EPA
United States
Environmental Protection
Agency

Exposure Assessment (EXA)
Course Series

General Concepts of Exposure Assessment

Reading Packet

EXA 401



EPA's Risk Assessment Training and Experience (RATE) Program

EXA 401: General Concepts of Exposure Assessment

READING PACKET

Exposure Assessment (EXA)
Course Series

EPA's Risk Assessment Training and Experience Program

EXA 401: General Concepts of Exposure Assessment

The objectives of this course are to provide participants with a basic foundation in the concepts and principles of human exposure assessment. Participants will be introduced to the various components of an exposure assessment as well as relevant key terminology. Fundamentals that will be covered include: intake, uptake, and dose; applied, potential, internal, and biologically effective dose; acute dose, average daily dose, and average lifetime dose; and dermal, oral, and respiratory dose. This course will familiarize participants with EPA's existing and soon-to-be updated *Exposure Assessment Guidelines* and other key exposure assessment resources.

Table of Contents

List of Figures

blank
page

1. INTRODUCTION TO GENERAL CONCEPTS OF EXPOSURE ASSESSMENT

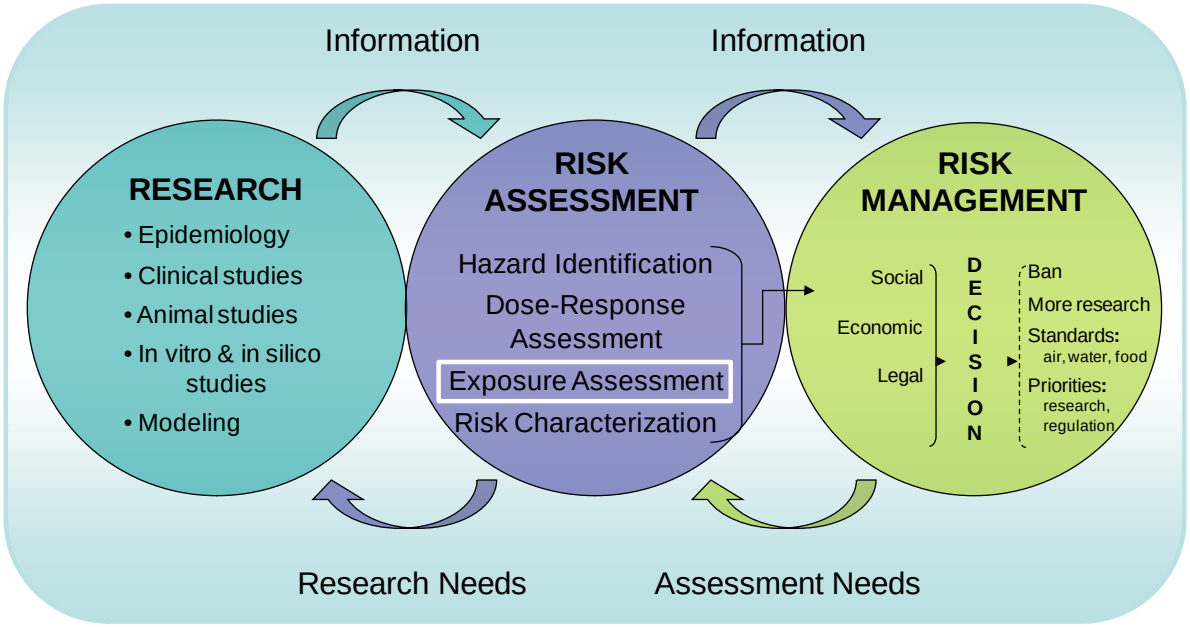
This course covers the basic concepts and principles involved in human exposure assessment. Specific topics covered in this course include:

- How exposure assessment relates to human health risk assessment
- Important elements of exposure assessment
- Introduce the concepts of uncertainty and variability in exposure assessment
- EPA resources and guidelines available for exposure assessors

Four hypothetical exposure examples will be used to illustrate how concepts and terminology related to exposure assessment are implemented.

The risk analysis paradigm is made up of three main areas: research, health risk assessment, and risk management evaluation. Exposure assessment is an important part of the paradigm and plays a key role in risk assessment.

Figure 1. Steps in the Risk Assessment Process



When a chemical or hazard is present, the possible pathways of exposure are identified and the potential effects on human health are evaluated. Potential human health risk is characterized through the synthesis of hazard identification, dose-response assessment, and exposure assessment. Human health risk can be defined as "the probability of adverse effects resulting from exposure to an environmental agent or mixture of agents" (U.S. EPA, 2003).

As stated above, risk is the probability that an adverse effect will occur. Risk is a function of both hazard (chemicals of concern) and exposure, as represented in the simplified equation below.

Hazard × Exposure = Risk

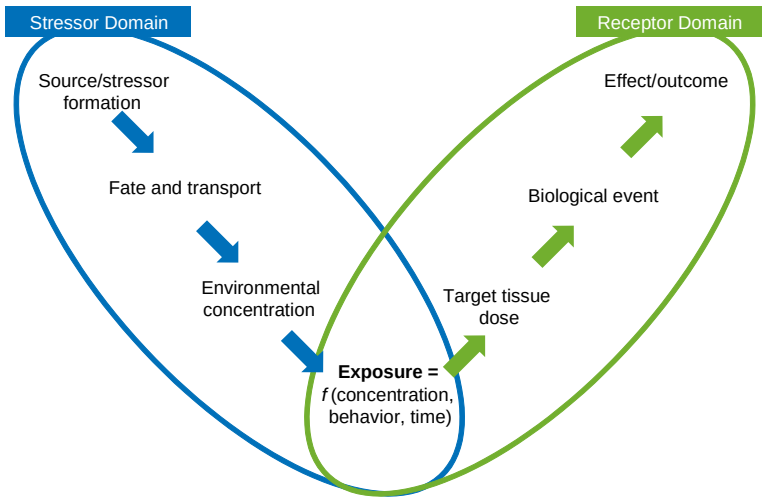
Even with a hazard present, **without exposure, there is no risk**. The fact that a chemical is hazardous does not mean that a release of that chemical from a source guarantees high risk. There may be contaminated soil from a chemical spill, but that area of soil may have been paved over. If there is no exposure because of the pavement, the exposure pathway is incomplete.

A phrase that represents this concept is "**the dose makes the poison**." This idea is first attributed to Paracelsus, a 16th century Swiss physician and chemist. He noted that all things can be poisonous if a person is exposed to an amount of the substance that produces a harmful effect. In the context of risk assessment, the amount of a toxic substance to which someone is exposed (concentration → dose) is a critical element of the equation used to estimate the harm that might occur.

Exposure assessment can be employed to inform decision-making about past and current exposures as well as potential future exposures. Implementing the risk assessment process allows the health risk to individuals or a population (i.e., human receptors) to be more accurately quantified based on the known parameters. However, there is always some uncertainty involved in estimating risk, and it is important to recognize the uncertainties that exist at every stage of the risk assessment process (NRC, 2009; IPCS, 2004; U.S. EPA, 1992b; NRC, 1983). Risk characterization based on exposure assessments ultimately informs risk management decisions, which can have substantial impacts on initiatives related to public health. These include bans or restrictions on chemicals or practices, updated standards for pollutants, and changes in research priorities or regulations.

The diagram in Figure 2 depicts a conceptual framework of exposure assessment beginning with release of the chemical from a source, chemical transport through environmental media, human exposure, and ultimately the effect or outcome that occurs from exposure.

Figure 2. Source-to-Effect Continuum (Williams et al., 2010a)



Consider exposure as the nexus between environmental contamination (i.e., the release of a toxic substance to the environment) and health effects that occur as a result of this contamination. Starting on the top left of the figure, a chemical or stressor originates from a source. Once in the environment, that chemical is subject to environmental fate and transport processes. These processes impact the amount and form of the chemical in the environment; some of the chemical may break down or a portion may become immobile or inactive. The changes that occur between the source and the site of exposure affect the resulting environmental concentration within each medium. **Exposure is therefore a function of the environmental concentration of the stressor, fate and transport processes, and time.** When exposure occurs, the chemical moves into the body of the exposed individual. The amount of the chemical that enters the body and comes into contact with a specific target organ or tissue in the body is the **target dose**, and it is dictated by interactions of the chemical with the metabolic processes of the body. When the chemical interacts with the biological target inside the body, a **biological event** can occur. Depending on the type and extent of the event created, an effect or outcome can occur. This effect might be minor—in the case of skin irritation from exposure to diluted chlorine bleach—or it might be major, such as nervous system dysfunction and shutdown due to overexposure to an organophosphate pesticide.

The exposure portion of this course will focus on the left side of Figure 2 ending at the exposure link at the bottom of the figure, with only a brief discussion on absorbed dose. The modules on health hazard further address the disposition and effects of contaminants in the body once exposure has occurred (Williams et al., 2010b; U.S. EPA, 1992b).

1.1 Exposure

Exposure is defined as contact made between a chemical, physical, or biological agent and the outer boundary of an organism. Typically, risk assessors consider exposure to occur by one of three exposure routes—inhalation, ingestion, or dermal contact. Exposure is quantified as the amount of an agent available at the exchange boundaries (also called absorption barriers) of the organism (e.g., lungs, gut, skin) (U.S. EPA, 1992b).

Exposure Assessment is the determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, and route of exposure.

The magnitude of exposure is a function of concentration, time, and behavior, as shown in the simplified equation below.

$$\text{Exposure} = f(\text{Concentration, Time, Behavior})$$

In this context, **concentration** refers to the medium-specific concentration of the substance. It is also important to consider how the concentration persists and changes over time. **Time** is the duration over which the exposure to the substance occurs. Exposure can be acute, meaning it occurs within a short time period, usually less than a day. Exposure could also be chronic, meaning it occurs either continuously or consistently over a longer time period, up to a lifetime. **Behavior** refers to how exposure to the substance occurs and the contact rate by inhalation, ingestion, or dermal contact (U.S. EPA, 1992b).

1.2 Dose

When a substance is taken into the body by one of the several routes of exposure discussed previously, the amount that gets into the body in a biologically available form is called the **dose**. Dose is defined as **"the amount of a substance available for interactions with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism"** (U.S. EPA, 2003).

There are a few different ways to measure dose:

- **Applied dose** is the amount of substance at an absorption barrier (e.g., skin, respiratory tract, stomach) that can be absorbed by the body.
- **Potential dose** is the amount of substance ingested, inhaled, or applied to skin, not all of which is actually absorbed.
- **Internal dose** is the amount of substance absorbed and available for interaction with biological receptors (e.g., organs, tissues).

The dose equation is a way to calculate dose from known or estimated data on exposure. The potential dose, absorbed dose, and internal dose can be calculated using these equations.

$$\text{Potential Dose} = \frac{C \times IR \times CF \times ED \times EF}{AT \times BW}$$

$$\text{Absorbed Dose} = \text{Potential Dose} \times AF$$

$$\text{Absorbed Dose} = \text{Internal Dose}$$

Where:

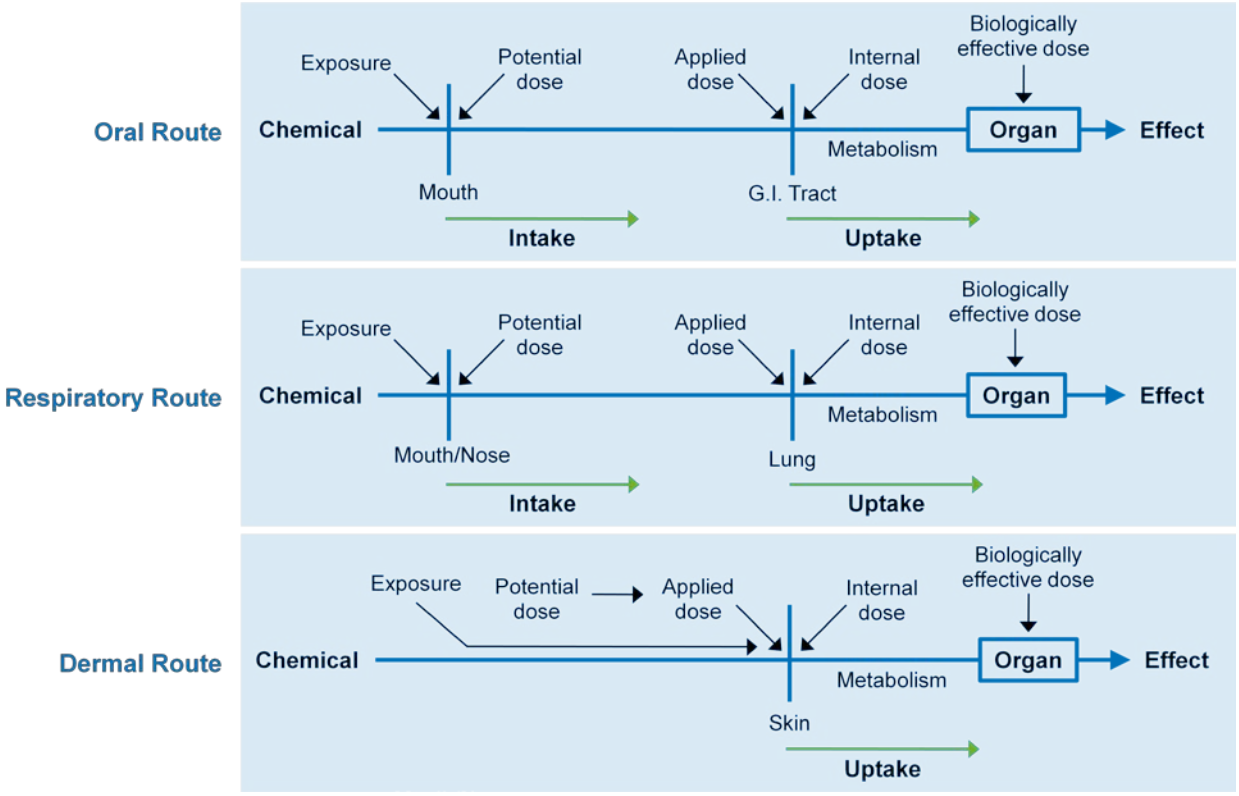
Term	Definition
C	Contaminant Concentration
IR	Intake Rate
CF	Contact Fraction
ED	Exposure Duration
EF	Exposure Frequency
AT	Averaging Time
BW	Body Weight
AF	Fraction of Potential Absorbed Dose

The **potential dose** of a contaminant is the product of the contaminant concentration, intake rate, contact fraction, exposure duration, and exposure frequency divided by the averaging time and body weight. Each of these terms is further discussed in EXA 403. The **absorbed dose** is the potential dose multiplied by the fraction of the potential dose that is absorbed by the body. The absorbed dose is also equivalent to the **internal dose**. Dose is generally expressed as mass of contaminant per unit body weight over time (e.g., mg/kg-day).

This equation is further discussed in subsequent EXA courses.

Figure 3 is based on figures included in EPA's Guidelines for Exposure Assessment (U.S. EPA, 1992b). It illustrates potential, applied, internal, and biologically effective doses. The specific meaning of these terms varies by exposure route.

Figure 3. Illustration of Dose



In terms of exposure by the **oral or respiratory route**, the potential dose is the amount of chemical that gets in the mouth or nose, and the applied dose is the amount that comes into contact with either the gastrointestinal (GI) tract or the lungs. The applied dose is usually smaller than the potential dose.

Dermal exposure routes are different from oral or respiratory exposure routes because of the absorption properties of the skin. The potential dose associated with dermal exposure involves the chemical and the matrix in which it is suspended (soil, for example) that could come into contact with the skin. It is unlikely that all of the potential dose will come into contact with the skin. The amount of chemical contained in soil that actually comes into contact with the skin is the applied dose.

In all cases, the **internal dose** is the amount of chemical that gets past the exchange boundary (e.g., skin, lungs, stomach) and into the blood, or the amount of the chemical that can interact with organs and tissues to cause biological effects (U.S. EPA, 1992b).

2. EXAMPLES OF EXPOSURE

This section uses four hypothetical exposure examples to introduce some important concepts in exposure assessment. This section will also help better define important exposure terminology.

Meet Jim. The next four sections follow Jim through a series of potential exposures to chemicals in and around his home. Jim has an older home and is retired. He spends most of his time cooking in the house or out in his garden. There are a number of ways that Jim could be exposed to chemicals in and around his home. These four specific exposure examples could occur by different exposure routes.

There are four possible hazards around Jim's home:

1) Benzene in Jim's drinking water
2) Elevated nickel and lead in Jim's garden soil
3) Smoke in Jim's kitchen
4) Pesticide residues on Jim's garden vegetables

2.1 Ingestion of Drinking Water Containing Benzene

Jim gets his water from a well on his property. Jim drinks about two liters (about a half gallon) of water per day and has done so for about 20 years. Jim's house is on a lot beside an old gas station, which has underground storage tanks that have started to degrade and leak.

Recall that an **exposure** occurs when a chemical or agent contacts the visible exterior of the person, making contact with the skin or openings into the body such as the mouth or the nose. Therefore, each time Jim drinks a glass of water from his well, he is exposed to the water and all of the potentially harmful contaminants in the water. Jim recently had his water tested, and the results showed levels of benzene above the maximum contaminant level (MCL) of 5 µg/L (U.S. EPA, 2009).

When Jim drinks the water, the process can be referred to as benzene **intake** because the substance enters Jim's body without passing through a barrier (U.S. EPA, 1992b). Intake in this situation only applies to exposure via ingestion. A related term, **uptake**, will be discussed in the second scenario.

Because Jim drinks 2 liters of water every day, as he has been doing for the last 20 years, he gets a small dose of benzene every day. In terms of exposure assessment, this would be considered a **chronic exposure**. Chronic exposures are repeated exposures by either ingestion, inhalation, or skin exposure for a period of time longer than approximately 10 percent of a person's lifespan (U.S. EPA, 2010).

The storage tank near Jim's house has not always leaked. In fact, when Jim tested his water, the inspectors estimated the tank had been slowly leaking for about seven years. To determine how much benzene Jim has

been exposed to each day, on average, the **average daily dose** (ADD) that was in Jim's water could be calculated.

In this situation, it may be difficult to determine how much benzene Jim was exposed to because it is not known exactly how long the tank has been leaking or how much benzene was in the water throughout the last seven years. However, some assumptions about benzene fate and transport and Jim's exposure can be used to estimate the ADD (in mg/kg-day) using the equation below.

$$ADD = \frac{C \times IR \times ED \times EF}{AT \times BW}$$

Where:

Term	Definition	Scenario Relevance
C	Contaminant Concentration	Concentration of benzene in Jim's water (mg/L)
IR	Intake Rate	How much water Jim consumes (L/day)
ED	Exposure Duration	How long Jim has been drinking the contaminated water (years)
EF	Exposure Frequency	How often Jim drinks water (days/year)
AT	Averaging Time	Time over which consumption is averaged (days)
BW	Body Weight	Jim's body weight (kg)

If nothing is done about the leaking tank, Jim could be exposed to benzene in his water for a long time. In cases where it is relevant to estimate the exposure to an individual or population over a lifetime, the **lifetime average daily dose** (LADD) is calculated.

The LADD can be calculated with the equation for ADD using a person's lifetime as the averaging time. Usually 80 years is assumed as an average lifespan. The LADD is commonly calculated when evaluating cancer risk because human health reference values often used to quantify cancer risk (e.g., oral cancer slope factor, inhalation unit risk estimate) are estimated with respect to a lifetime of exposure. In contrast, for noncancer risk, the averaging time is assumed to be the duration that exposure occurred. In Jim's case, this would be approximately seven years.

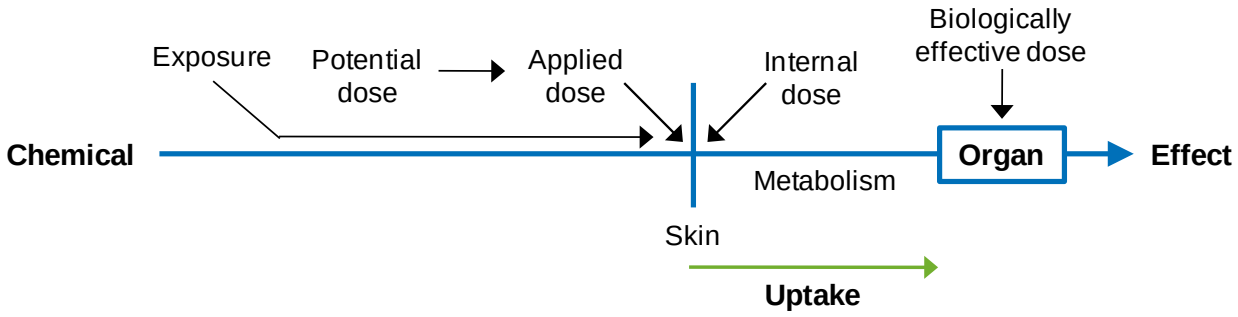
These dose equations are simplified to illustrate important exposure assessment concepts. In reality, concentration, intake, and body weight will change over time, and computer programs or modeling tools can be used to more accurately calculate these dose estimates. Exposures are not only calculated for individuals, they can also be estimated for a population, which introduces variability in these parameters. This will be discussed in detail during subsequent EXA courses.

2.2 Dermal Exposure to Soil Metals

Jim loves to garden in his backyard where he has been planting tomatoes and other vegetables for at least 20 years. He has four raised beds where he grows vegetables, and he likes to go out and get his hands dirty in the garden. Jim gardens for about nine months out of the year and rarely uses gardening gloves. Jim buys soil for his raised beds from a local mulch business, and recently found out that some of the "Garden Magic" soil that

he uses has elevated levels of nickel and lead. When Jim finishes gardening, he usually washes his hands and forearms, which are covered in dirt.

Figure 4. Illustration of Dose by Dermal Exposure Route



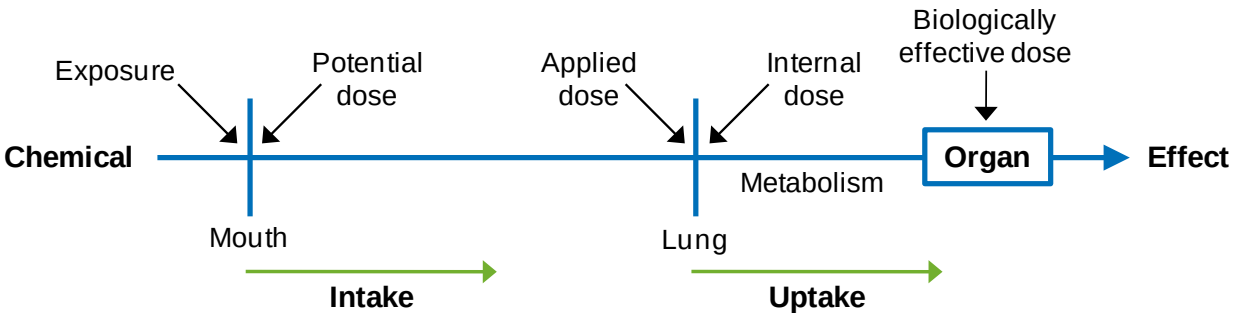
As shown in Figure 4, when Jim's skin contacts the soil, he is **exposed** to the soil and the contaminants in it. However, not all of the nickel and lead in the soil will be absorbed across the skin into Jim's body. In fact, lead is poorly absorbed across the skin (National Library of Medicine, 2010). Nickel can be taken into the body through the skin, but it is absorbed slowly (ATSDR, 2005). The soil containing nickel and lead that becomes caked on Jim's hands and arms is called the **potential dose**. The amount of nickel and lead in the soil which actually contacts Jim's skin is the **applied dose**. Not all of the nickel and very little of the lead in the applied dose will be absorbed into Jim's body. The amount of lead and nickel that crosses the skin's barrier and is absorbed into the body is the **internal dose**. Recall that the internal dose is equal to the **absorbed dose**. The process by which the contaminants are taken into the body across an absorption barrier is called **uptake**. This is contrasted with the intake of benzene associated with Jim's drinking water because the contaminant does not cross an absorption barrier before entering the body.

2.3 Inhalation of Kitchen Smoke

One of Jim's favorite things to do in the summer months is to have a homemade hamburger topped with fresh vegetables from his garden. If it is too hot or raining, Jim sometimes cooks the burgers inside instead of grilling outside. On one occasion, Jim was cooking burgers in his kitchen and went to answer the door. His neighbor had stopped by to chat. They had been talking for a while when Jim noticed the smell of smoke. He ran to the kitchen and realized the burgers were burning, and there was smoke everywhere. Jim breathed in a lot of smoke while attempting to clear the air.

Figure 5 shows that when Jim is **exposed** to the smoke, the potential dose is taken in through the mouth by **intake**, and the **applied dose** is absorbed through the lungs by **uptake**, to create an **internal dose**.

Figure 5. Illustration of Dose by Inhalation Exposure Route



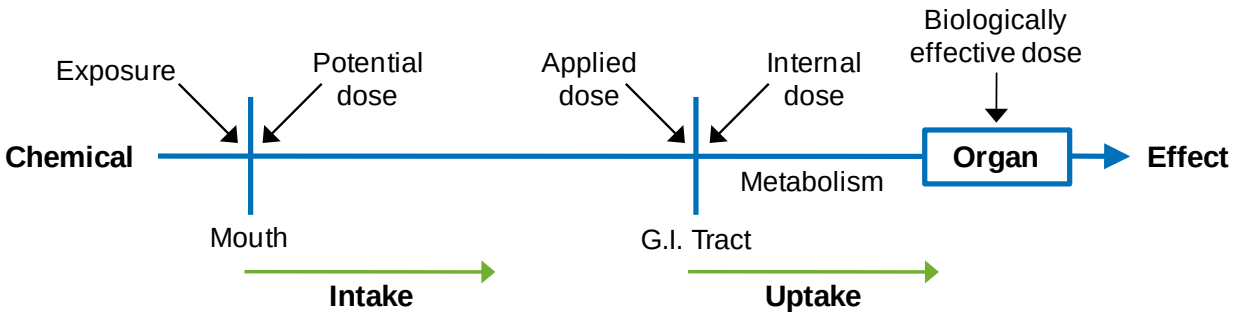
The smoke that Jim breathed in made him hoarse and slightly sick with a cough that cleared up after a few days. Jim's exposure to the smoke only lasted a few minutes, but was serious enough to affect his health at least temporarily. A short-term exposure like this is referred to as an **acute exposure**, and lasts less than 24 hours.

A wide range of contaminants might exist in smoke from cooking, including particulates, volatile compounds, and a complex mixture of combustion byproducts. This example differs from the first two in that it is not entirely clear what contaminants Jim inhaled. It is likely that Jim was exposed to all types of compounds when he breathed in the smoke.

2.4 Ingestion of Pesticide Residues

Jim loves to cook and eat homegrown vegetables, and each spring he looks forward to growing tomatoes and peppers in his garden. Jim does not usually use any pesticides, but occasionally he uses Malathion to control infestations of potato beetles. Jim loves fresh tomatoes right off the vine. He usually washes the produce once it is in the house, but has a habit of grazing on tomatoes and peppers while he is out in the garden. Sometimes he may eat two or three tomatoes while he is outside—they are just so good! Besides Jim's grazing of produce in the garden, Jim also eats store-bought vegetables that may have pesticide residues on them. Risk assessors may take into account residues on unwashed produce grown at home and purchased in the market in their risk assessments.

Figure 6. Illustration of Dose by Oral Exposure Route



As shown in Figure 6, Jim is **exposed** to the Malathion residue when he eats the food. Once the peppers and tomatoes are in Jim's stomach, the Malathion residue is absorbed by his stomach. In this case, the amount of Malathion in Jim's stomach is the **applied dose**, and the amount that makes it across his stomach lining and into his bloodstream is the **internal dose**. The applied dose is the amount of a substance that comes into contact with the absorption barriers of the body.

Malathion can affect the nervous system by interfering with the enzyme acetylcholinesterase. When Malathion reaches the nervous system from the blood, it can bind to acetylcholinesterase and prevent the nerves from firing properly. The amount of Malathion that completes the trip from the peppers and tomatoes, through Jim's stomach, and into the nervous system is the **biologically effective dose**. Biologically effective dose is defined as the amount of a substance that reaches the cells, sites, or membranes where adverse effects occur. This amount is usually much smaller than the applied dose because it has been filtered by the body, sometimes by multiple tissues.

These exposure examples involved different routes of exposure, exposure durations, sources of contaminants, and degrees of uncertainty. Each example evaluated a contaminant of concern, an exposure pathway, and factors affecting the type and extent of exposure.

The next section will focus on the elements of exposure and exposure factors.

3. EXPOSURE CONSIDERATIONS

This section focuses on important aspects of exposure that must be considered in the context of exposure assessment.

3.1 Population of Concern

Examples in this course have focused on one individual's exposure, but exposure assessment is frequently conducted for populations or groups of people. Public health professionals who conduct population-level risk assessments need data on a number of factors for the populations of concern to assess exposure accurately.

Some important characteristics of the population of interest are:

- Food and water intake rates
- Nondietary ingestions
- Inhalation rates
- Behavior patterns

It is possible that there are other factors relevant to the scenario as well. For example, immunodeficiency status could be important if the chemical of concern poses a hazard to the immune system. If the chemical of concern is a developmental hazard, then the number of women of child-bearing age would also be an important population-level exposure factor.

3.2 Elements of Exposure

The following elements of exposure must be characterized for any exposure assessment. Each of these elements was identified and discussed for the four exposure examples presented in Section 2.

- Pollutant sources
- Exposure pathways
- Contaminants
- Receptors

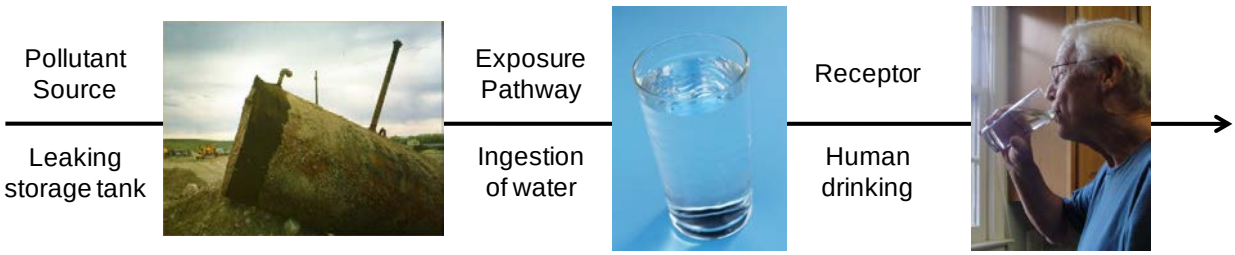
The **pollutant source** is critical to assessing exposure. To understand how individuals or a population is being exposed, one must first identify the source of contamination. Figure 7 shows that, in the first exposure example from Section 2, the pollutant source was the storage tank that was contaminating Jim's water supply.

Once the pollutant source has been identified, the specific **exposure pathways** of concern can be determined. An exposure pathway defines the process by which contaminants may come in contact with receptors. In the second example, the pathway of concern was dermal exposure to contaminated soil after Jim worked in the garden with his bare hands.

Once the exposure pathway is identified, it may be possible to determine which **contaminants** are of concern. In the first example, benzene from the leaking underground storage tank was the contaminant of concern in Jim's drinking water.

The term **receptor** can refer to an individual or population that is exposed. Characteristics of the receptors are needed in order to apply the appropriate exposure factors for quantitative exposure assessment. Our example exposure scenarios with Jim were helpful to understand exposure and some related concepts in exposure assessment. However, our discussion was limited only to one receptor—Jim.

Figure 7. Examples of Elements of Exposure



3.3 Exposure Factors

Exposure factors are quantifications of human behaviors and characteristics that affect exposure to environmental contaminants. Examples of exposure factors include body weight, skin surface area (for dermal exposure), life expectancy, inhalation rate, and ingestion rate. A detailed discussion of exposure factors is provided in EXA 406.

3.4 Uncertainty and Variability

Uncertainty and variability are important considerations for an exposure assessment. Both of these are discussed in more detail in other EXA courses, so only a brief overview is provided here and in Figure 8.

Uncertainty occurs due to a lack of knowledge stemming either from incomplete data or an incomplete understanding of a process. Uncertainty can often be reduced by collecting more data and better data (U.S. EPA, 2010). In exposure assessment, just as in other components of the risk assessment process, uncertainty is accounted for by using approximations and making assumptions.

Variability refers to heterogeneity or diversity within a data set; it is an inherent property of all data sets and it cannot be reduced or eliminated. Variability can, however, be characterized or described using more data (U.S. EPA, 2010).

One major difference between variability and uncertainty is that variability in the population may be known, but uncertainty is unknown. Although variability cannot be reduced or eliminated, it can be characterized. Uncertainty, on the other hand, exists because the information needed to completely characterize the situation is unknown.

Consider the concentration of a pollutant in a river and whether it is uncertain, variable, or both. If the concentration has not been measured, or if there is uncertainty in the measurement methods, then the

concentration is uncertain. The uncertainty can be reduced by taking more measurements or by using a better measurement method.

The concentration of the pollutant in the river might also be variable for a number of reasons. For example, the measured concentration of a pollutant in the water could vary depending on the amount of rainfall feeding into the river and flow rate at the time the river samples were collected. This variability cannot be reduced, but can be characterized and described by collecting data related to these variables during sample collection.

Figure 8. Uncertainty and Variability

Uncertainty 	**Variability** 
Lack of knowledge	Heterogeneity or diversity
Due to incomplete data or understanding of process	Inherent characteristic
Can be reduced with better data	Cannot be reduced
Is unknown and cannot be characterized	Can be characterized

The uncertainty and variability relating to one exposure factor, such as water intake rate, can impact an exposure assessment. For example, the distribution of water intake can vary within age groups or other population groups. These differences in intake can be due to participation in specific activities or due to climate pressures. Exposure factors can be used to account for this variability, but true variability of a population is difficult to capture. Variability can also include contaminant-related factors, such as contaminant concentrations in water. The contaminant concentrations can vary geographically or temporally.

Uncertainty in this situation exists in several areas, including water intake data for the population, concentration of the stressor in the media, or other exposure information for the population of concern. There may also be uncertainty about the geographic extent of the exposed population due to limited monitoring data or uncertainty about the fate and transport of the stressor in question.

3.5 Guidelines and Resources

EPA's Guidelines for Exposure Assessment were last revised in 1992. EPA is currently developing an updated version. These documents (U.S. EPA, 1992b) introduce general concepts in exposure assessment and provide guidance on planning exposure assessment. Chapters 3 and 4 describe gathering and developing data and provide details on using data to estimate exposure and dose. Chapter 5 discusses the assessment of uncertainty and variability in an exposure assessment, and Chapter 6 guides users through the presentation of results.

The Guidelines for Exposure Assessment is not the only resource to rely on when planning and conducting an exposure assessment. The remaining courses in this EXA series will cover the Exposure Assessment Guidelines as well as other key EPA resources, including but not limited to:

- EPA-Expo-Box **http://www.epa.gov/risk/expobox/**
- Exposure Factors Handbook (U.S. EPA, 2011)
- Child-Specific Exposure Factors Handbook (U.S. EPA, 2008)
- Example Exposure Scenarios (U.S. EPA, 2004a)
- Risk Assessment Guidance for Superfund (RAGS) (U.S. EPA, 2004b, 2001a, b, 1999, 1991a, b)
- Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants (U.S. EPA, 2005)
- Dermal Exposure Assessment: Principles and Applications (U.S. EPA, 1992a)

Additional resources on the topic of risk assessment are also available from EPA.

4. REVIEW

The basic concepts of this course are presented in this brief overview.

- Exposure assessment is an important part of the risk analysis paradigm and plays a key role in risk assessment.
- Exposure is a function of the environmental concentration of the stressor, fate and transport processes, and time.
- Exposure is defined as contact made between a chemical, physical, or biological agent and the outer boundary of an organism and can occur by inhalation, ingestion, or dermal contact.
- Dose is defined as the amount of substance available for interactions with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism. Dose can be measured in several ways:
    - Potential dose: Amount of substance ingested, inhaled, or applied to skin, not all of which is actually absorbed.
    - Applied dose: Amount of substance in contact with absorption barrier (e.g., skin, respiratory tract, stomach) that can possibly be absorbed by the body.
    - Internal dose: Amount of substance absorbed and available for interaction with biological receptors; equal to absorbed dose.
    - Biologically effective dose: Amount of substance that actually reaches and interacts with biological receptors.
- Some important aspects of exposure that must be considered in the context of an exposure assessment are:
    - Characteristics of the receptors
    - Elements of exposure—source of contamination, pathways of exposure, contaminants of concern, potential receptors, exposure routes
    - Exposure factors (e.g., ingestion rate, inhalation rate, body weights, activity patterns, skin surface area)
    - Uncertainty and variability at all aspects of exposure assessment

The basic concepts of exposure assessment introduced in this course will be repeated and reinforced during subsequent EXA courses and will include:

- Quantifying exposure and developing exposure scenarios to allow calculation of dose
- Following a chemical from source to receptor by exploring fate and transport and monitoring and modeling strategies
- Identifying exposure factor data and describing uncertainty and variability in exposure assessments
- Using biomonitoring data to improve confidence in exposure assessments
- Using practical case study examples—one on lead exposure and the other on dioxin

5. REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). (2005). Toxicological profile for nickel. Atlanta, GA: U.S. Centers for Disease Control and Prevention, Department of Health and Human Services. http://www.atsdr.cdc.gov/toxprofiles/tp15.pdf.

IPCS (International Programme on Chemical Safety). (2004). IPCS risk assessment terminology: Part 2: IPCS glossary of key exposure assessment terminology. Geneva, Switzerland: World Health Organization. http://www.who.int/ipcs/methods/harmonization/areas/en/ipcsterminologyparts1and2.pdf.

NRC. (1983). Risk assessment in the federal government: Managing the process. Washington, DC: National Academy Press. http://www.nap.edu/openbook.php?record_id=366&page=R1.

NRC (National Research Council). (2009). Science and decisions: Advancing risk assessment. Washington, DC: National Academies Press. http://www.nap.edu/catalog/12209.html.

U.S. EPA (U.S. Environmental Protection Agency). (1991a). Risk Assessment Guidance for Superfund (RAGS): Volume I - human health evaluation manual (part B, development of risk-based preliminary remediation goals): Interim. Washington, DC. http://www.epa.gov/oswer/riskassessment/ragsb/index.htm.

U.S. EPA (U.S. Environmental Protection Agency). (1991b). Risk assessment guidance for superfund: Volume I - human health evaluation manual (part C, risk evaluation and remedial alternatives): Interim, appendix C: Short-term toxicity values. (OSWER Dir 9285.7-01CFS). Washington, D.C. http://www.epa.gov/oswer/riskassessment/ragsc/index.htm.

U.S. EPA (U.S. Environmental Protection Agency). (1992a). Dermal exposure assessment: Principles and applications (pp. 389). (EPA/600/8-91/011B). Washington, DC: U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12188.

U.S. EPA (U.S. Environmental Protection Agency). (1992b). Guidelines for exposure assessment (pp. 139). (EPA/600/Z-92/001). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=15263.

U.S. EPA (U.S. Environmental Protection Agency). (1999). Risk Assessment Guidance for Superfund (RAGS): Volume I - human health evaluation manual, supplement to part A: Community involvement in Superfund risk assessments. (EPA/540-R-98-042). Washington, DC. http://www.epa.gov/oswer/riskassessment/ragsa/pdf/ci_ra.pdf.

U.S. EPA (U.S. Environmental Protection Agency). (2001a). Risk Assessment Guidance for Superfund (RAGS), Vol. I - Human health evaluation manual, Part D: Standardized planning, reporting and review of Superfund risk assessments. (OSWER9285747). Washington, DC. http://www.epa.gov/oswer/riskassessment/ragsd/index.htm.

U.S. EPA (U.S. Environmental Protection Agency). (2001b). Risk assessment guidance for superfund: Volume III - part A, process for conducting probabilistic risk assessment. (EPA 540-R-02-002). Washington, DC: U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. http://www.epa.gov/oswer/riskassessment/rags3adt/index.htm.

U.S. EPA (U.S. Environmental Protection Agency). (2003). Integrated Risk Information: Glossary of IRIS terms. Available online at http://www.epa.gov/iris/gloss8_arch.htm (accessed

U.S. EPA (U.S. Environmental Protection Agency). (2004a). Example exposure scenarios. (EPA 600/R03/036). Washington, DC. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=85843.

U.S. EPA (U.S. Environmental Protection Agency). (2004b). Risk Assessment Guidance for Superfund (RAGS), Volume I: Human health evaluation manual, (part E: Supplemental guidance for dermal risk assessment): Final. (EPA/540/R/99/005). Washington, DC. http://www.epa.gov/oswer/riskassessment/ragse/index.htm.

U.S. EPA (U.S. Environmental Protection Agency). (2005). Guidance on selecting age groups for monitoring and assessing childhood exposures to environmental contaminants (final) (pp. 50). (EPA/630/P-03/003F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. http://www.epa.gov/raf/publications/guidance-on-selecting-age-groups.htm.

U.S. EPA (U.S. Environmental Protection Agency). (2008). Child-specific exposure factors handbook (pp. 687). (EPA/600/R-06/096F). Washington, DC: U.S. Environmental Protection Agency, National Center for Environmental Assessment. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=199243.

U.S. EPA (U.S. Environmental Protection Agency). (2009). Drinking water contaminants. Available online at http://water.epa.gov/drink/contaminants/index.cfm (accessed August 23, 2010).

U.S. EPA (U.S. Environmental Protection Agency). (2010). IRIS site help and tools: Glossary, acronyms and abbreviations. Available online at http://www.epa.gov/ncea/iris/help_gloss.htm (accessed

U.S. EPA (U.S. Environmental Protection Agency). (2011). Exposure factors handbook 2011 edition (final). (EPA/600/R-09/052F). http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=236252.

Williams, P; Hubbell, BJ; Weber, E; Fehrenbacher, C; Hrdy, D; Zartarian, V. (2010a). An overview of exposure assessment models used by the U.S. Environmental Protection Agency. In G Hanrahan (Ed.), Modelling of pollutants in complex environmental systems (pp. 61-131). Hertfordshire, UK: ILM Publications.