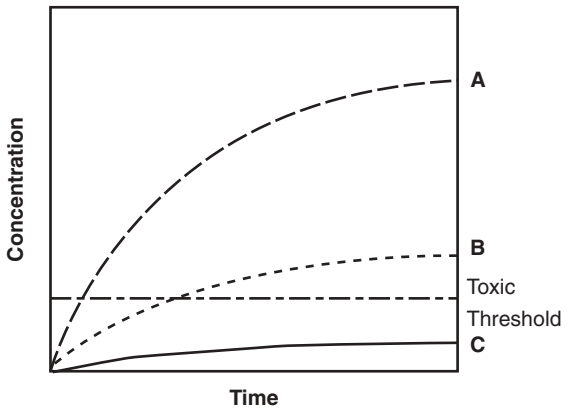

Figure 11.4 Dose-rate dependence of toxicity.

plotted on the left y-axis and exposure time is plotted on the x-axis. In this example, the body is assumed to eliminate the contaminant at a rate proportional to the amount of contaminant present in the body. In scenario A the contaminant is administered much faster than it can be eliminated, causing it to accumulate steadily at the active site, ultimately exceeding a toxic concentration. In scenario B, the contaminant is administered at a lower rate and accumulates at a slower rate, but eventually reaches a toxic level. In scenario C, on the other hand, the contaminant is administered at such a low rate that the elimination processes prevent it from reaching a toxic level. An analogous example could be developed to illustrate the effects of repair mechanisms, in which the elimination rate is replaced by a repair rate and the y-axis represents the extent of biological damage.

The quantitative relationship between dose and exposure duration is a subject of ongoing research. If sufficient data are available, pharmacokinetic models can be used to derive the relationship between exposure patterns and tissue concentrations at the target organ, providing the most realistic estimate of the effect of administered dose rate (Boyes et al. 2005). However, such models are not widely available, and the dose-rate dependence of toxicity therefore typically requires specification of the exposure duration. Exposure duration is generally specified qualitatively as acute, subacute, subchronic, and chronic. Acute exposures are effectively instantaneous, whereas chronic exposures persist over a large fraction of a lifetime. However, although there are moves to standardize terminology for toxicity testing, there is no hard-and-fast dividing line between the four exposure duration categories. Within an exposure duration category, adjustments for exposure duration are frequently based on Haber's law. This relationship was based on observations of acute toxicity of phosgene that suggested its toxicity to be proportional to the cumulative exposure, which was defined in Chapter 9 as the time integral of concentration. If concentration does not vary with time, cumulative exposure is the product of concentration and exposure time, or Ct_e. A linear relationship between toxicity and concentration is not universal, however, and simple scaling adjustments for exposure duration have been proposed that are based on power relationships (i.e., $C^n t$) (Eisenberg et al. 1975; ten Berge et al. 1986). The exponent n is a function of the contaminant and the exposure scenario. However, such relationships are highly approximate.

11.3.1.4 Route of Exposure The biokinetic behavior of a contaminant also helps to explain the strong dependence of toxicity on the route of exposure. The route through which a contaminant is administered may significantly affect the concentration at the target organ and thus the degree of toxic response. Substances that undergo detoxification or metabolic activation may have significantly different toxicities when exposure is through inhalation rather than through ingestion. This is because most chemical contaminants pass through the liver when taken orally. Since the liver is a site of a great deal of metabolic activity, there is a high chance of either activation or detoxification prior to entering systemic circulation. Extensive metabolism of an administered compound in the liver is referred to as the "first-pass" effect and can result in significant differences between ingestion toxicity and toxicity due to other intake pathways. Inhalation of contaminants, on the other hand, results in the entry of the substance to systemic circulation almost immediately. In addition, contaminants are absorbed into the body with different

efficiencies when administered via different routes. The effectiveness of the skin as a barrier to foreign substances tends to cause dermal toxicity to be fairly low for systemic toxins. Inhalation toxicities are therefore typically the highest, followed by ingestion and dermal.

► **Example 11.2**

DDT is a pesticide that was formerly used for pest control. Shortly after its development, it was used to combat malaria in Italy by dusting people directly with DDT powder, and no ill effects were observed. However, laboratory studies have shown that DDT is a neurotoxin. The reason that DDT was safe in this situation is because it is not readily absorbed through the skin, whereas it is relatively readily absorbed through the gut. Thus, the oral toxicity of DDT is much higher than the dermal toxicity.

11.3.2 Quantification of Responses

A precise and quantitative means of expressing the toxic endpoint is a prerequisite for conducting a quantitative dose–response analysis. Without a replicable and directly measurable toxicity endpoint, quantitative analysis cannot be performed. The toxic endpoint can be expressed in a variety of ways. **Discrete endpoints** are those that are either present or absent in a person: for example, the presence or absence of cancer. Discrete endpoints are thus binary (i.e., they have only two possible values). **Continuous endpoints** are those that vary with dose. Mathematically, they may take on any value. This distinction corresponds roughly to the stochastic–deterministic distinction introduced previously. Stochastic responses are normally discrete (e.g., the presence or absence of a tumor), whereas deterministic endpoints are normally continuous (e.g., increased liver weight or depression of white blood cell counts).

The most common endpoint is a fractional population response or incidence at a given dose level. Such an endpoint is obtained by estimating the fraction of an exposed population exhibiting some quantal response. Although quantification of discrete effects is relatively straightforward, quantification of continuous responses in a population is problematic. The usual approach is to convert a continuous endpoint into a discrete endpoint as illustrated in Example 11.3.

► **Example 11.3**

A researcher is interested in studying the effect of a particular contaminant on the liver. A common effect of contaminants on the liver is to cause an increase in liver weight. The implication is that the adverse effect of the contaminant is proportional to the increase in liver weight (i.e., a marginal increase in liver weight is likely to have negligible health effects, whereas a 50% increase may cause severe liver and circulatory dysfunction). However, each exposed person will exhibit a different dose-related increase in liver weight. A common way of approaching this problem

is to define (based on some knowledge of the implication of liver weight increase) a particular level of increased liver weight as deleterious and regard everything above that as damaging and everything below that as safe. For example, a 10% increase in liver weight might indicate the onset of hepatic toxicity. Thus, the quantal response is a 10% increase in liver weight; those animals exhibiting less than a 10% weight gain are considered not to display the response, whereas all of those exhibiting a 10% weight gain or greater can be considered to exhibit a deleterious response. The continuous response (liver weight increase) has thus been converted to a discrete response (negligible vs. adverse level of liver weight increase).

Toxicological response data are the goal for both epidemiological studies of human exposures and controlled laboratory experiments on animals. A study might yield data such as those presented in Table 11.2. Since many populations exhibit a lognormal distribution of sensitivity to contaminants, fractional responses are often sigmoidal when the cumulative response is plotted against the log of the dose. A plot of the data from Table 11.2, with a dose–response function fitted to the data points, is given in Figure 11.5. Data and curves such as these can be generated for both the deterministic and stochastic effects of contaminants. If the contaminant

TABLE 11.2 Hypothetical Dose–Response Data

Daily Dose (mg/kg)	Fractional Response	Daily Dose (mg/kg)	Fractional Response
1	0.022	100	0.619
3	0.073	300	0.803
10	0.198	1,000	0.927
30	0.382		

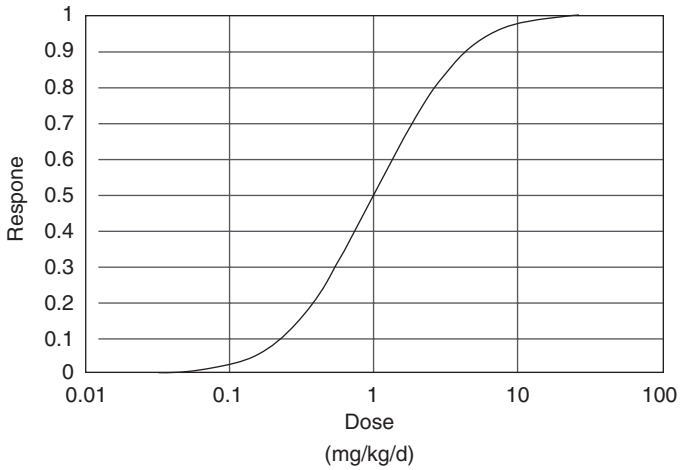

Figure 11.5 Dose–response curve.

has a threshold dose, there is a region where no response is expected. As the dose is increased above the threshold, there is a region of the curve at relatively low dose levels where a few susceptible people show a response. Following this is a region where a large fraction of the population exhibits symptoms of exposure. As the doses increase still further, the most resistant people in the population finally show the effects of exposure.

The types of data obtained are similar for both the stochastic and deterministic effects of contaminants; however, in the current practice of risk assessment the data are analyzed differently for the two types of effects. For deterministic effects, in which a threshold is assumed, the threshold dose is estimated. For stochastic effects, most of which are assumed to lack a threshold, a fractional response function is obtained. Understanding the way in which the fractional response function is quantified is necessary to understand the characterization of the risk from an exposure. This is discussed in more detail later in this chapter.

11.3.3 Sources of Dose–Response Data

11.3.3.1 Epidemiological Studies Human evidence for a causal link between a contaminant and a health effect and human data for developing a quantitative dose–response relationship are developed from epidemiological studies. For an epidemiological study to yield evidence of causality, several criteria related to the exposure and the effect must be met (Fleming and Bean 2000):

- Temporal relationship. The exposure must precede the effect.
- Plausibility. There must be a biological explanation for the relationship between the exposure and the effect.
- Dose–response relationship. The magnitude or incidence of the effect must vary in a systematic way in relation to the exposure.
- Statistical significance. The quantitative relationship between the exposure and effect should not be within the range of normal random variation.

Two types of observational epidemiological studies (cohort studies and case–control studies) can be used to establish dose–response relationships. In a **cohort study**, a contaminant exposure is identified, and the objective of the study is to identify health effects in the exposed population. This is accomplished by comparing the incidence of disease in the exposed population to that in a similar unexposed control population. A cohort study of the survivors of Hiroshima and Nagasaki has been, and is still being, used to develop dose–response relationships for the induction of cancer by radiation. Cohort studies are prospective in the sense that the exposure is identified first, and the exposed population is monitored for some period of time in the future to infer if effects occur.

► **Example 11.4**

Presented in Table 11.3 are data on the incidence of leukemia and other forms of cancer among survivors of the atomic bombings in Japan. Presented in the table

TABLE 11.3 Incidence of Leukemia and Other Forms of Cancer Among the Survivors of the Atomic Bombings in Japan

		Leukemia			Nonleukemia		
Dose (Sv)	Population	Observed	Expected	Ratio (Obs./Exp.)	Observed	Expected	Ratio (Obs./Exp.)
<0.01	34,272	58	88	0.66	2,443	2,593	0.94
0.01–0.1	23,321	38	61	0.62	1,655	1,688	0.98
0.1–0.5	11,500	32	20	1.6	927	866	1.07
0.5–1	3,500	19	6	3.2	329	273	1.2
1–2	2,000	23	3	7.7	218	147	1.5
2+	1,000	30	2	15.0	132	68	1.9

are six discrete dose ranges, the number of people in each dose range, the expected number of leukemias or other cancers based on a control population, the observed number (as of 1985) of cancers, and the ratio of observed to expected cancers. For example, it is seen here that there have not been any excess leukemia cases in the groups of survivors receiving a radiation effective dose of less than 0.1 Sv. However, there are more than seven times the number of expected leukemias in the group of survivors receiving radiation effective doses between 1 and 2 Sv. Data derived at high doses such as these are used to estimate the risk posed by lower levels of exposure.

In a **case–control study**, a group of subjects suffering from a specific disease is identified, and the objective of the study is to identify the cause, which could be a contaminant exposure, life style, heredity, or other factor. For a suspected contaminant, this is accomplished by comparing the exposure history of the affected population with that of an unaffected control population. Case–control studies are retrospective in that the effect is identified first and historical data are used to infer the cause. A case–control study established the link between DES (diethylstilbesterol) and vaginal cancer.

Other types of observational epidemiological studies are limited in their ability to generate dose–response information but can be used to generate hypotheses. Cross-sectional (or prevalence) studies can be used to compare the risks between exposed and unexposed (or greater- and lesser-exposed) populations. Ecologic studies are based on comparison of populations rather than individuals.

Epidemiological data are subject to uncertainty, due primarily to uncertainties in the estimated doses, the difficulty in obtaining appropriate control and experimental populations, and the presence of confounding factors such as other chemical exposures or life-style risk factors. Human epidemiological studies suitable for establishing dose–response relationships are limited to a very small number of contaminants. For this reason, dose–response relationships for most chemical contaminants must be derived from animal studies.

11.3.3.2 Animal Studies Several types of animal tests are used to quantify the deterministic toxicity of chemical contaminants. These are typically classified by their duration and the effect for which they test. Acute lethality tests establish the lethality of the contaminant under conditions of acute exposure. The contaminant is typically administered in a single exposure (duration less than 24 hours). A typical measure obtained from such a test is the median lethal dose, or $\mathbf{LD_{50}}$, which is defined as the single dose of a contaminant that results in the death of 50% of the test species under a specified set of conditions. Lethality tests can also be performed to establish the effects of extending the dose over a period of time (up to 30 days).

If a contaminant is selected for more extensive testing, longer-term studies may be performed to identify cumulative effects. Longer-term exposures are defined as those between 30 days and 10% of the animal lifetime. For typical laboratory species, this is approximately 90 days. These tests serve two functions: They generate estimates of a threshold dose if the contaminant is thought to have a threshold level of effect, and they allow for design of subsequent chronic tests by identifying

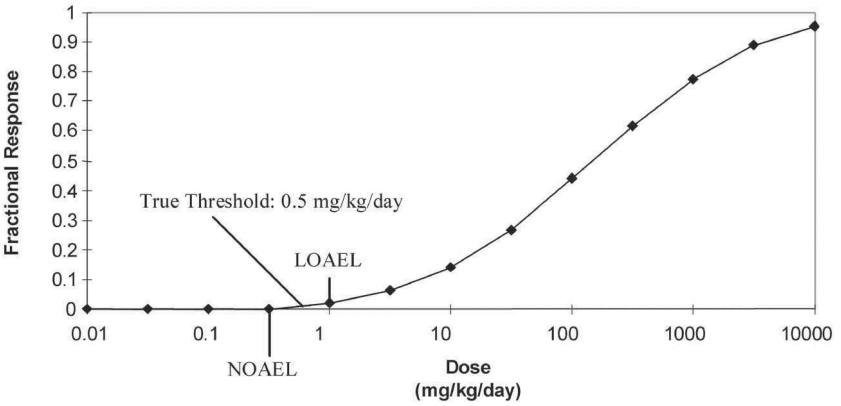

Figure 11.6 Relationship among the LOAEL, NOAEL, and threshold dose.

the **maximum tolerated dose** (MTD), the highest dose of a contaminant given over a specified period of time that does not result in increased overt toxicity. In practice, a commonly used indicator of overt toxicity is a suppression of body weight by more than 10%.

Chronic tests, which range anywhere from three months up to the lifetime of the animal, are used to obtain data on both dose thresholds and fractional response. Since the doses are increased discretely rather than continuously and since the entire population is not tested, the true threshold dose is not observable. Instead, there is a dose at which no statistically significant effects are observed in the exposed population, and the next highest dose yields detectable effects. The highest dose at which no statistically significant effects are observed in the exposed population is known as the **no observed adverse effects level** (NOAEL), and the lowest dose that results in detectable effects is known as the **lowest observed adverse effects level** (LOAEL). These two points bracket the true threshold dose in the exposed population, as illustrated in Figure 11.6. For carcinogenicity or other stochastic effects assumed to have no dose threshold, chronic tests provide fractional response data that form the basis for estimating responses at low doses.

► Example 11.5

In a study of the carcinogenic effects of acrylonitrile (Quast et al. 1980a), a particular species (Sprague–Dawley) of rats were fed acrylonitrile in drinking water at levels of 35, 100, and 300 ppm. A dose-dependent increase in tumors was observed. The data are given in Table 11.4 and the cumulative distribution (for nonzero dose levels) is plotted in Figure 11.7.

In another study (Quast et al. 1980b), the threshold for inhalation toxicity of acrylonitrile was evaluated. Groups of rats were exposed to 0, 20, and 80 ppm acrylonitrile. As a result of the exposure, inflammation and abnormal growth of nasal tissues were observed in some of the test animals. Results are given in Table 11.5 and plotted in Figure 11.8. For male rats, there was a significant toxic effect

TABLE 11.4 Stochastic Effects of Acrylonitrile on Sprague–Dawley Rats

Concentration (ppm)	Daily Dose (mg/kg)	Tumor Incidence
0	0	4/80
35	3.42	18/47
100	8.53	36/48
300	21.18	45/48

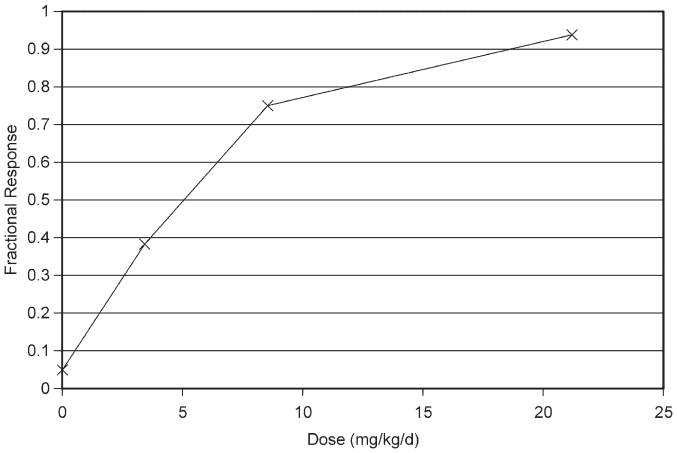

Figure 11.7 Fractional response curve for tumor incidence in rats exposed to acrylonitrile.

TABLE 11.5 Deterministic Effects of Acrylonitrile on Sprague–Dawley Rats

Concentration	Females (Nasal Inflammation)	Males (Abnormal Nasal Mucous Cell Growth)
0	2/11	0/11
20	6/10	4/12
80	7/10	10/10

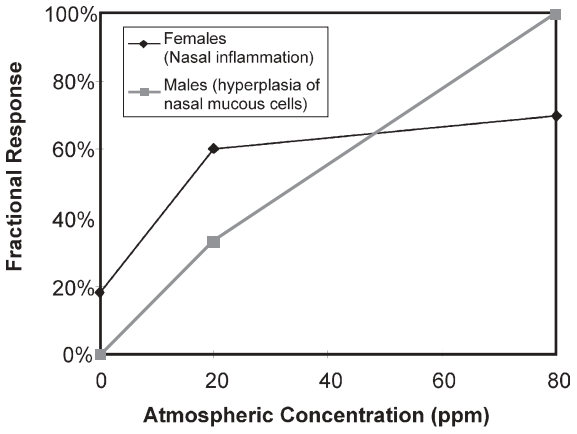

Figure 11.8 Dose–response curves for the effect of acrylonitrile on male and female rats.

at 20 ppm that was not evident at 0 ppm. Therefore, the threshold toxicity lies somewhere between 0 and 20 ppm. For female rats, there was an effect at 0 ppm, indicating the existence of a background incidence of the condition. However, the fraction of rats exhibiting the effect was greater at 20 ppm. Thus, for both male and female rats, 20 ppm is the LOAEL and a nonzero NOAEL was not observed.

Selection of dose levels for animal testing is constrained by two competing goals. On the one hand, doses are kept as low as possible to ensure that the toxicity of the chemical does not cause premature deaths. The MTD derived from subchronic tests is typically the highest dose used. Other doses are fractions of the MTD. In many animal studies the dose groups used are a control, 1/2 MTD, and MTD with each group consisting of 50 animals of each sex with both mice and rats, resulting in a minimum of 600 animals. The tests are run for two years and the animals are then sacrificed and examined. However, if dose levels are too low, it may be difficult to identify the level of response because of poor statistical resolution. Lowering the dose requires a corresponding increase in the numbers of animals required to provide a statistically adequate estimate of the response due to the chemical. A major factor driving the cost of a test is the number of animals used for the entire test. Obviously, the more animals that are needed, the greater are the costs involved in procuring the animals, caring for them, and performing the examinations. For example, subchronic and chronic studies using rats can range from hundreds of thousands to millions of dollars. With 50 animals per test group, a dose capable of causing an 8% ($p < 0.025$) increase in contaminant-induced tumors could result in the absence of tumors in the test group. To date, the largest study conducted, known as the ED01 study, was performed in the late 1970s by the National Center for Toxicological Research for the Food and Drug Administration. Designed to clarify the shape of the dose–response function at low levels, it used over 24,000 test animals and was designed to detect a 1% increase in tumor incidence. Animal testing at the regulatory target range 10^{-4} to 10^{-6} would require millions of animals and is not feasible.

Mere observance of a correlation between a toxic response and a particular chemical exposure does not itself justify a quantitative estimate of dose–response relationship. For example, an observed response attributed to a particular contaminant in a laboratory study may actually be due to a different agent (e.g., an impurity in the test solution). In animal studies, there is a great deal of control over the contaminant administered, and hence this is not typically a significant source of uncertainty. However, in epidemiological studies, it is considerably more difficult to associate a given effect with a given cause, although epidemiological studies can be buttressed by animal experimentation to identify the mechanism of action.

▶ Example 11.6

Consider a hypothetical epidemiological study in which workers who are exposed to an airborne organic solvent are tracked to determine if the exposure results in adverse effects. The study is performed and the exposed group is compared to a group of workers in a nearby town. If a statistically significant increase in lung

cancer is noted, the epidemiologist may then conclude that the solvent is a lung carcinogen. However, further suppose that the exposed population contains an unusually large population of smokers relative to the control population. Since cigarette smoke is carcinogenic, the smokers have a higher number of cancers. If these cancers are improperly attributed to the chemical, the carcinogenicity of the solvent will be overestimated.

A similar situation can arise even in controlled laboratory conditions. Consider another hypothetical situation in which laboratory studies are performed to determine the toxicity of the herbicide 2,4-D. In this study, the doses of 2,4-D are carefully measured to permit a precise quantification of the dose. However, unbeknown to the researcher, the herbicide is contaminated with minute levels of TCDD, a potent carcinogen. The carcinogenicity of the TCDD is so great that it can be responsible for most of the tumors resulting from the herbicide exposure. The researcher, unaware of the presence of the contaminant, may erroneously conclude that 2,4-D is a carcinogen based on the laboratory results.

11.4 DOSE–RESPONSE MODELING

The goal of dose–response modeling is to make a quantitative evaluation of the incidence of an adverse effect (such as cancer or systemic toxicity) that would be expected in a human population as a result of exposure to a particular amount of an environmental contaminant. From the perspective of scientific accuracy, this quantitative evaluation would ideally be based on evidence of adverse health effects arising from similar conditions of exposure and would be summarized in a mathematical relationship that expressed the fractional response of a population as a function of the dose received by that population. However, environmental exposures to chemical and radiological contaminants are often over long periods at low dose rates. Direct data on human health impacts are extremely limited for such exposures, since the effects may be statistically indistinguishable from the natural variability of the effect. Consequently, the quantitative estimation of human health effects due to environmental exposures usually requires two critical (and often controversial) extrapolations. If reliable data on health effects in human populations are not available from adequate epidemiological studies, dose–response relationships for humans must be inferred from animal studies, with the implicit assumption that animals are adequate models of human toxicological response. For stochastic responses, particularly those that have very low or nonexistent thresholds, a second extrapolation is required, which is potentially even more problematic. This is the extrapolation of effects at very high doses or dose rates to the very low doses or dose rates characteristic of environmental exposures.

Many dose–response analyses lack a firm foundation in biologically based models. The models that do exist are often highly simplified abstractions of the actual physiological processes involved in contaminant toxicity. In practice, such considerations dictate that effects be extrapolated from tests with similar durations and routes of exposure. If the dose rates or routes of contaminant exposure are substantially different from those used to develop dose–response data, the data must either be adjusted to reflect the changed conditions of exposure or the risk estimates must reflect the uncertainties associated with the extrapolation.

11.4.1 Animal-to-Human Extrapolation

Due to the large differences in size between humans and test animals, a given mass of contaminant does not have the same effect in humans as it does in animals. This is because the response is typically proportional to the concentration of a contaminant in a target tissue, not to the total amount of the contaminant in the organism. This necessitates the adjustment of the doses administered to laboratory animals to an equivalent human dose. The **equivalent human dose** is the dose that yields the same level of effect in human populations as that in animal populations. The methods that can be used to infer an equivalent human dose from an animal dose range from simple scaling laws to pharmacokinetic models that explicitly model the metabolism of the substance. If sufficient data are available, a pharmacokinetic model is the preferred method for scaling between humans and animal doses. These models are capable of providing a more accurate estimate of the equivalent human dose by determining the administered dose necessary to produce the same effective dose at the target organ in the test species and in humans.

Frequently, however, such models do not exist. In these cases, there are a variety of simple scaling relationships available for inferring equivalent human doses from animal doses, although there is considerable debate as to which relationship is most generally appropriate. The simplest relationship is based on body weight scaling. Early studies indicated that the LD_{50} was approximately equal in humans and animals when the mass of contaminant taken into the body was divided by the body weight. This observation led to the traditional chemical dose unit of mass of contaminant per unit body weight (BW), which is typically expressed in units of mg/kg (Dedrick 1973; Rhomberg and Wolff 1998). However, this relationship does not always hold true. The LD_{50} values are frequently found to be more nearly equal among species when a relationship based on body surface area is used. A mass scaled dose (in mg/kg) can be converted to a surface area scaled dose by noting that surface area is proportional to $BW^{2/3}$. Another simple scaling relationship endorsed by the U.S. EPA is based on relative metabolic rate, which is proportional to $BW^{3/4}$ (EPA 1992a). These relationships are reflected in the following general expression for estimating the equivalent human daily dose (D_{human}) corresponding to an animal daily dose (D_{animal}):

$$D_{\text{human}} = D_{\text{animal}} \left(\frac{\text{BW}_{\text{animal}}}{\text{BW}_{\text{human}}} \right)^{n} \tag{11.1}$$

where BW is the body weight in kilograms, D the dose in mg/(kg·d), and n the scaling factor. For surface area scaling, $n = 1/3$; for metabolic rate scaling, $n = 1/4$. These simple scaling relationships can be seen as highly simplified models of contaminant metabolism that are based on simple assumptions (e.g., that the most relevant physiological parameters are relative metabolic rates or surface areas of exchange boundaries) and require only readily available data (e.g., body weight).

11.4.2 High- to Low-Dose Extrapolation

For nonthreshold stochastic effects, the response at low doses is difficult to distinguish from the normal background incidence of the effect. This effect is exacer-

bated if the background incidence of the effect is relatively high, as is the case for many common cancers. Consequently, there are few, if any, actual dose–response data at low doses, and most human epidemiological studies and animal toxicological experiments are conducted at high doses and dose rates in an effort to yield statistically significant results in a relatively small group of exposed individuals. However, most environmental exposures are at much lower doses and dose rates than those characteristic of the data. This necessitates the use of models to predict the response at low doses and dose rates from data derived at high doses and dose rates. Several general types of models are used for low-dose extrapolation (Klaassen and Eaton 1991; van Leeuwen and Zonneveld 2001). These are **tolerance distribution models** for the distribution of individual toxic thresholds in a population, **mechanistic models** for the probability of developing cancer based on a theoretical understanding of the underlying carcinogenic processes, empirically derived **epidemiological models**, and **low-dose linearity** approaches. Tolerance distribution models are essentially parameterized statistical distributions, whereas mechanistic models draw on a theoretical understanding of the biology of the stochastic effect (usually, cancer) being modeled to predict the incidence. The underlying assumption of tolerance distribution models is that sensitivities are distributed in a population in a statistically random fashion. The underlying assumption of mechanistic models is that everyone is equally sensitive to cancer induction, and the probability of incidence is related to the dose. Epidemiological models are empirically based models in which functional relationship based on a biological understanding of how the contaminant may affect disease incidence is fitted to incidence data derived from observations of exposed human populations. Linear approaches rely simply on the assumption of low-dose linearity in the dose response function.

11.4.2.1 Tolerance Distribution Models For threshold effects, statistical models such as the probit (lognormal), the Weibull, and the log-logistic (logit) distributions and modifications of these, have been used to estimate the distribution of thresholds in a population. When applied to a population, these models yield an estimate of the fraction of people in a population exhibiting effects as a result of a particular dose. These models can be used for either deterministic or stochastic effects, provided that there is a threshold level of exposure required to induce the effect and that this threshold varies in a statistical manner among the exposed population. The mathematical forms of three common tolerance distribution models are given in Table 11.6.

The lognormal distribution is probably the most common statistical distribution in toxicology because it has been shown to fit experimental results for a wide array of contaminants. The Weibull and logit models are similar to the lognormal in their general shape; however, the logit model allows fitting data with a higher degree of variability. Since the Weibull and logit distributions predict threshold doses lower than those of the probit, they are typically considered to be more conservative.

11.4.2.2 Mechanistic Models of Carcinogenicity Mechanistic models are based on a biologically simple theoretical conceptualization of the underlying carcinogenic processes. Although such models can become mathematically complex, they are typically based on very simple biological assumptions. The most common models are “hit” models, multistage models, and cellular proliferation models.

TABLE 11.6 Statistical Tolerance Distribution Models

Model	Mathematical Form
Lognormal (log-probit)	$R(D) = \frac{1}{\sqrt{2\pi}} du \int_{-\infty}^{\rho_0 + \rho_1 \ln D} e^{-u^2/2}$
Log-logistic (logit)	$R(D) = [1 + e^{-(\rho_0 + \rho_1 \ln D)}]^{-1}$
Weibull	$R(D) = 1 - e^{-(\rho_0 + \rho_1 D^m)}$

TABLE 11.7 Mechanistic Models of Carcinogen Dose and Response

Model	Mathematical Form	Low-Dose Approximation ($\rho D << 1$)
One-hit	$R(D) = 1 - e^{-(\rho D)}$	$R(D) = \rho D$
Multistage	$R(D) = 1 - e^{-(\rho_0 + \rho_1 D + \ldots + \rho_k D^k)}$	$R(D) = \rho_0 + \rho_1 D$

The mathematical form of the one-hit and multistage models are presented in Table 11.7.

The **one-hit model**, first proposed in 1950 (Iverson and Arley 1950), is based on the hypothesis that exposure to even a single molecule of a carcinogenic substance (or a single particle of ionizing radiation) has the potential to cause cancer. At low doses, the probability of cancer increases approximately linearly as a function of dose. Multihit models (Cornfield 1977; Rai and Van Ryzin 1981) are a generalization of the one-hit model based on the hypothesis that two or more interactions must occur within a given time period to initiate a transformation. An important aspect of the hit models is that cancer susceptibility does not change with age.

► **Example 11.7**

The one-hit model can be fit to the data in Table 11.2 by estimating the parameter ρ for each of the data points and then averaging. The equation for the one-hit model is

$$R = 1 - e^{-\rho D}$$

where ρ is the cancer slope factor. This equation can be rearranged to yield

$$\rho = -\frac{\ln(1-R)}{D}$$

The daily average value of ρ for the data in Table 11.2 is 0.015 $(mg/kg)^{-1}$. Using this value in the one-hit model yields the fitted curve in Figure 11.9.

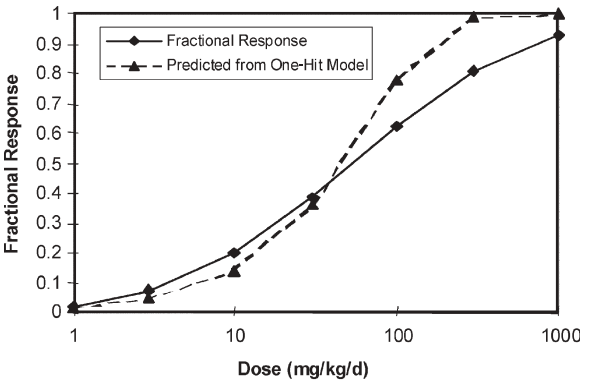

Figure 11.9 One-hit model fitted to the data in Table 11.2.

Another approach is based on the observation that age-specific incidence rates vary proportional to age raised to some power. Biologically, this suggests that a single cell must pass through several specific alterations prior to developing into a tumor. Because people who have lived longer are more likely to have cells that have passed through all the necessary stages, cancer susceptibility increases with age. **Multistage models** of carcinogenesis, proposed by Nordling (1953) and formalized mathematically by Armitage and Doll (1954), are based on the hypothesis that cancer results from several different events occurring in a fixed sequence, thereby causing the cell to pass through a fixed set of stages. This approach is similar to the multihit model, with the exception that the events must occur in a fixed sequence. If only one event is necessary to result in a tumor, the model reduces to the one-hit expression given in Table 11.7. Multistage models are useful because they fit a wide array of experimental data sets. At low doses, the approximate multistage model response is linear with respect to dose. A form of the multistage model that has been widely used in risk assessment is the linearized multistage model (Crump 1985), in which the linear portion of the multistage dose–response curve is constrained to lie at its 95th percentile confidence level. This approach has been used by the EPA to provide a conservative estimate of the cancer potency at low doses.

Both the hit model and the multistage model are based on interactions in single cells and do not account for proliferation and death in altered cells (van Leeuwen and Zonneveld 2001). The Armitage–Doll model was extended in 1957 (Armitage and Doll 1957) to account for cellular proliferation. A more complete treatment has been developed (Moolgavkar 1986; Moolgavkar et al. 1988) known as the **Moolgavkar–Venzon–Knudson** (MVK) **model**, in which the kinetics of cellular division and death are modeled explicitly for a two-stage carcinogenic process. Because the model can account for the dose-dependent effects on cell proliferation rates in addition to dose-dependent effects on genetic mutations, the model is particularly suited for examining the effect of nongenotoxic carcinogens.

11.4.2.3 Epidemiological Models of Radiological Risk In contrast to the theoretically derived mechanistic risk models discussed for chemical carcinogens,

empirically derived epidemiological risk models are the basis of risk estimates for exposure to radiation. Two general types of models are used, the absolute (or additive) risk model and the relative (or multiplicative) risk model. An **absolute** (or additive) **risk model** is used when the incremental risk due to radiation is independent of the background incidence. The absolute risk model assumes that the risk of a given type of tumor from exposure can be added to the natural background incidence of the tumor. Mathematically, this is expressed as

$$R(H) = r_0 + f(H)\,g(\beta) \tag{11.2}$$

A **relative** (or multiplicative) **risk model** is used when the incremental risk of cancer due to radiation depends on the background incidence in the population. The relative risk model assumes that the incremental risk due to exposure is a multiple of the natural background incidence of the tumor. Mathematically, this is expressed as

$$R(H) = r_0\,[1 + f(H)\,g(\beta)] \tag{11.3}$$

where H is the equivalent dose, r_0 the age-specific background risk of death due to a given type of cancer, $f(H)$ the dose–response model for radiation-induced cancer, and $g(\beta)$ a function that accounts for other important variables, such as gender, age, age at exposure, and time since exposure.

11.4.2.4 Linear Approaches to Low-Dose Extrapolation Linear approaches do not require any statistical or biological assumptions other than the low-dose linearity of the dose–response function. Because of this, they are sometimes termed "model-free" approaches. However, the approach is generally defended on the basis that the contaminant acts additively to some background rate of carcinogenesis (Crump et al. 1976; Hoel 1980). Such models are therefore related to the epidemiological approaches discussed in Section 11.4.2.3. Responses at low doses are defined simply by assuming that the dose–response curve is a straight line between some experimentally derived response at a given dose (e.g., the lowest dose with a statistically significant increase in tumors) and an assumption of zero response at zero dose. The approach was suggested in the 1980s (Gaylor and Kodell 1980; Van Ryzin 1980; Farmer et al. 1982; Krewski et al. 1984) and is reflected in current EPA guidance on carcinogenic risk assessment (EPA 2005) either when linearity can be demonstrated or when there is nothing known about the mode of action of the carcinogen.

11.4.2.5 Comparison of Risk Extrapolations Selection of an appropriate model can be problematic because current scientific knowledge is generally not available to identify the correct model conclusively. Often, several models can fit high-dose experimental data equally well, yet yield dramatically different estimates of the risk at low doses. The estimates of response based on extrapolations using the one-hit and multistage models can be several orders of magnitude greater than estimates based on extrapolations using the Weibull or multihit models, and the differences increase with decreasing dose. Selection of an appropriate model thus requires the exercise of professional judgment on the part of the analyst. Such

factors as biological plausibility, statistical goodness of fit, and desired degree of conservatism need to be taken into account in the selection of a model used to estimate the effects of a particular exposure.

11.5 RISK CHARACTERIZATION

As noted in Section 11.1, different metrics are used for threshold effects and nonthreshold effects. A margin of safety metric is typically used for threshold effects and a fractional response metric is typically used for nonthreshold effects. From the perspectives of regulatory policy and contemporary practice in risk assessment and risk management, all deterministic effects are categorized as threshold effects, and most stochastic effects are categorized as nonthreshold effects. However, from the perspective of science, the characterization of health effects associated with low doses is still an open question and is a topic of intense interest and study because of its implications with respect to regulatory policy.

11.5.1 Margin of Safety

Early methods in toxicological risk assessment were based on the observation of thresholds for many deterministic health effects. In general, if there is a population threshold dose associated with exposure to a given contaminant, it is possible to reduce exposures to a level protective of the entire population. The population margin of safety (MOS) is the ratio of the threshold dose to the actual dose:

$$\text{MOS} = \frac{\text{threshold dose}}{\text{actual dose}} \tag{11.4}$$

If the margin of safety is greater than 1, the threshold dose is greater than the actual dose, and no toxic effects are expected in the exposed population. If the margin of safety is less than 1, there is a possibility that effects may be observed. However, without knowledge of the shape of the dose–response curve, it is not possible to calculate a percentage of the population expected to display the toxic effect. Quantitative evaluation of risk when the margin of safety is less than 1 would require the use of an appropriate tolerance distribution model.

► **Example 11.8**

Based on experiments with mice, a human threshold dose for chronic ingestion exposure to an organic solvent is estimated to be 21 mg/(kg·d). An assessment of exposure to contaminated groundwater indicates that a reasonable maximum exposure level to the solvent in contaminated groundwater was 0.672 mg/(kg·d). The daily margin of safety at this level of exposure is thus (21 mg/kg·d)/(0.672 mg/kg·d) = 31. In other words, the exposure could be increased by a factor of slightly over 30 before toxic deterministic effects would be expected to occur.

One of the most significant limitations in the application of the MOS method of risk characterization is estimation of the threshold dose. It is extremely rare that data are available which give a reliable estimate of the threshold dose. One way of dealing with this is to use an experimentally derived estimate of the threshold dose adjusted by safety factors to calculate the margin of safety. This approach was suggested by Lehman and Fitzhugh (1954), who proposed that thresholds derived from toxicological studies be combined with a safety factor to identify an "acceptable daily intake", defined as an intake below which adverse health effects in human populations were expected to be extremely infrequent. In brief, the idea is that the threshold dose is estimated from epidemiological studies on human populations or from animal data. This estimate is used as a proxy for the true threshold and is then reduced by the use of safety factors. This is expressed as

$$\text{MOS} = \frac{\text{threshold dose estimate}}{\text{actual dose} \cdot \Pi \,\text{safety factors}} \tag{11.5}$$

The effect of including safety factors is to provide a more conservative estimate of the safety margin than would be achieved with an experimentally observed threshold alone.

The MOS approach is appropriate for quantifying exposures to a single contaminant. Exposure to a mixture of contaminants presents a challenge because information is needed on contaminant interactions in the body, but data on toxic interactions are extremely limited. Conceptually, the three types of interactions given in Table 11.8 may take place. An additive interaction is one in which the effect of simultaneous exposure to multiple contaminants is the sum of the individual effects. Unless there is information to suggest that synergistic or antagonistic reactions between contaminants may occur, responses are typically assumed to be additive. A synergistic interaction is one in which the effect of multiple contaminants is greater than the sum of the effects taken individually. Synergistic responses are of particular concern because of the potential to underestimate the risk posed by a mixture of contaminants. An antagonistic interaction is one in which the effect of multiple contaminants is less than the sum of the individual effects. Antagonistic interactions may result in an overestimate of risk. In most risk assessments, similar effects (those with similar mechanisms of toxicity) are assumed to be additive. Thus, for multiple contaminants with similar stochastic effects, the risk can simply be added. The professional judgment of a trained toxicologist is required when modeling contaminant mixtures that may have nonadditive interactions.

TABLE 11.8 Possible Effects of Chemical Interactions

Interaction	Effect
Additive	$R\left(\sum D_i\right) = \sum R_i(D_i)$
Synergistic	$R\left(\sum D_i\right) > \sum R_i(D_i)$
Antagonistic	$R\left(\sum D_i\right) < \sum R_i(D_i)$

11.5.2 Fractional Response

For contaminants that are assumed not to exhibit a threshold, an MOS approach is not possible, since any dose could result in some increased level of adverse effects. These contaminants are typically characterized by a fractional response obtained by estimating the dose and then determining, from the appropriate dose–response model, the fraction of the population that could be affected. This was illustrated in Figure 11.5, in which a dose of 1 mg/(kg·d) resulted in a response being observed in 50% of the population. In practice, this is not done graphically; rather, the dose–response models presented in Section 11.4 are used to estimate the response at a given exposure. A significant challenge in risk characterization lies in the selection of the appropriate model used to estimate the response. Ideally, this would be an appropriate mechanistic model that relates response to dose. Unfortunately, the mechanisms that lead to a physiological response are generally not known, particularly at the very low exposures characteristic of environmental contamination. Thus, the selection of a model is a mixture of scientific judgment and policy judgment. Typically (particularly for chemical carcinogens), the models and model parameters are conservative (i.e., they are biased to avoid underestimating the risk).

Although cancer is not the only possible stochastic effect of contaminant exposure, it has become the effect of greatest regulatory interest, and most work on characterization of stochastic health effects has been associated with cancer. A number of dose–response models for cancer risk or incidence were given in Section 11.4. These are based on either postulated mechanisms for carcinogenicity or epidemiological data. A significant characteristic of several of the models is that at low doses,[2] they may be approximated as linear functions of dose:

$$R \approx \rho D \tag{11.6a}$$

$$R \approx \rho E \tag{11.6b}$$

where R is the fraction of the population responding, ρ the cancer slope factor $[(\text{mg}/(\text{kg}\cdot\text{d}))^{-1}]$ or radiation risk factor (Sv^{-1}), D the chemical dose ($\text{mg}/(\text{kg}\cdot\text{d})$), and E the radiological effective dose (Sv).

Estimating the total incidence of the stochastic effect in an exposed population is straightforward. Given the fractional response due to a given dose and the total population exposed at that dose, the total incidence is

$$I = RP \tag{11.7}$$

where I is incremental incidence of effects in the exposed population, R the fractional response at dose D or H, and P the population receiving the dose. For a linear, nonthreshold dose–response model as expressed in Eq. 11.3, the incremental incidence can be estimated as

$$I = \rho D P \tag{11.8a}$$

[2] Actually, the approximation assumes low values of the product ρD, where ρ is the cancer slope factor (or radiation risk factor) and D is the dose. However, sufficiently low doses will usually cause this product to be small, regardless of the value of ρ.

$$I = \rho EP \tag{11.8b}$$

Estimating the risk to a specific person in the exposed population is difficult, for the reasons discussed in the introduction. Common practice in risk assessment is to assume that the risk faced by a given person is the same as the fractional response given by Eq. 11.1. This approximation holds given that no additional information is known about the person that might indicate their susceptibility to cancer relative to the entire exposed group. Hence, the risk is simply the chance that a person selected at random from that population has cancer as a result of the exposure (i.e., if every year, I persons out of a population of P contract cancer, the individual risk is $I/P = \rho D$).

► **Example 11.9**

A population is exposed to polychlorinated biphenyls at a dose of 7×10^{-5} mg/(kg·d) and dieldrin at a dose of 4×10^{-6} mg/(kg·d). Assume that the cancer slope factor for the PCBs is 4.34 $(\text{mg/(kg·d)})^{-1}$ and for dieldrin is 30 $(\text{mg/(kg·d)})^{-1}$. Assuming that the cancer risk from this mixture is additive, the combined risk from these two contaminants is calculated as follows:

$$\begin{aligned} R &= \rho_{\text{PCB}} D_{\text{PCB}} + \rho_{\text{Dieldrin}} D_{\text{Dieldrin}} \\ &= [4.34(\text{mg/(kg·d)})^{-1}]\,[7 \times 10^{-5}\,\text{mg/(kg·d)} + 30\ (\text{mg/(kg·d)})^{-1}] \\ &\quad [4 \times 10^{-6}\,\text{mg/(kg·d)}] \\ &= 3.1 \times 10^{-4} + 1.2 \times 10^{-4} \\ &= 4.3 \times 10^{-4} \end{aligned}$$

If 10,000 people are exposed at this level, an upper bound on the number of contaminant-induced cancers is

$$I = RP = 4.3 \times 10^{-4} \times 10{,}000 = 4.3$$

Although deterministic effects are not usually characterized using the fractional response method, it is possible to perform such a characterization using a method exactly analogous to that used for modeling stochastic effects. Threshold deterministic effects exhibit a distribution of thresholds across a population. If the average contaminant dose and distribution of thresholds were known, the fraction of the population that would be expected to experience adverse effects could be estimated.

11.6 REGULATORY IMPLEMENTATION

The preceding discussions were generic in nature. Because of the many uncertainties and judgments required in carrying out risk assessments, many of the judgments have been standardized in various regulatory programs. In the following

section we deal with some of the methods that are currently being used to implement the concepts discussed previously.

11.6.1 Deterministic (Noncancer) Endpoints

The acceptable daily intake concept was modified and adopted by the EPA, which has traditionally defined a quantity known as the **reference dose** (RfD). The reference dose is intended as a conservative measure of the dose that is expected to be without appreciable effects over a lifetime of exposure to the most sensitive member of a population. Because deterministic effects are typically assumed to be characterized by a threshold, the use of a reference dose is typically associated with the noncarcinogenic systemic effects of contaminants. These reference values are tabulated in the EPA Integrated Risk Information System (EPA 2006). They are typically derived from a NOAEL or LOAEL modified by the application of uncertainty factors as follows:

$$\mathrm{RfD} = \frac{\mathrm{NOAEL}}{\prod_{\mathrm{i}} \mathrm{UF}_i} \tag{11.9}$$

where RfD is the reference dose and the UF_i are uncertainty and variability factors. These factors are used as appropriate to account for a variety of extrapolations and uncertainties, including variability in sensitivity among human populations, uncertainties in extrapolating human sensitivity from animal data, extrapolation from subchronic to lifetime exposures, use of a LOAEL vs. a NOAEL, and use of incomplete databases (EPA 2002). Assignment of these factors is carried out by expert judgment prior to entry into the IRIS database and takes into consideration the extent of the experimental database supporting the reference value and the scientific quality of the studies comprising the database. Typically, each of these factors is a value of either 10 or 3 ($10^{0.5}$) as a default. Conceptually, this could give rise to a composite UF value of 100,000. However, the technical panel convened to review the reference dose and reference concentration process recommends that a combined UF of greater than 3000 suggests that the database is insufficient to derive a reference value.

There are significant limitations to the approach described above for calculating the RfD (Gaylor 1983; Kimmel and Gaylor 1988). Most notably, the value of the NOAEL or LOAEL is highly dependent on the experimental design. Since the doses in animal experiments are typically varied in discrete steps, the NOAEL must be one of the experimental dose levels; and determination of the NOAEL does not use any other dose–response information. Furthermore, the possibility that a larger study might result in a lower LOAEL cannot be excluded. In general, NOAEL/LOAEL approaches do not yield a consistent level of protection to exposed populations. To overcome this drawback, an alternative metric known as the benchmark dose (BMD) was proposed (Crump 1984, 1995). The **benchmark dose** is the dose corresponding to a specified level of response, the **benchmark response** (e.g., a 1%, 5%, or 10%). The benchmark dose is usually obtained by fitting a dose–response function to the data in the experimental range and determining the confidence interval on the dose at the benchmark response level. The

lower confidence interval on the dose corresponding to the specified level of response is the **benchmark dose lower limit** (BMDL). Alternative methods have been suggested for determining the BMDL in which the requirement for using a dose–response function is relaxed or eliminated (Bosch et al. 1996). A reference dose can then be computed on the basis of the BMDL rather than a NOAEL or LOAEL. Presented in Table 11.9 are reference doses for selected chemical contaminants along with the point of departure (NOAEL, LOAEL, or BMDL) for the assigned values.

Chronic inhalation toxicity is sometimes quantified with a no-effects atmospheric concentration rather than a no-effects inhalation dose. A measure similar to the reference dose can be computed by substituting the no-effects atmospheric concentration for the no-effects inhalation dose in Eq. 11.6. This yields a quantity known as the **reference concentration** (RfC). The reference concentration is not a measure of dose due to inhalation; rather, the concentration of the contaminant in air is used directly rather than computing intake from inhalation. Because the effects of acute exposure to airborne contaminants frequently depends more on momentary concentrations than on integrated exposures, use of the total dose over a period of time is not always appropriate for acute exposures. An exposure time correction (adjustment of the concentration to account for different exposure periods) can be performed as discussed in Section 11.3.1.3.

The quantitative measure of threshold effects follows a margin of safety approach and is termed the **hazard quotient** (HQ). It is derived from either a reference dose or a reference concentration as follows:

$$\mathrm{HQ} = \frac{D}{\mathrm{RfD}} \tag{11.10}$$

$$\mathrm{HQ} = \frac{C}{\mathrm{RfC}} \tag{11.11}$$

where D is the average daily dose (for either subchronic or chronic exposures), RfD the reference dose, RfC the reference concentration, and C the average airborne concentration. Strictly speaking, the hazard quotient is not a measure of risk. A hazard quotient of less than 1 indicates that the exposure is not expected to result in any adverse effects. A hazard quotient of greater than 1 does not suggest that adverse effects are expected but they are possible. Similarly, comparison of the hazard quotients for two different contaminants does not necessarily yield any information on the relative risk of the two different exposures.

To quantify the risk from exposure to multiple contaminants with deterministic effects, the hazard quotients for individual contaminants and/or multiple exposure pathways are summed to yield the **hazard index** (HI):

$$\mathrm{HI} = \sum_i \mathrm{HQ}_i \tag{11.12}$$

A hazard index of less than 1 indicates that no effects are likely to be observed. If the composite HI is greater than 1, the situation is a bit more complicated. It may be possible to segregate contaminants by critical effect (i.e., the first toxic response observed as the dosage is increased) and then sum HQs only for contaminants with

TABLE 11.9 Oral Reference Doses for Selected Contaminants

Contaminant	Critical Effect	Basis for Reference Dose	Point of Departure (mg/(kg·d))	Oral RfD (mg/(kg·d))
Aldrin	Liver toxicity	LOAEL	0.025	3×10^{-5}
Arsenic	Hyperpigmentation, keratosis, and possible vascular complications	NOAEL	0.0008	3×10^{-4}
Benzene	Decreased lymphocyte count	BMDL	1.2	4.0×10^{-3}
Cadmium	Significant proteinuria	NOAEL	0.005	5×10^{-4} (water)
Carbon tetrachloride	Liver lesions	NOAEL	0.71	7×10^{-4}
Chlordane	Hepatic necrosis	NOAEL	0.15	5×10^{-4}
Chlorine	No observed adverse effects	NOAEL	14.4	1×10^{-1}
Chloroform	Moderate to marked fatty cyst formation in the liver and elevated SGPT	BMDL10	1	1×10^{-2}
Chromium(VI) (18540-29-9)	None reported	NOAEL (ADJ)	2.5	3×10^{-3}
DDT	Liver lesions	NOEL	0.05	5×10^{-4}
Dieldrin	Liver lesions	NOAEL	0.005	5×10^{-5}
Ethylene dibromide	Testicular atrophy, liver peliosis, and adrenal cortical degeneration	LOAEL	27	9×10^{-3}
Heptachlor epoxide	Increased liver/body weight ratio in both males and females	LEL	0.0125	1.3×10^{-5}
Lindane	Liver and kidney toxicity	NOAEL	0.33	3×10^{-4}
Methylene chloride	Liver toxicity	NOAEL	5.85	6×10^{-2}
PCBs (Aroclor 1254, 11097-69-1)	Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger- and toenails, decreased antibody (IgG and IgM) response to sheep erythrocytes	LOAEL	0.005	2×10^{-5}
Tetrachloroethylene	Hepatotoxicity in mice, weight gain in rats	NOAEL	14	1×10^{-2}
Vinyl chloride	Liver cell polymorphism	NOAEL (HED)	0.09	3×10^{-3}

Source: EPA 2006.

similar critical effects. However, such estimation may be time consuming and complex and if performed improperly, may misrepresent the actual toxic response. EPA practice is to segregate the HQs according to exposure duration (i.e., acute, chronic, and subchronic exposures) and then report a separate hazard index for each exposure duration (EPA 1989). In a case where a composite HI of greater than 1 exists, the judgment of a trained toxicologist is typically necessary to properly interpret the effect of multiple chemical exposures.

► Example 11.10

A home garden is used for raising vegetables on a plot of land contaminated with aldrin and heptachlor epoxide. Doses from the consumption of produce from the garden are calculated to be 2.3×10^{-5} and 6.4×10^{-6} mg/(kg·d), respectively. From Table 11.9, the RfDs for aldrin and heptachlor epoxide are 3×10^{-5} and 1.3×10^{-5} mg/(kg·d), respectively. The hazard index for this scenario is found by the addition of the separate hazard quotients:

$$\text{HI} = \frac{2.3\times10^{-5}\ \text{mg/(kg·d)}}{3\times10^{-5}\ \text{mg/(kg·d)}} + \frac{6.4\times10^{-6}\ \text{mg/(kg·d)}}{1.3\times10^{-5}\ \text{mg/(kg·d)}} = 0.77 + 0.49 = 1.26$$

In this case, the hazard index is greater than 1, indicating that there may be a need for remediation. However, due to the application of safety factors, it is unlikely that there will be any observable effects.

Hazard quotients and hazard indexes are not calculated for the deterministic effects of radioactivity. Although reference doses per se are not defined for radionuclides, limits based on threshold doses for deterministic effects of exposure to ionizing radiation can be prescribed. Acute thresholds for deterministic effects (expressed as an equivalent dose) are 0.5 Sv to the bone marrow for depression of hematopoesis, 0.5 to 2 Sv to the lens of the eye for detectable opacity and 5 Sv for cataracts, 0.15 Sv to the testes for temporary sterility, and 3.5 to 6 Sv for permanent sterility. These values are reflected in U.S. Nuclear Regulatory Commission exposure limits for occupationally exposed workers (10 CFR 20.1201) that require radiological equivalent doses to individual organs to be less than 0.5 Sv (except for the lens of the eye, which is limited to less than 0.15 Sv). These values are based on prevention of deterministic effects. However, reference doses are developed for the chemical toxicity of several radioactive elements because some radionuclides, such as ^{238}U, may be present in the environment at sufficient concentrations to cause deterministic effects due to their chemical toxicity, even when the radiological effects are relatively minimal.

11.6.2 Stochastic Nonthreshold Endpoints

For contaminants that do not exhibit thresholds, incidence is the typical endpoint of concern. Cancer incidence is currently estimated using a linear, nonthreshold

approximation for exposure to both carcinogenic chemicals and ionizing radiation. The functional form of this relationship was given in Eq. 11.6. The key parameter in the equation is the cancer slope factor or the radiation risk factor.

For chemical carcinogens, slope factors are typically obtained from animal data, although human epidemiological data are preferred, if available. One approach is to fit a dose–response model such as those presented in Table 11.6 or 11.7 to the data with the slope factor being the slope of the curve at low doses. For many years, slope factors recommended and published by EPA were the upper 95% confidence level of the slope based on the linearized multistage (LMS) model. A second approach recently adopted by EPA (2005) uses a concept similar to benchmark dose defined in Section 11.6.1 as a **point of departure** for estimating the slope factor. For example, when linearity can be demonstrated or is assumed, the slope factor is the benchmark response divided by the benchmark dose. Slope factors published in EPA's Integrated Risk Information System (IRIS) are based on the LMS model, the benchmark dose, or both. These approaches probably provide a conservative estimate of the actual cancer risk. Slope factors for selected contaminants are given in Table 11.10.

For ionizing radiation, a unified framework for estimating the risk of stochastic effects has been developed by a variety of national and international agencies. This framework is based on the radiological effective dose introduced in Section 9.2.2. The effective dose is a single measure of dose that is comparable across a broad range of exposure scenarios. Current estimates of the risk of fatal cancer following exposure to ionizing radiation rely heavily on observations of cancer fatalities from survivors of the atomic bombings of Hiroshima and Nagasaki. The current recommended values for the risk coefficient are $0.04\,\mathrm{Sv}^{-1}$ for workers and $0.05\,\mathrm{Sv}^{-1}$ for the general public (ICRP 1991).

If cancer risks are high (i.e., over 0.01), the dose–response curves typically depart from linearity and Eq. 11.6 cannot be used. In such cases, the one-hit model may be used to provide a rough estimate of the risk. Fractional response according to the one-hit model is given by

$$R = 1 - e^{-\rho D} \tag{11.13a}$$

$$R = 1 - e^{-\rho E} \tag{11.13b}$$

However, cancer risks in the 1% range or higher are generally well above the levels of risk allowed in U.S. regulatory programs. Precise quantification of these risks is therefore not typically necessary in assessments conducted to support a regulatory decision. For assessments conducted for other purposes, contaminant-specific dose–response models are generally more appropriate than the generic, mechanistic one-hit model because they provide a more accurate quantification of the risk.

For exposure to multiple contaminants, carcinogenic risk is assumed to be additive and is summed as in Example 11.4. Aggregating carcinogenic risks is straightforward. For each receptor population, the risks due to each contaminant and exposure pathway are summed as in Example 11.9. If these risks are high, the risks may be summed as follows:

TABLE 11.10 Cancer Slope Factors for Selected Contaminants

Contaminant	Weight of Evidence	Tumor Type	Oral Slope Factor(s) $[mg/(kg \cdot d)]^{-1}$	Extrapolation Method
Arsenic	Human carcinogen	Skin cancer	1.5	LMS
Benzene	Human carcinogen	Leukemia	0.015–0.055	Linear extrapolation of human occupational data
DDT	Probable human carcinogen based on sufficient evidence of carcinogenicity in animals	Liver tumors, benign and malignant	0.34	LMS
Ethylene dibromide	Likely to be carcinogenic to humans based on strong evidence of carcinogenicity in animals and inconclusive evidence of carcinogenicity in an exposed human population	Forestomach tumors, hemangiosarcomas, thyroid follicular cell adenomas or carcinomas	2 (95% upper bound)	LMS
Vinyl chloride	Human carcinogen	Total of liver angiosarcoma, hepatocellular carcinoma, and neoplastic nodules	0.72 (adult exposures) 1.5 (lifetime exposures)	LMS LED 10/linear method

Source: EPA 2006.

$$R = 1 - e^{-\sum_i \rho_i D_i} \tag{11.14a}$$

$$R = 1 - e^{-\sum_i \rho_i E_i} \tag{11.14b}$$

where i is an index variable that ranges over all combinations of exposure pathways and all contaminants.

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PROBLEMS

11.1 The data in Table 11.11 are obtained in animal tests of a carcinogenic chemical.

(a) Determine the average carcinogenic risk factor for these data using the one-hit model and a mass extrapolation from animals to humans.

(b) Determine the carcinogenic risk factor using the point-of-departure approach. The benchmark response is 0.1, the corresponding BMD is 3.54 mg/kg·d, and there is evidence that the dose–response relationship is linear at low dose. Use the mass extrapolation from animals to humans.

TABLE 11.11 Animal Testing Data for Problem 11.1

Dose (mg/kg·d)	Number of Animals Contracting Cancer
0	5/50
10	18/50
20	24/50
40	37/50
80	45/50

11.2 The EPA maximum contaminant level in water for vinyl chloride is 1 μg/L. What is the cancer risk for a chronic exposure at this level?

11.3 An accident at a nuclear reprocessing plant in France results in the release of ^{85}Kr to the atmosphere. A resident in the vicinity of the plant is exposed to an airborne concentration of 1000 Bq/m^3 for a period of 2 hours. What are the deterministic and stochastic risks associated with the exposure?

11.4 As a result of the Chernobyl accident, the average concentration of ^{137}Cs in "total diet" samples in the Ovruch region of the Ukraine is 800 Bq/kg_{wet}. Using dietary uptake factors for fruits, vegetables, meat, and dairy from Table 9.1, estimate the yearly cancer risk to an adult due to consumption of food contaminated at this level.

11.5 Show that the animal to human extrapolation based on surface area yields the following relationship between animal dose, D_a, and human dose, D_h:

$$D_h \text{mg}/(\text{kg}\cdot\text{d}) = D_a \text{mg}/(\text{kg}\cdot\text{d})\left(\frac{\text{BW}_a}{\text{BW}_h}\right)^{1/3}$$

Approximate animals and humans as spheres.

11.6 Presented in Table 11.12 are average contaminant concentrations in brown trout from a hypothetical lake. Assuming risk to be additive, determine the cancer risk due to consumption of 120 g of fish per week by a 70-kg man for 70 years. Comment on the result.

TABLE 11.12 Contaminant Concentrations in Brown Trout for Problem 11.7

Contaminant	Concentration (mg/kg)
PCBs	1.76
Chlordane	0.13
Dieldrin	0.10
DDT	0.43

11.7 On the same graph, sketch the following: (**a**) linear, nonthreshold dose–response, (**b**) nonlinear, threshold dose–response, and (**c**) quadratic, nonthreshold dose–response.

11.8 In laboratory tests of a chemical, mice weighing 80 g ingest 2 mg of the chemical per day.

(a) What is the human equivalent dose based on a mass extrapolation?

(b) What is the equivalent human dose based on a metabolic rate extrapolation?

11.9 **(a)** In laboratory tests of a chemical, a net cancer incidence of 40% is observed among rats exposed at a dose of 25 mg/kg·d. What is the

carcinogenic risk factor based on the one-hit model and a metabolic rate extrapolation from animals to humans?

(b) If the BMDL (before extrapolation from animals to humans) for a carcinogenic contaminant is 0.7 mg/kg·d, the benchmark response is 0.05, and nothing is known about the carcinogenic mode of action of the contaminant, what is the carcinogenic risk factor? Use a metabolic rate extrapolation from animals to humans.

11.10 Estimate the risk associated with the consumption of aflatoxin in peanut butter sandwiches. Base the calculation on the consumption of one sandwich a day for 35 years. The average concentration of aflatoxin in peanut butter is 3 ppb and the average amount of peanut butter in a sandwich is 30 g. Assume that the carcinogenic slope factor for aflatoxin is 2900 $(mg/kg \cdot d)^{-1}$.

11.11 Assuming an inhalation cancer slope factor for benzene of 5×10^{-6} $(mg/kg \cdot d)^{-1}$, calculate the concentration in air corresponding to a risk of 10^{-5} for a lifetime exposure at a breathing rate of $20\,m^3/d$.

11.12 The average concentration of PCB's in fish in a hypothetical lake is approximately 3 ppm. Calculate the risk of fatal cancer to a person who eats fish from the lake three times a week for 20 years. The average serving size is 100 g. Assume a carcinogenic risk factor for PCBs of 8.9 $(mg/kg \cdot d)^{-1}$.

11.13 A medium–sized banana contains approximately 450 mg of potassium. Naturally occurring potassium contains ^{40}K, which is radioactive, at a concentration of approximately 31 Bq/g. Consider a person who ingests one banana per day for 30 years. Estimate his increased risk of fatal cancer as a result of ingestion of ^{40}K in those bananas that he has been consuming. The ingestion dose factor for ^{40}K is 5×10^{-9} Sv/Bq and the carcinogenic risk factor for fatal cancer is $0.05\,Sv^{-1}$. Should the person stop eating bananas?

11.14 Using a randomly selected number from 0 to 9, or a number assigned by the instructor, choose the chemical corresponding to the number according to Table 11.13. Use the IRIS database and/or another database found through TOXNET to obtain the following information:

TABLE 11.13 Contaminant List for Problem 11.14

Number	Contaminant
0	Aldrin
1	Arsenic
2	Benzene
3	Carbon tetrachloride
4	Ethylene dibromide
5	Lead
6	Methylene chloride
7	Polychlorinated biphenyls (PCBs)
8	Trichloroethylene (TCE)
9	Vinyl chloride

(a) Name of chemical and prior or current uses.

(b) Reference dose and principal deterministic effects (specify whether animals or humans) for (**i**) ingestion and (**ii**) inhalation.

(c) Carcinogenic slope factor and type of cancer (specify if based on humans or animals).

12 Uncertainty and Sensitivity Analyses

12.1 INTRODUCTION

Previous chapters have provided the technical basis for performing each of the four steps of the risk calculation process described in Chapter 1: (1) release assessment (Chapter 3), (2) transport assessment (Chapters 4 to 8), (3) exposure assessment (Chapter 9), and (4) consequence assessment (Chapters 10 and 11). It should be apparent that each of these steps involves uncertainty, often considerable uncertainty. It has long been recognized that the application of risk calculations requires a recognition and consideration of these uncertainties. Frequently, the uncertainties of the risk assessment play a major role in defining, evaluating, and choosing various risk management options. The objective of this chapter is to describe the different types of uncertainty, their sources, and methods used to describe and evaluate the uncertainties, both qualitative and quantitative. A fundamental goal of uncertainty analysis is to estimate the distribution of the risks associated with a given scenario. From this distribution a central measure of the risk, such as the mean or median, may be derived. A feature of some uncertainty analyses is the identification of factors that either dominate the risk (i.e., the risk drivers or parameters with great influence) or dominate the variability of the risk (i.e., those parameters that cause most of the variability in the result). The term **sensitivity analysis** is usually used to describe the process of identifying these factors. The risk analyst can use a sensitivity analysis to determine the most potent strategies for improving the risk calculation. These strategies include (1) obtaining more data to reduce the uncertainty in identified model parameters, (2) refining particular aspects of the analytical model, (3) revising the analytical goal to better suit the decision-making process, or (4) performing additional fundamental research.

12.2 TYPES AND SOURCES OF UNCERTAINTY

12.2.1 Qualitative and Quantitative Considerations

As discussed in previous chapters, risk assessment relies on models to estimate the assessment measure or performance measure(s) for a system of interest. As shown schematically in Figure 2.1, the problem statement and system description provide the framework for using a model to produce a numerical result, which is an estimate

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of the endpoint or performance measure. To avoid confusion and simplify the discussion, the term assessment measure is used in the chapter to describe this numerical result. The models introduced in previous chapters are largely **deterministic models**; that is, for each set of model input parameters, the model produces a single estimate of the assessment measure. Several models used in environmental risk assessment are **stochastic models**; that is, for each set of model input parameters, the model produces a statistical distribution of the assessment measures. This discussion of uncertainty focuses on deterministic models.

As described in Chapter 2, the system description step of model development yields both qualitative and quantitative information. Qualitative information is used to develop the model structure. For example, interviews with the inhabitants in the vicinity of a contaminated facility located next to a river reveal that they eat fish caught in the river. Consequently, an aquatic food chain pathway should be considered in modeling human health risk from the site. As an example of semiquantitative information, test cores of earth and rock from various locations around a site show a compact clay layer that starts at 20 m and extends down to 22 m. A model of contaminant transport by groundwater might incorporate this information by assuming an aquitard at a depth of 20 m. Thus, the nature and location of a boundary condition is determined. Quantitative data are frequently used to determine model input parameters. For example, the same test cores that identified the aquitard might be used to determine the hydraulic conductivity of the water table aquifer. Each core might yield a different value, so the modeler might choose the average value of the samples as the representative hydraulic conductivity.

The context in which uncertainty is considered is shown in Figure 12.1. Information ranges from very qualitative to very quantitative. Qualitative information influences the modeling approach; quantitative information typically influences the value of parameters used in the quantitative model, but may also influence the conceptual model. This system can be represented mathematically as

$$z = h(x_1, x_2, \ldots, x_n; I) \tag{12.1}$$

where z is the assessment measure of interest; h represents the analytical model, which as shown in Figure 1.6 may be a chain of linked models; the x_i are the parameters; and I symbolizes the qualitative information (in some mathematical literature, z is simply called the output variable and the x_i are the input variables). Uncertainty can attach to both the quantitative values of parameters and the qualitative information. Frequently, uncertainty in the assessment measure is determined by propagating the uncertainty in the parameters through the model. This is because several well-established methods are available to quantify the relationship. The influence of qualitative information on the assessment measure is treated less frequently because the methods are less definitive and harder to apply. However, the qualitative inputs can have a major impact on the assessment measure estimates, in both magnitude and type.

12.2.2 Sources of Uncertainty

Many schemes are available for categorizing uncertainty (van der Sluijs 1996). The sources of uncertainty in environmental risk analyses may be categorized as (1) parameter uncertainty, (2) model uncertainty, and (3) scenario uncertainty

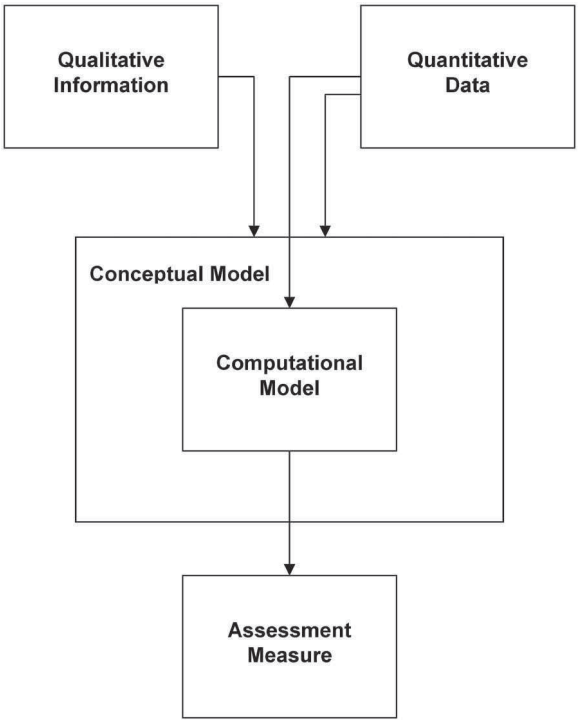

Figure 12.1 How quantitative and qualitative information are incorporated into the conceptual and computational models.

(Cranwell and Helton 1981; EPA 2001; Linkov and Burmistrov 2003). **Parameter uncertainty** can arise from variability of the parameter in space, time, or across a population. For example, percolation rate may be an important parameter affecting contaminant transport in the unsaturated zone. Percolation rate may vary spatially across a site because of variations in topology and vegetation. It also varies in time because rainfall and evapotranspiration are seasonal. Breathing rate and body mass are both important parameters in determining inhalation dose. Both parameters vary across a population, depending on the age, gender, and life-style of the people in the population. In addition, the breathing rate depends on the level of physical activity engaged in by each person.

Model uncertainty can apply to any of the modeling steps in Figure 2.1: conceptual, mathematical, or computational. Substantial errors can be and have been made in transforming conceptual models to mathematical models and subsequently, to computational models. For example, in groundwater transport of contaminants, numerical dispersion, an artifact of the numerical method, may overwhelm the actual physical dispersion being modeled (Bear 1987). However, as discussed in Chapter 2, several methods are available for assuring the quality of numerical models.

Considerations of model uncertainty tend to focus on the conceptual model. Most of the uncertainty in the conceptual model can be attributed to two general areas. The first is completeness (i.e., are all the important physical and chemical processes represented in the model?). The second is suitability (i.e., have the choices made regarding dimensionality, discretization, scale, time dependence, etc.

been chosen to provide meaningful results given the system characteristics and the problem statement?). As discussed in Chapter 2, each set of choices for key aspects of the model leads to a different conceptual model. Even if transparently unsuitable conceptual models are eliminated, several conceptual models appropriate for the problem statement and system description may remain.

Furthermore, a significant number of these models may be consistent with field measurements of the assessment measure or intermediate quantities. Consideration of model uncertainty often focuses on evaluation of these feasible alternative models. For example, Poeter and Anderson (2005) compare alternative models to field data by using well-defined quantitative comparison measures for hydrogeologic models. A more general approach to these issues is to perform **model validation**, wherein model predictions of the assessment measure are compared to field measurements in a more general fashion. However, this is difficult or impossible for many models used in environmental risk assessment; consequently, there is little agreement over how to define model validation in this context, much less how to accomplish it (Konikow and Bredehoeft 1992; Pescatore 1995; Eisenberg 1999). In the absence of field data for comparison to model predictions, methods for characterizing these uncertainties tend to be qualitative or, if quantitative, subjective.

The term **scenario uncertainty** is used in a variety of contexts with slightly different meanings. In many applications of risk analysis the term "scenario" is used to describe a particular set or sequence of events and conditions. For environmental risk analysis, a scenario may describe each qualitatively different type of release (as discussed in Example 12.2). In nuclear waste disposal problems the basis for formulating scenarios are often disruptive events external to the disposal system, such as volcanism, seismicity, human intrusion, and climate changes (Campbell and Cranwell 1988; Eisenberg et al. 1999). In this context, scenarios describe the future states of the system or the environment in which the system operates. In other cases, such as the remediation of contaminated sites (DOE 2005), the term "scenario" is used to describe hypothetical or generic individual exposures. For example, "building occupancy" and "resident farmer" scenarios describe generic exposure scenarios for decommissioned nuclear material sites (Kennedy and Strenge 1992). Other analysts use scenarios to describe certain initial defects in the system as well as future states (SKB 1999). For any of these meanings of "scenario", uncertainty may relate to the qualitative nature of the scenario, whether all scenarios have been included (completeness), or the likelihood of the various scenarios. However, the classification of uncertainties is not always as simple as the preceding discussion implies because these categories are not necessarily mutually exclusive. For example, hydrologic parameters such as permeability, porosity, and distribution coefficient for input into geosphere transport models are based on field and laboratory data. However, these parameter values are usually obtained by interpreting data according to a model of the physicochemical processes occurring in the tested material. Thus, parameter uncertainty may also contain a large element of model uncertainty.

12.2.3 Types of Uncertainty

Uncertainty has also been classified, based on the nature of the uncertainties, into two distinct categories: (1) uncertainties arising from the random nature of events,

processes, and systems, and (2) uncertainties arising from a lack of knowledge. In some literature (IAEA 1989; NCRP 1996), these two types of uncertainty are referred to as "type A" and "type B", respectively. Others (Apostolakis 1994) have adopted the more descriptive terms **aleatory** (related to chance) and **epistemic** (related to knowledge). Sometimes the less precise terms "variability" and "uncertainty" are used (EPA 2001). Still others use the terms "stochastic" and "subjective" (Helton 1994). These concepts can be illustrated through a hypothetical dice game. The outcome for each roll of the dice is random if the game is fair (unbiased). If a pair of dice is thrown, there are 11 possible values (2 to 12). The frequency with which each value occurs may be determined either by repeated trials or by calculation assuming that the six faces of each die are equally likely. However, the outcome of any particular roll is not predictable because processes generating the outcome are random. This is aleatory uncertainty. A separate issue is whether or not the game is, in fact, fair. This may depend on such factors as the venue (friend's house, casino, back alley), who brought the dice (yourself, the person rolling, the house), and who is rolling the dice (are the dice shaken or cupped?). In fact, the game is either fair or not; but one may only have a gut feel about the game's honesty, described by a "subjective probability." The likelihood of winning the bet depends on both types of uncertainties.

Aleatory uncertainty refers primarily to variability of risk assessment parameters spatially, temporally, or across a population. Examples include:

1. *Variability of meteorology or air dispersion characteristics.* These time-varying properties are often characterized by the joint frequency distribution of wind direction, wind speed, and stability class.
2. *Variability of hydrogeologic parameters across a site.* Properties such as hydraulic conductivity, dispersivity, hydraulic gradient, aquifer thickness, and distribution coefficient vary spatially and have a profound influence on contaminant transport.
3. *Variability of characteristics or responses across a population.* Properties such as the bioconcentration factor for fish, biouptake factor for plants, threshold dose for human response, and exposure parameters (breathing rate, consumption rate of green vegetables) vary across their respective populations.

Variability can be represented by a frequency distribution, which is a function that represents the relative frequency with which different values of a parameter are observed. Variability can sometimes be reduced by reducing the scale of the question or by disaggregating the inputs. For example, the variability in fish consumption can vary widely across the entire population, due in part to the presence in the overall population of subsistence fishermen who consume a great deal of fish. However, a site-specific survey might reveal that the rate of fish consumption in a subpopulation has less variability, and therefore the range in fish consumption rates in that subpopulation might be smaller than in the entire population.

Epistemic uncertainty arises from limitations of knowledge. This lack of knowledge may be related to parameters, models, and scenarios in an environmental risk assessment. The epistemic uncertainty associated with the value of a parameter may be expressed qualitatively or with varying degrees of quantification. For

example, the vegetative yield, Y, is a parameter required to calculate the concentration of a contaminant in vegetation from air deposition, as described by Eq. 8.11. Alternative treatments of the epistemic uncertainty in this parameter include:

1. Specification of a single estimate for a value based on limited data or expert judgment, accompanied by a description of the factors, such as rainfall, rate of fertilizer application, or insect infestation that could affect the value of the parameter
2. Specification of nominal, high estimate, and low estimate values based on limited data and expert judgment, thereby allowing calculation of a nominal, high, and low estimate of the assessment measure
3. Specification of a probability distribution for the value of Y, based on data and expert judgment, with epistemic uncertainty attached to the distribution type and statistics (e.g., mean and standard deviation)

Similarly, uncertainty in the distributions used to characterize aleatory uncertainty is another manifestation of epistemic uncertainty because it represents a lack of knowledge. For example, in an aqueous system with suspended solids, the concentration of a contaminant in aqueous and solid phases depends on the suspended solid concentration, SS, as shown in Eqs. 5.8, 5.9, and 5.10. The aleatory uncertainty in SS may be described by a probability distribution, but the distribution type and statistics (such as the mean and standard deviation) are uncertain due to lack of knowledge.

Epistemic uncertainty associated with models may include:

1. *Uncertainty regarding the completeness of the model.* Have all relevant processes and phenomena been included in the model?
2. *Uncertainty regarding the choices for various aspects of the model* (e.g., dimensionality, time dependence, boundary conditions, and initial conditions). Are these choices supported by field data? Do choices intended to produce pessimistic (i.e., conservative) results produce them under the entire range of modeled conditions? Does the model produce consistent results under nominal values of parameters as well as under extreme parameter values?
3. *Uncertainty regarding model validity.* Is there any evidence to support the application of the model in this context? Is extrapolation of the model valid (e.g., extrapolation of animal data to human toxicology)? Do existing site data support alternatives to the preferred model?

Epistemic uncertainty associated with scenarios may include:

1. *Completeness.* Have all significant potential scenarios (future states) been incorporated in the model?
2. *Representativeness.* Do the scenarios have enough range and variability to represent important occurrences?

Epistemic uncertainty is typically represented using a probability distribution, that is, a mathematical function that assigns a certain probability of occurrence to

each particular alternative. For example, each alternative conceptual model of a site could be assigned a probability representing the likelihood that the model best represents system behavior. For many environmental models, parameter values can be used to represent alternative models; in such cases, model uncertainty may be propagated as if it were parameter uncertainty. For example, hydraulic communication between a water table aquifer and a confined aquifer may be a key factor in determining where contamination in the confined aquifer originated. Total isolation of the two aquifers (i.e., an aquitard between them) could constitute one conceptual model, while total hydraulic coupling of the two aquifers could constitute a second conceptual model. Alternatively, a leakage parameter could be used to describe the degree of coupling (Freeze and Cherry 1979), and a continuous probability distribution could be applied to values of the leakage parameter to indicate the degree of belief in various degrees of hydraulic communication.

The distinction between aleatory and epistemic uncertainty has operational importance that goes beyond mere semantic differences. Depending on the type of uncertainty, different strategies are used to reduce uncertainty and decision makers treat types of uncertainty in risk assessments differently. Because aleatory uncertainties are inherent in the environmental system, strategies for reducing their impact on risk estimates are limited. One option is to modify the controllable portion of the system to minimize the impact of portions of the system dominated by aleatory uncertainty (e.g., reduce the likelihood of a release so that the uncertainties attendant to environmental transport are minimized). Epistemic uncertainties may often be reduced by obtaining more information. For the example above of the leakage between aquifers, additional data might be gathered by pump tests, tracer tests, core samples, or a combination of tests to determine more precisely the nature of the hydraulic communication between the two aquifers. Because decision makers evaluate aleatory and epistemic uncertainty differently while making decisions, it has become more of a conventional practice to display these uncertainties separately when presenting the results of risk assessments. However, classifying a particular model parameter as solely epistemic or aleatory may be difficult, if not impossible, because both aspects usually contribute.

12.3 STATISTICAL FUNDAMENTALS

Regardless of the sources or types of uncertainty in a given environmental risk assessment, they are described, quantified, and analyzed using the language, concepts, and mathematics of probability and statistics. Aleatory and epistemic uncertainties are both characterized by distribution functions. Although some authors (Kaplan 1991) carefully use frequency distributions to describe aleatory uncertainty and probability distributions to describe epistemic uncertainty, the distinction is not made here, except when the context requires it, since both probability and frequency distributions follow the same mathematical form. Although the mathematical formalism is the same, the meaning of the distribution is different for aleatory and epistemic uncertainty; this difference is important in interpreting results.

The following discussion is a brief introduction to some concepts of basic statistics. Because a complete exposition of these topics is beyond the scope of this book,

the reader is urged to consult any of the standard introductory textbooks on probability and statistics (e.g., Bowen and Bennett 1988; Ross 2004) for a full and complete development of this material.

12.3.1 Random Variables and Distribution Functions

Fundamental to probability and statistics is the concept of a **random variable**,[1] which is a real-valued function defined over a sample space with a probability measure. Random variables may be either continuous or discrete. An example of a continuous variable is the flow rate of a river over an extended period of time such as a year. Most parameters in risk assessments are treated as continuous variables. An example of a discrete variable is the final system state shown in the example of an event tree (Figure 3.6). Each release type (none, small, medium, large) has a certain probability of occurrence, which may be estimated using the event tree.

Any continuous random variable may be characterized by a **cumulative distribution function** (CDF), defined by

$$F(x) = \Pr\{\boldsymbol{X} \le x\} \tag{12.2}$$

$F(-\infty) = 0$ and $F(+\infty) = 1$. Closely related to the CDF is the **probability density function** (PDF), $f(x)$, which is a real-valued integrable function. The PDF is defined by the integral equation

$$F(x) = \int_{-\infty}^{x} f(x')\,dx' \tag{12.3}$$

If $F(x)$ is differentiable, this may be differentiated to yield

$$\frac{dF}{dx} = f(x) \tag{12.4}$$

By definition,

$$\int_{-\infty}^{+\infty} f(x')\,dx' = 1 \tag{12.5}$$

Also, $0 \le f(x') \le 1$.

The physical meaning of the PDF is difficult to conceptualize; however, $f(x)\,dx$ is the probability of finding the random variable in the infinitesimal interval $[x, x + dx]$. The probability of finding the random variable between a and b is

$$\Pr\{a \le \boldsymbol{X} \le b\} = \int_{a}^{b} f(x)\,dx \tag{12.6}$$

[1] To distinguish them from ordinary variables, many texts, this one included, denote random variables in boldface upper case type. In this discussion, $\boldsymbol{X}$ is the random variable and x is a particular value that the random variable may take. The notation $\Pr\{L(\boldsymbol{X})\}$ represents the probability of the term in braces, which is usually a logical relationship involving the random variable $\boldsymbol{X}$.

The **complementary cumulative distribution function** (CCDF)[2] is defined by

$$F^C(x) = \Pr\{\boldsymbol{X} > x\} = \int_x^{+\infty} f(x)\,dx \tag{12.7}$$

or

$$F^C(x) = 1 - F(x) \tag{12.8}$$

The CDF is a monotonically increasing function of x, while the CCDF is monotonically decreasing. Because the CDF is monotonic, its inverse is also a defined function; that is, if

$$F(x) = \alpha = \Pr\{\boldsymbol{X} \le G(\alpha)\} \tag{12.9}$$

then

$$x = G(\alpha) = G(F(x)) \tag{12.10}$$

The inverse of the CCDF may be defined similarly. Presented in Figure 12.2 are the PDF, CDF, and CCDF for the standard normal[3] distribution.

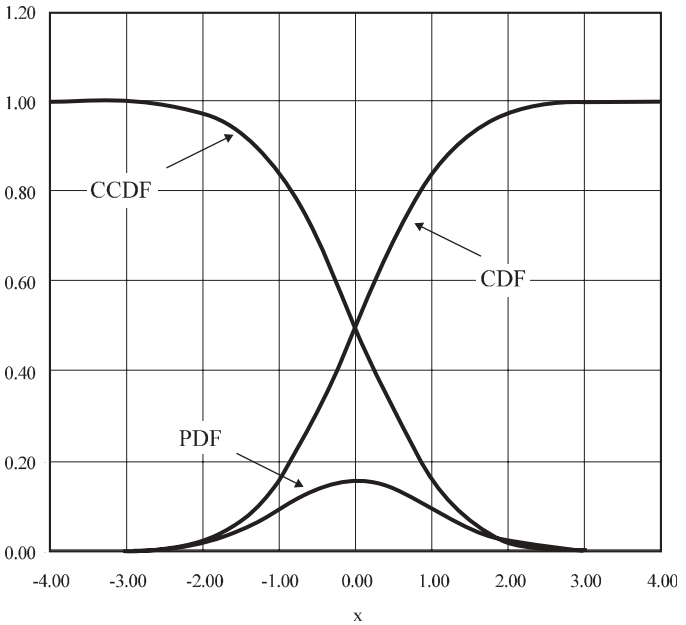

Figure 12.2 PDF, CDF, and CCDF for the standard normal distribution.

[2] The CCDF is sometimes termed the survival function in reliability studies.
[3] The normal distribution is also called the Gaussian distribution, which is the same function as that used to describe concentrations resulting from instantaneous point releases in the chapters on contaminant transport.

Random discrete variables may be characterized by a **probability mass function** (PMF) $p(x)$. The probability mass function is the parallel for discrete random variables of the PDF. It associates a probability with each possible discrete value x_i of the random variable, where the x_i are ordered from smallest to largest.

$$p(x) = \Pr\{\boldsymbol{X} = x_i\} \tag{12.11}$$

The discrete equivalent of Eq. 12.5 is

$$\sum_{i=1}^{N} p(x_i) = \sum_{i=1}^{N} \Pr\{\boldsymbol{X} = x_i\} = 1 \tag{12.12}$$

For a discrete random variable, the CDF and CCDF may be defined by Eqs. 12.3 and 12.7, respectively.[4] However, because the PMF has a finite number of discrete values, the CDF for a discrete random variable is simplified to be

$$F(x_n) = \Pr\{\boldsymbol{X} \le x_n\} = \sum_{i=1}^{n} p(x_i) \tag{12.13}$$

and the CCDF is

$$F^C(x_n) = \Pr\{\boldsymbol{X} > x_n\} = \sum_{i=n+1}^{N} p(x_i) \tag{12.14}$$

▶ Example 12.1

The sum of the numbers obtained by rolling a pair of dice is a discrete random variable. The sample space is the set of all the possible outcomes, $[a,b]$, for a pair of dice (i.e., {[1,1], [1,2], [2,1], . . .}) and the random variable $\boldsymbol{X}$ is the sum corresponding to each point in the sample space (i.e., {2, 3, 3, . . .}). The first column in Table 12.1 contains the x_i's (i.e., the sums), and the second column contains the

TABLE 12.1 Probability Mass Distribution, Cumulative Distribution Function, and Complementary Cumulative Distribution Function for the Sum of a Pair of Dice

Sum, x_i	$\Pr\{\boldsymbol{X} = x_i\}$	CDF ($\Pr\{\boldsymbol{X} \le x_i\}$)	CCDF ($\Pr\{\boldsymbol{X} > x_i\}$)
2	1/36	1/36	35/36
3	2/36	3/36	33/36
4	3/36	6/36	30/36
5	4/36	10/36	26/36
6	5/36	15/36	21/36
7	6/36	21/36	15/36
8	5/36	26/36	10/36
9	4/36	30/36	6/36
10	3/36	33/36	3/36
11	2/36	35/36	1/36
12	1/36	1	0

[4] The entire formalism for continuous random variables may be applied to discrete random variables if the use of the delta function and step function (introduced in Chapter 2) is allowed. Then the PMF is represented by a summation of delta functions, and the CCDF and CDF are represented by a summation of step functions.

probabilities for each sum (i.e., $\Pr\{\boldsymbol{X} = x_i\}$). The table also contains the CDF and the CCDF for the distribution.

12.3.2 Descriptive Statistics

Frequently, a distribution is characterized by descriptive statistics derived from the underlying distribution. The most common and most important of these statistics, the **arithmetic mean** (also average or first moment of the distribution), is defined by

$$\mu = \int_{-\infty}^{+\infty} x f(x)\, dx = E(\boldsymbol{X}) \tag{12.15}$$

where $E(\boldsymbol{X})$ denotes the expected value of the random variable $\boldsymbol{X}$. The **variance**, which is the second moment about the mean, is defined by

$$\sigma^2 = \int_{-\infty}^{+\infty} (x-\mu)^2 f(x)\, dx = \mathrm{var}(\boldsymbol{X}) \tag{12.16}$$

where $\mathrm{var}(\boldsymbol{X})$ denotes the variance of the random variable $\boldsymbol{X}$. In certain contexts the **standard deviation** σ, derived from the variance, is preferred:

$$\sigma = +\sqrt{\sigma^2} = +\sqrt{\mathrm{var}(\boldsymbol{X})} \tag{12.17}$$

Figure 12.2 shows the PDF, CDF, and CCDF for the standard normal distribution, which is defined to have a mean of zero and a standard deviation of 1. Thus, $x = -1$ corresponds to one standard deviation below the mean, $x = +1$ corresponds to one standard deviation above the mean, and so on. The value of the CDF at $x = -1$ is about 0.16, and the value of the CCDF is 0.84 (i.e., the probability of x falling between $-\infty$ and -1 is 0.16 and the probability it is greater than -1 is 0.84). For any normal distribution the probability that the random variable is between one standard deviation below the mean and one standard deviation above the mean, $\Pr\{\mu - \sigma \le \boldsymbol{X} \le \mu + \sigma\}$, is approximately 0.68.

Another commonly used statistic is the **median**, which may heuristically be defined as the value of the random variable such that values higher and lower are equally likely. For a continuous random variable this implies that $F(\text{median}) = 0.5$. In terms of Eq. 12.10,

$$\text{median} = G(0.5) \tag{12.18}$$

Many different kinds of distributions are used to describe random variables. Some of the distributions frequently used to describe random variables in environmental risk assessment are shown in Table 12.2.

12.3.3 Determination of Distributions

Given this brief introduction to basic statistics, the system representation shown in Eq. 12.1 may be reinterpreted to be a relationship among random variables,

$$\mathbf{Z} = h(\boldsymbol{X}_1, \boldsymbol{X}_2, \ldots, \boldsymbol{X}_n; \boldsymbol{I}) \tag{12.19}$$

TABLE 12.2 Properties of Distributions Commonly Used in Environmental Risk Analysis

Distribution	Parameters	PDF	Mean	Variance
Uniform	α, β real $\alpha < \beta$	$\frac{1}{\beta-\alpha}$, $\alpha \le x \le \beta$ 0 otherwise	$\frac{\alpha+\beta}{2}$	$\frac{(\beta-\alpha)^2}{12}$
Normal	μ, σ real $\sigma > 0$	$\frac{1}{\sigma\sqrt{2\pi}}\exp\left\{-\frac{(x-\mu)^2}{2\sigma^2}\right\}$ $-\infty < x < +\infty$	μ	σ
Lognormal	μ_Y, σ_Y $Y = \ln X$	$\frac{1}{\sigma\sqrt{2\pi}}\exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right]$ $-\infty < x < +\infty$	μ_X $\exp\left(\mu_Y + \frac{\sigma^2}{2}\right)$	σ_x^2 $[\exp(\sigma_Y^2) - 1]\exp(2\mu_Y + \sigma_Y^2)$
Beta	$\alpha, \beta > 0$	$\frac{\Gamma(\alpha+\beta)}{\Gamma(\alpha)\Gamma(\beta)}x^{\alpha-1}(1-x)^{\beta-1}$ for $0 < x < 1$ 0 otherwise	$\frac{\alpha}{\alpha+\beta}$	$\frac{\alpha\beta}{(\alpha+\beta)^2(\alpha+\beta+1)}$

where now the assessment measure $\boldsymbol{Z}$, the system parameters $\boldsymbol{X}_i$, and the qualitative information $\boldsymbol{I}$ may all be treated as random variables. Although every parameter may be treated as uncertain, in practice many variables have so little uncertainty that they are generally treated as constant. For example, in calculating the gravitational settling velocity of a particle, the gravitational acceleration, g, is treated as a constant even though it varies slightly with location. Constant parameters may be included in Eq. 12.19 by letting their variance be zero. In many cases, to simplify the notation, parameters considered to be constant are not included in Eq. 12.19 or are cited with their values in a separate table; however, it is usually important to document the rationale for considering certain parameters to be constant while considering others to be uncertain.

By considering the uncertainties represented by Eq. 12.19, the nature of the environmental risk assessment changes from determining the assessment measure as a function of parameters (Eq. 12.1) to one of determining the *distribution* of the assessment measure as a function of the *distributions* of the parameters. Thus, an important step in performing an environmental risk assessment with uncertainty is the determination of the distributions that characterize the input parameters of the assessment.

For generic assessments or site-specific screening assessments, generic distributions of parameters may be applied. Compilations of generic parameter distributions are published by various regulatory bodies (IAEA 1994; EPA 1997) or may be incorporated in computer programs (Napier et al. 2004). For detailed, site-specific assessments, site-specific parameter distributions should be used.

Both formal and informal methods are used to obtain site-specific parameter distributions, based on site-specific data. Some of the methods available to interpret site-specific data to obtain a representative distribution include the use of physical reasoning regarding the nature of the input parameter; an empirical CDF, given sufficient data; Bayesian updating with regional or generic distributions as the prior (Meyer et al. 1997); identification of several candidate distributions based on goodness of fit with available data; and a combination of methods (EPA 2001).

Typically, a combination of analytical methods and expert judgment by the analyst are used to develop a parameter distribution. Since site-specific data are generally sparse, there are usually insufficient data to select a probability distribution definitively. Furthermore, the model input parameter frequently represents the aggregate behavior of the system component, such as the effective hydraulic conductivity of a hydrogeologic unit or the average consumption rate of contaminated feed by cattle in a study area. Consequently, the distribution of measurements of the variable in the field is used to determine an average (arithmetic mean) or other representative value. However, in such cases the variability of the raw field data is likely to be larger than the derived variability in the average value.

■ Variability of the Mean of a Parameter

The amount of data available to determine model parameters in an environmental assessment are frequently sparse. For this reason it is important to distinguish between the aleatory distribution of values measured in the field and the epistemic uncertainty in a model parameter derived from field measurements (Meyer and Gee 1999). Consider a sample of n values of a physical variable measured in the field. For example, it might be K_D values measured by batch testing a few soil samples from a site. An appropriate modeling parameter is the mean of the K_D values for the entire population of small soil volumes across the site. The mean value is unknown, because only a small sample has been measured. However, a good estimate of the population mean is the sample mean:

$$\bar{X} = \sum_{i=1}^{n} \frac{X_i}{n}$$

where $\bar{X}$ is the sample mean, X_i are measurements of a particular environmental quantity, and n is the number of measurements. For repeated sampling, each sample of n measurements will yield a different sample mean. Because the sample mean averages over high and low values, the distribution of the sample means is narrower than of the underlying population, as shown in Figure 12.3.

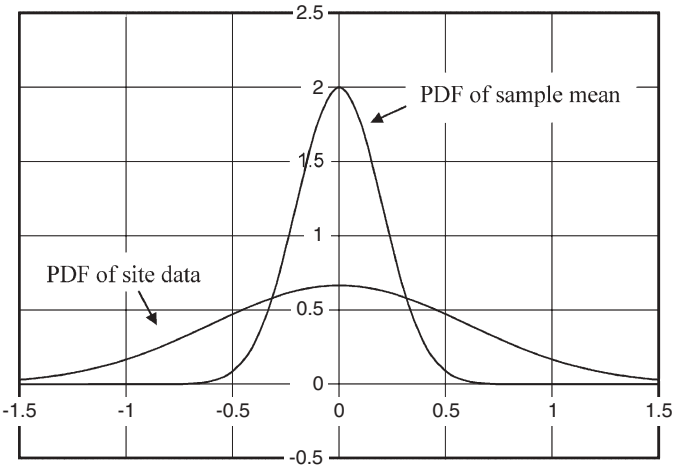

Figure 12.3 Example illustrating that variability of a parameter is greater than the uncertainty in the mean value of the variable.

Physical reasoning may be used to evaluate limited data and choose a representative distribution. For example, if the variable by its physical nature must be nonnegative, distributions possessing that property (e.g., the lognormal distribution) may be more appropriate candidates. Example 12.2 illustrates how physical arguments may be used to estimate parameter values based on the physical configuration of the system.

► Example 12.2

A common, simplifying approach in groundwater modeling is to combine several parallel stratigraphic units into a single hydrogeologic unit with an equivalent, composite hydraulic conductivity. The equivalent hydraulic conductivity is derived using physical reasoning that depends on the direction of groundwater flow (Bear 1972; Freeze and Cherry 1979). A hydrogeologic unit composed of n layers, each with thickness d_i and hydraulic conductivity $K_{H,i}$, is illustrated in Figure 12.4. The equivalent hydraulic conductivity of the entire unit, when the flow is perpendicular to the layers, is given by a type of harmonic mean:

$$K_{H,\text{equivalent}} = \frac{d_{\text{total}}}{\sum_{i=1}^{n} d_i / K_{H,i}}$$

where $d_{\text{total}} = \sum_{i=1}^{n} d_i$. However, when the flow is parallel to the layers, the equivalent hydraulic conductivity of the entire unit is given by the arithmetic mean of the transmissivity of each layer:

$$K_{H,\text{parallel}} = \frac{1}{d_{\text{total}}} \sum_{i=1}^{n} K_{H,i} d_i$$

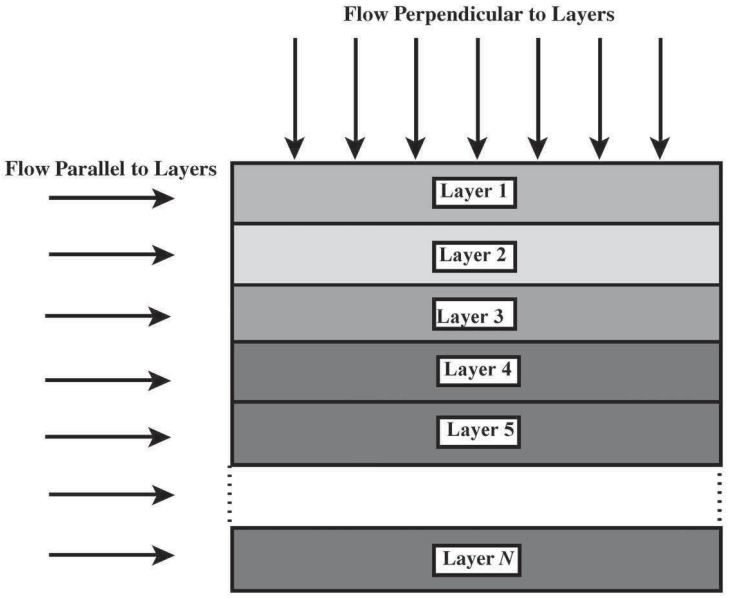

Figure 12.4 Hydrogeological unit for Example 12.2. The unit is composed of layers with differing hydraulic conductivities.

Thus, the value of the hydraulic conductivity used to represent the composite hydrogeologic unit depends on the principal direction of the flow, and the underlying distribution is based on physical reasoning.

Another approach to determining distributions for input parameters from sparse data is to use the principle of maximum entropy, first developed for information theory (Harr 1987). This method uses the type and amount of limited information available to determine the distribution, with the constraint that the uncertainty is maximized (Barnard et al. 1992; EPA 2001). This formalism makes the choice of the distribution objective. Depending on the type and amount of limited information available, the form of the distribution is constrained as shown in Table 12.3.

Increased use is being made of **expert elicitation** as another method for quantifying distributions, especially those related to epistemic uncertainty.

TABLE 12.3 Maximum Entropy Distributions for Various Types of Input Information

Information Provided	Resulting Probability Distribution
Range	Uniform
Expected value, standard deviation	Normal
Range, expected value, standard deviation	Beta

Expert elicitation is a formal, highly structured process that should be distinguished from expert judgment, which is generally informal and ubiquitous in environmental analysis. Expert judgment is used by virtually every analyst for every element of the analysis. It is used to make choices of great and small import, such as the choice of a particular conceptual model or computer code and the choice of the number of significant figures used to represent physical constants. Expert elicitation uses subjective probabilities (probabilities based on the expert's feelings as informed by knowledge) to describe the degree of belief in a particular state of nature. Expert elicitation is resource intensive, so it is usually employed to address issues that are very significant but when limited alternative approaches are available. For example, a sequence of studies (Budnitz et al. 1997; NAS–NRC 1997; Savy et al. 2002) addressing earthquake hazards to nuclear power plants in the eastern United States, including development of expert elicitation methods, deal with substantial uncertainties in geosciences data and models as well as disagreements among experts.

Several guides (Meyer and Booker 1990; Kotra et al. 1996; NCRP 1996) have been issued on the conduct of an expert elicitation. Although somewhat different approaches to handling various aspects of the expert elicitation are advocated (e.g., the role of the normative expert), the essential elements are similar. One approach (Kotra et al. 1996) divides the process into nine steps: (1) definition of objectives, (2) selection of experts, (3) refinement of issues and problem decomposition, (4) assembly and dissemination of basic information, (5) preelicitation training, (6) elicitation of judgments, (7) postelicitation feedback, (8) aggregation of judgments (including treatment of disparate views), and (9) documentation. As one might expect, the focus of the guidance is on procedural issues, since the technical input comes from the elicited experts and the assembled technical information on the subject.

12.4 UNCERTAINTY PROPAGATION

In practice, uncertainty analysis can be cast in terms of two closely related questions (Helton and Davis 2002):

1. Given the uncertainty in the input parameters, $\boldsymbol{X}_i$, what is the uncertainty in $\boldsymbol{Z}$?
2. What is the relative importance of each $\boldsymbol{X}_i$ in determining the behavior of $\boldsymbol{Z}$?

Both of these questions may be addressed by uncertainty propagation.

To simplify the problem of uncertainty propagation, the explicit reference to the qualitative information, I, is omitted from Eq. 12.19. Then the risk modeling sequence (or any part of it) yields a functional relationship between the input parameters, $\boldsymbol{X}_i$, and the uncertain assessment measure of interest, $\boldsymbol{Z}$:

$$\boldsymbol{Z} = h(\boldsymbol{X}_1, \boldsymbol{X}_2, \ldots, \boldsymbol{X}_n) \tag{12.20}$$

In general, the function h may be very complex, since it results from chaining models obtained in each of the fours steps of the risk assessment process. This is

especially true for situations where released contaminants may affect a receptor through transport in several media and by multiple exposure routes. An example would be a power plant releasing a contaminant such as mercury to air and water which is transported to the receptor by air, water, and aquatic and terrestrial food chains. Additional complexity arises when there is significant contaminant transfer from one medium to another. However, many of the models presented in this book are relatively simple algebraic forms that are products, quotients, and sums of input parameters. With the advent of ever more powerful computers and software, the standard methods for uncertainty propagation use numerical sampling techniques. Some analytical methods are presented here because they may still be used for simple environmental assessment problems and because of the insights they provide.

12.4.1 Concepts for Uncertainty Propagation

The deterministic form of Eq. 12.20, where the input parameters and assessment measure are ordinary, rather than random, variables is

$$z = h(x_1, x_2, \ldots, x_n) \tag{12.21}$$

The input variables, x_i, form a hyperspace ("hyper" because the space has $n + 1$ dimensions) and z is a surface in that hyperspace. For some nominal point in the hyperspace, P: $z', x_1', x_2', \ldots, x_n'$, the partial derivatives, $[\partial z/\partial x_i]_P$, are a quantitative measure of the effect of small variations in input parameters on the assessment measure. However, the values of these partial derivatives depend on the units of measurement for z and the x_i. Since essential behavior of the functional relation, h, is of primary interest, a normalized partial derivative sometimes termed a **sensitivity coefficient** is more useful:

$$S_i = \frac{x_i'}{z'}\left[\frac{\partial z}{\partial x}\right]_P \tag{12.22}$$

This is also called a sensitivity ratio or elasticity (in economic theory) (EPA 2001). If values of S_i are to be compared across a region in hyperspace or if z' or any x_i' is zero, nominal values of the variables (e.g., the upper bounds) should be used rather than the value at P. Since the value of S_i depends on P, it is a local property. Conceptually, the sensitivity coefficient is the fractional change in the assessment measure for a fractional change in an input variable. If the value of S_i is a small number, say less than 0.01, it may mean that variations in the parameter have little effect on the assessment measure. The sensitivity coefficient can be used to help prioritize the effort made in determining the uncertainty distributions to describe the x_i. Also, if the identification of the most significant input parameters does not comport with intuition, this may be an indication that something is amiss in model structure, linkage, or completeness. Recent practice has been to determine sensitivity coefficients by numerical methods, but an automated analytical approach (Pin et al. 1986) that examines the computer code representing the model to derive the partial derivatives has also been used.

► Example 12.3

Consider the following example of sensitivity analysis based on Example 6.1. Suppose that the system assessment measure of interest is groundwater travel time, t, which is given by

$$t = \frac{x}{u} = \frac{xn}{K_H |dH/dx|}$$

where x is the distance to the site boundary (nominal value 500 m), u the groundwater velocity, n the porosity (nominal value 0.33), K_H the hydraulic conductivity (nominal value 3×10^{-5} m/s), and dH/dx the hydraulic gradient (nominal value –0.01). The nominal value of groundwater travel time is 17 yr.

The sensitivity coefficients are obtained from Eq. 12.22. For example, the sensitivity coefficient for porosity is

$$\begin{aligned} S_n &= \frac{n}{t}\left(\frac{\partial t}{\partial n}\right) \\ &= \frac{K_H |dH/dx|}{x} \frac{x}{K_H |dH/dx|} \\ &= +1 \end{aligned}$$

The sensitivity coefficients for K_H and $|dH/dx|$ are –1. This example is typical of many problems in environmental risk assessment in that the mathematical model involves products and quotients, and the sensitivity coefficients are independent of the nominal values. For mathematical models that include special functions such as exponentials or error functions, the sensitivity coefficients generally are not constant and depend on the nominal values (i.e., the location in the hyperspace).

A different approach must be followed for the probabilistic form represented by Eq. 12.20. Clearly, the variance of the assessment measure, σ_Z^2, is related to the statistical properties of the input parameters, but a general formula for the relationship is not available. An approximate relationship can be developed by expanding h as a Taylor series about the mean values of the input parameters [i.e., the point, $(\mu_1, \mu_2, \ldots, \mu_n)$]. This yields

$$\boldsymbol{Z} \cong h(\mu_{X_1}, \mu_{X_2}, \ldots, \mu_{X_n}) + \sum_1^n \left[\frac{\partial h}{\partial \boldsymbol{X}_i}\right]_P (\boldsymbol{X}_i - \mu_{X_1}) \tag{12.23}$$

If the input parameters are statistically independent, the variance of the assessment measure can be shown to be (Bowen and Bennett 1988)

$$\sigma_{\boldsymbol{Z}}^2 \cong \sum_1^n \left[\frac{\partial h}{\partial \boldsymbol{X}_i}\right]_P^2 \sigma_{\boldsymbol{X}_i}^2 \tag{12.24}$$

If the input parameters are statistically dependent, a more complex form involving the covariance is obtained.

Equation 12.24 may be used to define an **uncertainty coefficient**, U, which represents the fraction of the variance of the assessment measure due to a particular input variable (Wescott et al. 1995):

$$U_i \cong \left[\frac{\partial h}{\partial \boldsymbol{X}_i}\right]_P^2 \frac{\sigma_{\boldsymbol{X}_i}^2}{\sigma_{\boldsymbol{Z}}^2} = \left(\left[\frac{\partial h}{\partial \boldsymbol{X}_i}\right]_P \left[\frac{\sigma_{\boldsymbol{X}_i}}{\sigma_{\boldsymbol{Z}}}\right]\right)^2 \tag{12.25}$$

The second form is similar to Eq. 12.22 because the partial derivative is normalized, but in this case by the ratio of the standard deviations (input parameter/assessment measure) and then squared.

Another useful strategy for identifying important aspects of an environmental system is to determine the fraction of the assessment measure caused by various environmental pathways, routes of exposure, or contaminants. In a tiered risk assessment, an early step may be to estimate assessment measures based on point estimates of the input parameters (EPA 2001). This is illustrated in Table 12.4, where the radiation dose is the assessment measure in retrospective risk assessment for a nuclear facility. Displayed in the table is the percentage of dose contributed by each of 13 exposure pathways for hypothetical scenarios.

Many other measures may be used to examine the sensitivity of environmental systems (EPA 2001), including (1) inspection of the risk equation; (2) sensitivity scores (weighted sensitivity ratios); (3) graphical techniques applied to the results of Monte Carlo simulations (e.g., scatter plots); (4) correlation coefficient or coefficient of determination, r^2 (e.g., Pearson product moment, Spearman rank); (5) normalized multiple regression coefficient; and (6) goodness-of-fit test for subsets of the risk distribution. In addition, methods have been suggested for evaluating the structural importance of various components of an environmental system (Eisenberg and Sagar 2000).

12.4.2 Methods for Uncertainty Propagation

Some overall measures that can be used to assess the significance of various input parameters were presented in Section 12.4.1. However, for a risk assessment that considers uncertainty, determination of the probability distribution that describes the assessment measure is a central goal. Uncertainty propagation methods determine the probabilistic behavior of the assessment measure, given the probability distributions of the input parameters.

12.4.2.1 Analytical Methods Analytical methods are usually applied only to simple problems. For example, the chain rule may be used to derive the probability distribution for an assessment measure that is a function of a single variable. If random variable $\boldsymbol{X}$ has a probability density function $f(x)$, and if $z = h(x)$ is either increasing or decreasing with x, the inverse function exists, $x = h^{-1}(z)$. In this case the probability density function of $\boldsymbol{Z} = h(\boldsymbol{X})$ can be shown to be given by

$$f(z) = f\left[h^{-1}(z)\right]\left|\frac{dx}{dz}\right| \tag{12.26}$$

TABLE 12.4 Results of a Dose Reconstruction for the Savannah River Site[a]

		Adult Female		Adult Male		Child Born in 1955		Child Born in 1964	
Route	Pathway	Dose (mSv)	%	Dose (mSv)	%	Dose (mSv)	%	Dose (mSv)	%
External	Air immersion	0.024	8.0	0.024	5.7	0.024	1.5	0.012	16.6
	Ground contamination	0.0024	0.8	0.0025	0.6	0.0024	0.1	9.3×10^{-5}	0.1
Ingestion	Beef	0.15	50.7	0.24	57.1	0.69	43.2	0.011	15.3
	Eggs	0.0014	0.5	0.0023	0.6	0.0021	0.1	0.0014	2.0
	Fruit	0.011	3.6	0.0105	2.5	0.043	2.7	0.0054	7.5
	Grain	0.0011	0.4	0.0013	0.3	0.0036	0.2	6.3×10^{-4}	0.9
	Leafy vegetables	0.018	5.9	0.018	4.2	0.030	1.9	9.7×10^{-4}	1.3
	Milk	0.049	16.1	0.065	15.3	0.70	43.9	0.026	35.4
	Poultry	0.0010	0.3	0.0012	0.3	0.0013	0.1	8.1×10^{-4}	1.1
	Root vegetables	0.0073	2.4	0.010	2.4	0.014	0.9	0.0044	6.1
	Soil[b]	4.9×10^{-7}	<0.1	4.9×10^{-7}	<0.1	7.4×10^{-6}	<0.1	8.1×10^{-8}	<0.1
Inhalation	Air inhalation	0.031	10.1	0.041	9.7	0.082	5.1	0.0091	12.6
	Resuspended soil	0.0040	1.3	0.0054	1.3	0.0037	0.2	7.9×10^{-4}	1.1
Total		0.30	100	0.42	100	1.59	100	0.0721	100

Source: CDC 2005.

[a] Contributions to total dose for a 39-year study period are shown for four hypothetical receptors in a particular exposure scenario. Doses based on point estimates of input parameters are disaggregated by exposure route and environmental pathway. Ingestion is easily identified as the most important exposure route in this case. Consumption of beef and milk are generally important pathways. Soil ingestion is always a minor contributor and can probably be eliminated from further consideration.

[b] Doses from the soil ingestion pathway were no more than 0.0005% of any family member's entire 39-year dose.

However, few risk assessment equations are dominated by the variability in a single parameter. For very simple functions of one or more random variables, such as sums and products, exact expressions exist for determining the mean and variance of the resulting function.

■ Lognormal Distributions in Environmental Risk Assessment

Environmental measurements are often found to follow a lognormal distribution. This observation has a theoretical basis. Consider the product of two lognormally distributed independent variables:

$$\boldsymbol{Z} = \boldsymbol{XY}$$

$$f(x) = \frac{1}{x\sigma_U\sqrt{2\pi}} \exp\left\{-\frac{[\ln(x) - \mu_U]^2}{2\sigma_u^2}\right\}$$

$$f(y) = \frac{1}{y\sigma_V\sqrt{2\pi}} \exp\left\{-\frac{[\ln(y) - \mu_V]^2}{2\sigma_v^2}\right\}$$

$$u = \ln(x), \qquad v = \ln(y)$$

By multiplying the distribution functions for X and Y and manipulating the result, it can be shown mathematically that the following obtains:

$$f(z) = \frac{1}{z\sigma_W\sqrt{2\pi}} \exp\left\{-\frac{[\ln(z) - \mu_W]^2}{2\sigma_w^2}\right\}$$

$$\mu_w = \mu_U + \mu_V \qquad \sigma_W^2 = \sigma_U^2 + \sigma_V^2$$

In other words, the product of two lognormally distributed independent variables is also a lognormally distributed random variable. It turns out that the quotient of two lognormal distributions is also lognormal. Many physical processes in environmental risk assessment are modeled through multiplication and division operations: for example,

$$C = \frac{\dot{S}_0}{Q}$$

$$C = \frac{\dot{S}_0}{\pi u \sigma_y \sigma_z}$$

$$R = \rho D = \frac{\rho \cdot \mathrm{CR} \cdot C}{\mathrm{BW}}$$

Thus, it is not surprising that lognormal distributions are used frequently in environmental risk assessment.

Exact expressions for statistically independent variables are given in Table 12.5. If a more general function is expanded about the mean values of the input parameters, as shown in Eq. 12.23, the mean value of the dependent variable is approximated by

TABLE 12.5 Exact Expressions for the Means and Variance of Simple Functions of Independent Random Variables

	Function $\boldsymbol{Z} = f(\boldsymbol{X}_1, \boldsymbol{X}_2, \ldots, \boldsymbol{X}_n)$	Mean μ_Z	Variance σ_Z^2
Constant	$\boldsymbol{Z} = a\boldsymbol{X} + b$	$a\mu_X + b$	$a^2\sigma_X^2$
Sum	$\boldsymbol{Z} = a_0 + \sum_{i=1}^{n} a_i \boldsymbol{X}_i$	$a_0 + \sum_{i=1}^{n} a_i \mu_{x_i}$	$\sum_{i=1}^{n} a_i^2 \sigma_{X_i}^2$
Product	$Z = XY$	$\mu_X \mu_Y$	$\mu_X^2 \operatorname{var}(X) + \mu_Y^2 \operatorname{var}(Y) + \operatorname{var}(X)\operatorname{var}(Y)$

$$\mu_z \cong f(\mu_{X_1}, \mu_{X_2}, \ldots, \mu_{X_n}) \tag{12.27}$$

and the variance is approximated by Eq. 12.24. These expressions, however, only provide approximate estimates of the mean and variance of the resulting function, not the entire distribution.

► Example 12.4

An abandoned industrial site is contaminated by beryllium and carbon tetrachloride. If the site were used for a residential farm, the point estimates of doses to a resident from these two contaminants are 2.2×10^{-3} and 2.3×10^{-3} mg/kg·d, respectively. The EPA reference doses for these contaminants are 0.005 and 0.007 mg/kg·d, respectively.

(a) What are the hazard quotients for Be and CCl_4?
(b) What is the hazard index for the site?
(c) Would this site be a candidate for immediate release based on these data and this exposure scenario?

Now consider that the doses stated above are not exact but represent the mean doses from a risk assessment. Suppose also that the variances associated with each contaminant dose are 9.3×10^{-6} $(\text{mg/(kg·d)})^2$ for Be and 6.5×10^{-6} $(\text{mg/(kg·d)})^2$ for CCl_4. Assume that the reference doses are exact, without uncertainty.

(d) Given this information, what is the mean value of the hazard index?
(e) What is the variance of the hazard index?
(f) What is the mean value of the hazard index plus one standard deviation? What added insight does this give to the problem?

Solution

(a) The hazard quotients are computed directly:

$$\text{HQ}_{\text{Be}} = \frac{2.2 \times 10^{-3}\ \text{mg/(kg·d)}}{0.005\ \text{mg/(kg·d)}} = 0.44$$

$$HQ_{CCl4} = \frac{2.3\times10^{-3}\ \text{mg/(kg·d)}}{0.007\,\text{mg/(kg·d)}} = 0.33$$

(b) The hazard index for the site is the sum of the hazard quotients:

$$HI = HQ_{Be} + HQ_{CCl4} = 0.44 + 0.33 = 0.77$$

(c) Based on this information, the HI < 1, so the site would be a candidate for immediate release.

(d) If the doses provided are the means of distributions, the formulas in Table 12.5 must be used to compute the mean values of first the HQs and then the HI. Since the reference doses are exact, the hazard quotient is of the form Z = *a*X, where X is the dose and *a* is the reciprocal of the reference dose. Hence the mean of the hazard quotient is the mean dose divided by the reference dose, just as in the deterministic case. The hazard index is of the form Z = X + Y, where X and Y are the hazard quotients for the two contaminants. Thus, the mean value of the hazard index is the sum of the mean values of the hazard quotient; again, it is the same as in the deterministic case.

(e) To compute the variance of the hazard index, the variances of the hazard quotients are needed. Application of the formulas in Table 12.5 gives

$$\sigma^2_{HQ} = \frac{\sigma^2_D}{(RfD)^2}$$

$$\sigma^2_{HQ_{Be}} = \frac{9.3\times10^{-6}\,(\text{mg/(kg·d)})^2}{\left(5\times10^{-3}\,\text{mg/(kg·d)}\right)^2} = 0.37$$

$$\sigma^2_{HQ_{CCl4}} = \frac{6.5\times10^{-6}\,(\text{mg/kg·d})^2}{\left(7\times10^{-3}\,\text{mg/(kg·d)}\right)^2} = 0.13$$

$$\sigma^2_{HI} = \sigma^2{}_{HQ_{Be}} + \sigma^2{}_{HQ_{CCl4}} = 0.37 + 0.13 = 0.50$$

(f) The standard deviation of the hazard index is the square root of the variance (i.e., $\sigma_{HI} = 0.71$). Adding one standard deviation to the mean value is 0.77 + 0.71 = 1.48. If a normal distribution is assumed for the hazard index, this means that there is about a 16% chance of exceeding an HI of 1.48. Based on the information from the uncertainty analysis, one might wish to perform additional studies before releasing the site.

12.4.2.2 Numerical Methods The Monte Carlo method is probably the most widely used technique for propagating uncertainty in environmental risk assessments. The simple Monte Carlo process, which is illustrated in Figure 12.5, is used to obtain an empirical distribution of the assessment measure from the probability distributions of the input parameters by repeatedly sampling the input parameter distributions and computing the assessment measure based on those samples.

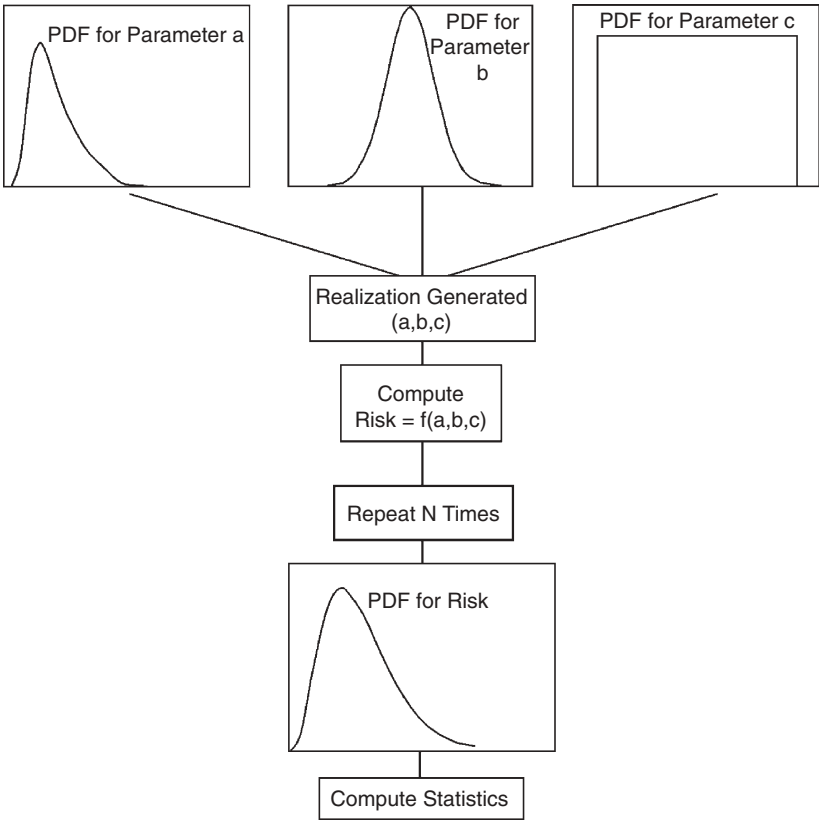

Figure 12.5 Propagation of uncertain parameters through a risk model by the Monte Carlo method. "Risk" may be an assessment measure such as dose.

Each realization (also called vector or scenario) of the n input parameters $\langle X_{1,k}, \ldots, X_{n,k} \rangle$ generates a single value for the assessment measure, Z_k. Standard statistical techniques may then be used to calculate summary statistics for the assessment measure distribution, such as the mean, variance, and percentiles. Since estimates of the summary statistics are based on a sample from the underlying distribution, the sampling error in these estimates may also be computed by standard methods.

In simple Monte Carlo sampling, values are selected at random from the distribution for each input parameter. Consequently, values of the parameter close to the mean are sampled much more frequently than extreme values. Example 12.5 shows how Monte Carlo methods may be used to derive a distribution of risk, based on distributions of the underlying parameters.

▶ Example 12.5

A simple hypothetical example of a Monte Carlo risk assessment is given in Thompson et al. (1992). In this paper, a case study of children playing in soils

contaminated with benzene and benzo[*a*]pyrene was developed. It was assumed that the children would play in the soil 1 day per week, 20 weeks per year, for a 10-year childhood. The soil ingestion pathway for benzene was modeled with the equation

$$\text{risk} = \frac{C_s \cdot \text{CR}\,(1\,\text{d/wk})(20\,\text{wk/yr})(10\,\text{yr})\left(10^{-6}\,\text{kg/mg}\right)\rho}{\text{BW}\,(364\,\text{d/yr})(70\,\text{yr})}$$

where C_s is the soil concentration, CR the soil contact (i.e. ingestion) rate, ρ the cancer slope factor, and BW the body weight. The parameters of the variable distributions are given in Table 12.6. The parameters of the normal distribution are the mean and standard deviation of the distribution, and the parameters of the lognormal distributions are the log mean and the log standard deviation, respectively.

The Monte Carlo simulation was carried out using Crystal Ball with approximately 10,000 samples from each distribution and yielded the frequency distribution and the CDF shown in Figure 12.6*a* and *b*. The summary statistics of these distributions are presented in Table 12.7.

TABLE 12.6 Parameter Distributions for Example 12.5

Parameter	Distribution
Body weight (kg)	Normal (47, 8.3)
Soil ingestion rate (mg/d)	Lognormal (3.44, 0.80)
Soil concentration (mg/kg)	Lognormal (0.84, 0.77)
Cancer slope factor $[(\text{mg/kg}\cdot\text{d})^{-1}]$	Lognormal (–4.33, 0.67)

TABLE 12.7 Summary Statistics for Example 12.5

Statistic	Value	Percentile	Risk Value
Trials	10,000	0% (min.)	1.57×10^{-12}
Mean Risk	3.75×10^{-10}	20%	5.46×10^{-11}
Median Risk	1.63×10^{-10}	40%	1.19×10^{-10}
Standard Deviation of Risk	7.33×10^{-10}	60%	2.29×10^{-10}
		80%	4.84×10^{-10}
		100% (max.)	1.70×10^{-8}

If knowledge of the extreme values of the assessment measure distribution (i.e., the distribution tails) is of interest, as is frequently the case, a very large number of realizations may be required to obtain that knowledge. A more efficient approach frequently employed is stratified sampling in which the input distributions are divided into intervals. Samples of the input parameter are selected from each interval, but logical rules limit the ability to resample an interval already sampled. A common form of stratified sampling is Latin hypercube sampling (LHS) (McKay et al. 1979; Iman and Shortencarier 1984). In LHS, each input distribution is

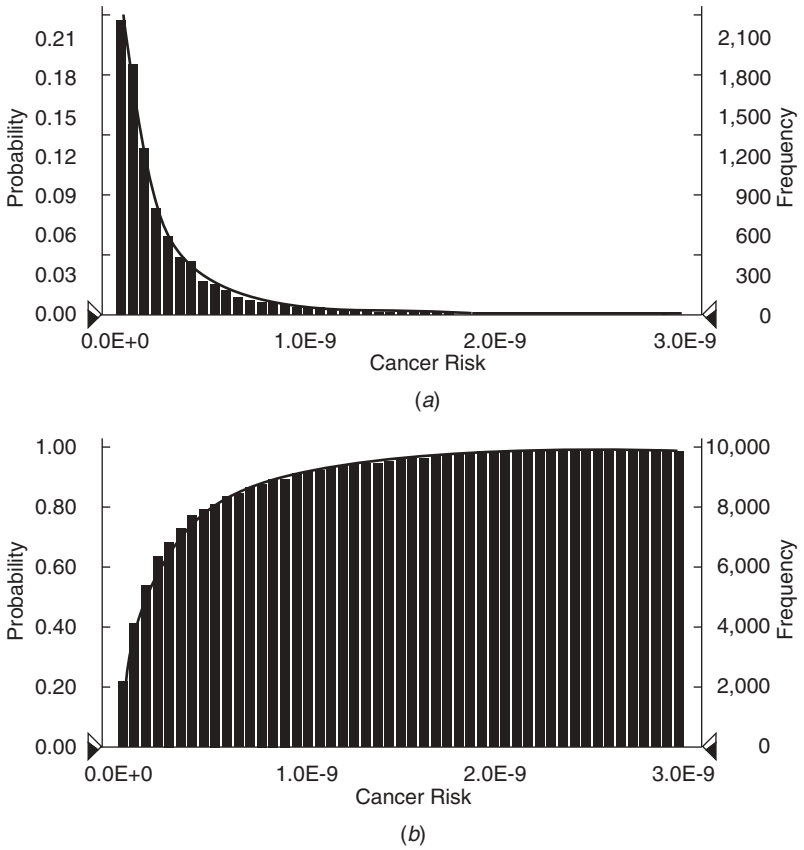

Figure 12.6 Monte Carlo results for Example 12.5: (*a*) probability and frequency of benzene risk; (*b*) cumulative distribution of benzene risk.

divided into m disjoint equiprobable intervals. For each input parameter $\boldsymbol{X}_i$, a sample is selected from each of its m intervals by simple random sampling, considering the underlying probability distribution, for a total of m samples for each parameter. Each realization is generated by randomly selecting one sample from the set of m samples for each parameter. Once the sample from the jth interval for the ith input parameter is used, it is not used again (sampling without replacement). At the end of the process, m realizations, each with sampled values for the n input parameters, is obtained. Since the sampling process forces samples to be obtained from all parts of the input distributions, the values for the sample statistics of the assessment measure are closer to the values of the population statistics than would be obtained from a set of simple random samples of the same size. However, the statistics for estimating the accuracy of the statistics are more difficult to compute and may yield an underestimate of the true precision.

Monte Carlo methods are not without difficulties. For environmental risk assessments with many input parameters and complex models (e.g., multiple transport media, multiple contaminants, variable environmental conditions) a great deal of computing power may be required to handle the large amount of data, including intermediate results, in a reasonable time. In addition, preparation of the input for such large problems requires considerable effort, for example, to develop the ratio-

nale for parameter distributions. Although the analysts may carefully select distributions for each input parameter, any given realization may be nonphysical because of the juxtaposition of incompatible values (often extreme values) for different parameters. This usually results from some correlation among input parameters that has not been effectively taken into consideration. Although most methods allow for the treatment of correlations among input parameters, such correlations are often difficult to characterize correctly.

A growing trend in quantitative risk analyses is to quantify and display aleatory and epistemic uncertainties separately, because it is recognized that decision makers consider these two types of uncertainty differently. Many approaches are used to accomplish this. One approach, using two iterations of Monte Carlo calculations,

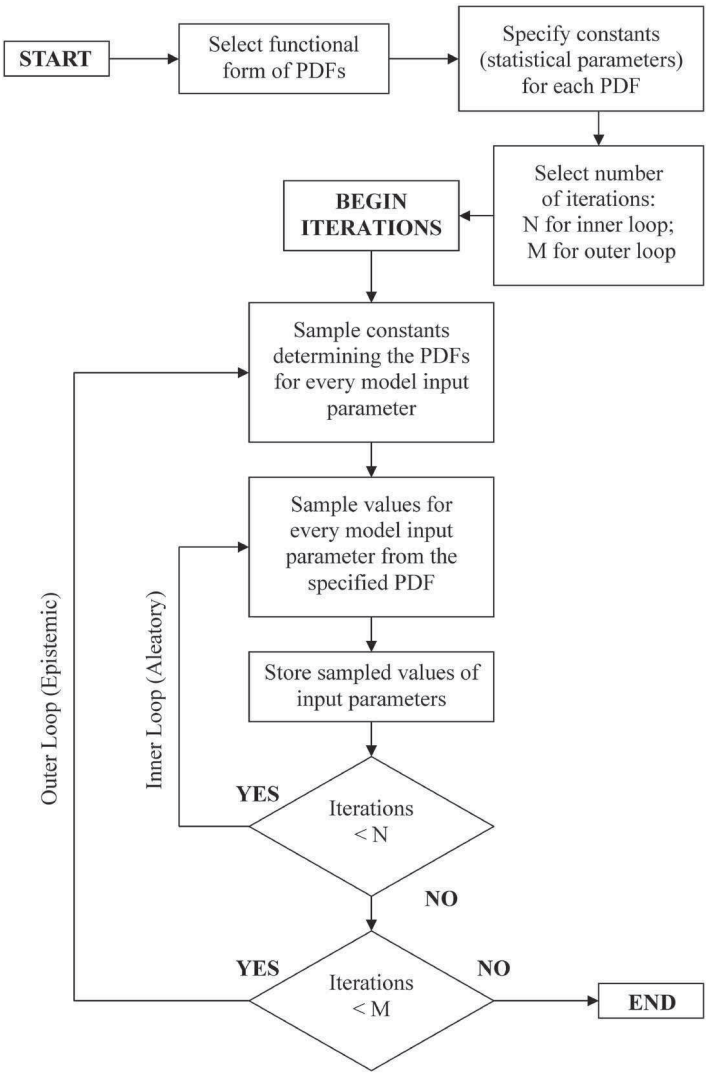

Figure 12.7 Flowchart to generate realizations that reflect both aleatory and epistemic uncertainty. (Based on EPA 2001.)

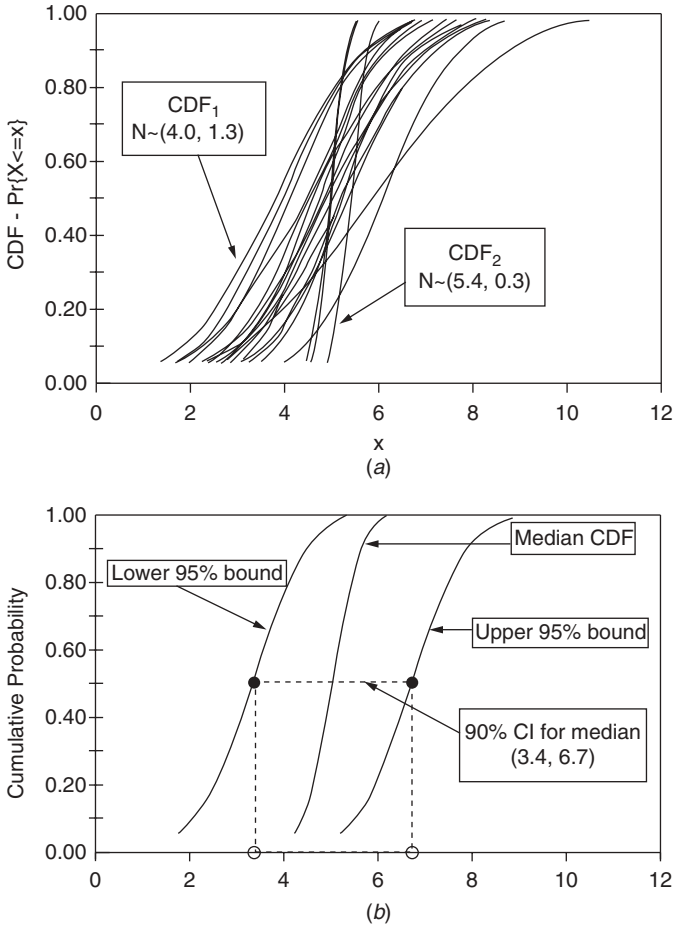

Figure 12.8 Example of propagation and display of aleatory and epistemic uncertainty: (*a*) aleatory uncertainty represented by CDFs for 20 sets of realizations of epistemic parameters (statistical parameters of the aleatory PDFs); (*b*) epistemic uncertainty:—upper and lower 95th percentiles of the CDFs in part (*a*). (From EPA 2001.)

is illustrated in Figure 12.7, with a hypothetical example shown in Figure 12.8. In this hypothetical example (EPA 2001) the input parameters of interest have aleatory uncertainty described by a probability distribution. However, the exact nature of these aleatory probability distributions is uncertain, so the constants determining these distributions (the statistical parameters) are treated as epistemic variables, also described by probability distributions. The objective of the process diagrammed in Figure 12.7 is to produce a set of realizations of input parameters for the risk assessment that reflect both aleatory and epistemic uncertainty. The outer loop selects sampled values for the constants (statistical parameters) describing the aleatory distributions. The inner loop generates samples of the input parameters based on the specified aleatory probability distributions using the constants sampled in the outer loop. The assessment measure is then calculated for each input parameter realization.

Figure 12.8 shows the result of a simple example in which the assessment measure is taken to be the same as a single input parameter, X. The aleatory uncertainty of this input parameter is described by a normal (Gaussian) distribution with an uncertain mean and standard deviation. The epistemic uncertainty in the mean is described by a normal distribution with a mean of 5 and a standard deviation of 0.5; the epistemic uncertainty in the standard deviation is described by a normal distribution with a mean of 1.0 and a standard deviation of 0.5. Twenty aleatory distributions of X are shown in Figure 12.8*a*. Each CDF represents a single Monte Carlo simulation of 2500 realizations, using fixed values for the mean and standard deviation of X. These values are identified for two of the curves. Figure 12.8*b* summarizes results for over 6 million Monte Carlo realizations. A total of 2500 realizations of the epistemic variables, the mean and standard deviation of X, are generated. Then, for each of these epistemic realizations, 2500 realizations representing the aleatory uncertainty in X are generated. Percentiles for each generated aleatory distribution may be determined; for example, the median (50th percentile), 95th percentile, and 5th percentile are of interest here. The three curves in Figure 12.8*b* represent the distributions describing the 5th, 50th, and 95th percentiles obtained from each epistemic realization. In Figure 12.8*a* each curve represents aleatory uncertainty, with no epistemic uncertainty (a particular choice of mean and variance of the aleatory distribution); in Figure 12.8*b* each curve represents epistemic uncertainty, with no aleatory uncertainty (a particular choice of aleatory percentile).

Although the preceding example used continuous PDFs to characterize the epistemic uncertainty in the aleatory distributions, this is not required. The same general sampling approach may be used: separating input parameters into aleatory and epistemic classes and sampling the two classes in separate stages of the analysis. However, some algorithm for assigning a probability to each choice of epistemic variables is required. For example, probabilistic analysis or expert elicitation may be used to assign a probability to each workable alternative conceptual model or scenario (Helton 1994).

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PROBLEMS

12.1 Classify the following examples in one or more of the following categories: (1) parameter uncertainty; (2) model uncertainty; (3) scenario uncertainty. State the rationale for the choice.

(a) It is not clear whether the dissolution of waste into groundwater at an abandoned waste site can be estimated by a single leaching coefficient or whether each species behaves differently.

(b) Bioconcentration factors for radioactive ^{137}Cs at the Savannah River Site, based on five fish muscle assays (piscivores), show a maximum value of 39,000, a minimum value of 908, and a mean value of 10,980 L/kg (Friday et al. 1996).

(c) Native Americans in the vicinity of the Hanford Reservation in the state of Washington practice a traditional life-style and consume significant quantities of wild rice and salmon from the Columbia River. These consumption habits are significantly different from those of the general population.

(d) Oily waste in a waste pit at an abandoned fleet service facility is slowly seeping into the groundwater; however, during some years spring rainfall floods the waste pit, floats the oily waste, and moves it over the ground surface.

(e) To estimate the risk from an acute, accidental release of a toxic gas from a processing plant, analysts have access to the joint frequency distribution of wind direction, wind speed, and atmospheric stability class. The joint frequency distribution is based on 5 years of hourly observations.

(f) The joint frequency distribution in part (e) was recorded at a local airport 5 miles from the processing plant. Criticisms of previous analyses raise the concern that the meteorological conditions at the processing plant are affected by changes in topography.

12.2 Classify the following examples as aleatory uncertainty, epistemic uncertainty, or both.

(a) The value of distribution coefficient is an important parameter for groundwater transport of contaminants. Soil samples taken from different locations on a contaminated site yield different values for this parameter.

(b) The mean value of measurements of the distribution coefficient is used to estimate contaminant transport at the contaminated site in part (a). Because the data are sparse, Student's t distribution is used to estimate confidence limits about this mean value.

(c) In modeling the fate of an accidental airborne release of contaminants at a facility located in a desert climate, wet deposition of contaminants is assumed to be zero.

(d) In modeling the fate of an accidental airborne release of contaminants all particles are assumed to have the same deposition velocity.

(e) Field measurements at several test wells across a contaminated site are interpreted using the Theis equation to determine transmissivity values.

(f) Laboratory experiments performed on rock and soil cores from the same locations as part (e) are interpreted using Darcy's law to determine the values of hydraulic conductivity.

12.3 The mathematical model for a simple risk problem is $Y = X_1 X_2 \exp(-X_2)$. Use the analytical methods for error propagation to estimate the mean and variance of Y if the means and standard deviations of X_1 and X_2 are μ_1 and μ_2 and σ_1 and σ_2, respectively.

12.4 The emission rate of dioxin from a paper mill to a river is 25 mg/s. The contaminant undergoes degradation with a first-order rate constant of $0.6\,d^{-1}$. The mean flow rate of the river is $5\,m^3/s$ and the mean water velocity is 0.5 m/s. Contaminant transport in the river can be adequately approximated by a one-dimensional advective model.

(a) Determine the concentration corresponding to the mean flow rate and mean water velocity at a downstream distance of 30 km.

(b) If the standard deviation of emission rate is 8 mg/s and the standard deviation of the flow rate is $2\,m^3/s$, estimate the standard deviation of contaminant concentration at 30 km.

12.5 Derive the probability density function of the lognormal distribution from the normal distribution by using the chain rule (Eq. 12.26).

12.6 The mathematical model of a risk assessment problem is $R = XY$. The uncertain parameter X is represented by a uniform distribution from 0.1 to 0.6, and the uncertain parameter Y is represented by a uniform distribution from 5 to 10. X and Y are independent.

(a) What are the mean and standard deviation of X?

(b) What are the mean and standard deviation of Y?

(c) What are the mean and standard deviation of R?

12.7 The uncertain function Z is a function of two random variables: $Z = X + Y$. Suppose that X has a uniform distribution with mean $\mu_x = 0.5$ and variance $\sigma_X^2 = 1/12$. Suppose that Y has a Gaussian distribution with mean $\mu_y = 1$ and variance, $\sigma_Y^2 = 1$. What are the mean and variance of Z?

13 Stakeholder Involvement and Risk Communication

13.1 INTRODUCTION

A fundamental premise of democracy is that the governed (the public) participate in making societal or governmental decisions. This is especially true for those directly affected by such decisions. For example, Article I of the U.S. Bill of Rights guarantees the public the right to "to petition the Government for a redress of grievances." Public involvement in matters involving environmental risk analysis has intensified over the last four to five decades. A variety of broad demographic, societal, technical, and institutional trends appear to be responsible for this, including:

1. Population has increased substantially, especially in suburban areas; consequently, facilities previously "remote" have become neighbors to substantial numbers of people.
2. The use of toxic or potentially toxic materials has increased, as industries and businesses have adopted advanced chemical processes or compounds.
3. The technical community and the public have become much more aware of the hazards posed to humans and other species by low concentrations of certain compounds in the environment.
4. Land used previously for industrial operations has been redeveloped for residential housing or community use. This creates a risk of exposure to residual contamination at such sites.
5. Environmental laws and governmental practices have mandated that highly technical government agencies involve the public in decisions involving environmental risk. At the same time the number of environmental laws, requirements, and regulations has increased substantially, creating more opportunities for involvement of the public.
6. There has been a growing awareness of the need to prevent poor and/or disadvantaged people from bearing an inequitable share of environmental risk from hazardous facilities, traditionally collocated with lower-socioeconomic residential areas.

Health Canada (2000) lists four reasons for an increased focus on public participation: globalization, transition to a knowledge-based society, decrease in

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societal trust of institutions, and fiscal pressure to expend governmental resources more efficiently. Federal agencies, in compliance with the Administrative Procedures Act (APA 2000), are required to inform the public about their decisions; however, the public appears to be increasingly opposed to many decisions made in this way. For example, building of new nuclear power plants declined in the United States in part because of increased public opposition. These difficulties spurred interest and scientific research in risk perception and risk communication. Although risk communication shares many attributes with communication in general, two aspects make it unique. First, risk, as defined in Chapter 1, is the probability that some harm will befall a member of the public, their family, and/or community. As one would expect from a rational person, this contemplation of potential harm almost always arouses negative thoughts and feeling, which promotes dissension rather than consensus in decision making. Second, as discussed in Chapters 1 to 13, the methods used to analyze environmental risk are highly technical and can be complex. Consequently, risk analysis results and methods are frequently very difficult for analysts to communicate to nontechnical, or even technical, audiences, so the audience frequently has difficulty accepting the results or the credibility of the analysis.

Although this discussion has used the term "public" loosely, it has become increasingly obvious that many "publics" participate as stakeholders in environmental risk analysis. One risk communication manual (NRC 2004) states: "External stakeholders [outside the NRC] include people who are organizationally impacted, personally impacted, and generally concerned as well as the media." For example, stakeholder groups include the owners of the facility or proposed facility, state government, local governments, Native American tribal governments, other federal agencies with overlapping authority, the industry associations to which the facility belongs, environmental activist organizations, religious groups, citizens' associations affected, labor unions, technical societies and experts with a special interest in the methods or objective of the risk analysis, technical advisory committees, the media (print and electronic), and individual citizens, among others.

The approach taken in this chapter for stakeholder participation and risk communication is similar to that taken in previous chapters. First, the motivation or need for the activity is discussed; second the scientific, technical, and, in this case, procedural basis for the activity is presented; third, methods useful in accomplishing the task are presented. Like other chapters, there is no intent here to cover the topics completely; rather, the goal is to present enough fundamental information to allow understanding of or participation in the activity.

13.2 STAKEHOLDER INVOLVEMENT

13.2.1 Potential Benefits and Detriments

Like many other topics in this book, stakeholder involvement in governmental or private projects, programs, or policies is an established scholarly discipline; consequently, only some fundamental considerations are presented here. Stakeholder involvement has become so embedded in democratic decision making that many

government agencies and international organizations (DOE 1998; EPA 2003; NEA 2004) have developed and published documents articulating the motivations, anticipated benefits, and methods for stakeholder involvement. Based on a paper by van den Hove (2003), the Nuclear Energy Agency (NEA 2004) has articulated three categories of benefits arising from stakeholder involvement:

1. *Substantive:* producing better decisions and outcomes
2. *Procedural:* changing the manner in which decisions are made or deliberations are conducted
3. *Contextual:* additional effects not directly related to decisions or the decision-making process, but benefiting the functioning of the society.

The primary substantive benefit is reaching a decision with better technical, environmental, economic, or social acceptance characteristics. Procedural benefits are (1) expanding the decision making basis with better information, a more complete set of factors and values affecting the decision, and more decision alternatives; and (2) enhancing the decision process with better information use, enhanced conflict management, increased legitimacy, greater empowerment of less organized parties, and improving the cost and time effectiveness of the process. Contextual benefits include better communication among stakeholders, including decision makers, increased confidence in institutions, reinforcement of democratic principles, and enhanced citizen involvement.

Somewhat parallel to the substantive and procedural benefits cited by the NEA, the EPA articulates purposes and goals for their Public Involvement Policy (EPA 2003), as shown in Table 13.1. The Presidential/Congressional Commission on Risk Assessment and Risk Management (1997a,b) provides a list of seven similar but more general benefits of stakeholder involvement: "(1) supports democratic decision-making; (2) ensures that public values are considered; (3) develops the understanding needed to make better decisions; (4) improves the knowledge base for decision-making; (5) can reduce the overall time and expense involved in decision-making; (6) may improve the credibility of agencies responsible for managing risks; and (7) should generate better accepted, more readily implemented risk management decisions."

The benefits cited by Health Canada (2000) for public participation in its program (generalized here) include the following: (1) more effective achievement of program goals; (2) improved program results and support for policies and regulations; (3) stronger public confidence in the agency; (4) a better informed and engaged public; (5) strengthened community organizations and leadership; (6) a more effective, responsive, citizen-oriented agency; (7) an agency more attuned to receiving input from a diverse population; (8) enhanced agency practices for decision making, risk management, effectiveness, and accountability; and (9) better training and development of employees.

All of these organizations recognize the negative impacts of public participation, primarily cost, resources, and possibly schedule delay; on balance, however, all favor public participation for the cited benefits and because an absence of effective public participation has too often led to disruption of important programs. Numerous cases of public participation in environmental decision making were evaluated

TABLE 13.1 Purposes and Goals of the EPA Public Involvement Policy

Purposes
Improve the acceptability, efficiency, feasibility, and durability of the Agency's decisions
Reaffirm EPA's commitment to early and meaningful public involvement
Ensure that EPA makes its decisions considering the interests and concerns of affected people and entities
Promote the use of a wide variety of techniques to create early and, when appropriate, continuing opportunities for public involvement in Agency decisions
Establish clear and effective guidance for conducting public involvement activities
Goals
Foster a spirit of mutual trust, confidence, and openness between the Agency and the public
Ensure that the public has timely, accessible, and accurate information about EPA programs in a variety of formats so that people can better understand the implications of potential alternative courses of action
Consult with interested or affected segments of the public and take public viewpoints into consideration when making decisions
Learn from individuals and organizations representing various public sectors and the information they are uniquely able to provide (community values, concerns, practices, local norms, and relevant history, such as locations of past contaminant sources, potential impacts on small businesses or other sectors, industry-conducted study results, etc.)
Solicit assistance from the public in understanding potential consequences of technical issues, identifying alternatives for study, and selecting among the alternatives considered
Keep the public informed about significant issues and changes in proposed programs or projects
Foster, to the extent possible, equal and open access to the regulatory process for all interested and affected parties
Understand the goals and concerns of the public, and respond to them
Anticipate conflict and encourage early discussions of differences among affected parties
Promote the public's involvement in the Agency's mission of protecting human health and the environment
Explain to the public how its input affected the Agency's decision

Source: EPA 2003.

using multiple attributes related to the participation process and substantive quality of the decisions (Beierle 2002; Bradbury et al. 2003). These evaluations showed that public participation generally provided positive benefits, but more intense public participation was correlated with better results. Many view this as a new paradigm for public interaction by agencies involved in environmental risk analysis (Kotra 2000): "The new dynamics of dialogue and decision making were characterized in discussion as a shift from the traditional 'decide, announce and defend' model, for which the focus was almost exclusively on technical content, to one of 'engage, interact and co-operate' for which both technical content and quality of process are of comparable import to a constructive outcome".

13.2.2 Scope of Stakeholder Involvement

Stakeholder involvement can cover a broad spectrum, from a minimal effort intended to inform, to a full-fledged partnership in making and implementing decisions. EPA (2000) articulates a spectrum of participation levels (from lowest to highest): (1) outreach, (2) information exchange, (3) recommendation, and (4) agreement. These are described briefly in Table 13.2. Similarly, Health Canada (2000) articulates five levels of public involvement (from lowest to highest): (1) inform or educate, (2) gather information and/or views, (3) discuss and/or involve, (4) engage, and (5) partner.

The fundamental criterion for choosing a particular level of stakeholder involvement is that the participation level supports the end goal of the program or activity. If a decision has already been made, describing it to the stakeholders is the appropriate participation level. Also in the case of a public health or safety emergency, the main goal of a government agency is to disseminate factual information. Information exchange is appropriate when a decision has not yet been made and stakeholders are invited to influence the outcome by providing factual information and value judgments regarding potential alternatives. In the United States, Executive Order 12866 (E.O. 12866 1993) requires each federal agency to "provide the public with meaningful participation in the regulatory process", especially for regulatory actions deemed "significant". This means that agencies are to seek participation prior to issuing regulations. Greater levels of participation, such as discussion and involvement and engaging stakeholders, may involve significant interactions among stakeholders instead of just interactions between each stakeholder and the primary agency. This may lead to negotiated approaches (e.g., negotiated rule making) or consensus approaches in which the stakeholders play an active role in reaching a final agreement. Finally, full partnering is appropriate when one or more stakeholders help to implement the decision. Regardless of which level of participation is chosen, it is important to articulate the level at the outset of the activity to prevent stakeholders from misunderstanding the potential impact of their involvement; such misunderstandings may be followed by stakeholder disappointment, anger, and opposition.

TABLE 13.2 EPA's Spectrum of Stakeholder Involvement

Phase	Participant Objective	EPA Objective
Outreach	Learn; become informed enough to determine whether to take more active interest or personal action	Build public awareness of environmental issues; provide materials that meet the needs of individuals and organizations
Information exchange	Provide input to decision	Understand more about issues, problems, values, perceptions; gather new information and data; better identify affected parties and their needs
Recommendation	Influence decision	Make a fully informed decision
Agreement	Help determine decision	Achieve mission and implementable decision

It should be noted that in the United States, legal or regulatory requirements may confer special status on certain stakeholders. Stakeholders with special status may include involved states, local governments, Native American tribal governments, and minority and/or low-income communities (EPA 2003).

13.2.3 Legal Basis and Requirements

Several laws encourage or mandate public participation and stakeholder involvement in environmental decisions by government agencies in the United States and other countries. Laying out the entire fabric of laws and requirements at federal, state, and local levels is beyond the scope of this book. However, a partial list of these mandates, based on a compilation by the EPA (2000), is shown in Table 13.3. At the federal level, mandates and requirements for public participation may be articulated in (1) legislation focused on a particular agency or activity, (2) executive orders that promulgate requirements generally applicable to all executive agencies,[1] or (3) policy statements by a particular agency. For example, the Nuclear Regulatory Commission, which has had a discretionary policy for open meetings since 1978, enhanced the policy in 2002 (NRC 2002) by clarifying the types of open meetings and participation permitted and by adopting Internet-based notification.

13.2.4 Methods and Approaches

Methods and approaches for stakeholder involvement should fit the scope of stakeholder involvement and must comply with all legal constraints. Some approaches for stakeholder involvement are listed in Table 13.4. This list, although not comprehensive, is intended to illustrate the range of methods available (Health Canada 2000; NEA 2004). It should be noted that higher levels of stakeholder involvement use higher-level involvement methods but usually also use one or more lower-level methods.

Implementation of a chosen approach requires conventional good management practices with attention to the special needs of public participation. Many organizations have prepared guides or road maps for public participation (EPA 1996, 2003; Health Canada 2000; Environment Council 2003; NRC 2004). A typical approach, listed by the EPA (2003), consists of the following steps:

1. Plan and budget for public involvement.
2. Identify the interested and affected public.
3. Consider providing technical or financial assistance.
4. Provide information and outreach.
5. Conduct public consultation and involvement activities.
6. Review and use input, and provide feedback to the public.
7. Evaluate public involvement activities.

[1] Quasijudicial agencies such as the Nuclear Regulatory Commission and the Securities Exchange Commission generally comply with executive orders even though there is no legal requirement to do so.

TABLE 13.3 Some Laws and Requirements Related to Stakeholder Involvement and Public Participation[a]

Name	Scope	Requirements for Public Participation
Administrative Procedure Act (APA)	Requires all federal agencies to follow certain standard procedures in promulgating regulations.	For informal rule making, requires the agency to publish a notice of proposed rule making in the *Federal Register* and provide an opportunity for interested parties to submit written data, views, or arguments. For formal rule making or formal adjudication, additional requirements for hearings include notice of planned hearings and an opportunity for input by interested parties (e.g., exceptions to agency decisions).
Federal Advisory Committee Act (FACA)	Constrains the formation and operation of advisory committees providing advice to federal agencies.	(1) Notice of upcoming meetings in the *Federal Register*; (2) permit public participation at meetings; (3) allow public access to committee reports and proceedings.
Regulatory Flexibility Act (RFA), as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA)	Requires federal agencies to assess the impact of regulations on small businesses, small governmental entities, and small organizations. For rules that may impose a significant economic impact on a substantial number of small entities, agencies must prepare a regulatory flexibility analysis of potential adverse impacts.	(1) Permit public comment on the draft regulatory flexibility analysis; (2) use techniques to facilitate participation by small entities, such as conferences, public hearings, and solicitation and receipt of comments via the Internet; (3) solicitation of advice and recommendations from small-entity representatives.
Unfunded Mandates Reform Act of 1995 (UMRA)	Requires federal agencies to assess the effects of a rule on state, local, and tribal governments and the private sector.	(1) For rules imposing significant intergovernmental mandates, federal agencies must develop a process to allow meaningful and timely input in the development of the regulatory proposal from elected officers of state, local, and tribal governments; (2) for rules imposing significant burdens on small governmental entities, agencies must develop a plan for informing these entities of the proposed rule and facilitating their input and for educating and advising these entities in regulatory compliance.
Executive Order 12898, "Federal Actions to Address	Requires each federal agency, to the greatest extent practicable and permitted by law, to make achieving	Requires ensuring meaningful public participation of minority and low-income populations by identifying potential effects and

TABLE 13.3 *Continued*

Name	Scope	Requirements for Public Participation
Environmental Justice in Minority Populations and Low-Income Populations"	environmental justice part of its mission.	mitigation measures and by improving accessibility by affected communities to public meetings, documents, and notices.
Executive Order 13175, "Consultation and Coordination with Indian Tribal Governments"	Requires most federal agencies to establish regular, meaningful consultation and collaboration with tribal officials in the development of federal policies that have tribal implications, to strengthen relationships between the U.S. government and Indian tribes, and to reduce the imposition of unfunded mandates upon Indian tribes.	Requires federal agencies to develop, utilize, and monitor implementation of an effective process that allows officials and representatives of Indian tribal governments to provide meaningful, timely input on regulations, legislative comments, proposed legislation, and policies that have substantial direct effects on one or more Indian tribes.
Executive Order 13132, "Federalism"	Requires agencies to evaluate and restrict policies and actions that have "substantial direct effects on the states, on the relationship between the national government and the states, or on the distribution of power and responsibilities among the various levels of government."	Each agency must have an accountable process to ensure meaningful and timely input by state and local officials in the development of regulatory policies that have federalism implications.
Executive Order 12866, "Regulatory Planning and Review"	Reforms and improves the efficiency of the regulatory process by enhancing planning and coordination with respect to both new and existing regulations; reaffirming the primacy of federal agencies in the regulatory decision-making process; restoring the integrity and legitimacy of regulatory review and oversight; and making the process more accessible and open to the public.	Wherever feasible, agencies should seek the views of appropriate state, local, and tribal officials before imposing regulatory requirements that might significantly or uniquely affect those governmental entities. Each agency should draft its regulations to be simple and easy to understand, with the goal of minimizing the potential for uncertainty and litigation arising from such uncertainty.
Executive Order 13166, "Improving Access to Services for Persons with Limited English Proficiency"	Each federal agency must prepare a plan to improve access to its federally conducted programs and activities by eligible persons limited in their English proficiency.	In carrying out this order, agencies should ensure that stakeholders, such as persons with limited English proficiency and their representative organizations, recipients, and other appropriate individuals or entities, have an adequate opportunity to provide input.

Source: Based on EPA 2000.

[a] The table includes the term *rule*, but the law or requirement may apply only to rules subject to formal rule-making requirements.

TABLE 13.4 Approaches for Stakeholder Involvement

Level of Stakeholder Involvement	Method	Description
Outreach (inform or educate)	Publications	Make available and/or distribute fact sheets, reports, and supporting documents, in hard copy, on the Internet, or in special collections (reading rooms); also videos, interactive CD ROMs, and Web-casts in appropriate media.
	Direct communication	Advertising, mailings, and public service announcements providing information and indicating availability of additional information.
	Press-mediated communication	Press kits, press releases, media events, and information articles or appearances in local media.
Information exchange (gather information and/or views)	Public meetings and hearings	Technical presentation of information by agency or consultants; input from attendees on technical analyses, information base, and value judgments.
	Deliberative polling	Polling of selected participants who have been provided information on the subject.
	Focus groups	Discussions by a small sample of persons selected to provide reactions to a proposal.
	Advisory groups	Groups of representatives of various stakeholders (industry, labor unions, neighboring communities, environmental activists) that present a particular policy point of view and comment on information basis.
Recommendation (discuss and/or involve)	Task force	Groups of representatives of various stakeholders (industry, labor unions, neighboring communities, environmental activists) that meet to make a set of recommendations to the agency.
	Workshops	Stakeholder representatives, technical experts, and governmental representatives meet together to explore a variety of issues and attempt to develop innovative approaches; may require multiple facilitators and sub group meetings.
Agreement (engage or partner)	Citizen advisory oversight groups	A standing committee of stakeholder representatives that monitors a particular site, activity, or study and provides ongoing feedback to the agency on methods, data, and results, and may recommend particular actions.
	Negotiated rule making	Stakeholder representatives work with regulatory agency to draft regulations.
	Referendum	All citizens are able to vote on a particular issue, necessarily stated simply.

Source: Data from Health Canada 2000; EPA 2003; NEA 2004.

13.3 RISK COMMUNICATION

Risk communication has many contextual meanings, including a dynamic sociological process, an academic field of study, a method employed by individuals and organizations to enhance effectiveness, and an approach to democracy. When practiced effectively, risk communication assures that important issues are identified and analyzed, assures that the risk analysis team has a common set of expectations and objectives, enhances acceptance of results by stakeholders, and is frequently mandated by law, procedures, or good practices of risk analysis.

There are several motivations for risk communication. A simple statement of the results (the truth as seen by the analyst) is seldom sufficient to communicate the analyst's view or to achieve the goals of the sponsoring organization. Human communication is imperfect. The message sent by the risk analyst may not be what the analyst intended. Furthermore, even if the message is sent relatively clearly, the message actually received by the audience may be quite different. In a digital age it is tempting to view risk communication as a mechanical transfer of information. However, because the communication is among humans, cultural, emotional, political, psychological, and other factors have a profound effect on how risk information is perceived and understood. The barriers to effective risk communication may be substantial. The discipline of risk communication attempts to alleviate and overcome these barriers.

13.3.1 Scientific Basis

13.3.1.1 Risk Perception Early studies in risk communication revealed that a number of factors influence a person's perception of any particular risk. Some important factors affecting risk perception are summarized in Table 13.5 (Fischoff et al. 1981; Covello and Merkhofer 1993). Examples illustrating the impact of these risk perception factors on a person's acceptance of risk are commonplace. A Hollywood actor may engage in risky behaviors such as skydiving and car racing but at the same time be strongly and vocally opposed to nuclear power. This may be due, in part, to the fact that the risky personal behaviors are voluntary, whereas exposure to nuclear power risks is imposed by the power company with the consent of the government. Risk of injury or death from airplane travel is about an order of magnitude less than that from automobile travel. This difference in risk acceptance is believed to result, in part, from the fact that the individual controls the automobile whereas the pilot controls the airplane.

The influence of perceived potential benefits is a major factor in acceptance of risks for individuals, businesses, and societies. For example, many individuals play state lotteries even though the expectation value of return[2] is negative. Undoubtedly, this is because the person buying the ticket focuses on the perceived potential benefit—winning a large prize—rather than the more likely outcome of no return on investment. An example common in environmental risk is the practice by haz-

[2] The expectation value of return from a lottery ticket is the expectation value of the prize minus the cost of the bet: $\overline{R} = (\sum_{i=1}^{N} V_i P_i) - C$, where P_i is the probability of winning an amount V_i and C is the cost of the lottery ticket. Usually, the most likely outcome (largest P_i) is nothing back, for which V_i is zero.

TABLE 13.5 Factors Affecting Risk Perception

Risk Perception Factor	Risks Perceived as Generally, *More* Acceptable	Risks Perceived as Generally, *Less* Acceptable
Volition	Voluntary	Imposed
Control	Under individual's control	Under another's control
Benefit	Clear, substantial benefit	Little or no benefit
Equity	Fairly distributed	Unfairly distributed
Cause	Natural	Anthropogenic
Distribution	Diffuse, broadly distributed in space or time	Catastrophic, concentrated in space or time
Source	Generated by a trusted source	Generated by a distrusted source
Familiarity	Familiar or historical	Exotic or new
Subject	Affecting adults	Affecting children or future generations
Nature of effect		
Severity	Slight	Major (death)
Agony	Painless	Painful
Duration	Instantaneous (e.g., accidental death)	Long, drawn-out latency (e.g., cancer fatality)

Source: Based on Fischhoff et al. 1981.

ardous facilities to provide the surrounding community with direct and indirect monetary benefits in consideration of increased risk. Often, this is accomplished by monetary payments to support the school system or other functions of local government and by providing employment opportunities for local residents. For example, the hazardous waste landfill operated at Emelle, Alabama provides direct and indirect benefits to the local community amounting to approximately $16.1 million per year, or about $950 per capita in Sumter County (Bailey et al. 1992); such benefits are significant in this economically depressed local community, where the per capita income is $8290. However, this study also found that community leaders and business leaders perceived much higher benefits than did the general public.

Because a perceived lack of fairness frequently leads to feelings of anger, equity issues are significant factors in risk communication. Facilities or environmental decisions that seem to put a certain group at risk while providing benefits to another group are frequently perceived in a very negative way. For example, a power plant, chemical plant, or waste disposal facility may put nearby residents at risk while enriching the owners and operators of such facilities. Natural risks are generally more readily acceptable than human-caused risks. Part of the emotional context for this appears to be the willingness to treat natural risks as purely random or "acts of God," whereas human-caused risks (even if accidental) are caused by persons deserving blame. For example, federal regulations strictly control the release of radionuclides generated in a nuclear reactor; however, the same radionuclides occurring naturally are generally not regulated by the federal government. Damage to property, death, and injury from hurricanes may be accepted as natural phenomena; however, these risks could be reduced if land-use policy prohibited

development of vulnerable coastal areas or stopped encouraging such development by federally subsidized flood insurance. Clearly, whether a particular risk is considered natural or human-caused depends on the observer.

Risks that are dispersed in time and/or space tend to be more readily accepted than risks that are considered catastrophes. One reason postulated for the aversion to catastrophes is that such events frequently cause substantial societal, economic, political, and emotional disruptions, with the attendant costs. Again, a possible cause for acceptable risk from air travel to be lower than the acceptable risk from automobile travel is that an air crash may involve the deaths of several hundred people, whereas automobile accidents seldom involve more than three or four deaths. Furthermore, air crashes occur in a single location and may affect a particular community since the travelers may be going to or coming from that location. Automobile crashes generally occur in widely separated locations and at different times. Regardless, the total number of annual deaths and the risk of death are much larger from automobile travel than from air travel. Risks generated or imposed by a trusted source are more readily accepted than risks from a distrusted source. For example, many people will accept risks of adverse side effects from medication because the medication is prescribed by a trusted physician; however, lower objective risks imposed, for example, by a public health official in a mandatory vaccination program may not be accepted. Familiar risks are more readily accepted than unusual risks. For example, the hazardous cargo causing most highway fatalities is gasoline in tanker trucks; however, the concern expressed about such traffic by the public and press appears to be minimal. On the other hand, concerns about trucks carrying radioactive material, including radioactive waste, appears to be very high, even though the number of fatalities from transporting radioactive material is either zero or very small (OTA 1986).

Risks to children and future generations are not accepted as well as equivalent risks to adults. This differentiation arises in part because most humans and most societies consider protection of children a high priority. In addition, because most children are not equipped to ensure their own safety, normal practice is for adults to provide additional, sometimes very stringent, levels of protection and safety. The manifestation of the risk usually affects the willingness to accept it. The severity of the manifestation is primary. Risk of minor irritation is usually better accepted than risk of injury, which is in turn usually better accepted than risk of death. Risk of a painful, lingering death (e.g., certain kinds of cancer) is generally less acceptable than a numerically equivalent risk for which death is instantaneous and painless.

13.3.1.2 Risk Communication Models A variety of approaches or models relevant to risk communication have been developed that take into account the various aspects unique to communicating risk: (1) technical, often complex, information; (2) hazard to human health, property, society, or the environment; and (3) negative feelings that influence risk perception. Lundgren and McMakin (1998) discuss eight approaches. The following expanded descriptions are based on selected categories.

Mechanistic Model In this approach (dubbed the communication process approach by Lundgren and McMakin) an analogy is made between physical com-

munication systems (e.g., cell phones) and human-to-human communication. A simple system construct is comprised of a source, transmission channel, and receiver. This provides a fundamental, albeit limited conceptual model for communication. Faulty communication can be attributed to unintended emissions by the source, distortion of the signal by the transmission channel, failure to receive the complete signal, and so on. The advantage of this approach is that well-established principles of (physical) communication theory and information theory may be applied. For example, Shannon (1948) and Shannon and Weaver (1963) applied the concept of entropy to analyze information content and noise in a signal (Figure 13.1). The disadvantage of this approach is that emotional characteristics of the source, transmission channel, and receiver may not be given the significant weight which experience shows that they deserve.

Public Policy Approaches The National Research Council and a specially formulated Presidential/Congressional Commission on Risk Assessment and Risk Management have produced several studies addressing risk communication from the viewpoint of public policy formulation. The earliest of these studies (NAS–NRC 1989) stressed that risk communication is a two-way interactive process among stakeholders, including the institutional sponsor. It countered a view held at that time by some governmental and scientific organizations that experts need only analyze an issue and announce the results.

A subsequent National Research Council study (NAS–NRC 1996) recommends use of an **analytic–deliberative process** that combines analysis (the systematic application of scientific theories and methods to an environmental problem) and deliberation (a process of discussion, reflection, and persuasion) to communicate and understand relevant issues and arrive at a substantive decision. The process is focused on reaching decisions that solve problems. It requires early, iterative interactions with stakeholders to help formulate the problem and provide information to aid in its solution. However, some practitioners caution that a close association of the analyst with solution options and those evaluating them may go beyond scoping the analysis to affect the analysis unduly (Yoe 2000).

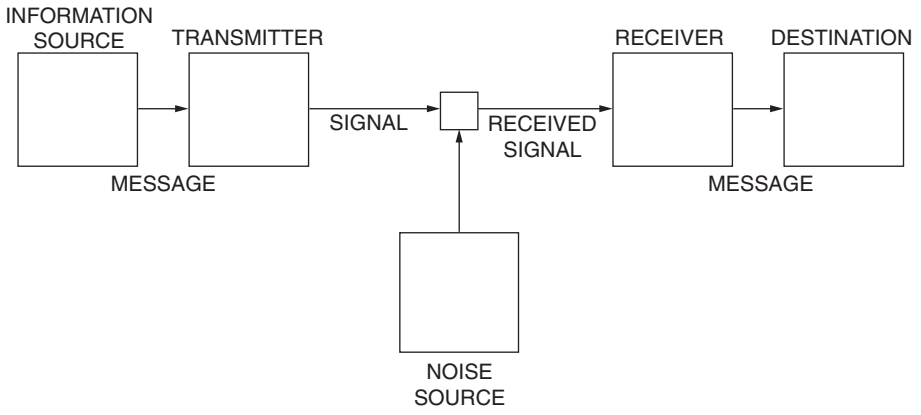

Figure 13.1 Conceptual model of communication. (From Shannon 1948; reprinted with permission of John Wiley & Sons, Inc.)

More recent studies (Presidential/Congressional Commission on Risk Assessment and Risk Management 1997a,b) outline a framework for conducting and applying risk assessment to issues of environmental health. A six-step process for risk management (which may be iterated) is advanced that requires stakeholder involvement at every step (Figure 13.2). The intention is to "emphasize the importance of collaboration, communication, and negotiation among stakeholders so that public values can influence risk management strategies." Volume 1 of the report provides guidelines for stakeholder involvement. Some of the more notable guidelines are as follows: (1) clearly state the extent to which stakeholder input will affect decisions so that false expectations are not raised; (2) give credit to stakeholders for their input and identify how it was used; (3) don't expect stakeholder involvement to end controversy; and (4) government agencies must adopt stakeholder participation as part of their mission by assigning staff and resources for this purpose.

Communication Theory Approaches General communication theory is a well established discipline (Griffin 2003; Miller 2004) with a variety of models for communication and techniques for enhancing delivery of messages through different media, including the mass media. One approach is to apply principles and practices based on general communication theory to risk communication (e.g., Rowan 1991; Langford et al. 2000; Jardine 2003; Santos and Chess, 2003). Jardine (2001) has suggested that development of theories and techniques specifically for risk communication has unnecessarily reiterated and "rediscovered" principles established in the broader discipline. Chess (2001) has indicated the importance of organizational theory in the development of risk communication approaches.

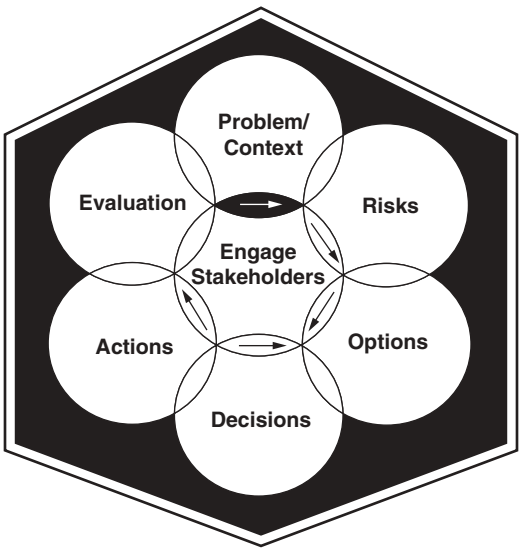

Figure 13.2 Paradigm for stakeholder involvement in environmental health risk assessment used by the Presidential/Congressional Commission on Risk Assessment and Risk Management (1997a,b).

Mental Models Approach A mental model is a psychological theory of cognition that attempts to represent how the human mind processes information, develops an understanding, and makes decisions (Gentner and Stevens 1983; Johnson-Laird 1983). This theory has been applied to the problem of risk communication (Atman et al. 1994; Bostrom, et al. 1994; Morgan et al. 2001). A key implication for risk communication is that information about a particular risk is processed in the context of a preexisting mental model that may include ideas about that particular risk, an approach to evaluating risks of this type, and an attitude regarding the source of the information. The mental models approach to risk communication emphasizes the need to explore, understand, and respond to the existing ideas and cognitive processes characteristic of the target audience. A five-step process has been suggested (Morgan et al. 2001): (1) use a suitably multidisciplinary team of experts to create an expert model of a hazard using influence diagrams; (2) conduct open-ended interviews to determine the mental models of the hazard, with a focus on how they may differ from the expert model; (3) conduct structured, precommunication interviews with people representative of the target audience to determine the prevalence of various mental models; (4) based on previous steps, develop a provisional risk communication approach focused on the high-priority differences between the expert model and mental models of the target audience by correcting erroneous beliefs and reducing lack of knowledge; and (5) test the effectiveness of the provisional approach on a sample of the target audience and refine it before use.

Hazard plus Outrage This paradigm, largely articulated by Peter Sandman, is that the response of a person to a risk depends on the technical hazard estimate and the feelings about that risk; hence, risk = hazard + outrage. This paradigm can explain high public concern over some risks that evoke a high emotional response but which technical experts estimate as minimal (e.g., transportation of nuclear waste or incineration of nerve agent). It can also explain lack of public concern over some risks (e.g., obesity or high blood pressure) that are objectively high but yield a low emotional response. This approach recognizes four major obstacles to effective risk communication (Covello and Sandman 2001): (1) "the uncertainty, complexity, and incompleteness of environmental data"; (2) distrust of experts, corporations, trade associations, and government agencies; (3) selective reporting by news media; and (4) psychological and sociological factors that influence how risk information is processed, including factors affecting risk perception (discussed, in part, in Section 13.3.1.1). An implication of this approach for risk management and risk communication is that to increase public acceptance, reduction of outrage factors is as important as reduction in technical risk.

13.3.2 Practical Considerations

Many authors have developed practical guides for effective risk communication that focus on organizational practices, guidelines for individual communicators, or both (NEHC, undated; Covello and Allen 1988; Lundgren and McMakin 1998; CDC 1999; ATSDR 2001; NRC 2004). These guides provide practical information for improving risk communication. For example, Table 13.6 lists the "seven cardinal rules of risk communication" developed by Covello and Allen for the EPA. The

TABLE 13.6 Seven Cardinal Rules of Risk Communication

Rule	Motivation
1. Accept and involve the public as a partner.	Your goal is to produce an informed public, not to defuse public concerns or replace actions.
2. Plan carefully and evaluate your efforts.	Different goals, audiences, and media require different actions.
3. Listen to the public's specific concerns.	People often care more about trust, credibility, competence, fairness, and empathy than about statistics and details.
4. Be honest, frank, and open.	Trust and credibility are difficult to obtain; once lost, they are almost impossible to regain.
5. Work with other credible sources.	Conflicts and disagreements among organizations make communication with the public much more difficult.
6. Meet the needs of the media.	The media are usually more interested in politics than risk, simplicity than complexity, danger than safety.
7. Speak clearly and with compassion.	Never let your efforts prevent your acknowledging the tragedy of an illness, injury, or death. People can understand risk information, but they may still not agree with you; some people will not be satisfied.

Source: Covello and Allen 1988.

NEHC Risk Communication Primer focuses on three aspects—the message, the messenger, and the medium—and provides detailed guidance for improving each aspect. An example of points related to the message are: (1) Structure and organize the key messages; (2) limit information to three key messages; (3) keep the key messages short (7 to 12 words); and (4) repeat the key messages. Examples of points related to the messenger are: (1) Factors determining trust and credibility are empathy and caring, honesty and openness, dedication and commitment, and competence and expertise, of which empathy and caring is dominant, as shown in Figure 13.3; (2) body language is very important and can determine the perception of honesty and openness; (3) to be perceived as dedicated and committed, be sure to keep all commitments and promises; and (4) to be perceived as competent, avoid using notes and dress professionally. An example of media guidelines is a three-option model for responding to questions: (1) Make an empathetic statement followed by the main conclusion and indication of future action; (2) state the main conclusion followed by two supporting facts; and (3) state the three key messages.

13.3.3 Unresolved Issues

Stakeholder participation augmented by the implementation of good risk communication practices has had a significant impact on democratizing environmental

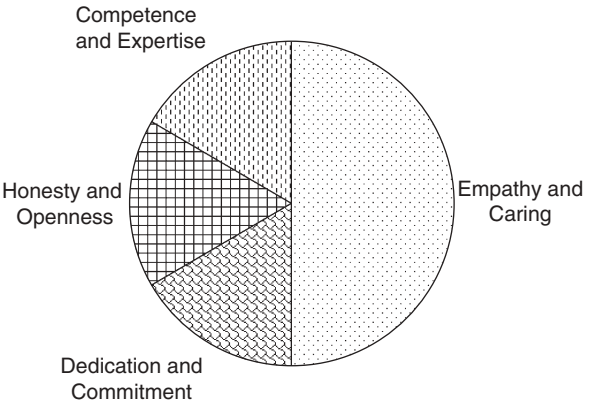

Figure 13.3 Importance of factors determining trust and credibility of a person communicating risk. (NEHC, undated; Covello 1992.)

decision making. However, with all the significant progress over several decades, some unresolved issues remain. In an increasingly fragmented society, stridently different points of view are likely. Although negotiations among stakeholders are an avenue for resolving differences and reaching agreement, one or more stakeholder may be unwilling to compromise. The result is an impasse, even though incentives may be offered to stakeholders to reach agreement. This may be a symptom of a more fundamental problem in which meeting the needs of the majority of society while protecting minority rights has become increasingly difficult. This has been exacerbated by the rise of many special-interest groups that have fixed beliefs which appear immutable and which drive their position.

When embraced fully and practiced effectively, risk communication promotes democracy and achieves better, more accepted decisions. However, some organizations may use risk communication techniques to put the best face on decisions or practices unpopular with stakeholders or to provide the appearance of seeking participation when decisions have already been made.

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PROBLEMS

13.1 Your company is planning to construct a factory that will produce nanoparticles for a variety of industrial uses. The planned factory site is a small agricultural community. Rate the perception of risks from the factory according to the criteria listed in Table 13.5. What types of actions might you recommend to the company management?

13.2 Research and discuss the effectiveness of stakeholder participation and/or risk communication in the following programs or events:

- **(a)** The development of a U.S. repository for high-level nuclear waste, since passage of the Nuclear Waste Policy Act of 1982, which mandates stakeholder involvement.
- **(b)** Advisory programs that warn against eating fish (certain species or fish from certain bodies of water) contaminated with mercury.
- **(c)** An anthrax threat such as that in the fall of 2001, when anthrax spores were discovered in several pieces of mail addressed to lawmakers and members of the news media, resulting in contamination of mail-sorting facilities and the deaths of several people.
- **(d)** Hazards to children from lead-based paint in older buildings.

14 Environmental Risk Management

14.1 INTRODUCTION

As described in Chapter 1, risk analysis is comprised of three components: risk assessment, risk communication, and risk management (Figure 1.1 and Section 1.1). The role of risk management is to articulate alternative actions and select one for implementation. This requires a two-way flow of information with risk assessment and risk communication. Information flows into risk management from risk assessment activities (e.g., relative risks of various alternatives) and from risk communication activities (e.g., value judgments of stakeholders regarding evaluation factors). Information flows from risk management regarding the decision to be made, potential alternative actions, the preferred alternative, and a clear statement of the rationale for the choice.

This chapter is concerned with both the risk management process (Section 14.2) and risk management methods and techniques (Section 14.3). However, in keeping with the treatment of risk assessment and risk communication, the emphasis is on methods; which are primarily decision analysis methods.

14.2 RISK MANAGEMENT PROCESS

The essential tasks of the risk management process, as defined by the American Chemical Society (ACS 1998), are (1) to identify risks that are higher or may be higher than a level acceptable to society, (2) to identify how such risks may be controlled, and (3) to decide on appropriate ways to mitigate or reduce these risks to an acceptable level. A conventional business management definition (Haimann et al. 1978) of a decision process includes (1) a definition of the objective; (2) a definition of alternative courses of action to achieve the objective; (3) an estimate of the outcomes, consequences, or results for each alternative; (4) a method, technique, or procedure by which one alternative course of action is selected; and (5) an evaluation of the selected alternative. Not surprisingly, the Presidential/Congressional Commission on Risk Assessment and Risk Management (1997) articulated a similar process for risk management related to government regulation of environmentally hazardous substances (also listed in Chapter 1). It consists of the following: (1) Define the problem and put it in context; (2) analyze the risks associated with the problem in context; (3) examine options for addressing the risks; (4) make decisions about which options to implement; (5) take actions to implement

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the decisions; and (6) conduct an evaluation of the action's results. A very similar six-step process is articulated by the U.K. Health and Safety Executive (HSE 2001).

In a very broad context, approaches for managing environmental risk may be prescriptive or goal-oriented. Such approaches include regulatory control, permitting, economic incentives, and voluntary action. Establishing a framework or context for the risk management issue is critical, because all remaining aspects of the process may experience difficulty because of misunderstandings by risk managers making the decision, by analysts involved in the risk assessment or decision analysis, and by stakeholders. Closing the loop on the risk management process by evaluating results is an essential step.

14.3 RISK MANAGEMENT METHODS

14.3.1 Approaches to Risk Management

As noted in Chapter 1, **environmental risk management** refers to the broad process of balancing risks, costs, and social values. The risk assessment provides estimates of risk. Economic analysis provides costs of various alternative actions. Social values can be provided by risk communication and stakeholder involvement. Risk management balances these inputs and chooses a particular alternative for action. Although an agency or decision maker responsible for taking action might decide the matter and announce the decision without stating the rationale behind it, such a practice is seldom used for environmental decisions having significant public impact and is generally prohibited by law. Instead, a two-stage approach to decision making is generally used (Morgan and Henrion 1990). The first stage involves development and articulation of strategies or criteria for decision making. The second involves the use of a mechanistic (mathematical) process to select a preferred alternative based on the stated criteria and inputs from risk assessment and stakeholder involvement. This approach avoids the invidious practice (or at least its appearance) of making a decision and then assembling a rationale to support it.

There are many criteria for decision making. Some criteria are mandated by law while others are developed by decision makers, frequently with stakeholder involvement. Morgan and Henrion (1990) identify four broad classes of decision criteria:

1. **Utility-based criteria** are those that involve some measure of the value or utility of the outcome. Utility-based criteria are frequently used for decisions where the outcome is described by a single variable, such as cost or the ratio of benefit to cost. However, utility-based criteria find substantial use for decisions where the outcome is described by multiple attributes, which are factors (cost, human health risk, ecological risk, worker risk, aesthetics, etc.) important to the decision maker. A key element of multiattribute utility analysis is the expression of the decision maker's preferences by mapping values of each attribute to a cardinal numerical scale. Utility-based criteria are appealing because they can be expressed quantitatively. Examples of such criteria are: (1) minimize the cost; (2) maximize the benefit/cost ratio; and (3) maximize the multiattribute utility. This permits a

succinct, unequivocal statement of the decision-making criterion and the use of powerful, well-established quantitative decision analysis tools. However, as explained in the next section, conversion of qualitative or quantitative attributes into utility requires value judgments expressed as preferences, about which there may be significant disagreement. This may be particularly evident for environmental decisions with significant stakeholder participation. Processes and decision methods for resolving these disagreements have been and continue to be the focus of considerable study.

2. **Rights-based criteria** stress natural rights over supposedly objective measures of collective utility, so are less concerned with the value of an outcome. Such criteria include zero risk or bounded risk, approval with compensation, and due process. Zero risk (or "zero discharge") means the complete elimination of any chance of harm from an activity, either by eliminating it or by preventing its introduction. Although this is sometimes possible with threshold contaminants, the complications introduced by nonthreshold contaminants (for which there is no level of exposure which carries no risk) and by the analysis of substitution risks (the risks imposed by the alternatives to the risk currently being addressed) make this criterion difficult to achieve in practice. This approach is often modified to the concept of bounded risk, in which a risk level is determined to be negligible and not actionable under certain regulatory standards. These negligible risk levels are sometimes termed "de minimis" levels, from the Latin phrase, "de minimis non curat lex" (the law does not concern itself with trifles). Zero risk and bounded risk are widely applied in U.S. legislation dealing with environmental hazards. For example, the Delaney clause of the Food Additives Amendment Act of 1958 [P.L. 85–929] required, in part: "No additive shall be deemed to be safe if it is found to induce cancer when ingested by man or laboratory animals." This zero risk requirement has since been modified [P.L. 104–170] so that de minimis levels based on human health risk are permitted. Approval with compensation is also a rights-based criterion, differing from zero risk in that it allocates the right of refusal to certain risks but allows negotiation and choice among a broader set of defined risks. Risks may only be imposed on a person or population on a voluntary basis, but such voluntary risk acceptance may be sought by offering compensation or mitigation of impacts.

3. **Technology-based criteria** are based on the idea of resolving the decision process by simply doing the best job currently possible and are used in several regulatory programs. Use of best available technology simplifies the decision process for managers of environmental risk. However, this approach may decrease incentives for innovation. As with other decision-making criteria, identifying the best available technology may be difficult and controversial, since generally the determination must consider the cost and availability of the technology as well as the efficiency with which it can reduce risks.

4. **Hybrid criteria** combine approaches for different magnitudes of risk. For example, rights-based criteria may be used to set an upper limit on risk, but utility-based criteria are used to optimize risks below the limit. For example, federal regulations limit the annual radiation dose to workers to 0.05 Sv at licensed facilities using radioactive material; however, the ALARA principle (i.e., that dose should be as low as reasonably achievable) is used to reduce doses below the annual limit in a cost-effective manner.

14.3.2 Fundamentals of Decision Analysis

As indicated in Section 14.3.1, a key element in risk management is choosing a preferred alternative action from several proposed alternatives. **Decision analysis** is the study of formal methods for making decisions, based on various criteria and constraints.

▶ Example 14.1

You go to your local restaurant for dinner and are presented with a variety of menu items. Your choice for dinner may depend on several attributes for each item, such as (1) cost, (2) preparation time, (3) quantity, (4) healthfulness, and (5) taste. Your decision may be dominated by one attribute or depend on a combination of factors. If you are in a hurry, you choose the item with the shortest preparation time. If you are very hungry but on a tight budget, you choose the item that provides the most food for the lowest cost.

14.3.2.1 Framework for Decision Analysis In general, decisions to which decision analysis methods are applied are more complex than indicated in Example 14.1. Modern approaches to decision analysis (Lifson 1972; French 1986) presume a rational decision maker confronted with a decision-making situation on a system in which a set of alternatives, $\{X_i\}$, is defined; a set of states of nature, $\{S_j\}$, is known; and an outcome, Z_{ij}, is associated with each alternative and each state of nature (Table 14.1). For simplicity, this description is limited to finite, discrete sets of alternatives and states of nature. In environmental risk management, alternatives are usually related to control of environmental emissions or remediation of contaminated sites. An example is a coal-fired power plant which is required to reduce ambient concentrations of SO_2 in a nearby town. Alternatives to accomplish this goal include (1) switching to use of coal with lower sulfur content, (2) increasing the height of the smoke stack, and (3) installation of SO_2 scrubbers. States of nature are conditions that prevail at some future time that do not depend on the choice of alternative. For the power plant decision, states of nature might include (1) no

TABLE 14.1 Paradigm for Decision Analysis

Alternative								
X_1	Z_{11}	Z_{12}	Z_{13}	...	Z_{1j}	...	Z_{1M}	
X_2	Z_{21}	Z_{22}	Z_{23}	...	Z_{2j}	...	Z_{2M}	
X_3	Z_{31}	Z_{32}	Z_{33}	...	Z_{3j}	...	Z_{3M}	
⋮	⋮	⋮	⋮	...	⋮	...	⋮	
X_i	Z_{i1}	Z_{i2}	Z_{i3}	...	Z_{ij}	...	Z_{iM}	
⋮	⋮	⋮	⋮	...	⋮	...	⋮	
X_N	Z_{N1}	Z_{N2}	Z_{N3}	...	Z_{Nj}	...	Z_{NM}	
	S_1	S_2	S_3	...	S_j	...	S_M	States of nature
	p_1	p_2	p_3	...	p_j	...	p_M	Probability

[a] For each alternative X_i, and each state of nature S_j, there is an outcome for the system Z_{ij}. Associated with each state of nature is a probability p_j.

change from currently prevailing conditions; (2) alteration of the joint frequency distribution of wind direction, stability class, and wind speed, by trends in meteorological conditions near the power plant; (3) increase in the cost of low-sulfur coal; (4) increase in the nominal sulfur content of low-sulfur and high-sulfur coal; and (5) various levels and combinations of (2), (3), and (4). States of nature are defined so they are mutually exclusive. Because future states cannot be predicted with certainty, there is usually a probability associated with each future state that indicates its likelihood of occurrence. States of nature are considered to exhaust all possibilities, so their probabilities sum to 1. Also, states of nature may incorporate model uncertainty and parameter uncertainty discussed in Chapter 12. Thus, a set of several alternative conceptual models can each generate a class of states of nature, characterized by the validity of a particular conceptual model. Similarly, a set of several nominal values for a model parameter (e.g., the hydraulic conductivity of an aquifer) each generates a class of states of nature, characterized by the particular nominal value or value range being representative for the system.

French (1986) points out that the outcome Z_{ij} associated with each alternative i and each state of nature j is essentially "a complete, holistic description of the possible consequences" given alternative i and state of nature j. A clear distinction is then made between Z_{ij} and a real, scalar quantity representing the value of this outcome to the decision maker. This value is defined so that it represents the preferences of the decision maker. That is, the value of Z_{ij} is greater than the value of Z_{kl} if and only if Z_{ij} is preferred to Z_{kl}. If this value function is constructed in a manner that satisfies certain other axiomatic properties, it is the **utility** of the outcome. Hereafter Z_{ij} is used to represent the value or utility associated with a particular outcome.

For very simple environmental decisions that consider only a single aspect of the outcome, the system attribute used to evaluate an outcome is usually transparent. For example, if the lowest-cost alternative is to be chosen, the cost of each alternative determines the utility. If the lowest risk alternative is to be chosen, the dose for each alternative determines the utility. However, for most environmental decisions, multiple attributes of the system are considered. For the power plant decision, attributes might include (1) ambient concentration of SO_2 in the nearby town, (2) peak concentrations of SO_2 in the nearby town, (3) cost of the alternative, (4) plant downtime required to implement a particular alternative, and (5) acceptability of an alternative to citizens in the nearby town. In general, attributes may be categorized (Lifson 1972) as related to the effectiveness of an alternative, the cost of an alternative, and the timing or schedule of an alternative. To accommodate multiple attributes, a utility function is generated for each attribute A_k that the decision maker uses in the decision. It is convenient to consider each outcome to be represented by a multidimensional vector $\bar{Z}_{ij}$ with components z_{ijk}. That is,

$$\bar{Z}_{ij} = \langle z_{ijk} \rangle \tag{14.1}$$

The components z_{ijk} represent the utility for system attribute k, alternative i, and state of nature j. The scalar system utility Z_{ij} is considered to be a function of the components of the corresponding vector representation of the outcome. Frequently, the function relating the components to the system utility Z_{ij} is taken to be a weighted sum of the utilities for each attribute. Although an attribute may be stated

qualitatively (e.g., preservation of natural beauty), the utility approach requires that each outcome be assigned a real number representing the preference of the decision maker. Some authors (Lifson 1972; French 1986) use the term "decision criteria" instead of system "attributes". However, these decision criteria, related to an outcome, are different and distinct from the decision-making criteria discussed in Section 14.3.1.

Decision analysis usually considers three categories of decisions (Lifson 1972; French 1986; Rapoport 1989): decisions under certainty, decisions under risk, and decisions under uncertainty. For decisions under certainty, the state of nature is considered to be known. This is equivalent to setting one of the probabilities of future states equal to 1 and the remainder equal to zero. In addition, for decisions under certainty, the outcome for each alternative is assumed to be known with no uncertainty. This reduces the matrix shown in Table 14.1 to the single column shown in Table 14.2. For decisions under certainty, the decision analytic problem is to select the most desirable alternative. For decisions under risk, the probability p_j of future states of nature and the outcomes z_{ijk} are known or assumed to be known with no uncertainty. For decisions under risk, the decision analytic problem is to select the most desirable alternative considering the probability of the various future states of nature and the attributes of the system under those conditions. For decisions under uncertainty, the states of nature, alternatives, and outcomes are defined, but the probability associated with each state of nature is unknown or unquantified.

Decision analysis methods attempt to reach the best decision given the information available at the time the decision is made. For decisions under risk and uncertainty, a good decision may lead to an undesirable outcome that becomes evident at some future time. This is because the actual state of nature has no good outcomes or because the alternative chosen yields the optimal outcomes on average, but a bad outcome for an unlikely state of nature. For example, a gambler is playing stud poker and based on the visible cards concludes that his opponent can win only if he draws the correct card to an inside straight. The gambler estimates the probability of that outcome to be less than 1 in 100. Based on the return expected, the gambler calls his opponent's bet. The opponent fills his inside straight and the gambler loses his bet—a good decision but a bad outcome.

TABLE 14.2 Decisions Under Certainty[a]

Alternative		
X_1	Z_{11}	
X_2	Z_{21}	
X_3	Z_{31}	
⋮	⋮	
X_i	Z_{i1}	
⋮	⋮	
X_N	Z_{N1}	
	S	State of nature
	1	Probability

[a] The matrix shown in Table 14.1 reduces to a single column for decisions under certainty.

▶ Example 14.2

Depending on the approach taken to make a decision, the decision may be the best, most rational decision under the circumstances, but the result may be disappointing. A chemical plant that uses several hazardous chemicals is to be sited in a valley subject to flooding. The state EPA requires the plant to be built above the 100-year floodplain. The plant owners, considering liability costs and replacement costs for damage to the plant, determine that the plant should be sited above the 500-year floodplain. This reduces the statistical chance of flooding the plant over a 40-year lifetime of operation to less than 7%. The plant is sited and built according to plan. In late August of the first year of operation a Gulf hurricane moves inland and dumps an enormous amount of rain into the valley, which floods. The plant is severely damaged and fines are assessed for the release of hazardous chemicals to the environment.

For decisions under certainty, the one-column matrix shown in Table 14.2 may be expanded into a **decision matrix** in which each outcome is expanded into its components representing the value of the system for a given attribute, as shown in Table 14.3. For decisions under certainty, it is often unnecessary to transform all outcomes into utilities. Instead, the quantitative or qualitative value of a given attribute for the outcome may be used and is called the "performance value" in this book. This decision matrix contains a column for each attribute A_k and a row for each of the viable alternatives X_i. The elements z_{ik} in the table are either the quantitative or qualitative performance value for the ith alternative with respect to the kth attribute or the utility of the ith alternative with respect to the kth attribute. The context indicates which is intended.

TABLE 14.3 Decision Matrix Showing the Evaluation z_{ik} of Each Alternative X_i for Each Attribute A_k

	Attribute						
Alternative	A_1	A_2	A_3	...	A_k	...	A_L
X_1	z_{11}	z_{12}	z_{13}	...	z_{1k}	...	z_{1L}
X_2	z_{21}	z_{22}	z_{23}	...	z_{2k}	...	z_{2L}
X_3	z_{31}	z_{32}	z_{33}	...	z_{3k}	...	z_{3L}
⋮	⋮	⋮	⋮	...	⋮	...	⋮
X_i	z_{i1}	z_{i2}	z_{i3}	...	z_{ik}	...	z_{iL}
⋮	⋮	⋮	⋮	...	⋮	...	⋮
X_N	z_{N1}	z_{N2}	z_{N3}	...	z_{Nk}	...	z_{NL}

■ Decision Criteria in the Superfund Program

Examples based on EPA decision criteria for the Superfund Program are used throughout this chapter; however, to be consistent with the terminology used in this book, these decision criteria are termed "attributes" in the examples that follow.

As discussed in Section 15.3, a core step in the CERCLA (Superfund) process is selection of a remedy based on the Remedial Investigation/Feasibility Study

(RI/FS). The RI/FS process is used to establish site characteristics, potential risks, and feasible cleanup options. This information is used to evaluate alternative remedies for a site and to select a preferred remedy. This selection and its rationale are documented in the Record of Decision. In the National Contingency Plan (EPA 2005), EPA has established nine evaluation criteria for remedy selection based on legal, policy, and technical considerations (EPA 1990). These criteria are divided into three groups of descending importance: (1) threshold criteria, (2) primary balancing criteria, and (3) modifying criteria. The two threshold criteria are regulatory compliance and protection of human and environmental health. Threshold criteria must be met for an alternative to warrant further consideration. The primary balancing criteria are long-term effectiveness; short-term effectiveness; reduction of toxicity; mobility and volume; implementability; and cost. Evaluations of an alternative for these quantitative criteria are derived from field and laboratory data, models, and economic analyses. The balancing criteria are used to identify important trade-offs among alternative remedies. The modifying criteria are state and public acceptance. These qualitative criteria may be used after the formal public comment period on the proposed plan and may modify the selection obtained by using the balancing criteria. This decision approach is an example of hybrid decision-making criteria, as discussed in Section 14.3.1. The threshold criteria establish a level of minimal performance required to assure an acceptable level of risk and are rights based. The remaining criteria are utility based and thus allow for optimizing the choice from among acceptable alternatives.

▶ Example 14.3

It has been determined that a solvent waste pit has been leaking PCE into the subsurface. To address this problem, a number of potential remediation plans have been submitted by the engineering department. Assuming that each plan meets the threshold attributes of the RI/FS protocol, the decision maker must select the alternative that best meets the five balancing attributes. As an initial step, each of the four alternatives (1, 2, 3, and 4) has been analyzed and rated for each of the five balancing attributes. In this and subsequent examples, these five attributes are abbreviated as follows: long-term effectiveness (LTE), short-term effectiveness (STE), reduction of toxicity, mobility, and volume (TMV), implementability (IMP), and cost (COST). LTE and STE are rated on a scale of 1 to 10, where 10 is the highest performance value. The performance values for each alternative are given in Table 14.4, which represents the decision matrix.

TABLE 14.4 Decision Matrix for Example 14.3[a]

	Attribute				
Alternative	1: LTE[b]	2: STE[b]	3: TMV	4: IMP	5: Cost
1	4	3	Poor	Moderate	$400,000
2	6	3	Fair	Very easy	450,000
3	8	9	Good	Moderate	600,000
4	5	8	Good	Easy	400,000

[a] Each entry is the rating for a given attribute and alternative.
[b] Scale of 0 to 10.

14.3.2.2 Scaling Methods Many decision-making techniques require that all the performance values in a decision matrix be translated to a [0,1] scale before comparison across alternatives. Simple scaling may be utilized to transform a range of quantitative and qualitative values to a [0,1] scale. Qualitative attributes such as implementability can be transformed by using Likert-type scaling (Nunnally and Bernstein 1994), which is a psychometric scaling method frequently used on questionnaires to determine attitudes. A generalized Likert scale is an ordered, symmetric array of statements characterizing attitudes toward an assertion, feature, object, event, and so on. The traditional five-point Likert scale of (1) strongly agree, (2) agree, (3) neither agree nor disagree, (4) disagree, and (5) strongly disagree is used in questionnaires to determine the respondent's attitude toward a variety of statements, such as "Nuclear power is dangerous." Likert-type scales with an even number of points force the respondent to be positive or negative, while scales with an odd number of points allow neutrality. For environmental decisions, the transformation of qualitative information into a Likert scale may be performed by a technical expert, stakeholders, or the decision maker (usually). For example, ease of implementing a particular alternative is almost always an important attribute in environmental decisions. An example of a five-point generalized Likert scale for implementability of a particular alternative is (1) very easy, (2) easy, (3) moderate, (4) difficult, and (5) very difficult, as shown in Figure 14.1. Implementability can be estimated using this scale by either a single decision maker or a group of stakeholders. Group evaluations can be aggregated using the arithmetic average of the equivalent quantitative ranking scores or by other methods of aggregation.

Quantitative attributes that are represented by a range of numerical values can be scaled using a normalization procedure. For attributes where higher numerical values are preferred (e.g., long-term effectiveness), the following transformation performs the scaling:

$$r_{ik} = \frac{P_{ik} - P_{ik}^{\min}}{P_{ik}^{\max} - P_{ik}^{\min}} \tag{14.2}$$

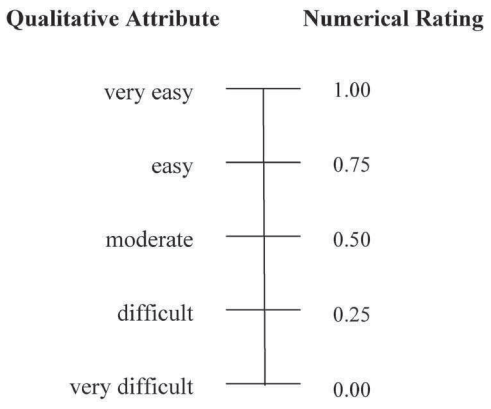

Figure 14.1 Generalized Likert scale for a criterion of implementability. The more desirable qualitative attributes are given higher numerical ratings.

For attributes where lower numerical values are preferred (e.g., cost), the following transformation performs the scaling:

$$r_{ik} = 1 - \frac{P_{ik} - P_{ik}^{\min}}{P_{ik}^{\max} - P_{ik}^{\min}} \tag{14.3}$$

where r_{ik} is the normalized performance value, P_{ik} the unscaled performance value of the ith alternative for the kth attribute, P_{ik}^{max} the maximum unscaled performance value for the kth attribute, and P_{ik}^{min} the minimum unscaled performance value for the kth attribute. For example, the scaled performance value for cost of alternative 2 in Example 14.3 is computed by using Eq. 14.3:

$$P_{2,5} = 1 - \frac{\$450{,}000 - \$400{,}000}{\$600{,}000 - \$400{,}000} = 0.75$$

► **Example 14.4**

The scaling methods can be used to transform the decision matrix for the waste pit problem discussed in Example 14.3. The resulting decision matrix is given in Table 14.5. For TMV, the performance scale is as follows: poor, 0; fair, 0.25; good, 0.5; very good, 0.75; and excellent, 1.0. For IMP the performance scale is shown in Figure 14.1.

TABLE 14.5 Decision Matrix for Waste Pit Problem in Example 14.3 with Scaled Ratings

	Attribute				
Alternative	1: LTE	2: STE	3: TMV	4: IMP	5: Cost
1	0.4	0.3	0.0	0.5	1.0
2	0.6	0.3	0.25	1.0	0.75
3	0.8	0.9	0.5	0.5	0.0
4	0.5	0.8	0.5	0.75	1.0

Performance scaling is a useful tool for decision analysis. However, the analyst and decision maker should be aware that scaling may magnify or minimize differences among alternatives. In Example 14.4 the scaled cost of alternative 2 is 0.75, compared to a value of 1.0 for alternatives 1 and 4. However, this 25% drop in scaled value for alternative 2 is produced by an increase in cost of only 12.5%, which could easily be within the uncertainty in the cost estimates.

As discussed in Section 14.3.2.1, an important concept in scaling outcomes is that of utility. A utility function maps values of an attribute to a dimensionless scale expressing the decision maker's preferences. Utility theory (Raiffa 1968; Lifson 1972) is considered by many to be the basis of modern decision analysis.

The performance scaling discussed in the preceding paragraphs may be considered to be utility functions. However, the functional relationships represented by Eqs. 14.2 and 14.3 are linear with respect to the magnitude of the variable (e.g., cost in the example). A generalized utility function may be nonlinear with the magnitude of the performance value and therefore better represent the decision maker's preferences, including risk aversion and risk seeking. This attribute makes utility theory an especially useful approach for decisions under risk. Homeowners' insurance provides an example. The insured pays a premium and therefore suffers a loss. In return, the insured is reimbursed for a range of potential damage or losses due to accidents or natural events. A common feature of such insurance is a deductible, an amount that the insured must pay for any covered loss. If the insured valued the risk of all losses the same, the insured would demand a zero deductible. This is because a broken window or scratch in the siding that occurs with some frequency, costing $100 to repair, would pose the same incremental risk as serious fire damage, costing $200,000 to repair but occurring with a frequency lower by a factor of 2000. In fact, most rational decision makers would elect a lower premium and only cover losses over a nominal deductible, perhaps $250. Such choices show an aversion to risks with higher consequences. Such preferences are exhibited in the environmental arena when high costs are paid to minimize risks with large consequences but very low risks are permitted. For example, nuclear power plants are allowed routinely to release small amounts of radioactive material to the environment, but extraordinary measures at large cost are used to prevent and mitigate significant releases of radioactivity in the case of a reactor accident.

14.3.3 Methods for Decision Analysis Under Certainty

The extensive literature on decision analysis methods cannot be incorporated here. Instead, examples of five different utility-based decision-making approaches are illustrated. The five approaches are dimension reduction, dominance, sequential decision making, attitude-based, and dimensional scoring. Dimension reduction simplifies the selection process by expressing all attributes in terms of one common measure, such as monetary value. Dominance is a screening method used to reduce the number of alternatives to be considered. Sequential elimination examines alternatives in order of attribute importance. This technique is particularly useful when only one attribute is controlling. Attitude-oriented methods utilize the attitude that the decision maker has to make a selection. Dimensional scoring computes the system value or utility by assigning a weight, proportional to the importance of each attribute, to the performance value for each outcome. The approach chosen depends on the nature of the decision to be made and the preferences of the decision maker. There is no universally applicable or correct approach. Different approaches may yield different decisions.

14.3.3.1 Dimension Reduction Dimension-reducing techniques simplify the decision-making process by transforming all attribute performance values to quantitatively comparable terms. Cost–benefit analysis is the most common example of this type of technique. This technique reduces all monetary and nonmonetary attributes into numerical values representing the costs and benefits associated with

each. Cost–benefit analysis can be a very effective decision-making method if the costs and benefits are easily quantifiable. However, the estimation of the costs and benefits for an alternative can be very difficult and often is controversial. An inherent assumption is that different attributes can be evaluated in commensurable terms. Although commensurability can almost always be forced, doing so may lose credibility with stakeholders. A classic example is assigning a dollar value to the loss of human life in a public decision-making context. In many cases, cost–benefit methods are not sufficient because they cannot easily address subjective or qualitative issues such as political support or public acceptance. Also, questions about the value of risk of human life or suffering and the values of remediation to a Greenfield versus a Brownfield status are not easily resolved. These issues begin to enter into the complex realm of social economics and may require the determination of trade-off values. Trade-off values are a quantification of the social and economic benefits associated with different alternatives.

► **Example 14.5**

Example 14.3 introduced a problem involving remediation of a leaking waste pit. The costs in dollars were estimated using standard engineering cost estimation procedures. Since human health impacts can result in such quantifiable effects as lost workdays, increased medical costs, and long-term illnesses, it is conceptually possible to estimate the financial impact of these health effects. In addition, remediation may result in an increase in property values or a reduction in expected liability from suits or regulatory actions as the site is cleaned up. A numerical estimate of total benefits, in dollars, for each alternative based on these factors is given in Table 14.6.

TABLE 14.6 Dollar Value of Benefits and Cost/Benefit Ratio for Each Alternative as Explained in Example14.5

Alternative	Benefit	Cost	Cost/Benefit Ratio
1	$390,000	$400,000	1.03
2	460,000	450,000	0.98
3	520,000	600,000	1.15
4	500,000	400,000	0.80

Based on the cost/benefit ratio, the best option is alternative 4 and the worst is alternative 3. However, it is important to note that this analysis is highly sensitive to the quantification of the human health impacts.

14.3.3.2 Dominance Dominance is the simplest of the decision-making approaches presented. Dominance analysis is a screening approach useful in reducing the number of alternatives to be considered. In this approach, each pair of alternatives is compared to determine whether the quantitative and qualitative attribute performance values for one are better than those of another. If the performance value for each attribute of one alternative is better than or equal to the corresponding performance value of another alternative, it is said to dominate.

► **Example 14.6**

To apply the dominance approach, the performance values for a pair of alternatives are compared on a pairwise basis for each attribute. Results for the attribute values in Table 14.5 are presented in Table 14.7.

TABLE 14.7 Pairwise Comparison of Alternatives for Each Attribute in the Decision Matrix for Example 14.6

Row	Alternative Pair	Attribute				
		1: LTE	2: STE	3: TMV	4: IMP	5: Cost
1	Alt. 1 vs. alt. 2	<	=	<	<	>
2	Alt. 1 vs. alt. 3	<	<	<	=	>
3	Alt. 1 vs. alt. 4	<	<	<	<	=
4	Alt. 2 vs. alt. 3	<	<	<	>	>
5	Alt. 2 vs. alt. 4	>	<	<	>	<
6	Alt. 3 vs. alt. 4	>	>	=	<	<

For each pair of alternatives, alternative U vs. alternative V, a table entry of ">" indicates $x_{uk} > x_{vk}$, a table entry of "=" indicates $x_{uk} = x_{vk}$, and a table entry of "<" indicates $x_{uk} < x_{vk}$. To meet the dominance criterion, a row must have only < and = or > and =. Any row with both < and > does not meet the criterion. As this table shows, only the third row, the comparison of performance values for alternatives 1 and 4, meets the dominance test. This indicates that alternative 1 may be eliminated, since alternative 4 is better in all regards except cost, where it is equal. Although this screening process is helpful in reducing the number of alternatives that must be considered, the dominance technique usually does not yield a single preferred choice.

14.3.3.3 Sequential Decision Making The primary sequential decision-making method is termed lexicography. This method considers one attribute at a time to examine alternatives for elimination. The procedure for this method consists of three steps. First, the decision maker subjectively ranks the attributes in descending order of importance. Second, if the performance value for the most important attribute is highest for a single alternative, that alternative is preferred. If several alternatives have the same highest performance value for the most important attribute, they all remain feasible alternatives. Third, the remaining alternatives are evaluated sequentially for each attribute in order of descending importance until only one alternative remains. The order in which the alternatives are eliminated can provide a ranking of the desirability of the various alternatives.

► **Example 14.7**

A decision matrix for a waste pit similar but not identical to Example 14.3 is given in Table 14.8. The priority ranking of the attributes is (1) STE, (2) TMV, (3) LTE, (4) IMP, and (5) COST. The decision matrix in Table 14.8 shows the attributes in

TABLE 14.8 Decision Matrix for Example 14.7

	Attribute				
Alternative	1: STE	2: TMV	3: LTE	4: IMP	5: Cost
1	3	Poor	4	Moderate	$400,000
2	2	Fair	6	Very easy	450,000
3	8	Good	8	Moderate	600,000
4	8	Good	5	Easy	400,000

their order of importance. Using these rankings, the performance values of the alternatives are first considered with respect to STE. Alternatives 3 and 4 have equal, preferred performance values. Therefore, only these two alternatives are considered further. Comparing these alternatives according to the second-most-important attribute, TMV, indicates that both alternatives are still equal. Comparison of the third attribute, LTE, indicates that alternative 3 is the best choice.

This method is very simple and straightforward to use since it considers only one attribute at a time. This is also one of its drawbacks. When all of the attributes for alternatives 3 and 4 are considered, it becomes obvious that while alternative 3 is more effective in the long term, it is also more expensive and more difficult to implement.

14.3.3.4 Attitude-Based Decision Making A number of techniques have been developed for situations where the attitude of the decision maker is known to be pessimistic, optimistic, conservative, fatalistic, and so on. Since environmental decision makers are generally required to make conservative decisions, a technique that minimizes the worst outcome is often useful. The mini-max (also maxi-min) approach, for a conservative or pessimistic decision maker, determines the worst performance associated with an alternative and then selects the alternative that minimizes the worst of these bad outcomes. Using the terminology introduced previously, the minimum z_{ik} for each alternative A_i is determined; then the alternative with the greatest minimum z_{ik} is selected. For applications with a different value system (e.g., venture capital) the decision maker may wish to achieve the best possible outcome; in this case the maxi-max approach is employed and the alternative with the largest best outcome is selected (e.g., highest return on investment).

► **Example 14.8**

The mini-max approach can be illustrated using the decision matrix presented in Table 14.5. The minimum performance value for each alternative is indicated in boldface type in Table 14.9. The minimum performance values for each alternative are as follows:

- *Alternative 1:* TMV = 0.0
- *Alternative 2:* TMV = 0.25
- *Alternative 3:* COST = 0
- *Alternative 4:* LTE = TMV = 0.5

TABLE 14.9 Decision Matrix for Example 14.8

	Attribute				
Alternative	1: LTE	2: STE	3: TMV	4: IMP	5: Cost
1	0.4	0.3	**0.0**	0.5	1.0
2	0.6	0.3	**0.25**	1.0	0.75
3	0.8	0.9	0.5	0.5	**0.0**
4	**0.5**	0.8	**0.5**	0.75	1.0

Since alternative 4 has the largest minimum value (the best of the worst outcomes), it is selected. This technique does not consider the importance of an attribute in selecting the preferred alternative.

14.3.3.5 Dimensional Scoring Simple additive weighting is probably the most commonly used dimensional scoring method. The relative merit of each alternative is determined using an additive function, which sums the performance values for each attribute. The function that yields the composite-weighted score is known as the merit function. If different attributes have different degrees of importance, appropriate weights must be applied to the performance values. The merit function is given by

$$\mathrm{MF}_i = \sum_{k=1}^{L} w_k z_{ik} \tag{14.4}$$

where i takes on values from 1 to N, MF_i is the value of the merit function for alternative A_i, w_k the weighting constant for attribute k, and z_{ik} the scaled performance value for alternative i and attribute k. It is further required that

$$\sum_{1}^{L} w_k = 1 \tag{14.5}$$

A potential problem in applying this technique is that the relative weights for each attribute may be controversial and difficult to determine. Since numerical values of the weights must be determined rather than just the order of importance of the attributes, these issues are more difficult than for the sequential method.

► **Example 14.9**

Using the same priorities presented in Example 14.7 for the sequential method, weights are assigned to each attribute in descending order of priority: short-term effectiveness (0.25), reduction of toxicity, mobility, and volume (0.225), implementability (0.2), long-term effectiveness (0.175), and cost (0.15). Using the same decision matrix as Example 14.4, the merit function values for each alternative may be computed as shown in Table 14.10. As indicated by Eq. 14.4, each merit function is the sum of products. For example, for alternative 1:

TABLE 14.10 Decision Matrix for Example 14.9

k:	Attribute					Merit Function,
Weight, w_k:	1: LTE	2: STE	3: TMV	4: IMP	5: Cost	$\sum_1^4 w_k z_{i,k}$
Alternative	0.175	0.250	0.225	0.200	0.150	
1	0.4	0.3	0.0	0.5	1.0	0.3950
2	0.6	0.3	0.25	1.0	0.75	0.5488
3	0.8	0.9	0.5	0.5	0.0	0.5775
4	0.5	0.8	0.5	0.75	1.0	0.7000

$$\begin{aligned} \mathrm{MF}_1 &= (0.175)(0.4) + (0.25)(0.3) + (0.225)(0.1) + (0.2)(0.5) + (0.15)(1) \\ &= 0.07 + 0.075 + 0.0225 + 0.1 + 0.15 \\ &= 0.3950 \end{aligned}$$

The merit function is highest for alternative 4, indicating that it is the preferred alternative using this method with these weights.

14.3.4 Methods for Decision Analysis Under Risk

A brief introduction to this highly researched topic is presented here. Returning to the decision analysis paradigm shown in Table 14.1, there are a total of N alternatives and M states of nature. Each alternative has an outcome, Z_{ij}, under each state of nature. Each state of nature has an associated probability, which may be a classical probability, a frequency, a subjective probability, or some combination of these. Multiattribute utility analysis is frequently used for this type of situation (DOE 1986). A set of utility functions is used to map each outcome to a corresponding utility value:

$$U_{ij} = F_{ij}^U \left(Z_{ij} \right) \tag{14.6}$$

For outcomes described by values for multiple attributes, a utility function is defined for each attribute:

$$u_{ijk} = G_{ijk}^U \left(z_{ijk} \right) \tag{14.7}$$

However, the utility functions $F_{i,j}^U$ and $G_{i,j,k}^U$ must possess certain mathematical properties. Frequently, simple functions known to possess the appropriate mathematical properties are used. One such function is the simple additive weighting function described in Section 14.3.3.5. An extension of Eq. 14.4 is

$$U_{ij} = \sum_{k=1}^{L} w_k z_{ijk} \tag{14.8}$$

where the z_{ijk} are defined so that preferred values are larger. It can be shown that the best alternative is the one with the largest expected utility. The expected utility for each alternative is defined as

$$\bar{U}_i = \sum_{j=1}^{M} p_j U_{ij} = \sum_{j=1}^{M} p_j \sum_{k=1}^{L} w_k z_{ijk} \tag{14.9}$$

This concept may be extended to a continuous set of future states, in which case the first summation in Eq. 14.9 becomes an integral.

▶ Example 14.10

Methods for decisions under risk are illustrated by extending Example 3.2. The event tree identifies four end states for the ammonia storage facility: no release, small gradual release, medium gradual release, and large instantaneous release. Quantification of the event tree yields probabilities for each end state based on the initiating event, as shown in Table 14.11. However, the plant operators are interested in mitigating accident consequences, so to simplify the analysis, only the end states with releases are considered in the decision. Since the no-release end state is eliminated, the probabilities are renormalized as shown in the table.

The operators of the plant are considering three alternatives to reduce consequences should a release of ammonia occur. Alternative 1 is to make no changes. Alternative 2 is replacement of the HVAC vent on the containment roof with a 20-m stack, which would reduce consequences to persons outdoors on the plant site. Alternative 3 is the purchase of residences and business on or near the site boundary, so the public exclusion zone around the plant would be extended. Attributes of interest for this decision are serious injury or death to employees (WDI), serious injury or death to members of the public (PDI), and cost. Table 14.12 provides estimates for each of these three attributes for each of the three alternatives and each of the three plant end states considered (states of nature).

The high stack reduces the number of worker deaths but does not change worker survivability inside the plant. The high stack does not affect consequences for a large release. Extending the site's boundary eliminates public deaths. Table 14.13 provides performance value estimates scaled according to Eqs. 14.2 and 14.3.

The decision maker states the following rule for determining weights for the three attributes: Public deaths and injury are weighted 10 times more heavily than worker death and injury, which is weighted nine times more heavily than cost. Hence, the weights for public deaths, worker deaths, and cost are 0.90, 0.09, and 0.01, respectively. Using these weights and the scaled performance values in Table 14.13, utilities for each alternative and end state are computed using Eq. 14.8 and are shown in Table 14.14.

TABLE 14.11 Probabilities for the Event Tree End States Shown in Example 3.2 Based on the Initiating Event

End State	Conditional Probability	Renormalized Probability
No release	0.9545	0
Small gradual release	0.045	0.989011
Medium gradual release	0.0004	0.008791
Large instantaneous release	0.0001	0.002198

TABLE 14.12 Performance Values for Three Attributes for Each Plant End State Considered and Each Alternative

	State of Nature		
	Small Gradual Release	Medium Gradual Release	Large Instantaneous Release
Probability	0.989011	0.008791	0.002198
Alternative 1: no change	WDI = 1 PDI = 0 Cost = 0	WDI = 5 PDI = 0 Cost = 0	WDI = 50 PDI = 5 Cost = 0
Alternative 2: high stack	WDI = 1 PDI = 0 Cost = $100,000	WDI = 1 PDI = 0 Cost = $100,000	WDI = 50 PDI = 5 Cost = $100,000
Alternative 3: extend site boundary	WDI = 1 PDI = 0 Cost = $1M	WDI = 5 PDI = 0 Cost = $1M	WDI = 50 PDI = 0 Cost = $1M

TABLE 14.13 Scaled Performance Values for Three Attributes for Each Plant End State Considered and Each Alternative

	State of Nature		
	Small Gradual Release	Medium Gradual Release	Large Instantaneous Release
Probability	0.989011	0.008791	0.002198
Alternative 1: no change	WDI = 0.02 PDI = 1 Cost = 1	WDI = 0.1 PDI = 1 Cost = 1	WDI = 0 PDI = 0 Cost = 1
Alternative 2: high stack	WDI = 0.02 PDI = 1 Cost = 0.1	WDI = 1 PDI = 1 Cost = 0.1	WDI = 0 PDI = 0 Cost = 0.1
Alternative 3: extend site boundary	WDI = 0.02 PDI = 1 Cost = 0	WDI = 0.1 PDI = 1 Cost = 0	WDI = 0 PDI = 1 Cost = 0

TABLE 14.14 Utilities Computed Using the Weights Specified

	State of Nature			
	Small Gradual Release	Medium Gradual Release	Large Instantaneous Release	Expected Utility
Probability	0.989011	0.008791	0.002198	
Alternative 1: no change	0.9118	0.9190	0.0100	0.90988
Alternative 2: high stack	0.9190	0.9910	0.0010	0.9176
Alternative 3: extend site boundary	0.9018	0.9090	0.9000	0.90185

The expected value of the utility for each alternative is computed according to Eq. 14.9. For example, the expected utility for alternative 1 is computed as follows

$$\bar{U}_1 = \sum_{j=1}^{3} p_j U_{1j} = (0.989)(0.9118) + (0.00879)(0.919) + (0.0022)(0.01)$$
$$= 0.90988$$

The expected utilities for alternatives 1, 2, and 3 are, respectively, 0.90988, 0.91761, and 0.90185. Based on this analysis, alternative 2 has the highest expected utility, so the high stack is built.

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PROBLEMS

14.1 Derive a five-interval weighting scale (from 0 to 1) for quantifying community attitude toward tritium releases. (Let higher numbers show worse performance.)

14.2 Consider the following unscaled performance values for two hypothetical attributes, LTC and TR.

	Unscaled Performance Value	
Alternative	LTC	TR
1	1.5	1.1
2	0.3	1.3
3	1.8	0.75
4	0.75	0.5

Normalize the performance values for each attribute to a (0,1) scale. A large number reflects a high performance value for a given attribute.

14.3 Consider the decision matrix in Table 14.15. Using the simple additive weighting (SAW) method, select the optimal alternative.

TABLE 14.15 Decision Matrix for Problem 14.3

	Attribute				
Weight:	Cost	Exposure	Feasibility	Time	Compliance
Alternative	0.25	0.25	0.1	0.2	0.2
1	0.4	0.3	0.1	0.7	1.0
2	0.6	0.3	0.3	0.9	0.75
3	0.8	0.9	0.5	0.5	0.0
4	0.5	0.8	0.5	0.7	1.0

15 Environmental Laws and Regulations

15.1 INTRODUCTION

Environmental laws and regulations are included in this textbook on environmental risk analysis, because it is in this legal and regulatory context that environmental risk analyses and risk assessments find their most frequent application. These applications of environmental risk assessment are motivated by the need to resolve environmental issues in a broad societal context. Environmental laws, regulations, guidance, and other formal constraints play a key role in both motivating and structuring environmental risk assessments.

15.2 GENERAL LEGAL AND REGULATORY STRUCTURE FOR ENVIRONMENTAL PROTECTION

15.2.1 U.S. Governmental Structure

In the United States a governmental system has been developed in which power and authority are shared among different governmental jurisdictions: the federal government, the states, counties, and cities. In addition, at each jurisdictional level, power and authority are divided among the legislative, executive, and judicial branches of government. These frequently overlapping spheres of authority are a manifestation of a system of checks and balances that serves to limit governmental authority and preserve individual liberty. However, this complex governmental structure creates a complex system for environmental protection and the application of environmental risk analysis. Most important federal environmental legislation has specific provisions for involving certain stakeholders, such as states and Indian tribes. Most of this chapter is devoted to the description of major federal environmental laws and regulations, including the mandate created by them for risk analysis and risk assessment; however, it should be recognized that state and local requirements may also mandate such analyses. Furthermore, international treaties, agreements, and standards promulgated by international agencies may require risk assessments.

A simple view of government is the following: the legislature enacts laws governing various activities affecting the environment; the executive enforces those laws

Quantitative Environmental Risk Analysis for Human Health, by Robert A. Fjeld, Norman A. Eisenberg, and Keith L. Compton

through a variety of regulatory, licensing, and enforcement activities; and the courts adjudicate a variety of issues, including the determination of guilt or innocence of parties accused by the executive of violating environmental laws. Risk analysis is used to support environmentally related activities of all three major branches of government. It is used to identify environmental problems and the need for corrective legislation. Generic risk analyses may be used by executive agencies to formulate regulations mandated by enacted legislation. Risk assessments are routinely used by regulatory agencies to support licensing decisions for regulated facilities. Frequently, risk analyses and risk assessments are incorporated into the environmental impact statements which are required to support major federal actions. Finally, risk analyses are used in court cases to support claims of noncompliance with regulations, civil liability, or criminal culpability. The assignment of responsibility for the release of contaminants to the environment and subsequent damage to individuals and ecosystems is the focus of the rapidly growing legal discipline of toxic torts.

15.2.2 Regulatory Hierarchy

At the federal level there is considerable flexibility, with corresponding variability, in the manner in which various agencies and departments implement requirements for environmental protection and associated risk analyses. Most regulatory regimes begin with passage of an act by Congress and signing it into law by the President. Once enacted, U.S. law is codified in the United States Code. Federal agencies have wide latitude in implementing federal law. A typical first step is issuance of regulations (sometimes called rules) through a public rule-making process, specified by the Administrative Procedures Act (APA 2000). Once promulgated, these regulations have the force of law and are codified in the Code of Federal Regulations (CFR). This process allows broad policies for environmental protection to be set by the Congress, while federal agencies use their technical expertise to develop regulations implementing those policies. Agencies with specific regulatory authority (e.g., EPA, Nuclear Regulatory Commission, and Federal Energy Regulatory Commission) may engage in an additional step of licensing facilities (e.g., chemical plants, nuclear power reactors, liquefied natural gas terminals). Licensing a facility usually consists of the federal agency evaluating a license application, which may contain a risk analysis or risk assessment, and subsequently, inspecting the operational safety of the facility. As a further step in regulation, a federal agency may issue guidance, which provides detailed instructions on acceptable methods for demonstrating compliance with specific regulatory provisions. For example, several guidance documents developed by the EPA for Superfund risk assessments are noted in Section 15.4.

15.3 MAJOR FEDERAL ENVIRONMENTAL LAWS AND REGULATIONS

Federal law is often enacted to solve a particular problem or to respond to the concerns of a particular group of stakeholders. Laws enacted in this fashion to address specific issues have the potential for establishing overlapping authority

and/or diverse, possibly conflicting goals among several federal agencies. Federal agencies with overlapping authority for environmental matters frequently reduce the resultant conflicts through an agreement called a memorandum of understanding. For example, five separate laws require the Department of Transportation to regulate the transport of radioactive material, and three additional laws require the Nuclear Regulatory Commission to regulate certain aspects of the transport of radioactive material. To avoid conflicting regulations, the two agencies developed a memorandum of understanding on the transportation of radioactive material (Memorandum of Understanding 1979).

Major environmental laws are presented in Table 15.1. Because environmental laws were developed and enacted to address specific problems, there is no planned structure or hierarchy among them. Stimson et al. (1993) suggests three broad classifications of laws: premanufacturing and marketing controls, pollution controls, and liability controls. Premanufacturing and marketing controls attempt to keep environmentally hazardous chemicals from being used in manufacturing or distributed to consumers. If such chemicals are prevented from entering the system, they cannot pose problems by being dispersed in the environment or abandoned as waste. Pollution controls attempt to limit the amount of contaminant released to air, water, and soil, generally through a permitting system focused either on release amounts or the concentrations in environmental media that result from a release. Liability controls attempt to assign responsibility for cleanup of contaminated sites to current or previous owners and operators. Since the cost of site cleanup can be

TABLE 15.1 Major Environmental Laws

1. National Environmental Policy Act of 1969 (NEPA); 42 U.S.C. 4321–4347
2. Chemical Safety Information, Site Security and Fuels Regulatory Relief Act Public Law 106–40, Jan. 6, 1999; 42 U.S.C. 7412(r); Amendment to Section 112(r) of the Clean Air Act
3. Clean Air Act (CAA); 42 U.S.C. s/s 7401 et seq. (1970)
4. Clean Water Act (CWA); 33 U.S.C. s/s 121 et seq. (1977)
5. Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund); 42 U.S.C. s/s 9601 et seq. (1980)
6. Emergency Planning and Community Right-to-Know Act (EPCRA); 42 U.S.C. 11011 et seq. (1986)
7. Endangered Species Act (ESA); 7 U.S.C. 136; 16 U.S.C. 460 et seq. (1973)
8. Federal Insecticide, Fungicide and Rodenticide Act (FIFRA); 7 U.S.C. s/s 135 et seq. (1972)
9. Federal Food, Drug, and Cosmetic Act (FFDCA); 21 U.S.C. 301 et seq.
10. Food Quality Protection Act (FQPA); Public Law 104–170, Aug. 3, 1996
11. Freedom of Information Act (FOIA); U.S.C. s/s 552 (1966)
12. Occupational Safety and Health Act (OSHA); 29 U.S.C. 651 et seq. (1970)
13. Oil Pollution Act of 1990 (OPA); 33 U.S.C. 2702–2761
14. Pollution Prevention Act (PPA); 42 U.S.C. 13101 and 13102, s/s et seq. (1990)
15. Resource Conservation and Recovery Act (RCRA); 42 U.S.C. s/s 321 et seq. (1976)
16. Safe Drinking Water Act (SDWA); 42 U.S.C. s/s 300f et seq. (1974)
17. Superfund Amendments and Reauthorization Act (SARA); 42 U.S.C. 9601 et seq. (1986)
18. Toxic Substances Control Act (TSCA); 15 U.S.C. s/s 2601 et seq. (1976)

substantial, this is an important cost-recovery capability for federal, state, and local governments. Some of the laws in Table 15.1 are categorized according to this scheme in Table 15.2.

Of the laws listed in Table 15.1, seven are considered to provide substantial motivation for environmental risk assessments:

1. National Environmental Policy Act (NEPA)
2. Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)
3. Resource Conservation and Recovery Act (RCRA)
4. Superfund Amendments and Reauthorization Act (SARA)
5. Toxic Substances Control Act (TSCA)
6. Clean Air Act (CAA)
7. Clean Water Act (CWA)

Each of these laws and the type of risk assessment mandated are described in the following sections.

15.3.1 National Environmental Policy Act

The National Environmental Policy Act (NEPA) of 1969 was signed into law on January 1, 1970. Although earlier laws focused on particular aspects of environmental protection (e.g., the Clean Air Act of 1955, the Solid Waste Disposal Act of 1965), NEPA declares protection of the environment to be national policy. The policy goals stated in NEPA include "[to] encourage productive and enjoyable harmony between man and his environment; to promote efforts which will prevent or eliminate damage to the environment and biosphere and stimulate the health and welfare of man; to enrich the understanding of the ecological systems and natural resources important to the Nation." In the context of quantitative risk analysis for human health, it should be noted that NEPA specifically states as policy: "Attain the widest range of beneficial uses of the environment without degradation, risk to health or safety, or other undesirable and unintended conse-

TABLE 15.2 Environmental Laws Categorized According to Main Function

Premanufacturing and marketing control
Toxic Substances Control Act (TSCA)
Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Pollution control
Clean Air Act (CAA)
Clean Water Act (CWA)
Resource Conservation and Recovery Act (RCRA)
Safe Drinking Water Act (SDWA)
Liability control
Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)
Superfund Amendments and Reauthorization Act (SARA)
Emergency Planning and Community Right-to-Know Act (EPCRA)

quences." However, NEPA defines environmental protection very broadly to include protection of aesthetics, history, and culture.

The main mechanism articulated by NEPA for achieving environmental policy goals is the requirement that planning, decision making, and taking action by federal agencies consider environmental impacts. Thus, NEPA requires that federal agencies prepare Environmental Impact Statements (EISs) on those federal actions significantly affecting the quality of the human environment. NEPA also creates the Council on Environmental Quality (CEQ) within the Executive Office of the President. The CEQ assists the President by reviewing the environmental programs at federal agencies and by assisting in the preparation of the annual Environmental Quality Report submitted to Congress.

Environmental Impact Statements summarize all the foreseeable environmental impacts of a particular major federal action such as granting an operating license for an industrial facility or opening federal land for mineral exploration. Impacts may be described in qualitative or quantitative terms. An EIS is required by law to state in detail: "(i) the environmental impact of the proposed action, (ii) any adverse environmental effects which cannot be avoided should the proposal be implemented, (iii) alternatives to the proposed action, (iv) the relationship between local short-term uses of man's environment and the maintenance and enhancement of long-term productivity, and (v) any irreversible and irretrievable commitments of resources which would be involved in the proposed action should it be implemented." Although NEPA mandates consideration of environmental impacts and documentation of a decision, it does not require adoption of a particular decision-making strategy, such as minimizing adverse impacts. Federal agencies are given broad latitude to make decisions based on a variety of factors.

Although risk assessments and risk analyses are not required by NEPA, they are frequently performed as part of the NEPA process and are used to support an Environmental Impact Statement. For facilities such as fossil-fueled power plants or landfills which routinely release contaminants to the environment during normal operations, risks to members of the public in the vicinity of the facility are usually estimated. For those facilities, such as nuclear power plants or oil refineries, where accidents may have consequences beyond the boundary of the site, consideration of public safety and health risks are usually estimated.

▶ Example 15.1

Draft and Final Environmental Impact Statements (DOE 1999, 2002) were prepared for the disposal of high-level radioactive waste at the proposed Yucca Mountain Repository. These EISs estimate many impacts, including impacts to land use and ownership, air quality, hydrology, biological resources and soils, cultural resources, occupational and public health and safety, and socioeconomic impacts. A dose and risk assessment was performed to estimate radiological doses and health impacts to the public during the repository operational (preclosure) phase, consisting of construction, operation, monitoring, and closure.

The major radionuclide released during the operation period is expected to be ^{222}Rn, a naturally occurring radionuclide that is released from rock in the underground excavation and brought to the surface by repository ventilation. The amount

of ^{222}Rn released from the subsurface facility depends on the temperature of the repository, because a cooler repository must be designed with more spacing between waste canisters, hence more excavated volume and greater releases. Depending on the repository temperature, the release of ^{222}Rn from the excavation is estimated to range from 0.78 to 1.7 PBq (21,000 to 46,000 Ci) over a 24-year period of operations. In addition, fuel-handling operations release a small quantity of ^{85}Kr; however, the radiological impact from this release is dwarfed by that from ^{222}Rn.

To estimate the consequences of these radioactive noble gases, DOE has used the GENII computer code (Napier et al. 1988, 2004). For this study, dispersion factors were calculated based on a joint frequency distribution of wind speed, wind direction, and stability class for the five-year period from 1993 to 1997.

DOE estimated doses from these releases to a maximally exposed individual (MEI) located at the site boundary (about 20 km from the releases). DOE also estimated a population dose for a population of about 76,000 people located within 80 km (50 miles) of the repository. The dose to the MEI is estimated to be 0.12 to 0.20 mSv for the nominal 24-year period of operation, depending on the thermal regime. The dose to the MEI was also estimated for the case where the waste was aged for an additional 26 years (increasing the operation period to 50 years). For this case the dose to the MEI is estimated to be 0.43 mSv. The integrated population dose is estimated to be 2.3 to 3.9 person-Sv for the nominal 24-year period of operation, increasing to 8.3 person-Sv for the waste-aging scenario.

15.3.2 CERCLA and SARA

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) was enacted by Congress on December 11, 1980. CERCLA, commonly known as the Superfund Act, has been amended by the Superfund Amendments and Reauthorization Act (SARA), 1986. The main focus of CERCLA is the cleanup of hazardous waste sites; however, some aspects of CERCLA require the federal government to respond to and effect the cleanup of any releases of hazardous substances. CERCLA may be described by four main elements: (1) identification of hazardous waste sites and prioritization of these sites for cleanup, (2) establishment of federal authority to respond to spills of hazardous substance and to remediate contaminated sites, (3) establishment of liability of persons responsible for releases of hazardous waste or hazardous substances to the environment, and (4) creation of a trust fund to finance remediation of abandoned waste sites.

The trust fund was financed by a newly created tax on the chemical and petroleum industries which generated $1.6 billion during the first five years. The law authorizes two types of response actions: short-term removals for emergency situations requiring prompt response and long-term remedial response actions. The National Contingency Plan includes guidelines and procedures for response and the National Priorities List (established by the Act), which is a list of Superfund sites with a priority for remediation. Under the act, EPA may require responsible parties to clean up contaminated sites through a variety of enforcement measures. In the event that responsible parties cannot be identified, no longer exist, or fail to act, the EPA may arrange for cleanup; however, EPA is empowered to attempt cost recovery, whenever possible, from responsible parties.

► Example 15.2

Superfund sites have been designated in every state. EPA provides information on the location and nature of these sites as well as information on planned remediation activities. On its Web pages, EPA has links to Superfund sites (current and delisted sites) for each state. As an example, Figure 15.1 is a map of Superfund sites for the state of Idaho.

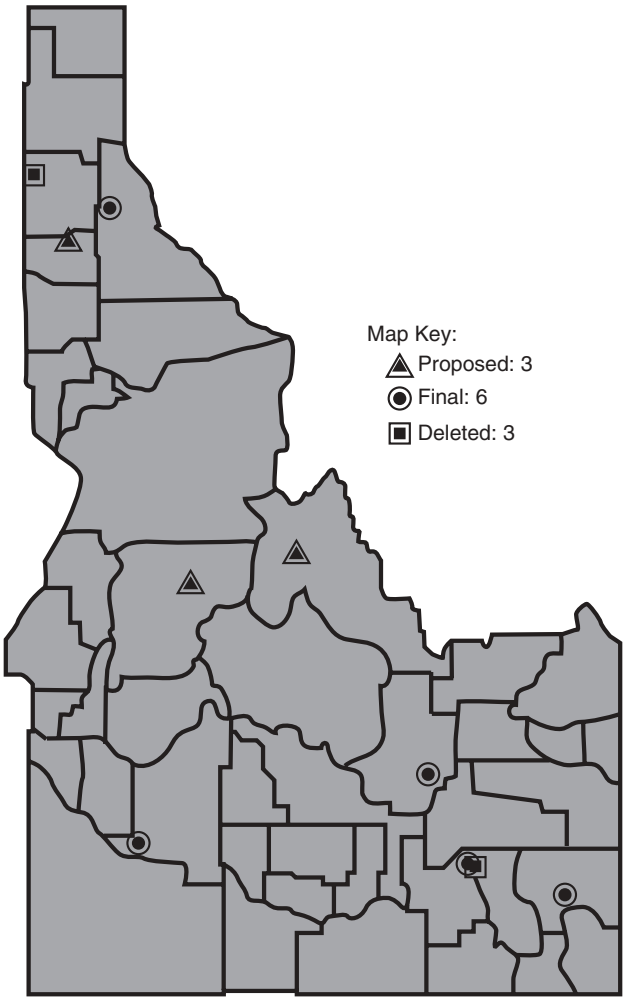

Figure 15.1 Superfund sites in Idaho as of October 8, 2004.

Environmental remediation activities carried out under the requirements of CERCLA use the risk assessment and risk management methods described in earlier chapters as substantial inputs to decision making. EPA has developed

substantial guidance for applying risk analysis in this context. Therefore, the framework for environmental remediation under CERCLA is presented in detail in Section 15.4 as an extended example of how risk methods are applied in practice.

The Superfund Amendments and Reauthorization Act (SARA) was enacted on October 17, 1986. The SARA amendments to CERCLA were based, in part, on the experience gained from administering the Superfund programs begun in 1980. These amendments do the following:

1. Mandate public notice of pending Superfund actions, including the decision rationale and solicitation of public input on the action.
2. Authorize grants to groups of affected individuals for technical assistance.
3. Apply Superfund requirements to federal facilities.
4. Require that proposed remediation actions comply with other applicable federal and state laws and regulations.
5. State a preference for remediation actions that enhance their long-term effectiveness by reducing significantly the volume, toxicity, or mobility of contaminants at the site.
6. Provide for flexibility in reaching agreements with parties for cleanup of contaminated sites, including partial funding by the government, negotiated agreements, and consent decrees.
7. Authorize reimbursement of costs to affected local governments for temporary emergency measures, not to exceed $25,000 for a single response.
8. Require that the governing body of an Indian tribe shall be afforded substantially the same treatment as a state for many important provisions of the acts.
9. Authorize grants for states and Indian tribes to establish or enhance response and remediation capabilities.
10. Allow the trust fund to be as large as $8.5 billion.

SARA also required EPA to revise the Hazard Ranking System (EPA 1992) to ensure that it assessed accurately the relative degree of risk to human health and the environment posed by uncontrolled hazardous waste sites that may be placed on the National Priorities List (NPL).

► **Example 15.3**

The following description is derived from the Record of Decision (EPA 1987) of June 1, 1987. The 5-acre Geiger (C&M oil) site is in Charleston County, South Carolina, approximately 10 miles west of the city of Charleston and within 1 mile of tidal wetlands. In 1987, approximately 40 people lived within the immediate site vicinity. Starting in March 1969, a prior owner was permitted to incinerate waste oil at the site. Eight unlined lagoons were constructed to hold waste oil. In late 1971 in response to complaints, the South Carolina Pollution Control Authority (SCPCA) ordered the owner to cease all incineration and waste deposition

activities and to take action to prevent spillage, leakage, or seepage of oil from the site. In April 1974, in response to evidence of recent oil dumping and overflowing, the Charleston County Health Department (CCHD) ordered the site closed. In March 1982, George Geiger purchased the site. In 1983, the lagoons were filled with local soils after approval was denied to excavate and dispose of the oil-contaminated soil. Subsequently, the site was used to store equipment. The primary contaminants of concern were arsenic, toluene, organics, PCBs, and heavy metals (lead, chromium, mercury).

The remedial alternative selected and funded by EPA included extraction and on-site treatment of contaminated groundwater with discharge to an off-site stream; excavation and on-site thermal treatment of soil to remove organic contaminants; solidification and/or stabilization of thermally treated soil, if necessary, to reduce mobility of metals; and backfilling of excavated areas with treated soil, followed by grading and covering with gravel. The estimated capital cost for site remediation was about $7M.

The impacts of SARA and risk considerations on the decision are illustrated by the following quote from the full text of the ROD (EPA 1987):

> The conclusion of the above discussion is that a no-action alternative for groundwater would be out of compliance with section 121 of SARA, which requires clean-up of contaminated groundwater to levels which are protective of human health and the environment. Classification of the groundwater and the potential future use of the groundwater indicate that present contaminant levels in the groundwater are not acceptable.
>
> Indicator chemicals were used to establish cleanup goals for groundwater. Indicator chemicals were selected on the basis of which chemicals pose the greatest potential health risk at the Geiger site. These indicator chemicals include those developed in the public health evaluation. Toluene and 1,1-dichlorobenzene were included because maximum concentrations for these compounds have been established based on aquatic life chronic toxicity values.
>
> For carcinogenic contaminants, a 10^{-5} risk level was deemed appropriate for groundwater remediation. EPA's draft "Guidance on Remedial Actions for Contaminated Groundwater at Superfund Sites" (October 1986) specifies that groundwater remediation should achieve a level of protection in the 10^{-4} to 10^{-7} excess cancer risk range, with 10^{-6} being used as a point of departure. Groundwater in the contaminated surficial aquifer is not used by human receptors immediately downgradient of the site and natural attenuation will lower contaminant concentrations before groundwater migrates from the site to existing residential wells or sensitive wetlands. Therefore, a 10^{-5} risk level is sufficient for protection of human health and the environment. A higher risk level would not be acceptable because of the possibility that wells may be placed near the site. The Geiger site is in a lightly populated area, but residences are located near the site.

15.3.3 Resource Conservation and Recovery Act

The Resource Conservation and Recovery Act (RCRA), enacted in 1976, amended the Solid Waste Disposal Act of 1965. Major amendments to RCRA include the Hazardous and Solid Waste Amendments of 1984, the Federal Facilities Compli-

ance Act of 1992, and the Land Disposal Program Flexibility Act of 1996. The stated goals of RCRA include:

1. Protection of the environment and human health from waste disposal hazards
2. Reduction in waste by treatment, reuse, and recycling material
3. Cooperation among the federal, state, and local governments for better waste management
4. Enhancement of waste management practices through research, development, and training

Specific provisions to accomplish these broad goals include (1) a prohibition on future open land dumping and conversion of existing open dumps to safer facilities, and (2) promulgation of guidelines for solid waste collection, transport, separation, recovery, and disposal practices and systems.

RCRA recognized that effective waste management practices needed to control the generation of waste rather than just the disposal of wastes. Major regulatory mandates of RCRA address management of solid waste, management of hazardous waste, and regulation of underground storage tanks. CERCLA addresses unused and abandoned hazardous facilities, while RCRA focuses on current and proposed facilities. As a preventive measure, the Hazardous and Solid Waste Amendments of 1984 prohibits future land disposal of untreated hazardous wastes and requires timely closure of existing facilities of this type.

15.3.4 Toxic Substances Control Act

The Toxic Substances Control Act (TSCA), enacted October 11, 1976, gives EPA the authority to regulate the manufacture, importation, distribution, and use of the large number of industrial chemicals in the United States. EPA evaluates industrial chemicals for their potential risk to human health or the environment. Users of potentially harmful chemicals may be required to perform tests to better define their risk. If EPA determines that a chemical poses an unreasonable risk, it is authorized to ban the manufacture, processing, use, disposal, or import of that chemical. TSCA also requires EPA to track the development of new industrial chemicals or new substantial uses of industrial chemicals. If such developments pose an unreasonable risk, EPA is required to restrict the use of such chemicals. TSCA supplements other federal statutes, including the Clean Air Act and the Toxic Release Inventory under the Emergency Planning and Community Right-to-Know Act (EPCRA).

► Example 15.4

Early on, EPA identified aniline compounds as a concern for health effects in workers and the public. Aniline compounds are used to make dyes and are a feed material for a large number of organic chemical products, including isocyanates, rubber processing chemicals, hydroquinone (used to manufacture drugs),

herbicides, synthetic fibers, and photographic chemicals. In 1988 EPA issued "Testing Consent Orders" (EPA 1988) for aniline and seven substituted anilines (anilines in which halogen atoms are substituted for hydrogen atoms) pursuant to its authority under TSCA. The Testing Consent Order mandates testing for various categories of effects, depending on the compound. Testing includes the following types of effects: chronic health effects with emphasis on blood and nervous system disorders, teratogenicity, carcinogenicity, mutagenic effects, epidemiology studies, and chemical fate and environmental effects testing. The testing requirements for aniline (CAS: 62-53-3) were withdrawn July 27, 1994 (53 FR 31804). However, testing results for various aniline compounds have been compiled and listed on their Web site (EPA 2005a). Table 15.3 is a partial listing of the results available for aniline.

15.3.5 Clean Air Act

The Clean Air Act (CAA) was originally passed in 1970, although it amended previously passed laws aimed at controlling air pollution. The main provisions of the 1970 CAA are:

1. EPA must promulgate National Ambient Air Quality Standards for contaminants identified as harmful to human health or the environment. Standards are required for six common air pollutants: (1) carbon monoxide, (2) nitrogen dioxide, (3) sulfur dioxide, (4) ozone, (5) lead, and (6) particulates with an aerodynamic diameter less than or equal to 10μm.
2. EPA must establish two types of air quality standards: primary standards to protect human health and secondary standards to protect the public welfare.
3. The use of lead as an additive in gasoline must be discontinued.
4. New stationary sources of air pollution must meet strict emission limitation standards.
5. Mobile sources of air pollution (vehicles, such as automobiles) must limit emissions of contaminants.

Amendments to the CAA in 1977 extended the deadlines for meeting certain standards. Major amendments to the CAA were passed in 1990 to reduce emissions causing acid rain, set deadlines for reduced automotive emissions, encourage the use of low-sulfur coal and alternative fuels, require the use of "best available control technology" to reduce toxic emissions, encourage reduction in the use of chlorofluorcarbons to prevent ozone depletion, and encourage state cooperation to address regional air pollution problems. Listed in Table 15.4 are the standards for the seven air pollutants for which ambient air quality limits have been established.

15.3.6 Clean Water Act

The Clean Water Act (CWA) was passed originally in 1972. It supplemented previously passed laws aimed at controlling water pollution. The main goal of the 1972

CWA is "to restore and maintain the chemical, physical and biological integrity of the nation's water." The main provisions of the 1972 CWA and its major amendments of 1977 and 1987 are:

1. Establishes the National Pollutant Discharge Elimination System (NPDES), a permitting system, as the basic mechanism for regulating water discharges of pollutants
2. Reaffirms requirements for states to set water quality standards for all surface waters under their jurisdiction
3. Establishes penalties for violations of NPDES permits
4. Requires states to list bodies of water with impaired quality and plan for their restoration
5. Establishes the requirement that waters not be further degraded
6. Establishes a program to control nonpoint sources of water pollution
7. Provides funding activities for specific state water pollution control activities

15.4 CERCLA PROCESS

Extensive documentation has been prepared by the EPA to describe Superfund activities, methods, and guidance. For example, documents describe a history of the Superfund program (EPA 2000), guidance for human health evaluation (EPA 1989), and guidance for conducting probabilistic risk assessments (EPA 2001).

15.4.1 Remedial Actions Under CERCLA

The remedial process under CERCLA consists of 13 steps (EPA 2002):

1. Discovery
2. Preliminary assessment/site investigation
3. Proposed listing on the National Priorities List (NPL)
4. Final listing on the NPL
5. Remedial investigation/feasibility study (RI/FS) begins
6. FS completion and proposed plan
7. Notice and comment on consent decree (if needed)
8. Pre-record of decision (ROD) significant changes (if needed)
9. ROD
10. Post-ROD significant changes (if needed)
11. Remedial design/remedial action
12. Operation and maintenance
13. Proposed NPL deletion and final NPL deletion in the *Federal Register*

The main steps of concern in the context of this textbook are steps 2 and 5, in which risk assessment plays a significant role, and step 6, in which risk assessment

TABLE 15.3 Selection of Results of Aniline Testing

Chemical Name	CAS No.	Study Code/ Type	Protocol/ Guidelines	Species
4-Nitroaniline	100-01-6	HEGTOXCHRM: mammalian bone marrow micronucleus assay	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	Mice
4-Chloroaniline	106-47-8	HEGTOXCHRM: mammalian bone marrow micronucleus assay	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	Mice
Aniline	62-53-3	EEATOX: acute aquatic invertebrate toxicity	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	*Gammarus fasciatus* (amphipod)
	62-53-3	EECTOX: chronic aquatic toxicity—crustacean	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	*Daphnia magna*
	62-53-3	EECTOX: chronic aquatic toxicity—crustacean	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	*Daphnia magna*
	62-53-3	HEGTOXCHRM: mammalian bone marrow micronucleus assay	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	Mice
2-Nitroaniline	88-74-4	HEGTOXCHRM: mammalian bone marrow micronucleus assay	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	Mice
2-Chloroaniline	95-51-2	EEATOX: acute fish toxicity	Non-TSCA Protocol/Guideline (docket OPTS-42054B)	Rainbow trout

Source: EPA 2006.

TABLE 15.3 *Continued*

Exposure	Dose/ Concentration	No. per Group	Results	Reference
Intraperitoneal injection, 2×, 24 hours apart	0, 80, 400, 800 mg/kg/day	5 to 6/sex	No evidence of clastogenicity was found in any treatment group.	54 FR 42034; 10/13/89 OTS0532109
Oral (gavage), single dose	0, 50, 100, 200 mg/kg body weight	5 male; 5 female	The incidence of micronucleated polychromatic erythrocytes in the test animals treated with 4-chloroaniline were within normal range. The number of normochromatic erythrocytes containing micronuclei was not increased. The ratio of Polychromatic to normochromatic erythrocytes in both male and female test animals remained unaffected. Results indicated that the test material was not mutagenic.	53 FR 45385; 11/9/88 OTS0519119
Flow-through, 96 hours	0.18, 0.38, 0.70, 1.4, 2.7 mg/L (measured)	20 (10/replicate)	Exposure of the test animals to the test material (aniline) resulted in a 96-hour LC_{50} of 2.3 mg/L (1.9 to 3.1 mg/L). The no-observed-effect concentration (NOEC) based on survival was 1.4 mg/L.	54 FR 25167; 6/13/89 OTS0519116
Flow-through, 21 days	0.006–0.040 (measured)	20 (10/replicate)	No effects were noted at 0.016 mg/L. At 0.027 mg/L and higher, reproduction was significantly decreased as compared to controls. The maximum allowable toxicant concentration (MATC) was 0.021 mg/L.	54 FR 33772; 8/16/89 OTS0532105
Flow-through, 21 days	0.006–0.040 mg/L	20 (10/replicate)	Decreased reproduction occurred at 0.027 mg/L and higher. No effects were noted at 0.016 mg/L. The MATC was 0.021 mg/L, measured concentration.	54 FR 33773; 8/16/89 OTS0532105
Intraperitoneal injection, 2×, 24 hours apart	0, 30, 100, 300 mg/kg/day	3/sex	Increased incidence of micronucleated polychromatic erythrocytes were seen in the high-dose groups for both sexes.	54 FR 33773; 8/16/89 OTS0532103
Intraperitoneal injection, 2×, 24 hours apart	0, 50, 250, 500 mg/kg/day	5 to 6/sex	No evidence of clastogenicity was noted in any dose group.	54 FR 42034; 10/13/89 OTS0532108
Flow-through, 96 hours	0.30, 0.58, 1.1, 2.0, 4.3 mg/L (measured)	20 (10/replicate)	The test material had an LC_{50} value (and a 95% confidence limit) of 1.0 mg/L (0.82 to 1.4 mg/L). Altered body coloration and erratic swimming were noted.	54 FR 25167; 6/13/89 OTS0519118

TABLE 15.4 Standards for the Seven Air Pollutants for Which Ambient Air Quality Limits Have Been Established

Pollutant	Primary Standard	Averaging Time	Secondary Standard
Carbon monoxide	9 ppm (10 mg/m^3)	8-h[a]	None
	35 ppm (40 mg/m^3)	1-h[a]	None
Lead	1.5 μg/m^3	Quarterly average	Same as primary
Nitrogen dioxide	0.053 ppm (100 μg/m^3)	Annual[b]	Same as primary
Particulate matter			
PM_{10}	50 μg/m^3	Annual[c]	Same as primary
	150 μg/m^3	24-h[a]	
$PM_{2.5}$	15.0 μg/m^3	Annual[d]	Same as primary
	65 μg/m^3	24-h[e]	
Ozone	0.08 ppm	8-h[f]	Same as primary
Sulfur oxides	0.03 ppm	Annual[b]	—
	0.14 ppm	24-h[a]	—
	—	3-h[a]	0.5 ppm (1300 μg/m^3)

[a] Not to be exceeded more than once per year.
[b] Arithmetic mean.
[c] To attain this standard, the 3-year average of the weighted annual mean PM10 concentration at each monitor within an area must not exceed 50 μg/m^3.
[d] To attain this standard, the 3-year average of the weighted annual mean PM2.5 concentrations from single or multiple community-oriented monitors must not exceed 15.0 μg/m^3.
[e] To attain this standard, the 3-year average of the 98th percentile of 24-hour concentrations at each population-oriented monitor within an area must not exceed 65 μg/m^3.
[f] To attain this standard, the 3-year average of the fourth-highest daily maximum 8-hour average ozone concentrations measured at each monitor within an area over each year must not exceed 0.08 ppm.

and decision-making methods play a significant role. The process starts when the existence of a site is discovered. A preliminary assessment of the potential for harm from the site is prepared based on review of land-use records and a limited site investigation. Based on this assessment, the site is evaluated for inclusion on the National Priorities List (NPL), primarily through the use of the Hazard Ranking System, a risk-based scoring system (EPA 1992). EPA conducts a rule making to enter a site on the NPL.

Once a site has been listed, a short-term removal process may be undertaken to prevent, minimize, or mitigate damage to the public or the environment when a release has occurred or a release is likely. The remedial investigation and feasibility study (RI/FS) process is the long-term response to site contamination and the threat it poses. Important activities in the RI/FS process are project scoping, data collection, risk assessment, treatability studies, analysis of alternatives, and identification of applicable or relevant and appropriate requirements (ARARs). ARARs are requirements, criteria, and regulations imposed by state or federal law. The remedial investigation is a structured assessment process to characterize site contamination and to identify, evaluate, and select appropriate remedial action alternatives. The feasibility study is a management process to define and analyze alternatives, cleanup approaches, and desired end states against nine specified evaluation criteria. The RI and FS are related, are contemporaneous, and result in

a picture of existing conditions at the site and an array of alternative cleanup options. Risk assessment methods play a significant role in the RI/FS process by developing the baseline risk assessment for the unremediated site and estimates of effectiveness for potential remediation alternatives.

After the RI/FS has been completed, a single preferred alternative is selected for implementation and publicly announced as a proposed plan. After receiving public comment on the plan proposed, a final decision on the appropriate remedial action is made and documented in the Record of Decision (ROD). Detailed planning on the remediation strategy is carried out and the strategy is implemented in the remedial design/remedial action step, resulting in final action to remediate environmental contamination at the site. After the remedial action has been initiated, the action is reviewed at least every five years while hazardous contaminants remain at the site to determine whether the remedial action selected is still sufficiently protective of human health and the environment.

15.4.2 Risk Assessment in the RI/FS Process

The RI/FS process has two major objectives: characterization of the nature and extent of risks posed by uncontrolled hazardous waste sites and evaluation of potential remedial options. These objectives are realized in a three-step process consisting of scoping activities, a remedial investigation, and a feasibility study. In the remedial investigation, the site is characterized and treatability investigations are performed. In the feasibility study, remedial alternatives are developed, screened, and analyzed.

A central element of the remedial investigation is development of a baseline risk assessment to document the risks from current conditions at the unremediated site. The baseline risk assessment process under CERCLA consists of four steps:

1. *Contaminant identification*. Contaminant identification consists of the process of selecting contaminants of concern (COCs). Selection of a contaminant as a COC can be based on its inherent toxicological properties, the amount or extent of the contamination at the site, or identified or potential contamination of critical exposure pathways. The assessment process can sometimes be simplified by the selection of indicator chemicals. These are chemicals that are representative of a larger set of chemicals on the basis of similar properties of toxicity, mobility, and persistence. As such, they may be used as surrogates for other chemicals in the analysis.

2. *Exposure assessment*. Exposure assessment involves the process of characterizing the environmental fate and transport of the contaminants and the human exposure to these contaminants. In CERCLA, this is a three-step procedure:

   a. Characterize the exposure setting. The exposure setting is characterized by two factors: (1) the nature and extent of actual or potential contaminated media, and (2) characteristics of the potentially exposed populations, such as demographics, behavior patterns affecting exposure, spatial distribution of the surrounding population, and sensitive subpopulations such as children or subsistence farmers.

b. Identify the exposure pathways. Identification of exposure pathways involves the construction of a conceptual site model and the use of measured or calculated concentrations in environmental media.

c. Quantify the exposure. Estimates of the level of exposure (in the form of dose and dose-rate information) and the nature of exposure (e.g., exposure route and duration) are used to make quantitative estimates of exposure.

3. *Toxicity assessment*. Toxicity assessments have three elements: an evaluation of the types of health effects due to exposure to a particular chemical, the relationship between the magnitude of exposure and the magnitude of response, and a discussion of the associated toxicological uncertainties. Although procedures for performing toxicity assessments are discussed in Chapter 10, in practice, extensive original assessments are not carried out. Rather, information on the contaminants of concern obtained through a review of the literature is used to perform the toxicity assessment. The EPA also publishes summary toxicological information in two primary sources available on the Internet: the Integrated Risk Information System for chemicals (EPA 2005b) and the Health Effects Assessment Summary Tables for radionuclides (EPA 2005c).

4. *Risk characterization*. In the final phase of the baseline risk assessment, the results of the toxicity and exposure assessments are reviewed and integrated to produce an estimate of the risk posed by the unremediated site. Risks are quantified using the methods discussed in Chapter 11. Risks are first calculated for individual substances and for chemical mixtures, and these are then aggregated across different exposure pathways and different chemical sources to yield an integrated picture of the site-specific risk as well as a comparison of the risks posed by individual contaminants or sources. At this point, human epidemiological studies for populations near the site may be reviewed to provide an independent check on the risk assessment. However, unless the estimated risks indicate a very high probability of detectable health effects or unusual symptoms, it is unlikely that epidemiological studies will have the power to demonstrate low levels of risk. Furthermore, recent exposures to contaminants that cause health effects with long latency periods are unlikely to be confirmed by epidemiological studies.

15.5 ADDITIONAL REGULATIONS

The major environmental laws discussed in previous sections have led to issuance of multiple environmental regulations, principally by EPA in Title 40 of the Code of Federal Regulations. However, other laws enable or require various federal agencies to issue, enforce, and/or comply with environmental regulations. Two main situations arise. In the first, agencies with regulatory authority issue regulations assuring compliance with environmental law and standards within the sphere of influence designated for the agency. In the second, agencies that operate facilities issue regulations or internal rules (known by various designations, such as "orders") governing the facilities operated by the agency.

Agencies that issue environmental regulations include the Department of Housing and Urban Development (e.g., requiring HUD-funded projects to use sites free of hazardous material—24 CFR Part 58), the Nuclear Regulatory Commission

(environmental compliance of power reactors, licensees using radioactive material, and radioactive waste sites—10 CFR Part 50), and the Federal Energy Regulatory Commission (environmental compliance of natural gas facilities—18 CFR Part 380). Agencies that operate facilities and require that they be operated in compliance with environmental standards include the Department of Defense (e.g., operation of bases by the Army—32 CFR 650 and 651, operation of ships by the Navy Department—32 CFR 775), the Department of Energy (operates several laboratories and industrial facilities using radioactive and other hazardous chemicals—10 CFR 1021), and the Bureau of Land Management (permits some mining operations on public land—43 CFR 3460).

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PROBLEMS

15.1 For the nominal and waste aging cases mentioned in Example 15.1, what is the cancer risk corresponding to the estimated doses for the MEI?

15.2 Referring to Example 15.3, what concentrations in drinking water would correspond to a 5×10^{-5} risk for toluene and 1,1-dichlorobenzene? Assume an average adult consumption rate of 1.5 L/d.

15.3 Consider the data provided in the third row (CAS 62-53-3) of Table 15.3. Based on these data, estimate the concentration at which 10% of the population would die.

15.4 The EISs cited in Example 15.1 estimate radiation doses to workers and the public and doses from normal operations and accidents. Based on the discussion in Chapter 13, what are some of the factors that might influence the perception and evaluation of these risks?

APPENDIX A
Mathematical Tools

A.1 SPECIAL FUNCTIONS

A.1.1 Dirac Delta Function

The **Dirac delta function** is a mathematical way of representing point quantities. In the context of risk assessment, it can be used to represent emissions that occur at an instant in time (i.e., an instantaneous release) or at a point in space (i.e., a point source). The temporal delta function is defined as

$$\delta(t-a) \equiv \begin{cases} 0 & t \neq a \\ 1 & t = a \end{cases} \tag{A.1}$$

and has units of (time^{-1}). It has the property

$$\int_{t_1}^{t_2} \delta(t-a)\,dt = \begin{cases} 1 & t_1 \leq a \leq t_2 \\ 0 & \text{otherwise} \end{cases} \tag{A.2}$$

Thus, the emission rate for a release of S_0 kilograms of contaminant at time t_1 is expressed as $\dot{S}(t) = S_0\delta(t - t_1)$.

The spatial δ-function is similarly defined as

$$\delta(x-a) \equiv \begin{cases} 0 & x \neq a \\ 1 & x = a \end{cases} \tag{A.3}$$

It has units of (length^{-1}) and the property

$$\int_{x_1}^{x_2} \delta(x-a)\,dx = \begin{cases} 1 & x_1 \leq a \leq x_2 \\ 0 & \text{otherwise} \end{cases} \tag{A.4}$$

The emission rate for a release of S_0 (kg) of contaminant at location x_1 is expressed as $\tilde{S}(t) = S_0\delta(x - x_1)$.

A.1.2 Heaviside Unit Step Function

The **Heaviside unit step function** is a mathematical way to turn functions on and off at discrete times or locations. The temporal step function is defined as (Kreysig 1967; Hildebrand 2002)

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$$h(t-a) \equiv \begin{cases} 0 & t < a \\ 1 & t \geq a \end{cases} \tag{A.5}$$

The step function is a dimensionless on-switch that can be used to turn a function on at $t = a$. To represent a finite step, it is necessary to use two step functions, one with a positive sign to turn it on at the appropriate time and a second with a negative sign to turn it off at the appropriate time. For example, depicted in Figure A.1 is a finite step release which begins at t_1 and ends at t_2. Mathematically, the emission rate is expressed by $\dot{S}(t) = \dot{S}_0[h(t - t_1) - h(t - t_2)]$.

The spatial step function is similarly defined as

$$h(x-a) \equiv \begin{cases} 0 & x < a \\ 1 & x \geq a \end{cases} \tag{A.6}$$

A.1.3 Error Function and Complementary Error Function

Mathematically, the **error function** is defined as

$$\operatorname{erf}(z) = \frac{2}{\sqrt{\pi}} \int_0^z \exp\left(-z'^2\right) dz' \tag{A.7}$$

and $\operatorname{erf}(-z) = -\operatorname{erf}(z)$. The integrand in Eq. A.7 is a Gaussian distribution with $\sigma = \sqrt{2}/2$, and the error function is one-half the area of the distribution between 0 and z or, equivalently, the area between $-z$ and $+z$ (Figure A.2).

The **complementary error function** is defined as $\operatorname{erfc}(z) = 1 - \operatorname{erf}(z)$. The error function and the complementary error function are shown in Figure A.3.

A.2 LAPLACE TRANSFORMS

A.2.1 Definitions and Notation

Laplace transforms are a powerful mathematical tool that can be used to solve solve initial value problems (i.e., differential equations for which the initial conditions are specified). Laplace techniques are of particular utility in environmental transport because many environmental transport problems are of the initial value type (i.e., given concentration or emission rate at some initial time and/or some initial location, the problem is to predict concentration at elapsed times and distant locations).

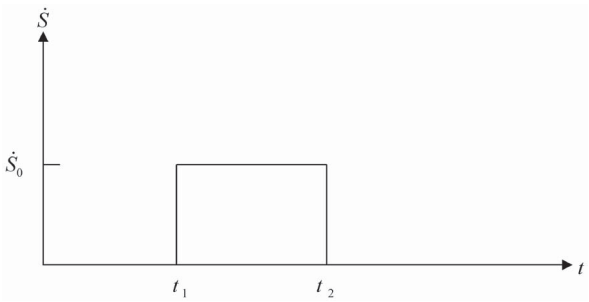

Figure A.1 Finite step release.

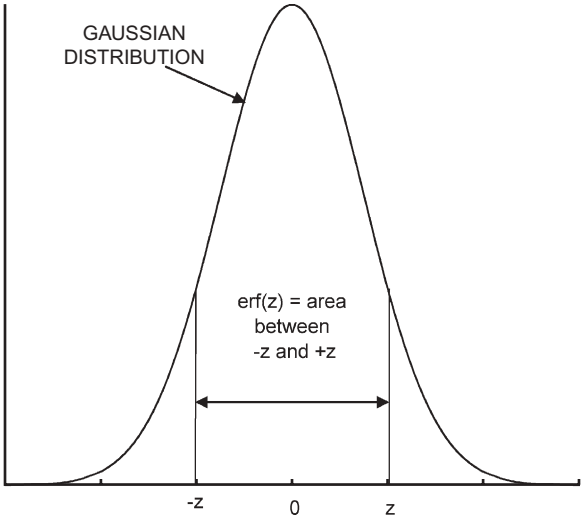

Figure A.2 Relationship between the error function and the Gaussian distribution.

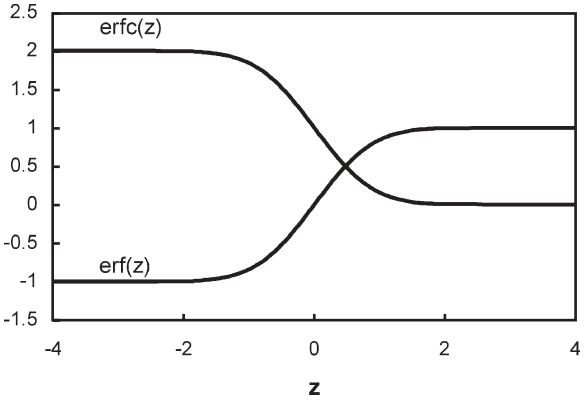

Figure A.3 Plots of the error function and the complementary error function.

In mathematics, a transform is a mathematical operation that changes a mathematical expression into another form. They are useful in that the new form of the expression may be easier to manipulate than the original form. In particular, Laplace transforms can be used to convert a differential equation into an algebraic equation. The algebraic equation can be solved for the dependent variable in transform space. Taking the inverse transform then yields the solution to the differential equation.

The Laplace transform of a time-dependent function is defined by

$$\mathcal{L}[f(t)] \equiv \int_0^\infty e^{-st'} f(t')\,dt' \tag{A.8}$$

Similarly, the Laplace transform of a one-dimensional spatial function is defined by

$$\mathcal{L}[g(x)] \equiv \int_0^\infty e^{-px'} f(x')\,dx' \tag{A.9}$$

For simplicity, the following notation is adopted:

$$\mathcal{L}[f(t)] = F(s) \qquad \mathcal{L}[g(x)] = G(p) \tag{A.10}$$

The inverse transforms are denoted by

$$\mathcal{L}^{-1}[F(s)] = f(t) \qquad \mathcal{L}^{-1}[G(p)] = g(x) \tag{A.11}$$

Transforms useful in risk assessment are included in Table A.1, and tables focused on the contaminant transport equation are given by van Genuchten and Alves (1982). Spatial transforms are obtained by simply replacing t by x and s by p.

TABLE A.1 Laplace Transforms

$f(t)$	$F(s)$	Equation Number
1	$\frac{1}{s}$	(A.12)
$\delta(t-a)$	e^{-as}	(A.13)
$h(t)$	$\frac{1}{s}$	(A.14)
$h(t-a)f(t-a)$	$e^{-as}F(s)$	(A.15)
$e^{at}f(t)$	$F(s-a)$	(A.16)
e^{at}	$\frac{1}{s-a}$	(A.17)
t^n	$\frac{n!}{s^{n+1}}$	(A.18)
$\frac{1}{\sqrt{t}}$	$\sqrt{\frac{\pi}{s}}$	(A.19)
$t^n e^{at}$	$\frac{n!}{(s-a)^{n+1}}$	(A.20)
$e^{at} - e^{bt}$	$\frac{a-b}{(s-a)(s-b)}$	(A.21)
$\sin at$	$\frac{a}{s^2+a^2}$	(A.22)
$\cos at$	$\frac{s}{s^2+a^2}$	(A.23)
$\sinh at$	$\frac{a}{s^2-a^2}$	(A.24)
$\cosh at$	$\frac{s}{s^2-a^2}$	(A.25)
$\frac{e^{-a^2/4t}}{\sqrt{\pi t}}$	$\frac{e^{-a\sqrt{s}}}{\sqrt{s}}$	(A.26)

A.2.2 Basic Transforms and Properties

$$\mathcal{L}[af(t)] = a\mathcal{L}[f(t)] \tag{A.27}$$

$$\mathcal{L}[f_1(t) - f_2(t)] = \mathcal{L}[f_1(t)] - \mathcal{L}[f_2(t)] \tag{A.28}$$

$$\mathcal{L}\left[\frac{df(t)}{dt}\right] = sF(s) - f(0) \tag{A.29}$$

$$\mathcal{L}\left[\frac{d^2f(t)}{dt}\right] = s^2F(s) - sf(0) - f'(0) \tag{A.30}$$

A.2.3 Solution of Differential Equations with Laplace Transforms

The procedure for solving differential equations with Laplace transforms is as follows:

1. Take the Laplace transform of each side of the equation.
2. Solve for the dependent variable in transform space.
3. Take the inverse of the transformed dependent variable.

The process utilizes Eqs. A.29 and A.30, where $f(0)$ and $f'(0)$ are **starting values**. In many environmental transport problems, an abrupt change occurs at $t = 0$ (or similarly, at $x = 0$). Examples would be a release beginning at $t = 0$ or occurring at $x = 0$. It is very easy to represent these changes that occur at either $t = 0$ or $x = 0$ in the contaminant transport equation, usually in the generation term. When this approach is followed, the starting values are usually zero. The advantage of this approach is that it is not necessary to figure out the initial conditions because they appear in the solution. The alternative is to specify the initial conditions as $f(0)$ and $f'(0)$.

► **Example A.1**

This procedure can be illustrated for the first-order removal model (Eq. 2.8). The differential equation is

$$\frac{dC(t)}{dt} = -kC(t)$$

with the starting value $C(0^+) = C_0$.

Taking the Laplace transform of both sides of the differential equation, we have

$$\mathcal{L}\left[\frac{dC(t)}{dt}\right] = \mathcal{L}[-kC(t)]$$

Applying Eqs. A.29 and A.27 yields

$$sC(s) - C(0) = -kC(s)$$

Substituting the starting value and solving for $C(s)$ gives us

$$C(s) = \frac{C_0}{s+k}$$

Taking the inverse transform, we obtain

$$\begin{aligned} C(t) &= \mathcal{L}^{-1}\left[\frac{C_0}{s+k}\right] \\ &= C_0\mathcal{L}^{-1}\left[\frac{1}{s+k}\right] \end{aligned}$$

Applying Eq. A.17 from Table A.1 yields

$$\mathcal{L}^{-1}\left[\frac{1}{s-a}\right] = e^{at}$$

Here $a = -k$ and

$$\mathcal{L}^{-1}\left[\frac{1}{s+k}\right] = \mathcal{L}^{-1}\left[\frac{1}{s-(-k)}\right] = e^{-kt}$$

Thus,

$$C(t) = C_0 e^{-kt}$$

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APPENDIX B
Degradation and Decay Parameters

TABLE B.1 Degradation Half-Lives of Organic Contaminants[a]

	Soil		Air		Surface Water		Groundwater	
Contaminant	High	Low	High	Low	High	Low	High	Low
Aldrin	1.6 y	3 w	9 h	1 h	1.6 y	3 w	3 y	1 d
Benzene	16 d	5 d	21 d	2 d	16 d	5 d	2 y	10 d
Carbon tetrachloride	1 y	6 m	18 y	1.8 y	1 y	6 m	1 y	7 d
Chlordane	3.8 y	9.3 m	2 d	5 h	3.8 y	9.3 m	7.6 y	1.6 y
Chloroform	6 m	4 w	8.7 m	26 d	6 m	4 w	5 y	8 w
DDD	16 y	2 y	7 d	18 h	16 y	2 y	31 y	70 d
DDE	16 y	2 y	7 d	18 h	6 d	15 h	31 y	16 d
DDT	16 y	2 y	7 d	18 h	1 y	7 d	31 y	16 d
Ethylene dibromide	6 m	4 w	4 m	11 d	6 m	4 w	4 m	20 d
Heptachlor	6 m	1 d	10 h	1 h	6 d	1 d	6 d	1 d
Heptachlor epoxide	1.5 y	1 m	2.5 d	6 h	1.5 y	1 m	3 y	1 d
Hexachlorocyclohexane	8 m	2 w	4 d	10 h	8 m	2 w	8 m	6 d
Methyl isocyanate	20 m	9 m	20 h	2 h	20 m	9 m	20 m	9 m
Tetrachloroethylene	1 y	6 m	5 m	16 d	1 y	6 m	2 y	1 y
Trichloroethylene	1 y	6 m	11 d	1.1 d	1 y	6 m	4.5 y	11 m
Vinyl chloride	6 m	4 w	4 d	10 h	6 m	4 w	8 y	8 w

Source: Howard PH (1991). *Handbook of Environmental Degradation Rates.* Chelsea, MI: Lewis Publishers.

[a] h, hours; d, days; w, weeks; m, months; y, years.

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