DRAFT
TOXICOLOGICAL PROFILE FOR AMERICIUM

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

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UPDATE STATEMENT

Toxicological profiles are revised and republished as necessary, but no less than once every three years. For information regarding the update status of previously released profiles, contact ATSDR at:

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Division of Toxicology/Toxicology Information Branch
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FOREWORD

This toxicological profile is prepared in accordance with guidelines developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). The original guidelines were published in the *Federal Register* on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects;

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the Federal, State, and local levels; interested private sector organizations and groups; and members of the public. We plan to revise these documents in response to public comments and as additional data become available. Therefore, we encourage comments that will make the toxicological profile series of the greatest use.

Comments should be sent to:

Agency for Toxic Substances and Disease Registry
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Background Information

The toxicological profiles are developed by ATSDR pursuant to Section 104(i) (3) and (5) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund) for hazardous substances found at Department of Energy (DOE) waste sites. CERCLA directs ATSDR to prepare toxicological profiles for hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL) and that pose the most significant potential threat to human health, as determined by ATSDR and the EPA. ATSDR and DOE entered into a Memorandum of Understanding on November 4, 1992 which provided that ATSDR would prepare toxicological profiles for hazardous substances based upon ATSDR's or DOE's identification of need. The current ATSDR priority list of hazardous substances at DOE NPL sites was announced in the Federal Register on July 24, 1996 (61 FR 38451).

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel and is being made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

Jeffrey P. Koplan, M.D., M.P.H.
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QUICK REFERENCE FOR HEALTH CARE PROVIDERS

Toxicological Profiles are a unique compilation of toxicological information on a given hazardous substance. Each profile reflects a comprehensive and extensive evaluation, summary, and interpretation of available toxicologic and epidemiologic information on a substance. Health care providers treating patients potentially exposed to hazardous substances will find the following information helpful for fast answers to often-asked questions.

Primary Chapters/Sections of Interest

Chapter 1: Public Health Statement: The Public Health Statement can be a useful tool for educating patients about possible exposure to a hazardous substance. It explains a substance's relevant toxicologic properties in a nontechnical, question-and-answer format, and it includes a review of the general health effects observed following exposure.

Chapter 2: Relevance to Public Health: The Relevance to Public Health Section evaluates, interprets, and assesses the significance of toxicity data to human health.

Chapter 3: Health Effects: Specific health effects of a given hazardous compound are reported by *type of health effect* (death, systemic, immunologic, reproductive), by *route of exposure*, and by *length of exposure* (acute, intermediate, and chronic). In addition, both human and animal studies are reported in this section.

> ***NOTE:*** Not all health effects reported in this section are necessarily observed in the clinical setting. Please refer to the Public Health Statement to identify general health effects observed following exposure.

Pediatrics: Four new sections have been added to each Toxicological Profile to address child health issues:

Section 1.7 How Can (Chemical X) Affect Children?
Section 1.8 How Can Families Reduce the Risk of Exposure to (Chemical X)?
Section 3.7 Children's Susceptibility
Section 6.6 Exposures of Children

Other Sections of Interest:

Section 3.8 Biomarkers of Exposure and Effect
Section 3.11 Methods for Reducing Toxic Effects

ATSDR Information Center

Phone: 1-888-42-ATSDR or (404) 498-0110 ***Fax:*** (404) 498-0057
E-mail: atsdric@cdc.gov ***Internet:*** http://www.atsdr.cdc.gov

The following additional material can be ordered through the ATSDR Information Center:

Case Studies in Environmental Medicine: Taking an Exposure History—The importance of taking an exposure history and how to conduct one are described, and an example of a thorough exposure history is provided. Other case studies of interest include *Reproductive and Developmental Hazards; Skin Lesions and Environmental Exposures; Cholinesterase-Inhibiting Pesticide Toxicity;* and numerous chemical-specific case studies.

Managing Hazardous Materials Incidents is a three-volume set of recommendations for on-scene (prehospital) and hospital medical management of patients exposed during a hazardous materials incident. Volumes I and II are planning guides to assist first responders and hospital emergency department personnel in planning for incidents that involve hazardous materials. Volume III—*Medical Management Guidelines for Acute Chemical Exposures*—is a guide for health care professionals treating patients exposed to hazardous materials.

Fact Sheets (ToxFAQs) provide answers to frequently asked questions about toxic substances.

Other Agencies and Organizations

The National Center for Environmental Health (NCEH) focuses on preventing or controlling disease, injury, and disability related to the interactions between people and their environment outside the workplace. *Contact:* NCEH, Mailstop F-29, 4770 Buford Highway, NE, Atlanta, GA 30341-3724 • Phone: 770-488-7000 • FAX: 770-488-7015.

The National Institute for Occupational Safety and Health (NIOSH) conducts research on occupational diseases and injuries, responds to requests for assistance by investigating problems of health and safety in the workplace, recommends standards to the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), and trains professionals in occupational safety and health. *Contact:* NIOSH, 200 Independence Avenue, SW, Washington, DC 20201 • Phone: 800-356-4674 or NIOSH Technical Information Branch, Robert A. Taft Laboratory, Mailstop C-19, 4676 Columbia Parkway, Cincinnati, OH 45226-1998 • Phone: 800-35-NIOSH.

*The National Institute of Environmental Health Sciences (*NIEHS) is the principal federal agency for biomedical research on the effects of chemical, physical, and biologic environmental agents on human health and well-being. *Contact:* NIEHS, PO Box 12233, 104 T.W. Alexander Drive, Research Triangle Park, NC 27709 • Phone: 919-541-3212.

Radiation Emergency Assistance Center/Training Site (REAC/TS) provides support to the U.S. Department of Energy, the World Health Organization, and the International Atomic Energy Agency in the medical management of radiation accidents. A 24-hour emergency response program at the Oak Ridge Institute for Science and Education (ORISE), REAC/TS trains, consults, or assists in the response to all kinds of radiation accidents. *Contact:* Oak Ridge Institute for Science and Education, REAC/TS, PO Box 117, MS 39, Oak Ridge, TN 37831-0117 • Phone 865-576-3131 • FAX 865-576-9522 • 24-Hour Emergency Phone 865-576-1005 (ask for REAC/TS) • e-mail: cooleyp@orau.gov • website (including emergency medical guidance): http://www.orau.gov/reacts/default.htm

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Referrals

The Association of Occupational and Environmental Clinics (AOEC) has developed a network of clinics in the United States to provide expertise in occupational and environmental issues. *Contact:* AOEC, 1010 Vermont Avenue, NW, #513, Washington, DC 20005 • Phone: 202-347-4976 • FAX: 202-347-4950 • e-mail: aoec@dgs.dgsys.com • AOEC Clinic Director: http://occ-env-med.mc.duke.edu/oem/aoec.htm.

The American College of Occupational and Environmental Medicine (ACOEM) is an association of physicians and other health care providers specializing in the field of occupational and environmental medicine. *Contact:* ACOEM, 55 West Seegers Road, Arlington Heights, IL 60005 • Phone: 847-818-1800 • FAX: 847-818-9266.

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THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1. Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying end points.

2. Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific minimal risk levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

3. Data Needs Review. The Research Implementation Branch reviews data needs sections to assure consistency across profiles and adherence to instructions in the Guidance.

PEER REVIEW

A peer review panel was assembled for americium. The panel consisted of the following members:

1. Raymond Guilmette, Ph.D., Manager, Radiation Toxicology Program, Lovelace Respiratory Research Institute, Albuquerque, NM

2. Ronald Kathren, C.H.P., D.E.E., Professor of Pharmaceutical Science, Washington State University; President, The Kathren Group, Inc., Richland, WA

3. Raymond Lloyd, Ph.D., Professor Emeritus of Radiobiology, University of Utah School of Medicine, Taylorsville, UT

4. Melvin Sikov, Ph.D., Scientist Emeritus, Pacific Northwest National Laboratory, Richland, WA

These experts collectively have knowledge of americium's physical and chemical properties, toxico-kinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(I)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about americium and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Elevated americium levels have been found in at least 8 of the 1,585 current or former NPL sites. However, the total number of NPL sites evaluated for this substance is not known. As more sites are evaluated, the sites at which americium is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are normally exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact. However, since americium is radioactive, you can also be exposed to its radiation if you are near it.

If you are exposed to americium, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS AMERICIUM?

Americium (pronounced, ˈam-ə-ˈris(h)-ˈē-əm) is a man-made, radioactive, actinide element with an atomic number of 95. It was discovered in 1945. Actinides are the 15 elements, all of whose isotopes are radioactive starting with actinium (atomic number 89), and extending to lawrencium (atomic number 103). When not combined with other elements, americium is a silvery metal. Americium has no naturally occurring or stable isotopes. There are two important isotopes of americium, americium 241 and americium 243, also written as ^{241}Am or ^{243}Am, and read as

americium two-forty-one and americium two-forty-three. Both isotopes have the same chemical behavior in the environment and the same chemical effects on your body. However, these isotopes have different atomic weights and different radioactive properties, such as their half-life (see next paragraph) and the energy of radiation they give off. Each radioactive isotope of an element, including americium, constantly gives off radiation, and this changes it into an isotope of a different element or a different isotope of the same element. This process is called radioactive decay. ^{241}Am and ^{243}Am give off alpha particles (sometimes referred to as alpha radiation) and turn into neptunium 237 (^{237}Np) and neptunium 239 (^{239}Np), respectively. Neptunium is also radioactive, so these isotopes give off radiation and change into other elements. This process continues and eventually ends when stable isotopes of bismuth and lead are formed.

Half-life is the term used to describe the speed of the decay process. Specifically, the half-life is the time it takes for half of the atoms of an isotope to give off its radiation and change into a different isotope. The half-life of ^{243}Am is 7,370 years. The half-life of ^{241}Am is 432 years. An isotope's half-life together with its mass tell how much radioactivity there is in something, such as your body. Specific activity is the term used to tell how much radioactivity is contained in 1 gram of the material itself. If you have 1 gram of each americium isotope side by side, the ^{241}Am would be about 17 times more radioactive (i.e., its specific activity would be 17 times higher) than the ^{243}Am. ^{241}Am is of particular interest as more of it is produced from plutonium 239 (^{239}Pu) in nuclear reactors and nuclear explosions, and it has a higher specific activity than ^{243}Am. It should be noted that ^{239}Pu is produced from uranium 238 (^{238}U), the most abundant isotope of uranium in nuclear power reactor fuel, when it is exposed to neutrons that are present in nuclear reactors or when atomic bombs explode. The intermediate products are uranium 239 (^{239}U) and neptunium 239 (^{239}Np). Consequently, various uranium, neptunium, plutonium, and americium isotopes may be present simultaneously. These would have to be separated to obtain pure ^{241}Am. ^{243}Am is produced from ^{241}Am, but has to be manufactured intentionally.

Quantities of americium, as well as other radioactive elements, are measured in units of mass (grams) or radioactivity (curies or becquerels). The becquerel (Bq) is a new international unit

and the curie (Ci) is a traditional unit; both are used currently. A Bq is the amount of radioactive material in which 1 atom transforms every second, and a Ci is the amount of radioactive material in which 37 billion atoms transform every second.

In order for ^{241}Am to be produced from ^{239}Pu, there must be a source of neutrons. This is the case in nuclear reactors or nuclear explosions. The immediate precursor or parent of ^{241}Am is plutonium 241 (^{241}Pu) (half-life 14 years), which radioactively decays into ^{241}Am; this step does not require an external source of neutrons or other particles and, thus, any ^{241}Pu that has been produced will decay into ^{241}Am. Therefore, new ^{241}Am is constantly being made from the decay of ^{241}Pu. Once all of the ^{241}Pu decays, no new ^{241}Am will be produced. As mentioned above, ^{241}Am also transforms or decays into another element and gives off radiation. The element it decays into is neptunium (Np) and in the process, it gives off a particle called an alpha particle (i.e., helium atom) or alpha radiation. High energy electromagnetic radiation, known as gamma radiation (or gamma rays), is also given off. The alpha radiation cannot pass through solid objects, such as paper or human skin, but the gamma radiation is very penetrating. ^{241}Am is called the parent, and ^{237}Np is called the transformation product or daughter. When one radioisotope decays through a series of different radioactive materials, the series of reactions is called a decay chain. The series ends when a stable, nonradioactive element is formed. For more information on radiation, see Appendix D and the glossary at the end of this profile or the ATSDR *Toxicological Profile for Ionizing Radiation*.

The predominant commercial use of ^{241}Am is in ionization-type smoke detectors. This application relies on the high energy alpha particles produced when the isotope decays as an ionization source. A typical household smoke detector contains 0.9 microcuries (μCi) (33,000 Bq) of ^{241}Am (a microcurie is one millionth of a curie), and 1 gram of americium dioxide is sufficient to make 5,000 smoke detectors. ^{241}Am is also used for industrial gauging applications and in medical diagnostic devices. There are no commercial applications for ^{243}Am. For more information about the properties and uses of americium, see Chapters 4, 5, and 6.

1.2 WHAT HAPPENS TO AMERICIUM WHEN IT ENTERS THE ENVIRONMENT?

^{241}Am is a long lived, manmade radioactive chemical that is produced in nuclear reactors and nuclear explosions. Very low levels of ^{241}Am may be present in soil and water as a result of atmospheric nuclear weapons testing that predominantly took place between 1945 and 1963 when a nuclear test ban treat was enacted. As a result of these tests, ^{241}Am and its precursor, ^{241}Pu, were injected into the stratosphere and were dispersed throughout the world. These and other products of uranium and plutonium fission that were produced as a result of nuclear weapons testing are referred to as fallout products; the radiation resulting from the testing is referred to as fallout background radiation. The radionuclides and the radiation thus produced are often referred to as fallout background. In the environment, ^{241}Am will undergo radioactive decay (half-life 432 years). Since ^{241}Pu (half-life 14 years) is released along with ^{241}Am, the amount of ^{241}Am will slowly increase for centuries even in the absence of additional ^{241}Am releases. Americium released to the environment would generally occur as a chemical compound, where it is combined with other elements, such as oxygen, rather than in metallic form. In addition to radioactive decay, americium may change its chemical form in the environment or become attached to or separated from particles.

^{241}Am released to the atmosphere will be associated with particles and will settle to earth or be washed from the air in precipitation (e.g., rain, snow). ^{241}Am from atmospheric nuclear weapons tests is injected into the stratosphere and may remain in the atmosphere for decades, traveling all around the world and only slowly settling to earth. ^{241}Am released in nuclear accidents, like Chernobyl, stays in the lower atmosphere where it can begin settling out near the site from which it is released. Larger particles will settle out more quickly and over a smaller area; smaller particles may remain in the atmosphere for several months and travel far from where they are released. Precipitation scrubs particles out of the air more rapidly and deposits them in areas where the precipitation occurs.

^{241}Am released into water from nuclear facilities will stick to particles in the water or the sediment at the bottom of the body of water into which it was released, and some may be carried

along by the current. ^{241}Am deposited on soil is so strongly attached to soil particles that it does not travel very far into the ground. Ultimately, most americium ends up in the soil or sediment.

While plants may take up ^{241}Am from the soil, the amount taken up is small, especially in the parts of the plant that you eat most often like the fruit, grain, and seeds. While fish may take up ^{241}Am, very little builds up in the flesh. Most of the ^{241}Am found in shellfish like shrimp or mussels is attached to the shell, rather than in the edible parts of the animal. For more information about what happens to americium in the environment, see Chapter 6.

1.3 HOW MIGHT I BE EXPOSED TO AMERICIUM?

You may be exposed to a substance by eating food, drinking water, breathing air, or by skin contact. Children may also be exposed by eating dirt. Exposure of the general population to ^{241}Am in air, water, soil, and food is generally very low. The concentrations of ^{241}Am in these media, often referred to as fallout background levels, are what we have inherited from atmospheric nuclear testing that predominantly took place in the 1940s–1960s. They are called background levels of fallout products as radioactive substances produced by these tests have spread all over the globe. In soil and sediment, these levels will increase, reaching a maximum in about the year 2035, as ^{241}Pu decays to ^{241}Am; afterwards levels will slowly decline, with less than 1% remaining after 3,000 years.

People working at sites where nuclear waste is stored, nuclear power plants, or other nuclear facilities that handle ^{241}Am may be exposed to higher levels of americium. People living near these sites may also be exposed to elevated levels of americium. People who produce or handle ^{241}Am in smoke detectors or other devices may also be exposed to higher levels. Exposure can also occur as a result of a nuclear accident. In such cases, exposure would be by breathing or eating dust containing ^{241}Am. Even at moderately contaminated sites, enough ^{241}Am is not generally taken up into plant and animal tissue so as to be of much concern. You can find more information on how you may be exposed to americium in Chapter 6.

1.4 HOW CAN AMERICIUM ENTER AND LEAVE MY BODY?

Americium can enter your body from the air you breathe, the food you eat, the water you drink, or from contact with your skin. After you breathe in americium, some forms of americium that do not dissolve easily in water might stay in your lungs for months or years. Other forms that dissolve easily in water stay in your lungs for hours or days. Some americium that enters your lungs may get into your blood. A very small amount of americium that enters your digestive tract may also enter your blood. Most of the americium entering your blood leaves your body in your urine and feces. Of the small amount that stays in your body, most goes to your bones where it can stay for many decades. A small amount goes into your liver and other organs, where it may stay only for a few years.

1.5 HOW CAN AMERICIUM AFFECT MY HEALTH?

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body. In the case of a radioactive chemical, it is also important to gather information concerning the radiation dose and dose rate to the body. For some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

The radiation from exposure to americium is the primary cause of adverse health effects from absorbed americium. Upon entering the body by any route of exposure, americium moves relatively rapidly through the body and is concentrated in bones where it remains for a long time. While in the bone, americium atoms decay, releasing alpha particles and gamma-rays. Alpha particles travel very short distances within the body. They collide with, and give energy to,

nearby cell matter. This can cause changes in the genetic material of such cells that could result in the formation of bone cancers. The gamma-rays released by decaying americium can travel much farther before hitting cellular material, and some of these gamma-rays can even leave the body without damaging any cell matter. Exposure to extremely high levels of americium, as has been reported in some animals studies, has resulted in damage to other organs such as the lungs, liver, and thyroid. However, americium is accumulated in these organs for only a relatively short time, and it is unlikely that you would be exposed to amounts of americium large enough to cause harmful effects in these organs.

1.6 HOW CAN AMERICIUM AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans. Potential effects on children resulting from exposures of the parents are also considered.

Americium accumulates in the bone, and remains there for a long time. In the bone, americium emits radioactive alpha particles that cause damage to the surrounding tissue. The body normally repairs all of the damage, but if it fails, it could cause bone cancer after many years. If exposure occurs to children, the exposure to the radiation from americium would be for a longer time, causing a larger radiation dose over their lifetime and increasing the likelihood of producing cancer. In addition, in young children the bone is rapidly growing and this may cause americium to stay in the bone longer. Also, children may absorb more of the inhaled or ingested americium than adults. This would let the americium build up to higher levels and give a larger radiation dose to the bone. However, there are no actual data showing that children are more susceptible than adults to radiation-induced americium toxicity. A developing baby could be affected by americium while in the womb if the pregnant mother has americium in her blood.

1.7 HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO AMERICIUM?

If your doctor finds that you have been exposed to significant amounts of americium, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

It is possible that higher-than-normal levels of americium may be in soil near a nuclear waste site, nuclear reactor, or plant that manufactures ionization smoke detectors or gauges containing americium. Some children eat a lot of dirt. You should prevent your children from eating dirt. Make sure they wash their hands frequently, and before eating. If you live near a nuclear waste site, nuclear reactor, or plant that manufactures ionization smoke detectors or gauges containing americium discourage your children from putting their hands in their mouths or from engaging in other hand-to-mouth activities.

Discharge water and emission from facilities that make americium smoke detectors or gauges or produce plutonium for nuclear weapons may contain americium. These operations are strictly regulated, but you can check local health advisories before consuming fish or other seafood from these waters. Nuclear reactors are not expected to discharge measurable amounts of americium.

Ionization smoke detectors that contain americium are safe when installed. However, you should not allow your children to play with these detectors and they should be promptly returned to the manufacturer for disposal if they are damaged or no longer being used.

1.8 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO AMERICIUM?

Most physicians do not test for americium in their offices, but they can collect samples and send them to special laboratories. Since americium is radioactive, it is normally measured by its radiation emissions. These emissions are used to tell the amount of americium (in curies or Becquerels) and the radiation dose it gives to your body (in Sieverts or rem). Radiation detectors measure the radiation that is released from objects or materials, including the whole body. If

americium were to enter your body from americium-contaminated air or food, these radiation detectors could measure radiation in your blood, feces, urine, tissue samples, or teeth. Special radiation detectors could also be used to measure the radiation that leaves your whole body from americium that is inside your body.

1.9 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Food and Drug Administration (FDA), and the Nuclear Regulatory Commission (NRC).

Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR), the National Institute for Occupational Safety and Health (NIOSH), and the FDA.

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for americium include the following:

The Nuclear Regulatory Commission (NRC) established limits for levels of the americium radioisotope, ^{241}Am, in air and water that can be released from licensed facilities. The levels, in radiation units, are 0.00000000000002 microcurie per milliliter (2×10^{-14} μCi/mL) for air and

0.00000002 microcurie per milliliter ($2x10^{-8}$ μCi/mL) for water. Colorado has set a groundwater limit for ^{241}Am of 0.00000000000015 picocurie per liter ($1.5x10^{-10}$ μCi/mL). The FDA has set a food contamination limit of 2 becquerel per kilogram ($5.4x10^{-5}$ μCi/kg) for ^{241}Am to protect 3-month-old children, the most sensitive population group.

More information on regulations and guidelines is available in Chapter 8.

1.10 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department, your regional Nuclear Regulatory Commission office, or

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop E-29
Atlanta, GA 30333

* Information line and technical assistance

Phone: 1-888-42-ATSDR (1-888-422-8737)
Fax: (404) 498-0057

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

* To order toxicological profiles, contact

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: 1-800-553-6847 or 1-703-605-6000

2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO AMERICIUM IN THE UNITED STATES

Americium is a man-made radioactive actinide element; it has no stable isotopes. The only isotope used commercially is ^{241}Am, several kilograms of which are produced annually. The most common application of americium is in ionization smoke detectors, a typical home smoke detector containing about 0.9 µCi (33.3 kBq) of radioactivity. One gram of $^{241}AmO_2$ provides enough active material for more than 5,000 smoke detectors. The isotope ^{241}Am is formed when by the action of neutrons on ^{239}Pu is exposed to neutrons such as is the case in nuclear reactors and nuclear explosions. The immediate precursor is ^{241}Pu, which itself is formed when ^{238}U (the major uranium isotope used in nuclear power reactor fuel) is exposed to neutrons, as occurs in a nuclear reactor or nuclear explosion. The only other americium isotope produced in macroscopic quantity (10–100 g annually) is ^{243}Am, which is produced directly by the capture of two neutrons by ^{241}Am. Americium is also used in a wide range of industrial gauging applications.

Very low levels of ^{241}Am may be found in environmental media worldwide, a legacy of atmospheric nuclear weapons testing that predominantly took place between 1945 and 1963. In nuclear reactors, ^{241}Am has been detected in primary coolant water, stack aerosols, and waste water. Because ^{241}Am is produced from the decay of ^{241}Pu (half-life 14.4 years), releases of ^{241}Pu result in an ingrowth of ^{241}Am (decay of plutonium to americium in the sample matrix). The maximum concentration of ^{241}Am following an accidental release of ^{241}Pu occurs 70–80 years post release. Consequently, the impact of ^{241}Am from atmospheric nuclear weapons testing will reach its peak in about the year 2035. Environmental levels of ^{241}Am will subsequently decline according to the ^{241}Am rate of decay. Exposure of the general population to ^{241}Am via air, water, soil, and food are generally very low; these 'background' levels are a result of fallout from past atmospheric nuclear weapons tests. Since 1973, ^{241}Am air concentrations have been less than 1 aCi/m^3 (0.037 µBq/m^3) and are continuing to decline. Levels around nuclear power plants are indistinguishable from fallout background. ^{241}Am levels in surface seawater of the North Sea and North Atlantic Ocean have stayed around 10 Bq/m^3 (270 pCi/m^3) between 1976 and 1988, but may be considerably higher near discharges from nuclear facilities. ^{241}Am in soil resulting from cumulative deposition and ingrowth in the New York region should reach a maximum of 0.88 mCi/km^2 (33 MBq/km^2) in the year 2035. In the Food and Drug Administration (FDA) Total Diet

Studies in 1983–1986 and 1987–1992, the concentrations of radionuclides were sufficiently low as to require no specific action or simple monitoring.

2.2 SUMMARY OF HEALTH EFFECTS

No information was located regarding the potential for adverse effects in humans or animals based on heavy metal properties of americium. Reported adverse effects related to overexposure to americium appear to be the result of ionizing properties of radiation. Both ^{241}Am and ^{243}Am release alpha particles and gamma rays during decay. The radioactivity of ^{241}Am is approximately 17 times greater than that of ^{243}Am. Its higher level of radioactivity and greater abundance makes ^{241}Am of greater health concern than ^{243}Am. Due to the ionizing properties of radiation from ^{241}Am and ^{243}Am, increased cancer rates would be expected among individuals exposed to sufficiently large quantities. Such exposures are not likely among the general population, and studies of cancer risk specifically associated with exposure of humans to radioactive americium isotopes were not located. Animal studies indicate increased risk of bone cancer in areas of bone containing relatively high levels of americium. Depressed white blood cell counts have been observed in the few cases of accidental overexposure of humans and in a number of animal studies. Signs of radiation-induced damage have been observed in liver, thyroid, blood, and kidneys of experimental animals exposed to large amounts of americium that resulted in relatively high americium burdens within these tissues. Inhaled americium has also damaged the respiratory system of exposed animals.

Cancer. Animal studies demonstrate that internal exposure to the radioactive isotope, ^{241}Am, results in the development of cancer in the tissues that sequester this element. Increases in bone cancers occurred in dogs that inhaled $^{241}AmO_2$. Increased cancer incidences were noted in laboratory animals observed for a lifetime following intraperitoneal or intravenous injection of ^{241}Am. In an analysis of all soft tissue tumors in dogs exposed by intravenous injection, both positive and negative associations between exposure and tumor incidence were apparent in a variety of soft tissues, but the development of these tumors appeared to be influenced by the competing risk of death from bone cancer or other severe radiation effects. In dogs administered single intravenous injections of ^{241}Am, the incidence of death was dose-related and predominantly due to bone cancer, although the incidence of liver tumors was higher than that of bone tumors in dogs exposed to lower levels of ^{241}Am. Offspring of rats, administered a single intravenous injection of ^{241}Am on gestation day 16, developed osteosarcomas; none were observed in a control group.

Hepatic Effects. Degenerative changes in the liver have been observed in laboratory animals exposed to ^{241}Am via inhalation or intravenous injection.

Immunological and Lymphoreticular Effects. Depressed white blood cell counts have been observed in humans and animals exhibiting relatively high body burdens of ^{241}Am following accidental or experimental exposure.

Hematological Effects. Accidental exposure to ^{241}Am resulted in lymphopenia and thrombocytopenia in a single human case. Animal studies indicate that inhaled or intravenously-injected ^{241}Am may cause hematological effects that include elevated hematopoietic activity, leukopenia, decreased hematocrit and platelets, and increased cellularity in bone marrow. *In vitro* assays of bone marrow cells from mice administered ^{241}Am parenterally indicate a decreased ability to support hematopoiesis and reduced colony-forming capacity. It appears that damage to hematopoietic stem cells is more readily identified through changes in colony forming capacity than by changes in peripheral white blood cell counts.

Musculoskeletal Effects. Accidental exposure of a worker to ^{241}Am resulted in histological signs of fibrosis, bone cell depletion, and bone marrow atrophy. Degenerative changes in bone have also been observed in animals acutely exposed to ^{241}Am via inhalation and intravenous administration.

Endocrine Effects. Degenerative changes of the thyroid have been noted in dogs acutely exposed to ^{241}Am by inhalation. Severe thyroid damage has also been shown to occur following repeated intravenous injection of ^{241}Am.

Renal Effects. Animal studies indicate that degenerative changes in the kidneys may result from acute inhalation exposure to ^{241}Am. Repeated intraperitoneal injection of ^{241}Am resulted in histopathologic changes in the kidneys of rats.

Respiratory Effects. Inhalation exposure of dogs to ^{241}Am resulted in respiratory insufficiency and pneumonia as well as histopathologic changes in the lungs. Inhalation exposure of rats resulted in radiation pneumonitis.

2.3 MINIMAL RISK LEVELS

Inhalation MRLs

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for americium due to the lack of suitable human or animal data regarding health effects following inhalation exposure to americium.

Oral MRLs

No acute-, intermediate-, or chronic-duration oral MRLs were derived for americium due to the lack of suitable human or animal data regarding health effects following oral exposure to americium.

3. HEALTH EFFECTS

3.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective on the toxicology of americium. It contains descriptions and evaluations of toxicological studies and epidemiological investigations and provides conclusions, where possible, on the relevance of toxicity and toxicokinetic data to public health.

A glossary and list of acronyms, abbreviations, and symbols can be found at the end of this profile.

3.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals and others address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure (inhalation, oral, dermal, and external) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELS have been classified into "less serious" or "serious" effects. "Serious" effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). "Less serious" effects are those that are not expected to cause significant dysfunction or death, or those whose significance to the organism is not entirely clear. ATSDR acknowledges that a considerable amount of judgment may be required in establishing whether an end point should be classified as a NOAEL, "less serious" LOAEL, or "serious" LOAEL, and that in some cases, there will be insufficient data to decide whether the effect is indicative of significant dysfunction. However, the Agency has established guidelines and policies that are used to classify these end points. ATSDR believes that there is sufficient merit in this approach to warrant an attempt at distinguishing between "less serious" and "serious" effects. The distinction between "less serious" effects and "serious" effects is considered to be important because it helps the users of the profiles to identify levels of exposure at which

major health effects start to appear. LOAELs or NOAELs should also help in determining whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the Levels of Significant Exposure (LSE) tables and figures may differ depending on the user's perspective. Public health officials and others concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAELs) or exposure levels below which no adverse effects (NOAELs) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels or MRLs) may be of interest to health professionals and citizens alike.

A User's Guide has been provided at the end of this profile (see Appendix B). This guide should aid in the interpretation of the tables and figures for Levels of Significant Exposure and the MRLs.

Americium is a man-made element that has no stable form; all isotopes of americium are radioactive (see Section 4.1, Chemical Identity). Americium isotopes vary with respect to decay rates, daughters produced, and specific activity (see Section 4.2, Physical, Chemical, and Radiological Properties). Of particular interest is ^{241}Am, a decay product of ^{241}Pu. Americium is produced in small quantities (see Section 5.1), and information regarding human exposure is limited. Although this Toxicological Profile is not limited to health and pharmacokinetics data for any particular radioisotope of americium, available studies primarily involve the isotope ^{241}Am and, to a much lesser extent, ^{243}Am. Accidental occupational or environmental exposures to ^{241}Am in humans are usually associated with co-exposures to ^{241}Pu and other radioactive isotopes comprising the major sources of radiation associated with nuclear fallout from nuclear explosions and releases from processing and fuel reprocessing plants (see Chapter 6, Potential for Human Exposure). Occasional, unusual accidental exposures to household products containing ^{241}Am (e.g., ingestion of ^{241}Am-containing parts of smoke detectors) have resulted in atypically high exposures to ionizing radiation from americium. Available information on the health effects of americium is based entirely on effects observed in humans and animals from radioactivity attributable, at least in part, to americium exposures. The animal studies discussed below involve exposures to relatively high levels of ionizing radiation from uptake of ^{241}Am. As with other substances, uptake and disposition of americium in the body (and hence the potential for specific health effects from internalized americium) depend upon the chemical properties of the particular americium compound to which one may be exposed, as well as the route of exposure (see Section 3.4 for more information regarding uptake and disposition of americium). Based on available toxicokinetic information and the emissions of americium, the major

health concern is radiation damage from internalized americium. Inhalation, ingestion, or dermal absorption of americium compounds (or compounds that generate americium via radioactive decay) in amounts large enough to pose a chemical health risk would be expected to result in much more significant radiation toxicity.

Radioisotopes of americium primarily emit alpha particles (high energy particles that can travel short distances, but are not capable of penetrating the skin) and gamma rays (that can penetrate the entire body). Gamma rays originating from outside the body are the main source of external radiation hazard to internal organs. Once americium is internalized, the alpha particles are the major cause of damage to cells in the immediate vicinity of decaying americium atoms. Cellular damage results from the ionization of molecules that are encountered by ionizing (e.g., alpha, beta, gamma) radiation. In radiation biology, the term "dose" refers to the amount of energy that radiation transfers to an organ or tissue that it encounters. Absorbed dose is the amount of energy absorbed per unit mass, measured in units called rad or Gray (Gy) (see Appendix D for a detailed description of principles of ionizing radiation).

3.2.1 Inhalation Exposure

Available information from human exposures indicates that airborne americium-containing particles are deposited in the respiratory tract, cleared to some extent via mucociliary action, and swallowed or expelled (Edvardsson and Lindgren 1976; Fry 1976; Newton et al. 1983; Sanders 1974; Toohey and Essling 1980). Descriptions of human respiratory tract models that can be used for radiation protection also include relevant information regarding biokinetics of inhaled particles (ICRP 1994b, 1995; NCRP 1997). Quantitative data are not available, however. Supporting animal studies include inhalation exposure to aerosols of americium (Buldakov et al. 1972; DOE 1978; Gillett et al. 1985; Sanders and Mahaffey 1983; Talbot et al. 1989; Thomas et al. 1972) or intratracheal instillation of americium compounds (Moushatova et al. 1996).

3.2.1.1 Death

No reports were located regarding death in humans resulting from acute-, intermediate-, or chronic-duration inhalation exposure to americium.

Death was noted within 6 months in an unspecified number of dogs following acute exposure to ^{241}Am aerosols (as americium nitrate) resulting in inhaled activity of 1.5 µCi/kg (55.5 kBq/kg) (Buldakov et al.

1972). Significant early mortality, attributed to radiation pneumonitis, was noted in rats following acute inhalation of $^{241}AmO_2$ particles (AMAD 0.75–1.39 μm) resulting in initial lung burdens of approximately 1.3 μCi (48 kBq) (Sanders and Mahaffey 1983).

3.2.1.2 Systemic Effects

No data were located regarding gastrointestinal effects, endocrine effects, dermal effects, or ocular effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

Respiratory Effects. No reports were located regarding respiratory effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding respiratory effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Respiratory insufficiency and pulmonary pneumonia were reported in a group of five dogs following acute exposure to ^{241}Am aerosols (as americium nitrate) resulting in an inhaled activity of 1.5 μCi/kg (55.5 kBq/kg) (Buldakov et al. 1972). In another study, gross and microscopic lung lesions, including pleural thickening, fibrosis, mineralization, and cell proliferation, were noted in dogs sacrificed from 127 to 1,022 days following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be moderately soluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposures resulted in estimated initial ^{241}Am body burdens between 38 and 51 μCi (1.4 and 1.9 MBq). Increased respiratory frequency and decreased tidal volume were observed in two of the early sacrifices (Thomas et al. 1972). Radiation pneumonitis was observed in rats exposed once (nose-only) to $^{241}AmO_2$ aerosols resulting in a lung tissue activity level of approximately 650 nCi (24 kBq), but not at lung tissue activity levels #31 nCi (1.15 kBq) (DOE 1978).

Cardiovascular Effects. No reports were located regarding cardiovascular effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding cardiovascular effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Various effects on the cardiovascular system, including shifts in hemodynamics (blood pressure and flow), electrocardiographic changes, and myocardial ishchemia were reported in a group of five dogs following acute exposure to ^{241}Am aerosols (as americium nitrate) resulting in inhaled activity of 1.5 μCi/kg (55.5 kBq/kg) (Buldakov et al. 1972).

Hematological Effects. Persistent clinical lymphopenia and thrombocytopenia were observed in a 64-year-old man exposed to ^{241}Am when an ion-exchange column containing about 100 g of ^{241}Am exploded in his face (Filipy et al. 1995). The explosion resulted in contact exposure through the intact and lacerated skin and presumed inhalation exposure (see Sections 3.2.3.1 and 3.2.3.2 for additional information regarding this accident).

No reports were located regarding hematological effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Leukopenia and elevated hematopoietic activity were reported in a group of five dogs following acute exposure to ^{241}Am aerosols (as americium nitrate) resulting in inhaled activity of 1.5 μCi/kg (55.5 kBq/kg) (Buldakov et al. 1972). Hemotocrit, total white blood cell counts, neutrophils, lymphocytes, and platelets were decreased from pre-exposure values in dogs following a 10-minute inhalation exposure to aerosols of ^{241}Am (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC), resulting in estimated initial ^{241}Am body burdens between 38 and 51 μCi (1.4 and 1.9 MBq) (Thomas et al. 1972). These values remained lower than pre-exposure values in the dogs individually sacrificed 127, 256, and 512 days post exposure; the only apparent recovery occurred in the platelet count of the dog sacrificed 1,022 days post exposure, reaching near pre-exposure values approximately 900 days post exposure.

Musculoskeletal Effects. Information regarding musculoskeletal effects in humans is limited to the findings of bone marrow peritrabecular fibrosis and decreased cellularity in bone samples taken from the corpse of a 64-year-old man who had been exposed to ^{241}Am (11 years earlier) when an ion-exchange column containing about 100 g of ^{241}Am exploded in his face (Priest et al. 1995). The explosion resulted in contact exposure through the intact and lacerated skin and presumed inhalation exposure (see Sections 3.2.3.1 and 3.2.3.2 for additional information regarding this accident).

No reports were located regarding musculoskeletal effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Pathologic findings in the bone, including fibrosis with focal osteoid thickening, focal resorption, and cartilage degeneration, were observed in dogs (initial body weights between 8.4 and 10.6 kg) sacrificed between 127 and 1,022 days following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposures resulted in estimated initial ^{241}Am body burdens between 38 and 51 μCi (1.4 and 1.9 MBq) (Thomas et al. 1972).

Hepatic Effects. No reports were located regarding hepatic effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding hepatic effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Changes were observed in the livers of male rats 30 days after a single intratracheal instillation (0.2 mL) of ^{241}Am (as americium nitrate), resulting in an activity dosage of 0.05 μCi (1.8 kBq); the effects were predominantly hepatic parenchyma degeneration and focal necrosis with the effect observed in areas where blood entered the organ (Moushatova et al. 1996). Liver lesions (fatty changes in the centri-lobular region) were also observed in two beagle dogs 512 or 1,022 days following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposures resulted in estimated initial ^{241}Am body burdens between 38 and 51 μCi (1.4 and 1.9 MBq) (Thomas et al. 1972).

Renal Effects. No reports were located regarding renal effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding renal effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Kidney lesions (generalized glomerular mesangial thickening with obliteration) were observed in two beagle dogs 512 or 1,022 days following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposures resulted in estimated initial ^{241}Am body burdens between 38 and 51 μCi (1.4 and 1.9 MBq) (Thomas et al. 1972).

Endocrine Effects. No reports were located regarding endocrine effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding endocrine effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Thyroid gland lesions (gross atrophy, with fibrosis) were observed in two beagle dogs 512 or 1,022 days following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposures resulted in estimated initial ^{241}Am body burdens between 38 and 51 μCi (1.4 and 1.9 MBq) (Thomas et al. 1972).

Body Weight Effects. No reports were located regarding body weight effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding body weight effects in animals following intermediate- or chronic-duration inhalation exposure to americium. One of four dogs lost approximately 15% of its body weight during the time period of 365 days to sacrifice at 512 days following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposure resulted in an estimated initial body burden of 38 µCi (1.4 MBq) (Thomas et al. 1972).

Metabolic Effects. No reports were located regarding metabolic effects in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding metabolic effects in animals following intermediate- or chronic-duration inhalation exposure to americium. Increased B-glucuronidase activity was reported in mice receiving a single 30-minute nose-only exposure to aerosols of ^{241}Am (as americium nitrate) that resulted in a lung-averaged cumulative radiation dose of 2,000 rad (20 Gy) (Talbot et al. 1989).

3.2.1.3 Immunological and Lymphoreticular Effects

Lymphopenia was noted in a 64-year-old man exposed to ^{241}Am when an ion-exchange column containing about 100 g of ^{241}Am exploded in his face (Filipy et al. 1995) (see Sections 3.2.3.1 and 3.2.3.2 for additional information regarding this accident)

Leukopenia was reported in a group of five dogs following acute exposure to ^{241}Am aerosols (as americium nitrate) resulting in inhaled activity of 1.5 µCi/kg (55.5 kBq/kg) (Buldakov et al. 1972). Total white blood cell counts, neutrophils, and lymphocytes were decreased from pre-exposure values in dogs following a 10-minute inhalation exposure to aerosols of ^{241}Am dioxide (presumed to be relatively insoluble, having been generated by heating ^{241}Am oxide to 600 EC). Exposures resulted in estimated initial ^{241}Am body burdens between 38 and 51 µCi (1.4 and 1.9 MBq) (Thomas et al. 1972). These values remained decreased in the dogs individually sacrificed 127, 256, and 512 days post exposure. However, no reports were located regarding immunological consequences of reduced leukocyte counts in humans or animals exposed to americium.

No reports were located regarding the following health effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to americium:

3.2.1.4 Neurological Effects

3.2.1.5 Reproductive Effects

3.2.1.6 Developmental Effects

3.2.1.7 Cancer

No reports were located regarding cancer in humans following acute-, intermediate-, or chronic-duration inhalation exposure to americium.

No reports were located regarding cancer in animals following intermediate- or chronic-duration inhalation exposure to americium. Osteosarcomas developed in 4 of 15 dogs surviving more than 1,000 days following a single inhalation exposure to aerosols of $^{241}AmO_2$ that resulted in initial lung burdens of approximately 2–6 μCi (74–222 kBq) (Gillett et al. 1985). Among the four dogs that developed osteosarcomas, the radiation dose to the skeleton, calculated to 1,000 days post exposure, ranged from 96 to 410 rad (0.96 to 4.1 Gy).

3.2.2 Oral Exposure

No reports were located regarding the following health effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to americium:

3.2.2.1 Death

3.2.2.2 Systemic Effects

3.2.2.3 Immunological and Lymphoreticular Effects

3.2.2.4 Neurological Effects

3.2.2.5 Reproductive Effects

3.2.2.6 Developmental Effects

3.2.2.7 Cancer

3.2.3 Dermal Exposure

3.2.3.1 Death

No reports were located regarding death in humans that could be associated with acute-, intermediate,- or chronic-duration dermal exposure to americium. An explosion and accidental exposure of a 64-year-old man to 1–5 Ci (37–185 GBq) of ^{241}Am was followed by immediate treatment to reduce contamination to approximately 6 mCi (222 MBq) and to 1 mCi (37 MBq) after the first day (Thompson 1983; Toohey and Kathren 1995). Survival was likely the result of intense and long-term chelation therapy, such as with diethylenetriaminepentaacetic acid (DTPA). The patient lived until natural death 11 years after the accident (see Section 3.2.3.2 for more detailed information regarding this accidental exposure and subsequent treatment and follow-up).

3.2.3.2 Systemic Effects

No data were located regarding respiratory effects, cardiovascular effects, gastrointestinal effects, hepatic effects, renal effects, endocrine effects, dermal effects, ocular effects, body weight effects, or metabolic effects in humans or animals following acute-, intermediate-, or chronic-duration dermal exposure to americium:

Hematological Effects. Information regarding hematological effects in humans following dermal exposure to americium is limited to the case of a 64-year-old man exposed to ^{241}Am when an ion-exchange column containing about 100 g of ^{241}Am exploded in his face. Numerous studies relate various aspects of this accident as well as subsequent treatment and follow-up (Breitenstein and Palmer 1989; Filipy et al. 1995; Jech et al. 1983; McMurray 1983; Palmer et al. 1983; Robinson et al. 1983; Thompson 1983; Toohey and Kathren 1995). The explosion resulted in contact exposure through the intact and lacerated skin and presumed inhalation exposure, evident from external chest measurements of radioactivity. The amount of activity initially deposited on the man and his clothing was estimated to be 1–5 Ci (37–185 GBq). Immediate treatment reduced contamination to approximately 6 mCi (222 MBq). By the end of the first day post accident, the activity had been reduced to 1 mCi (37 MBq). Intense and long-term chelation therapy was employed to reduce body burden. Signs of adverse effects included clinical lymphopenia and thrombocytopenia that persisted for 5 years. The lymphopenia had rapid onset and a slow recovery, although pre-exposure values were not attained by 11 years post exposure, at which time, the worker died of unrelated causes. Significant, but transient effects on erythropoiesis were

indicated by a decrease in erythrocyte concentration, hemoglobin concentration, and packed red cell volumes (see Section 3.4.1.3 for additional information regarding toxicokinetics).

No reports were located regarding hematological effects in animals following acute-, intermediate-, or chronic-duration dermal exposure to americium.

Musculoskeletal Effects. Histopathological examination of bone at autopsy of a worker who, 11 years prior to death, received a large exposure to ^{241}Am from an industrial explosion showed pathologic changes including peritrabecular fibrosis, bone cell depletion, and bone marrow atrophy (Priest et al. 1995). These effects could not be directly related to estimated radiation dose or bone turnover rates (see Section 3.2.3.2 Hematological Effects for a more detailed account of this accidental exposure to americium).

No reports were located regarding musculoskeletal effects in animals following acute-, intermediate-, or chronic-duration dermal exposure to americium.

3.2.3.3 Immunological and Lymphoreticular Effects

Leukopenia has been observed following ^{241}Am exposure (Filipy et al. 1995), but no reports were located regarding associated immunological consequences.

No reports were located regarding the following health effects in humans or animals following acute-, intermediate-, or chronic-duration dermal exposure to americium:

3.2.3.4 Neurological Effects

3.2.3.5 Reproductive Effects

3.2.3.6 Developmental Effects

3.2.3.7 Cancer

3.2.4 External Exposure

External exposure to americium is not expected to be of particular health concern because the high-energy alpha particles released from americium are not capable of penetrating the skin. Americium also emits gamma rays that can penetrate the entire body. However, it is unlikely that humans would be exposed to

an external americium source with enough gamma radiation to pose a significant threat to internal organs and tissues.

No reports were located regarding the following health effects in humans or animals following acute-, intermediate-, or chronic-duration external exposure to americium:

3.2.4.1 Death

3.2.4.2 Systemic Effects

3.2.4.3 Immunological and Lymphoreticular Effects

3.2.4.4 Neurological Effects

3.2.4.5 Reproductive Effects

3.2.4.6 Developmental Effects

3.2.4.7 Cancer

3.2.5 Other Routes of Exposure

3.2.5.1 Death

No reports were located regarding death in humans resulting from acute-, intermediate-, or chronic-duration exposure to americium by routes other than inhalation, oral, dermal, or external exposure.

Dose-related decreased long-term survival was observed in beagle dogs following single intravenous injections of ^{241}Am at average activity levels ranging from 1.9 to 2,900 nCi/kg (0.07 to 107 kBq/kg) (Taylor et al. 1991, 1993a).

3.2.5.2 Systemic Effects

No data were located regarding systemic effects in humans following acute-, intermediate-, or chronic-duration exposure to americium by routes other than inhalation, oral, dermal, or external exposure. No data were located regarding respiratory effects, cardiovascular effects, gastrointestinal effects, renal effects, dermal effects, ocular effects, or metabolic effects animals following exposure to americium by routes other than inhalation, oral, dermal, or external exposure.

Hematological Effects. A limited study reported dose-related depressions in white blood cell counts in dogs that were administered single intravenous injections of ^{241}Am at activity levels of 0.1–2.8 μCi/kg (3.7–29.6 kBq/kg) (Dougherty 1970). Maximum depression in granular leukocytes and monocytes was reached approximately 1 month post injection. Depression of lymphocytes occurred more slowly; minimal values were reached 1 year or more post injection. Depression of red blood cells was observed only groups of dogs injected with ^{241}Am at activity levels of 0.9 μCi/kg (33.3 kBq/kg) or greater.

Hepatic Effects. Degenerative liver changes, severe reduction in liver weight, and early death (typically attributed to bone cancer) were observed in dogs following single intravenous injections of ^{241}Am at activity levels of approximately 2.9 μCi/kg (107 kBq/kg) (Taylor et al. 1991). The average radiation dose to the liver in these dogs was 590 rad (5.9 Gy). Degenerative liver changes and early death were also reported in dogs administered single intravenous injections of ^{241}Am at a similar activity level (2.8 μCi/kg or 104 kBq/kg) (Lloyd et al. 1970; Taylor et al. 1993a).

Endocrine Effects. Thyroid weights were significantly lower in adult beagle dogs administered single intravenous injections of ^{241}Am at activity levels ranging from approximately 0.3 to 2.75 μCi/kg (11 to 102 kBq/kg) than those of controls (Taylor et al. 1993a). Histologic examination revealed follicular hyperplasia and degenerative cellular changes. Lower serum thyroxine levels were noted in the peripheral blood of dogs in the two highest exposure groups.

3.2.5.3 Immunological and Lymphoreticular Effects

A limited study reported dose-related depressions in white blood cell counts in dogs that were administered single intravenous injections of ^{241}Am at activity levels of 0.1–2.8 μCi/kg (3.7–29.6 kBq/kg) (Dougherty 1970). Maximum depression in granular leukocytes and monocytes was reached approximately 1 month post injection. Depression of lymphocytes occurred more slowly; minimal values were reached 1 year or more post injection.

3.2.5.4 Neurological Effects

No reports were located regarding neurological effects in humans or animals following acute-, intermediate-, or chronic-duration exposure to americium by routes other than inhalation, oral, dermal, or external exposure.

3.2.5.5 Reproductive Effects

No reports were located regarding reproductive effects in humans following acute-, intermediate-, or chronic-duration exposure to americium by routes other than inhalation, oral, dermal, or external exposure.

Dose-related increased incidences of death were reported among 14- and 17-day-old fetuses of rats that had been administered single intravenous injections of ^{241}Am between 1 and 10 days prior to mating (Moskalev et al. 1969). In this study, ^{241}Am was administered at activity levels ranging from 0.2 to 96 μCi/kg (7.4 to 3,552 kBq/kg). During gestation, concentrations of ^{241}Am were 9–15 times higher in placental tissues than in fetuses. The investigators indicated that death may have been the result of placental changes.

3.2.5.6 Developmental Effects

No reports were located regarding reproductive effects in humans following acute-, intermediate-, or chronic-duration exposure to americium by routes other than inhalation, oral, dermal, or external exposure.

Decreased fetal weight and increased fetal death were reported following single intravenous injection of pregnant rats with ^{241}Am (activity level 90 μCi/kg or 3,330 kBq/kg) on gestation day 9 (Rommerein and Sikov 1986). The investigators indicated a tendency toward increased incidences of fetuses with anomalous ribs.

3.2.5.7 Cancer

In a study of dogs administered single intravenous injections of ^{241}Am (Taylor et al. 1991), the incidence of death was dose-related and predominantly due to bone cancer, although the incidence of liver tumors was higher than that of bone tumors in dogs exposed to lower levels of ^{241}Am. Increases in bone cancers occurred in mice (Schoeters et al. 1991; Taylor et al. 1983; Van Den Heuvel et al. 1995), rats (Carter et al. 1951), and dogs (Jee et al. 1985; Lloyd et al. 1994a, 1994b) observed for a lifetime following the administration of a single intraperitoneal or intravenous injection of ^{241}Am. In an analysis of all soft tissue tumors in the dogs treated by intravenous injection, both positive and negative associations between

exposure and tumor incidence were apparent in a variety of soft tissues, but the development of these tumors appeared to be influenced by the competing risk of death from bone cancer or other severe radiation effects (Lloyd et al. 1995).

3.3 GENOTOXICITY

No reports were located regarding genotoxic effects in humans following acute- or intermediate-duration inhalation exposure to americium. Chromosomal aberrations in lymphocyte preparations were elevated for an entire group of seven nuclear fuel production workers who were exposed for 11–22 years to external gamma radiation, with an additional internal exposure of six of these workers to alpha emitting ^{241}Am 5 years prior to the analysis (Bauchinger et al. 1997). In five of these six cases, the internalized accumulated effective dose equivalent from ^{241}Am represented 5–25% of the total exposure, the main contribution coming from external exposure to gamma radiation. Internalized ^{241}Am represented 66% of the total exposure in the other worker. Another case involved a radiation worker and his wife, college-age daughter, and 10-year-old son. They were exposed for several years in their house to elevated levels of ^{241}Am that resulted in body burdens of 6.5–89 nCi (0.24–3.3 kBq) from a source used by the father for private experiments. Chromosomal aberrations in isolated leukocytes were noted to be similar to those observed in other cases of accidental or therapeutic exposure to external radiation sources (Kelly and Dagle 1974). The cytogenetic damage observed was low and only grossly comparable to historical control values available to the authors. The limited human data do not indicate a significant genotoxic response to inhaled americium.

No reports were located regarding genotoxic effects in animals following intermediate- or chronic-duration inhalation exposure to americium. A single report regarding genotoxic effects following acute inhalation exposure to americium indicated decreased numbers of pulmonary alveolar macrophages (PAMs) (maximum decrease at day 21), increased numbers of micronuclei, and multinucleated cells in mice receiving a single 30-minute nose-only exposure to ^{241}Am (as americium nitrate) producing a cumulative radiation dose of 2,000 rad (20 Gy) to lungs (Talbot et al. 1989). The initial alveolar deposition was 32.4 nCi (1.2 kBq), but rapidly declined to about 8.1 nCi (300 Bq) by 21 days and to approximately 2.7 nCi (100 Bq) by the end of the study (day 98). Following exposure to ^{241}Am, essentially all PAMs contained radiolabel; extensive labeling was still observed at each sacrifice time period between postexposure days 3 and 98.

No reports were located regarding genotoxic effects in humans or animals following acute-, intermediate-, or chronic-duration oral or dermal exposure to americium.

3.4 TOXICOKINETICS

3.4.1 Absorption

3.4.1.1 Inhalation Exposure

Evidence for absorption of inhaled americium to blood of humans is provided by several cases of accidental exposure in which workers were exposed to airborne ^{241}Am (Edvardsson and Lindgren 1976; Fry 1976; Newton et al. 1983; Sanders 1974; Toohey and Essling 1980). Although these cases do not provide a complete quantitative description of the absorption of inhaled americium in humans, they clearly demonstrate that inhaled americium oxides (e.g., AmO_2) can be absorbed, as indicated by the detection of ^{241}Am radioactivity in the chest region followed by its transfer to other regions of the body, mainly liver and bone, and excretion in urine and feces. In one case, an adult worker inhaled aerosols of plutonium dioxide (PuO_2) and $^{241}AmO_2$ (Newton et al. 1983). Thoracic counts of radioactivity that were made with an external radiation detector starting 7 days after the exposure showed that ^{241}Am was cleared from the chest area with biological clearance half-times of 11 (80%) and 920 days. These estimates were assumed to reflect clearance from the lung to systemic compartments. However, fecal excretion was estimated to be approximately 50% of the estimated initial deposit of ^{241}Am in the lung, suggesting that extensive mechanical transport to the esophagus and subsequent ingestion of the deposited americium, typical of inhaled large particles, may have occurred. External counting also detected ^{241}Am in the skull area on day 913 post accident, suggesting that ^{241}Am may have been transferred to the skeleton.

In a second case, an adult worker inhaled mixed oxides of curium dioxide ($^{244}CmO_2$) and $^{241}AmO_2$ (^{244}Cm:^{241}Am airborne ratio, 3:1) which resulted in the deposition of approximately 450 nCi (16.6 kBq) of radioactivity in the lungs, or approximately 112 nCi (4.1 kBq) of ^{241}Am (Sanders 1974). Chest counts of the subject indicated that total radioactivity was eliminated from the lungs with a biological half-time (i.e., decay-corrected) of 28 days; however, data on the elimination of each isotope from the lung was not reported. Most of the elimination from the chest area was accounted for by recovery of radioactivity in the feces, consistent with mechanical clearance of the deposited activity to the gastrointestinal tract. Further evidence for systemic absorption in this case was the detection of ^{241}Am in urine; approximately

1.1 nCi (41 Bq, 1% of the deposited activity) of ^{241}Am was excreted in urine in 365 days, with a half-time of 35 days.

In a similar case, two adults accidentally inhaled $^{241}AmO_2$ (particle size not specified), which resulted in the deposition of approximately 15 nCi (555 Bq) of activity in the lungs (Fry 1976). Chest activity measured over a period of 200–1,500 days after the accident indicated that the biological half-time for ^{241}Am in the lungs is approximately 900–1,400 days. Activity measured over the chest, liver, and extremities (knees and ankles, reflecting primarily bone activity) indicated that, 324 days after the accident, approximately 41% of the whole-body activity was in the lung, 47% was in the liver, and 12% was in bone. After 1,392 days, lung activity decreased to 18%, liver activity was unchanged (47%), and bone activity had increased to 35%.

Additional evidence for absorption of inhaled americium, and more quantitative estimates of the extent of and rates of absorption, are provided by animal experiments. The lung deposition and absorption of inhaled aerosols of americium nitrates and oxides have been studied in experimental animals; the results of representative studies are provided in Table 3-1. In general, exposures were nose-only (although this was not always specified in the reports) to relatively well-characterized aerosols of ^{241}Am compounds. In most studies, the actual exposure concentration of americium was not reported. Estimates of lung retention half-times reflect the rate of loss of radioactivity from the lung following any initial (3–8 days) loss of americium that may have been transferred to the gastrointestinal tract and excreted without absorption. In some studies, absorption half-times were estimated, which reflect the loss of americium from the lung to the systemic tissues (usually blood to a summation of blood and other tissues other than the gastrointestinal tract). In either case, the estimates were based on curve-fitting of the data on lung radioactivity-time profiles and other assumptions. Thus, some of the differences in estimates undoubtedly reflect differences in particle size (known to significantly affect deposition location within and mucociliary clearance from the respiratory tract), the curve-fitting method, duration of observations, and intra-study variability; nevertheless, general trends are evident. Retention and systemic absorption can be described with two or three rate functions that represent fast and slower processes for transfer of americium from the lung (albeit with considerable differences in the rates reported in various studies, probably reflecting differences in animal species and exposure scenarios). For example, in monkeys exposed to aerosols of $^{241}AmO_2$ (activity median aerodynamic diameter [AMAD] 1.4 μm), retention half-times of ^{241}Am were 0.1 days (32% of the initial deposited) and 168 days (68%) (Mewhinney and Muggenburg 1985). Stanley et al. (1982) also observed both fast and slower phases of lung retention in monkeys exposed to $^{241}AmO_2$. Mewhinney et al. (1982) found that in dogs exposed to aerosols of

Table 3-1. Retention and Absorption Estimates in Animals Exposed to Americium Compounds by Inhalation

Animal model	Am species	Exposure	Exposure concentration[a]	Aerosol AMAD (μm±GSD)	Lung deposition (percent of dose)	Lung retention or absorption half-time[b]	Reference
Monkey, adult	$^{241}AmO_2$ (dust)[c]	Aerosol	No data	1.5±1.6	67 nCi 21 ng	47 days (89%) 550 days (11%) (retention)	Stanley et al. 1982
Monkey, 3–5 years	$^{241}AmO_2$	Aerosol, nose-only	No data	1.4 μm	75 μCi/kg 23 μg/kg	0.1 days (32%) 160 days (68%) (retention)	Mewhinney and Muggenburg 1985
Dog, adult	$^{241}AmO_2$	Aerosol, nose-only, 20 min	No data	0.8±1.2	41% 60–250 nCi/kg 18–77 ng/kg	9 days (89%) 290 days (11%) (retention)	Mewhinney et al. 1982
			No data	1.8±1.07	39% 39–240 nCi/kg 12–74 ng/kg	13 days (76%) 170 days (24%) (retention)	
			No data	3.5±1.06	30% 30–190 nCi/kg 12–59 ng/kg	25 days (69%) 310 days (31%) (retention)	
Dog, adult	$^{241}AmO_2$ (dust)[c]	Aerosol	No data	2.2±1.8	29% 190 nCi 59 ng	39 days (67%) 5,000 days (33%) (retention)	Stanley et al. 1982
Dog, adult	$^{241}AmO_2$	Aerosol, nose only 10 minutes	2.18 Ci/m^3 673 mg/m^3	0.8–1.0±1.5	50% 2.1–3.1 μCi/kg 0.7–1.0 μg/kg	20–30 days (retention)	Thomas et al. 1972
Dog, adult	$^{241}AmO_2$	Aerosol, nose-only	42–336 Ci/m^3 13–104 mg/m^3	1.40±1.69	ND	~60 days (retention)	Craig et al. 1975

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Table 3-1. Retention and Absorption Estimates in Animals Exposed to Americium Compounds by Inhalation *(continued)*

Animal model	Am species	Exposure	Exposure concentration[a]	Aerosol AMAD (μm±GSD)	Lung deposition (percent of dose)	Lung retention or absorption half-time[b]	Reference
Dog, adult	$^{241}AmO_2$	Aerosol, nose-only	0.66 mCi/m^3 0.20 mg/m^3	0.63±2.64	13% 1,2 nCi 0.38 ng	20–30 days 120 days 400 days (retention)	Craig et al. 1979
			42 mCi/m^3 13 mg/m^3	1.25±1.77	30% 120 nCi 37 ng		
			340 mCi/m^3 105 mg/m^3	1.35±1.74	35% 1,150 nCi 355 ng		
Hamster, adult	$^{241}AmO_2$	Aerosol	No data	1.9±2.0	45 nCi/kg 14 ng/kg	11 days 200 days (retention)	Stather et al. 1979
Rat, adult	$^{241}AmO_2$ (dust)[c]	Aerosol	No data	2.3±1.7	7.8 nCi 2.4 ng/kg	95 days (89%) 2,800 days (11%) (retention)	Stanley et al. 1982
Rat, adult	$^{241}AmO_2$ (dust)[d]	Aerosol, nose-only, 1-hour	No data	2.1±1.78	0.14 nCi/kg 0.042 ng/kg	2,000 days >10,000 days (absorption)	Stradling et al. 1992, 1994
Rat, adult	^{241}Am nitrate	Aerosol, nose-only 1–2 hour	No data	0.3±2.5	75 nCi/kg 23 ng/kg	49 days 315 days (absorption)	Stradling and Stather 1989
	$^{241}AmO_2$ (dust)[e]		No data	1.4±2.0	26 nCi/kg 8.1 ng/kg	7,000 days 7,000 days (absorption)	

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Table 3-1. Retention and Absorption Estimates in Animals Exposed to Americium Compounds by Inhalation *(continued)*

Animal model	Am species	Exposure	Exposure concentration[a]	Aerosol AMAD (μm±GSD)	Lung deposition (percent of dose)	Lung retention or absorption half-time[b]	Reference
Rat, adult	^{241}Am nitrate (dust)[f]		No data	3.8±1.6	0.51 nCi/kg 0.16 ng/kg	170 days 1,200 days (absorption)	Stradling and Stather 1989
	^{241}Am chloride (dust)[g]		No data	1.8±2.4	0.11 nCi/kg 0.035 ng/kg	47 days 1,700 days (absorption)	
Rat, adult	^{241}Am (dust)[h]	Aerosol, nose-only	No data	1.26±1.29	11 nCi/kg 3.6 ng/kg	14,000 days >17,000 days (absorption)	Moody et al. 1991
				0.21±1.61	89 nCi/kg 27.6 ng/kg	5,800 days 8,600 days (absorption)	
				1.74±1.94	47 nCi/kg 14 ng/kg	4,600 days >14,000 days (absorption)	
	^{241}Am (dust)[i]			1.63±1.76	2.3 nCi/kg 0.7 ng/kg	170 days 290 days 380 days (absorption)	

[a]Were only activity was reported, mass concentration of Am was calculated assuming a specific activity of 3.24 Ci/g.
[b]Retention reflects total elimination from lung; absorption reflects transfer to blood
[c]Powder collected from plutonium oxide milling operation
[d]Dust from a weapons test site
[e]Dust from corrosion of PuO_2 wire
[f]Dust from corrosion of industrial process line equipment, mainly Am chlorides
[g]Dust from electrorefining process residues
[h]Dust derived from ignition of Pu (900–1,000 EC)
[i]Dust derived from air oxidation of molten Pu.

AMAD = activity median aerodynamic; GSD = geometric standard deviation

americium dioxide (AMAD 0.8–3.5 μm), approximately 30–50% of the inhaled activity was deposited in the lung; at an AMAD of 0.8 μm, the estimated retention half-times were 9 and 290 days for 89 and 11% of the lung burden, respectively. Within 8 days of exposure, 32% of the initial lung burden was detected in extrarespiratory tissues, mainly the liver (21%) and skeleton (11%). Fractional deposition in the lung decreased and retention half-time increased with increasing aerosol AMAD, which is consistent with larger particles being deposited in the upper airways, from which mechanical clearance to the gastrointestinal tract would be more pronounced. The observations of rapid and slow phases of retention of americium initially deposited in the respiratory tract are consistent with the results of other studies of dogs exposed to $^{241}AmO_2$ aerosols (Craig et al. 1975; Thomas et al. 1972). Studies in rodents indicate that inhaled americium nitrates and chlorides are absorbed more rapidly than americium oxides (Stather et al. 1979b; Stradling and Stather 1989; Stradling et al. 1994). Americium oxides in dusts from weapon sites and industrial facilities were absorbed particularly slowly, with absorption half-times of 5–25 years, although this may also vary depending on the process that leads to the production of the dust. For example, americium oxides in dust formed during the ignition of plutonium was absorbed from the lung with a half-time of 0.5–1 year, whereas americium oxides produced from the air oxidation of molten Pu had absorption half-times exceeding 12 years (Moody et al. 1991). This likely reflects differences in the oxide species formed under the different conditions (e.g., temperature, etc.).

Information on the absorption of americium from the lung can also be derived from studies in which americium compounds were instilled directly into the trachea. Although intratracheal instillation does not precisely mimic inhalation exposure, rates of absorption of the deposited americium have been shown to be comparable for both routes of exposure. For example, Stradling et al. (1992) estimated nearly identical half-times for absorption of ^{241}Am in rats that received either intratracheal instillations of ^{241}Am oxide-containing dust or were exposed to aerosols of the same dust (2,000 days, >10,000 days). Estimates of absorption half-times in rats that received intratracheal instillations of $^{241}AmO_2$ in various types of dusts from weapons or fuel facilities indicate a slow and fast component to absorption, with the fast component ranging from 1 to 5 years, and rates of absorption following intratracheal instillation of americium nitrate citrate or hydroxide are higher than after instillation of americium oxides (Crawley et al. 1976; Müller et al. 1989; Stradling et al. 1989, 1992, 1994).

3.4.1.2 Oral Exposure

Data on the absorption of ingested americium in humans are limited to reports from accidents in which mixed inhalation, oral, and dermal exposures were experienced. However, studies that have been

reported indicate that <0.1% of the ingested activity is absorbed, which is consistent with numerous observations that have been made in experimental animals. Absorption of americium contained in shellfish (molluscs) has been studied in humans. Eight adult subjects (six males and two females) ingested molluscs (winkles) containing ^{241}Am that were collected from marine waters near the British Nuclear Fuels facility at Sellafield, Cumbria (Hunt et al. 1986a, 1986b). The range in the ingested activity of ^{241}Am was 18–76 Bq (0.49–2.1 nCi, 0.15–0.63 ng). Serial 24-hour urine samples were collected from each subject for up to 10 days after they ingested the molluscs. The fraction of the activity absorbed was estimated as the ratio of the observed cumulative urinary excretion of ^{241}Am to that of the excretion predicted to occur if absorption had been complete. The latter was predicted using a kinetic model of excretion of absorbed americium described by Takada et al. (1984). The reported geometric mean absorption for the eight subjects was 0.6×10^{-4} (geometric standard deviation [GSD], 0.1; range $0.4–2.1 \times 10^{-4}$), or 0.006% of the ingested activity. Seven of the initial eight subjects and one new subject participated in a subsequent study (six males and two females) in which the same protocol was implemented and the ^{241}Am activity range was 10–25 Bq (0.27–0.68 nCi, 0.083–0.21 ng) (Hunt et al. 1990). The arithmetic mean absorption fraction was 0.8×10^{-4} (range, $0.4–1.5 \times 10^{-4}$) or 0.008% of the ingested activity. When the results of the two studies were combined, the arithmetic mean absorption fraction was 0.9×10^{-4} (range, $0.3–2.5 \times 10^{-4}$) or 0.009% of the ingested activity, and absorption fractions for males and females were similar. Three of the initial eight subjects and three new subjects participated in a third study (five males and one female) in which the same protocol was implemented; however, the subjects ingested cockles collected from Ravenglass, Cumbria for a range of ingested activity of 15–17 Bq (0.41–0.46 nCi, 0.13–0.14 ng) (Hunt 1998). The arithmetic mean absorption fraction was 1.2×10^{-4} (range, $0.3–2.6 \times 10^{-4}$) or 0.012% of the ingested activity.

Additional information on the absorption of ingested AmO_2 is provided from accidental oral exposures. In one case, a worker ingested a ceramic particle containing approximately 2.85 μCi (105 kBq, 0.88 μg Am) of $^{241}AmO_2$. During the first 8 days after the particle was ingested, approximately 4 pCi (0.15 Bq, 0.00014% of the estimated) activity was excreted in the urine (Smith et al. 1983). In a second case, a worker swallowed two 2 mm (diameter) silver disks used in the manufacture of smoke detectors, which contained approximately 4.22 μCi (156 kBq, 1.3 μg) of ^{241}Am. The subject excreted the disks in feces on days 16 and 24 post ingestion. Urinary ^{241}Am excretion during this time was approximately 3 pCi (0.11 Bq, 0.7 ng Am) or 0.0007% of the ingested activity (Rundo et al. 1977). The time lapse between ingestion and fecal excretion of the disks was related to suspected delay in esophageal transit and infrequent bowel movements by the subject.

Studies in nonhuman primates provide additional evidence that <0.1% of an ingested amount of americium is absorbed. Ham et al. (1994) estimated the gastrointestinal absorption of americium in marmosets by comparing the retention of ^{241}Am in the liver and carcass after intraperitoneal injection of a citrate solution containing ^{241}Am (1.6 Bq, 43 pCi, 0.012 ng Am) or after gastric intubation with ^{241}Am mixed with potato powder (250 Bq, 6.7 μCi, 2.0 ng Am). The absorption fraction of ^{241}Am mixed with potato powder was estimated to be approximately $6x10^{-4}$ or 0.06% of the administered activity.

Gastrointestinal absorption of americium has also been estimated in pigs, guinea pigs, mice, and rats. The results of representative studies are provided in Table 3-2. While these studies differ in the methodology used to estimate absorption, the studies reveal certain important trends. In general, the animal studies reveal a relatively low absorption of ingested americium across species (<1% in adult animals). In the pig, guinea pig, hamster, and rat, absorption is higher by a factor of 30–200 in neonatal animals compared to adults and/or progressively decreases in magnitude with age after birth (to a factor of 4 at 30 days for the guinea pig) (Bomford and Harrison 1986; David and Harrison 1984; Sullivan et al. 1985). Americium appears to be absorbed to a similar extent when it is ingested as an aqueous solution of relatively water soluble nitrates or citrates or when it is incorporated into foods, such as molluscs, potato, or liver tissue (Bulman et al. 1993; Ham et al. 1994; Harrison et al. 1988; Hisamatsu and Takizawa 1987; Stather et al. 1979a). Absorption of relatively water insoluble oxides of americium is lower by a factor of 4–10 compared to the absorption of americium citrate, whose absorption factor is a factor of 3–6 lower than that of americium nitrate (Stather et al. 1979a; Sullivan 1980a, 1980b). Similarly, in rats, the absorption of americium in surface dust from a weapons site was lower by a factor of 10–50 compared to americium oxide or nitrate (Harrison et al. 1994; Sullivan 1980b). A species difference was noted, guinea pigs absorbing 5 times more americium dust than rats. Other factors that appear to increase absorption in rats include fasting compared to the fed state and iron deficiency compared to the iron replete state (Sullivan and Ruemmler 1988; Sullivan et al. 1986). Concurrent oral exposure to Fe^{3+} and americium also appears to increase the absorption of ingested americium; the latter effect may result from redox reactions in the gastrointestinal tract catalyzed by Fe^{3+} (Sullivan et al. 1986). While the exact site and chemical species of americium that is absorbed from the gastrointestinal tract is not known, chemical speciation models applied to saliva and gastric fluid predict that the major species in saliva will be citrate and phosphate complexes, whereas in gastric fluid, the major species will be Am^{3+} (Webb et al. 1998). Speciation models for americium in the small intestine have not been reported; however, complexes with citrate, lactate, and phosphate would be expected based on equilibrium constants for these weak acid complexes and their abundance in intestinal fluid (Webb et al. 1998).

Table 3-2. Absorption Estimates in Animals Exposed to Americium Compounds by Ingestion

Animal model	Dose and media[a]	Absorption (percent of dose)	Basis for estimate of absorption	Reference
Marmoset, adult	^{241}Am in potato powder single gavage dose, 2.0 ng	0.06	Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intraperitoneal dose	Ham et al. 1994
Pig, adult	^{241}Am citrate aqueous solution, single gavage dose, 190 μg/kg	0.16	Absorption estimate based on maximum whole-body ^{241}Am (8 days), without adjustment for excretion	Eisele et al. 1987
Pig, miniature	^{241}Am citrate aqueous solution, single gavage dose, 75 μg/kg	0.06		
Pig, neonate	^{241}Am nitrate aqueous solution, single gavage dose	~3	Absorption estimate based on carcass ^{241}Am (5 days), without adjustment for excretion	Sullivan and Gorham 1983
Guinea pig, neonate—adult	^{241}Am citrate aqueous solution, single dose applied to tongue, 0.6–0.9 μg/kg	% / Age 1.1 / 0.5 d 0.55 / 1 d 0.19 / 5 d 0.17 / 10 d 0.06 / 15 d 0.03 / 20 d 0.02 / 30 d 0.005 / adult	Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intraperitoneal dose (neonates) or intravenous dose (adults), with adjustment for excretion	Bomford and Harrison 1986
Guinea pig and rat, adult	^{241}Am in three surface dust samples, single dose applied to tongue (GP, 5–15 ng/kg) or fed (rat, 12–49 ng/kg)	GP / Rat 0.002 / 0.0003 0.005 / 0.001 – / 0.001	Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intraperitoneal dose, with adjustment for excretion	Harrison et al. 1994
Guinea pig, adult	^{241}Am nitrate, aqueous solution, single gavage dose, 2–4.4 μg	0.017–0.031	Absorption estimate based on sum of liver, skeletal and urine ^{241}Am	Sullivan 1980a

Table 3-2. Absorption Estimates in Animals Exposed to Americium Compounds by Ingestion *(continued)*

Animal model	Dose and media[a]	Absorption (percent of dose)		Basis for estimate of absorption	Reference
Hamster, neonate—weaning	^{241}Am nitrate, aqueous solution, single dose applied to tongue, 1.6–2.5 ng	%	Age	Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intraperitoneal dose, with adjustment for excretion	David and Harrison 1984
		4.5	1 d		
		1.70	4 d		
		0.50	7 d		
		0.006	22 d		
		0.02	30 d		
Hamster adult	^{241}Am nitrate, aqueous solution, single gavage dose, 2.8 ng/kg	0.054		Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intravenous dose, with adjustment for excretion	Stather et al. 1979
	^{241}Am citrate aqueous solution, single gavage dose, 2.6 ng/kg	0.012			
	^{241}Am dioxide, aged aqueous suspension, single gavage dose, 19 ng/kg	0.0058			
	^{241}Am in liver from exposed hamsters, single dose, 2.9 ng/kg	0.0034			
Mouse, adult	^{241}Am nitrate, aqueous solution, single gavage dose, 32 μg/kg	0.0028		Absorption estimate based on sum of ^{241}Am in carcass (excluding the gastrointestinal tract) and liver	Hisamatsu and Takizawa 1987
	^{241}Am citrate, aqueous solution, single gavage dose, 43 μg/kg	0.015			
	^{241}Am in liver from exposed mice, single gavage dose	0.0024–0.0026			
Rat, adult	^{241}Am in wild molluscs or ^{241}Am nitrate injected into mollusc tissue, twice weekly for 8 weeks	0.026–0.12 (wild mussels)		Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intravenous dose, with adjustment for excretion, twice weekly for 8 weeks	Harrison et al. 1988
		0.016–0.040 (injected mussel)			

Table 3-2. Absorption Estimates in Animals Exposed to Americium Compounds by Ingestion *(continued)*

Animal model	Dose and media[a]	Absorption (percent of dose)	Basis for estimate of absorption	Reference
Rat, adult	^{241}Am in potatoes exposed to ^{241}Am, or injected into potatoes, single dose or daily for 23 days	0.12–0.16	Absorption estimate based on comparisons with liver and carcass ^{241}Am after an intravenous or intraperitoneal dose, with adjustment for excretion	Bulman et al. 1993
Rat, adult	^{241}Am nitrate, aqueous solution, single gavage dose, 5 μg/kg	0.063	Absorption estimate based on sum of liver, skeletal and urine ^{241}Am	Sullivan 1980a
Rat, adult	^{241}Am oxide, aqueous suspension, single gavage dose, 33 μg/kg	0.015		
Rat, adult	^{241}Am nitrate, aqueous solution, single gavage dose, 12 μg/kg; fed or fasted, with or without Fe^{3+}	0.017 (fed) 0.124 (fast) 1.19 (fast w/Fe^{3+})	Absorption estimate based on sum of ^{241}Am in carcass (excluding the gastrointestinal tract) and urine, Fe^{3+} gavage dose before ^{241}Am dose	Sullivan et al. 1986
Rat, neonate	^{241}Am nitrate, aqueous solution, single gavage dose, 0.44 μg	4.6	Absorption estimate based on ^{241}Am in carcass, excluding the gastrointestinal tract	Sullivan 1980b
	^{241}Am oxide, aqueous solution, single gavage dose, 0.59 μg	0.32	Absorption estimate based on ^{241}Am in carcass, excluding the gastrointestinal tract	
Rat, neonate or adult	^{241}Am nitrate, gavage single dose, 285 μg/kg (adult), 1,380 μg/kg (neonate)	% / Age 5.7 / 2 d 0.0062 / adult	Absorption estimate based on ^{241}Am in carcass, excluding the gastrointestinal tract	Sullivan et al. 1985
	^{241}Am citrate, aqueous solution, single gavage dose, 285 μg/kg (adult) 1,480 μg/kg (neonate)	% / Age 5.9 / 2 d 0.047 / adult		
Rat, weanling	^{241}Am nitrate, aqueous solution, single gavage dose, Fe deficient or replete, 7–9 μg/kg	4.8 (Fe deficient) 1.8 (Fe replete)	Absorption estimate based on sum of ^{241}Am in carcass, excluding the gastrointestinal tract and urine	Sullivan and Ruemmler 1988

[a]Where the chemical form and/or dose are not presented, these were not provided in the referenced report. Where only activity was reported, mass concentration of Am was calculated assuming a specific activity of 3.24 Ci/g.

d = days; Fe = iron; GP = guinea pig

3.4.1.3 Dermal Exposure

Information on the dermal absorption of americium in humans or animals is extremely limited. Some qualitative information is available from accidental exposures that included other routes of exposure. One of the most well studied was an accident in which a worker received facial wounds from projectile glass (and other debris) and nitric acid during an explosion of a vessel containing ^{241}Am (McMurray 1983). The subject also inhaled ^{241}Am released to the air as dust and nitric acid aerosols, which was evident from external chest measurements of radioactivity (Palmer et al. 1983). Accurate estimates of dermal absorption of americium cannot be made from this case because of the complex exposure scenario and because of the occurrence of acid and projectile wounds to the skin which might have greatly affected the penetration of any americium that was deposited on the surface of the skin. Nevertheless, it is possible to conclude from measurements of the dermal ^{241}Am activity soon after the accident and measurements of long-term excretion of ^{241}Am that a substantial fraction of the initial radioactivity deposited in the area of the wounds was absorbed. Over the first 60-day period after the accident, dermal ^{241}Am activity decreased from >5 mCi (185 MBq, 1.5 mg) to approximately 0.15 mCi, (5.5 MBq, 0.046 mg), largely as a result of skin decontamination procedures, although normal epidermal sloughing would be an expected contributor as well (Robinson et al. 1983). Cumulative urinary and fecal excretion of ^{241}Am over an observation period of 5 years was estimated to be 1 mCi (37 MBq, 20% of the initial deposited activity, and peak body burden, excluding the skin, was estimated to have been approximately 0.08 mCi (3 MBq, 1.6% of the estimated initial deposited activity). Thus, dermal absorption, under these extreme conditions of mechanical and chemical skin wounds, may have been as high as 20%.

3.4.1.4 Other Routes of Exposure

Several cases of accidental exposure to americium as a result of wound penetrations have been reported (Thompson 1983). These exposures have resulted in ^{241}Am burdens in the liver and skeleton, indicating absorption and distribution from the wound site (McInroy et al. 1989).

3.4.2 Distribution

3.4.2.1 Inhalation Exposure

The United States Transuranium and Uranium Registries (USTUR) collects data on the americium content of human tissues obtained from autopsies. Recent data summaries reported from the USTUR

show that after occupational inhalation exposures, ^{241}Am activity resides primarily in the respiratory tract, skeleton, liver, and muscle (see Table 3-3). In three cases in which occupational exposures were believed to have been primarily, if not exclusively by inhalation, the following tissue distribution was observed at autopsy: skeleton, 48%; respiratory tract, 35%; muscle, 10%; liver, 3%; and other issues, 4% (McInroy et al. 1989). Analyses of larger sets of USTUR cases have shown that, excluding the respiratory tract and other *site of entry* tissues (e.g., wound, gastrointestinal tract), approximately 80–90% of the americium in the body is associated with the skeleton, liver, and muscle, the skeletal:soft tissue burden ratio is approximately 3, and the highest soft-tissue concentrations are observed in the liver (Filipy and Kathren 1996; Filipy et al. 1994; Kathren et al. 1988). Whole-body counting of workers who were chronically exposed to plutonium and americium aerosols yielded a similar distribution between the skeleton and soft tissue americium burden. In 20 workers, the geometric mean skeletal:liver burden ratio was 4.4 (2.4 GSD, range 1.2–17) (Badjin and Molokanov 1998). Further evidence that the skeleton and liver are the primary sites of accumulation of absorbed americium is provided by inhalation studies conducted in monkeys (Stanley et al. 1982), dogs (Craig et al. 1975, 1979; Mewhinney et al. 1982; Thomas et al. 1972), and rats (Stradling et al. 1992, 1994). Americium is also taken up by teeth. In rats, americium accumulates in the dental pulp of developing teeth and eventually is incorporated into the mineralized dentin (Hammerström and Nilsson 1970b).

Information on the distribution of americium in blood after exposures to americium is available from an accident victim who was exposed by inhalation, dermal deposition, and wound penetration as a result of an explosion of a vessel containing ^{241}Am (McMurray 1983). The internal ^{241}Am activity was sufficiently high in this case to provide reliable estimates of blood and serum ^{241}Am concentrations. Essentially all of the ^{241}Am in blood was in the serum (Robinson et al. 1983). Studies in animals provide further evidence that americium is confined largely to the plasma portion of blood. In baboons, dogs, and rats, >95% of americium in blood is associated with plasma. A variety of human and animal studies (*in vivo* and *in vitro)* have shown that americium in plasma binds extensively to proteins (80–90%), including albumin and transferrin (Boocock and Popplewell 1966; Bruenger et al. 1969; Cohen and Wrenn 1973; Cooper and Gowing 1981; Durbin 1973; Popplewell and Boocock 1967; Turner and Taylor 1968). When americium was incubated with human serum *in vitro*, <1% of the americium was ultrafilterable; however, essentially all of the americium could be rendered ultrafilterable by the inclusion of 50 mM citrate in the serum incubate, suggesting reversible binding of americium to serum proteins (Chipperfield and Taylor 1972).

Table 3-3. Tissue Americium Levels from Human Autopsies

Tissue	Mean percent of body burden	Mean percent of systemic burden (excluding respiratory tract)
	Based on three inhalation cases[a]	
Respiratory tract	34.8	–
Liver	3.3	6.3
Skeleton	47.6	70.3
Muscle	10.0	16.1
Other	24.3	7.2
Tissue	**Soft tissue:liver concentration ratio[b] (number of mixed exposures)**	
Brain	0.08 (8)[c]	
Heart	0.123 (6)	
Kidneys	0.06 (38)	
Muscle	0.056 (8)	
Pancreas	0.03 (4)[c]	
Spleen	0.06 (38)	
Testes	0.029 (23)	
Thyroid	0.03 (21)	

[a]McInroy et al. 1989
[b]Filipy et al. 1994
[c]Calculated from Filipy and Kathren 1996

As noted above, bone is a primary site of accumulation of americium in the skeleton. In human autopsy samples of bone, ^{241}Am is found primarily at the cortical and trabecular bone surfaces (Priest et al. 1995; Schlenker et al. 1989). Americium that is observed in the deeper regions of human bones is thought to result from burial of historic surface deposits by newly accreted bone. Supporting evidence for accretion being a mode of redistribution of americium in bone comes from studies of monkeys, rats, and dogs that received single injections of ^{241}Am (Durbin 1973; Herring et al. 1962; Lloyd et al. 1972; Nenot et al. 1972; Polig 1976; Priest et al. 1983). In these studies, the initial deposition of ^{241}Am occurred at bone surfaces, including the periosteal and endosteal surfaces of trabecular bone and along the vascular channels of cortical bone, with more pronounced deposition at surfaces that were undergoing resorption, and with subsequent overlay of new bone (Priest et al. 1983). Because most americium in bone is associated primarily with the bone surface, the americium concentration in bone (per g bone ash) tends to vary with the bone ash content, a reflection of the variability in bone surface-to-volume ratio in a human who was occupationally exposed to americium (Kathren et al. 1987, 1990; Stevens et al. 1977).

When residence times for the americium measured in autopsy cases are taken into consideration (time between exposure and autopsy), the measured skeletal burdens are consistent with elimination half-times of approximately 50 years (McInroy et al. 1989, 1995). This value represents an average; however, in an individual, exchanges between bone and soft tissue stores of americium may be more rapid during periods of active bone metabolism such as infancy and childhood, pregnancy, and menopause (Lloyd et al. 1999). Studies in animals provide evidence for this concept (Durbin 1973). After an intravenous injection of americium citrate, skeletal tissue accumulated approximately 76% of the injected activity in neonatal dogs and 29% in adult dogs (Stevens et al. 1977). Higher skeletal uptakes of injected americium have also been observed in neonatal rats and mice, relative to adult animals (Hollins et al. 1973; Schoeters et al. 1990) and in young rats (3 months of age) compared to old rats (13 months of age) (Sontag 1983). On the other hand, americium uptake into maternal bone of lactating rats was similar to that of nonlactating rats, while concurrent calcium uptake into bone was lower in lactating rats (Hollins and Durakovic 1972). Thus, active mobilization of bone mineral, *per se*, may not always promote release of americium from bone.

The liver is the primary soft tissue site of initial accumulation of absorbed americium in humans. Americium taken up into skeletal muscle appears to have a longer retention half-time than americium in the liver. Therefore, at long times after exposure (years), the skeletal muscle will contain a larger fraction of the systemic americium burden than the liver (Filipy and Kathren 1996; Kathren 1994; McInroy et al. 1995). Information on the distribution of americium within the human liver is not available; however,

studies in dogs and rats have shown that within a few days after an intravenous injection, hepatic ^{241}Am is associated with lysosomes (Gruner et al. 1981; Lindenbaum and Rosenthal 1972; Seidel et al. 1986; Sütterlin et al. 1984). Americium in the hepatic cytosol is bound to ferritin and other unidentified proteins (DOE 1984; Stover et al. 1970). It is not known whether americium reacts with metallothionein; however, since americium forms stable complexes with polycarboxylate compounds such as DTPA (Lloyd et al. 1975a, 1975b), stable complexes with sulfhydryl groups in proteins such as metallothionein would not be expected. However, binding of ^{241}Am to an unidentified low molecular weight protein fraction in a rat liver has been reported (DOE 1984).

Information on the placental transfer of inhaled americium in humans is not available. Studies in animals that received parenteral injections of americium show that absorbed americium is transferred to the fetus. To some extent, therefore, this distribution pathway would be expected after inhalation exposure (Sikov 1987; Sikov and Kelman 1989; Stather et al. 1992). In baboons that received a single intravenous injection of americium citrate during the 5th month of pregnancy, approximately 0.4% of the activity was transferred to the fetus within 7 days post administration (Paquet et al. 1998). The fetal:maternal whole-body ^{241}Am concentration ratio was approximately 0.1 and the tissue distribution in the fetus was similar to that observed in adult animals, with the skeleton and liver accounting for most of the ^{241}Am activity in the body. Transfer of injected americium to the fetus has also been quantified in mice and rats that received intravenous injections of americium citrate during pregnancy (Sikov 1987). In both rats and mice, the fetal skeleton and liver were the major sites of accumulation of americium (Hisamatsu and Takizawa 1983; Sasser at al. 1986; Schoeters et al. 1990; DOE 1986; Van Den Heuvel et al. 1992; Weiss et al. 1980). In rats that received an injection of ^{241}Am citrate on days 18 or 19 of pregnancy, fetal transfer was approximately 0.01% of the amount of americium administered to the dams. The fetal:maternal ^{241}Am concentration ratio in rats was approximately 0.02 (Hisamatsu and Takizawa 1983). In mice that received an injection of ^{243}Am citrate (0.01 μCi) on day 16 of pregnancy, fetal transfer was approximately 0.04% of the administered maternal amount per gram of fetus; the ratio of fetal:maternal ^{243}Am activity at the time of dissection was 0.029 (Weiss et al. 1980).

Information on the distribution of absorbed americium to mammary milk in humans is not available. Numerous studies in animals have shown that transfer to milk occurs and that neonates can be exposed to americium during lactation. These studies include experiments in which the animals were exposed by intravenous injection or oral administration of americium; however, the implications are relevant to any route of exposure, including inhalation. In rats that received an intravenous injection of americium citrate during lactation, approximately 0.1% of the injected activity was transferred to the nursing pups (Sasser et

al. 1986). Oral and intravenous exposure to americium has resulted in the transfer of americium to mammary milk of cows, goats, and sheep (McClellan et al. 1962; Sutton et al. 1979). In a study of sheep that received americium chloride intravenously, the concentration of americium in mammary milk was 2–3 times that of maternal plasma (McClellan et al. 1962).

3.4.2.2 Oral Exposure

Information on the distribution of ingested americium in humans is not available. However, it is reasonable to suggest that the distribution of americium absorbed into the systemic circulation from the gastrointestinal tract would be similar to that absorbed after inhalation (see Section 3.4.2.1). Studies in experimental animals support this; the major sites of americium accumulation after ingestion of americium are the skeleton, liver, and muscle. In pigs, ingesting americium citrate once, the distribution of the americium body burden 8 hours post ingestion, when the highest total body burdens were observed, was as follows: bone, 56%; liver, 29%, and muscle, 5% (Eisele et al. 1987). The skeleton and liver accounted for 40% (range, 8–67%) and 29% (range, 7–30%) of the body burden of ^{241}Am, respectively, 7 days following a single gavage administration of americium citrate or nitrate to rats (Sullivan et al. 1985, 1986). Although it is possible that americium that is absorbed from the gastrointestinal tract might be taken up preferentially by the liver because of the hepatic portal blood flow, there is no evidence that a *first pass effect* substantially alters the overall tissue distribution of absorbed americium. For example, in marmosets that received with an intravenous injection of americium citrate, the liver accounted for 27% of the total body burden of americium (excluding the gastrointestinal tract); whereas after a single gavage administration of americium in potato powder, the liver accounted for 31% of the body burden (oral:intravenous ratio of 1.14) (Ham et al. 1994). A similar comparison made in hamsters resulted in an oral:intravenous ratio for the fractional liver burden of 0.9 (Stather et al. 1979a), and comparisons made in guinea pigs and rats resulted in ratios <1 (Bomford and Harrison 1986; David and Harrison 1984; Harrison et al. 1994).

3.4.2.3 Dermal Exposure

The distribution of americium that is absorbed from the skin would be expected to be similar to that from other routes, with the exception of retention of americium at the site of dermal exposure. Evidence for this is provided from an accident in which a worker received facial wounds from projectile debris and nitric acid during an explosion of a vessel containing ^{241}Am (McMurray 1983). The subject also inhaled ^{241}Am released to the air as dust and nitric acid aerosols, which was evident from external chest

measurements of radioactivity (Palmer et al. 1983). However, since the peak tissue burdens (lung, liver, and bone) of ^{241}Am, measured 3 days after the accident, were collectively only 0.08 mCi, whereas the 5-year cumulative excretion of ^{241}Am was approximately 1 mCi, it is almost certain that the source of most of the absorbed ^{241}Am was the skin and/or skin wounds. The bone:liver ratio of ^{241}Am 3 days after the exposure was approximately 3, and was 17–23 at autopsy 11 years after the accident. This suggests that bone and liver would be the major sites of accumulation of americium absorbed from the skin, as is the case for americium that is absorbed by other routes. The bone:liver ratio at 3 days was probably strongly affected by the aggressive DTPA therapy being employed at that time, since americium can be more easily removed from soft tissue deposition sites than from bone.

3.4.2.4 Other Routes of Exposure

Various cases of internal exposure to americium have been reported in which the exposures resulted from skin punctures with materials also containing plutonium. Information on the distribution of americium in these cases has been derived from the analysis of autopsy tissues. In most cases, the largest fraction of the ^{241}Am activity measured in the body was associated with tissues near the puncture wound. In one case, 18 years after a puncture wound to the left hand resulting in the deposition of a splinter of plutonium metal, 80% of the measured ^{241}Am activity (a product of plutonium decay) was associated with the left arm axillary lymph nodes and left hand, 12% was measured in the skeleton, and 1% was measured in the liver (Popplewell and Ham 1989). The large amount of activity associated with the left arm lymph nodes and left hand reflects the local accumulation of americium near the wound site. However, if such local accumulations are subtracted from the total body activity, then most of the systemic americium measured after puncture wound exposures has been associated with the skeleton (80–90%) while liver, muscle, and spleen account for the remainder (5–10%), with skeletal:liver ratios of approximately 9:1 (Lagerquist et al. 1972; McInroy et al. 1989; Popplewell and Ham 1989). These observations are consistent with those made from autopsy analyses after inhalation exposures (McInroy et al. 1989).

A large degree of variation is apparent in retention rates for americium in the liver among various animal species (Durbin 1973), as indicated by measured or estimated liver clearance half-times of approximately 5–16 days in rats, 152 days in baboons, 1–10 years in dogs, and 10 years in Chinese hamsters. A liver clearance half-time of 2 years has been estimated for humans (Griffith et al. 1983). Refer to Section 3.5.1 for information regarding toxicokinetic mechanisms that may play a role in interspecies differences in liver retention of americium.

3.4.3 Metabolism

3.4.3.1 Inhalation Exposure

The metabolism of americium consists of binding interactions with proteins and probably complex formation with various inorganic anions such as carbonate and phosphate, and carboxylic acids such as citrate and lactate (Durbin 1973; Taylor 1973; Webb et al. 1998). These types of interactions would be expected for all routes of exposure.

3.4.3.2 Oral Exposure

See Section 3.4.3.1.

3.4.3.3 Dermal Exposure

See Section 3.4.3.1.

3.4.4 Elimination and Excretion

3.4.4.1 Inhalation Exposure

Inhaled americium that is deposited in the nasopharyngeal or tracheal-bronchial regions of the respiratory tract will undergo extensive mucociliary transport to the esophagus with subsequent ingestion and fecal excretion. Evidence for this in humans is provided by cases of accidental inhalation exposure of americium oxides in which a substantial fraction (>50%) of the estimated initially deposited activity was found to be excreted in feces during the first days to weeks after the exposure (Edvardsson and Lindgren 1976; Newton et al. 1983; Sanders 1974). Once absorbed from the lung, americium is excreted in feces and urine. The relative contribution of each in humans is difficult to assess from existing data because most data are derived from accidental exposures, after which the victims were subjected to chelation therapy which would have increased the urinary excretion of americium. Following an accidental inhalation exposure in which approximately 1.22 μCi (45.1 kBq) of ^{244}Cm and ^{241}Am (in the form of mixed oxides) were deposited in the respiratory tract, the cumulative 1-year excretion of radioactivity was approximately 1,174 μCi (43.4 kBq, 96% of the deposited activity) in feces and 5.4 nCi (200 Bq, 0.4% of the deposited activity) in urine (Sanders 1974). The equivalent fractions of ^{241}Am, based on

measurements of the ^{244}Cm:^{241}Am ratios in feces and urine, were similar, 96% in feces, and 0.4% urine. The subject received 14 treatments with the chelating agent, DTPA, which may have influenced rates of excretion of americium.

Studies of excretion in animals after inhalation exposures also indicate a relatively large contribution of fecal excretion to the total elimination of americium that deposits in the respiratory tract. In dogs that inhaled an aerosol of AmO_2, fecal and urinary excretion of americium during the first 10 days after exposure were 30 and 0.7% of the initial body burden, respectively, and were 50 and 6% of the initial burden, respectively, by 810 days post exposure (Craig et al. 1979). Mechanical clearance from the upper respiratory tract to the gastrointestinal tract would be expected to make a substantial contribution to fecal excretion during the first few weeks after exposure and may continue for longer periods. The observation that fecal excretion continued to be the main route of excretion months after the exposure suggests two possibilities: (1) mechanical clearance from the lower respiratory tract (e.g., lung) continues to contribute to fecal excretion for longer periods of time (ICRP 1994b), and (2) absorbed americium is excreted in feces, suggesting a role for biliary excretion.

Lung deposition patterns and the relative contributions of the fecal and urinary pathways vary with the aerosol particle size. Evidence for this is provided by a study in which dogs inhaled AmO_2 aerosols having different size distributions. As the particle size AMAD increased from 0.8 μm (±1.2 GSD) to 3.5 μm (±1.06 GSD), the urine:fecal ratios of cumulative excretion (from days 8 to 730 after the exposure) decreased from 4 to 0.3 (Mewhinney et al. 1982). These observations are consistent with an effect of particle size on deposition patterns in the respiratory tract. Larger particles will be deposited in the tracheo-bronchial airways and will be subjected to mucociliary transport to the esophagus where they can enter the gastrointestinal tract. Therefore, the fraction of the deposited activity transferred to the gastrointestinal tract and excreted in the feces would be expected to increase with increasing mean particle size of the inhaled aerosol. The general trend of increased fecal excretion with increasing particle size would be expected to occur in humans, although the exact relationship between particle size and deposition patterns will vary across species because of species differences in airway geometry, breathing patterns, and air flow rates in the respiratory tract (see Section 3.4.5).

Once absorbed into the general circulation, regardless of the route of exposure, americium is excreted in both feces and urine. Evidence for this derives from experiments in animals that received an intravenous or intramuscular injection of americium (see Section 3.4.4.4).

3.4.4.2 Oral Exposure

Urinary excretion of ingested americium has been measured in humans who ingested shellfish (molluscs) that had assimilated americium from their aquatic environment. In eight adult subjects (six males and two females) who ingested molluscs (winkles) containing ^{241}Am (activity range 10–25 Bq, 0.27–0.68 nCi, 0.083–0.21 ng), the mean cumulative urinary excretion during the 7 days post ingestion was approximately 0.0005% and the estimated fraction of the absorbed activity excreted in the urine was 0.07% (Hunt et al. 1990). In a similar study of six subjects (five males and one female) who ingested cockles collected from Ravenglass, Cumbria (activity range of 15–17 Bq, 0.41–0.46 nCi, 0.13–0.14 ng), the mean cumulative urinary excretion during the 7 days post ingestion was approximately 0.0009% of the ingested americium and the estimated portion of the absorbed activity excreted in the urine was 0.08% (Hunt 1998). Three of these subjects had participated in the earlier study with winkles (Hunt et al. 1990); urinary excretion during 3 days prior to ingestion of the cockles was used to correct for internalized americium from prior exposures. Measured ^{241}Am activity in the cockles was indistinguishable from the cumulative activity measured in fecal samples collected from the subjects during the 7 days post ingestion.

Additional information on the excretion of ingested americium derives from an accident in which a worker ingested a ceramic particle containing $^{241}AmO_2$ (Smith et al. 1983). Five days after the accident, the particle, containing approximately 2.85 μCi (105 kBq) of ^{241}Am (0.88 μg Am), was excreted in the feces. Previous fecal samples and one subsequent fecal sample collected the day after the particle was excreted had essentially no ^{241}Am activity. During the first 8 days after the particle was ingested, approximately 4 pCi (0.15 Bq, 0.00007% of the estimated activity) was excreted in the urine.

Studies in rats and guinea pigs have shown that in these species, urinary excretion is the major route of excretion of americium absorbed from the gastrointestinal tract during the first week after exposure. In rats that were administered americium nitrate or citrate by gavage, 30–80% of the estimated absorbed activity was excreted in urine during the first 7 days post administration (Sullivan 1980a; Sullivan et al. 1985). The wide range may reflect the uncertainty in the estimates of the relatively low fraction absorption of the americium. The fraction of the absorbed activity that was excreted in urine was lower in iron-deficient rats (35%) than in iron replete rats (78%) (Sullivan et al. 1985). In guinea pigs, 35–50% of the absorbed activity was excreted in urine during the first 7 days after a single gavage dose of americium nitrate (Sullivan 1980a).

Once absorbed into the general circulation, regardless of the route of exposure, americium is excreted in both feces and urine. Evidence for this derives from experiments in animals that received an intravenous intramuscular injection of americium (see Section 3.4.4.4).

3.4.4.3 Dermal Exposure

Information on the excretion of americium after dermal exposure in humans or animals is extremely limited. Some qualitative information is available from an accidental exposure in which a worker received facial wounds from projectile debris and nitric acid during an explosion of a vessel containing ^{241}Am (McMurray 1983). The subject also inhaled ^{241}Am released to the air as dust and nitric acid aerosols, which was evident from external chest measurements of internal radioactivity; thus, excretion estimates reflect combined inhalation, dermal, and wound penetration exposures (Palmer et al. 1983). Measurements of cumulative fecal and urinary excretion of ^{241}Am during the first years after the accident, and periodic measurements made from day 10 to 11 years post accident indicated a fecal:urine excretion ratio of approximately 0.2–0.3, although the ratio was approximately 1 on day 3 post accident (Breitenstein and Palmer 1989; Robinson et al.1983). However, the ratio was almost certainly affected by initial chelation with Ca-DPTA, followed by daily intravenous therapy with the chelating agent, Zn-DPTA, treatments that would have increased the urinary excretion of americium (Breitenstein and Palmer 1989). The above not withstanding, the observations made on this subject demonstrate that fecal excretion was an important pathway of excretion in this subject long after mechanical clearance of americium from the respiratory tract would have been complete. This is consistent with observations made in nonhuman primates that show that americium is excreted into bile (see Section 3.4.4.4). However, the extent to which the biliary excretion pathway in humans might resemble that of nonhuman primates is not known.

Once absorbed into the general circulation, regardless of the route of exposure, americium is excreted in both feces and urine. Evidence for this derives from experiments in animals that received an intravenous intramuscular injection of americium (see Section 3.4.4.4).

3.4.4.4 Other Routes of Exposure

Measurements of fecal and urinary excretion of americium after an inhaled or ingested dose do not provide all of the information needed to assess the relative importance of either route in the excretion of absorbed americium. This is because inhaled americium is subject to mucociliary transport and ingestion,

and only a small fraction of ingested americium is absorbed. Studies in which animals were parenterally administered americium have provided quantitative information on the routes of excretion of absorbed americium. The relative contribution of the fecal and urinary pathways appear to vary with species and time post administration; however, in general, the fecal pathway predominates. In monkeys, urinary excretion of radioactivity following intravenously administered americium citrate was the major route of excretion only during the first few weeks post administration, at which time, cumulative urinary and fecal excretion accounted for 10 and 3% of the administered activity, respectively (Durbin 1973). Long-term cumulative excretion (months to years), however, was primarily in feces, with 37% of the administered activity excreted in feces and 20% in urine. A similar pattern of urinary and fecal excretion over time was evident in baboons (Cohen and Wrenn 1973; Durbin 1973; Guilmette et al. 1980). Similarly, in rats, dogs, and hamsters, urinary excretion of radioactivity following intramuscularly or intravenously injected americium citrate or chloride was the major route of excretion only during the first few days to weeks, with a fecal:urine excretion ratio of approximately 0.11–0.15; however, over a 1–2 year period, the fecal:urine ratio was approximately 5–9 (Durbin 1973; Stather et al. 1979a).

The large contribution of the fecal route to excretion of absorbed americium appears to be the result of excretion of americium into the bile. In monkeys that received an intravenous injection of americium citrate, ^{241}Am was detected in gall bladder bile and its concentration increased as the relative rate of fecal excretion increased over time post injection (Durbin 1973). Durbin (1973) estimated that at bile production rates similar to humans, biliary excretion could have accounted for most, if not all, of the fecal excretion of americium observed in the monkeys.

3.4.5 Physiologically Based Pharmacokinetic (PBPK)/Pharmacodynamic (PD) Models

Physiologically based pharmacokinetic (PBPK) models use mathematical descriptions of the uptake and disposition of chemical substances to quantitatively describe the relationships among critical biological processes (Krishnan et al. 1994). PBPK models are also called biologically based tissue dosimetry models. PBPK models are increasingly used in risk assessments, primarily to predict the concentration of potentially toxic moieties of a chemical that will be delivered to any given target tissue following various combinations of route, dose level, and test species (Clewell and Andersen 1985). Physiologically based pharmacodynamic (PBPD) models use mathematical descriptions of the dose-response function to quantitatively describe the relationship between target tissue dose and toxic end points.

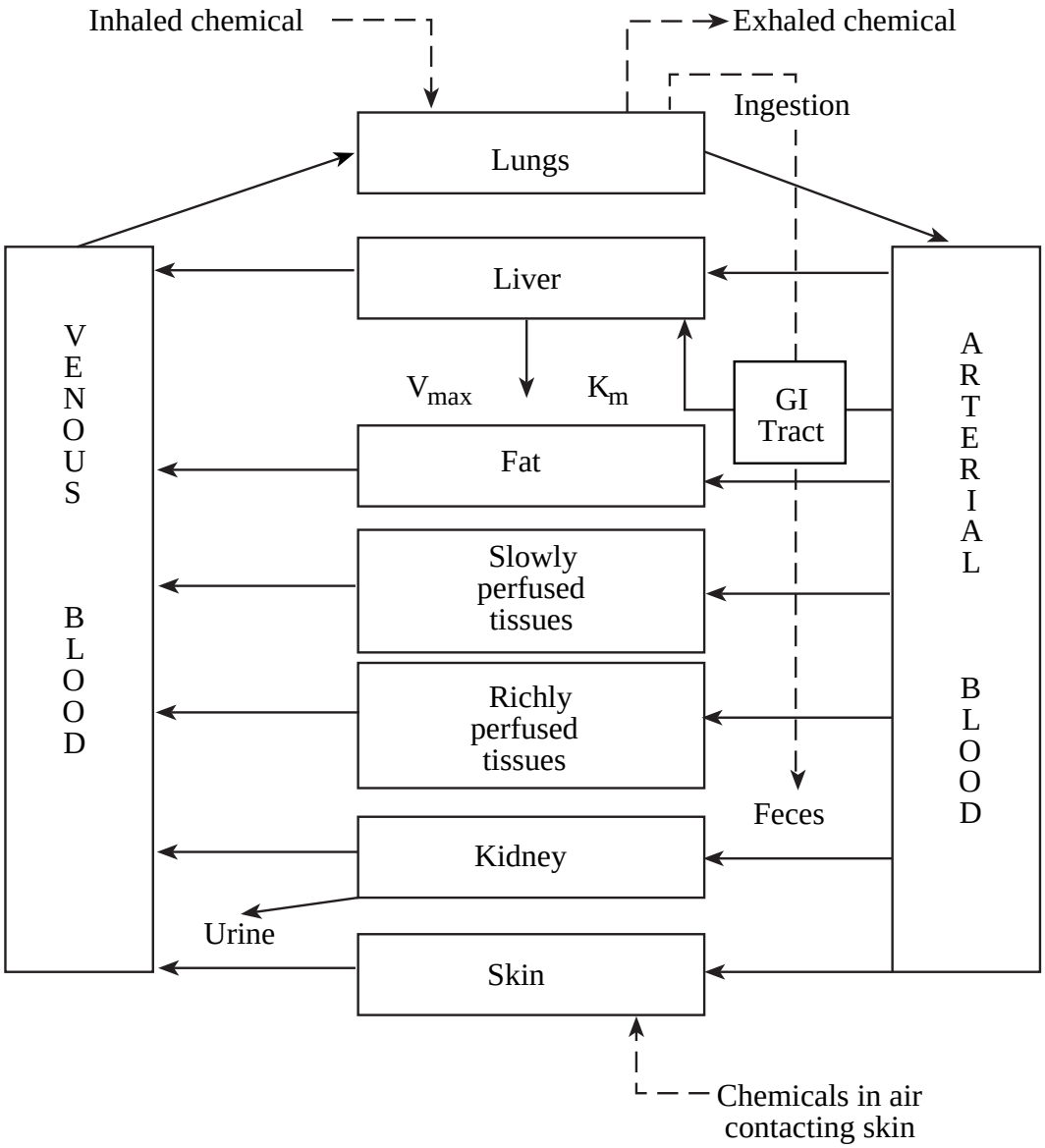
PBPK/PD models refine our understanding of complex quantitative dose behaviors by helping to delineate and characterize the relationships between: (1) the external/exposure concentration and target tissue dose of the toxic moiety, and (2) the target tissue dose and observed responses (Andersen et al. 1987; Andersen and Krishnan 1994). These models are biologically and mechanistically based and can be used to extrapolate the pharmacokinetic behavior of chemical substances from high to low dose, from route to route, between species, and between subpopulations within a species. The biological basis of PBPK models results in more meaningful extrapolations than those generated with the more conventional use of uncertainty factors.

The PBPK model for a chemical substance is developed in four interconnected steps: (1) model representation, (2) model parametrization, (3) model simulation, and (4) model validation (Krishnan and Andersen 1994). In the early 1990s, validated PBPK models were developed for a number of toxicologically important chemical substances, both volatile and nonvolatile (Krishnan and Andersen 1994; Leung 1993). PBPK models for a particular substance require estimates of the chemical substance-specific physicochemical parameters, and species-specific physiological and biological parameters. The numerical estimates of these model parameters are incorporated within a set of differential and algebraic equations that describe the pharmacokinetic processes. Solving these differential and algebraic equations provides the predictions of tissue dose. Computers then provide process simulations based on these solutions.

The structure and mathematical expressions used in PBPK models significantly simplify the true complexities of biological systems. If the uptake and disposition of the chemical substance(s) is adequately described, however, this simplification is desirable because data are often unavailable for many biological processes. A simplified scheme reduces the magnitude of cumulative uncertainty. The adequacy of the model is, therefore, of great importance, and model validation is essential to the use of PBPK models in risk assessment.

PBPK models improve the pharmacokinetic extrapolations used in risk assessments that identify the maximal (i.e., the safe) levels for human exposure to chemical substances (Andersen and Krishnan 1994). PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals in humans who are exposed to environmental levels (for example, levels that might occur at hazardous waste sites) based on the results of studies where doses were higher or were administered in different species. Figure 3-1 shows a conceptualized representation of a PBPK model.

Figure 3-1. Conceptual Representation of a Physiologically Based Pharmacokinetic (PBPK) Model for a Hypothetical Chemical Substance



Note: This is a conceptual representation of a physiologically based pharmacokinetic (PBPK) model for a hypothetical chemical substance. The chemical substance is shown to be absorbed via the skin, by inhalation, or by ingestion, metabolized in the liver, and excreted in the urine or by exhalation.

Source: adapted from Krishnan et al. 1994

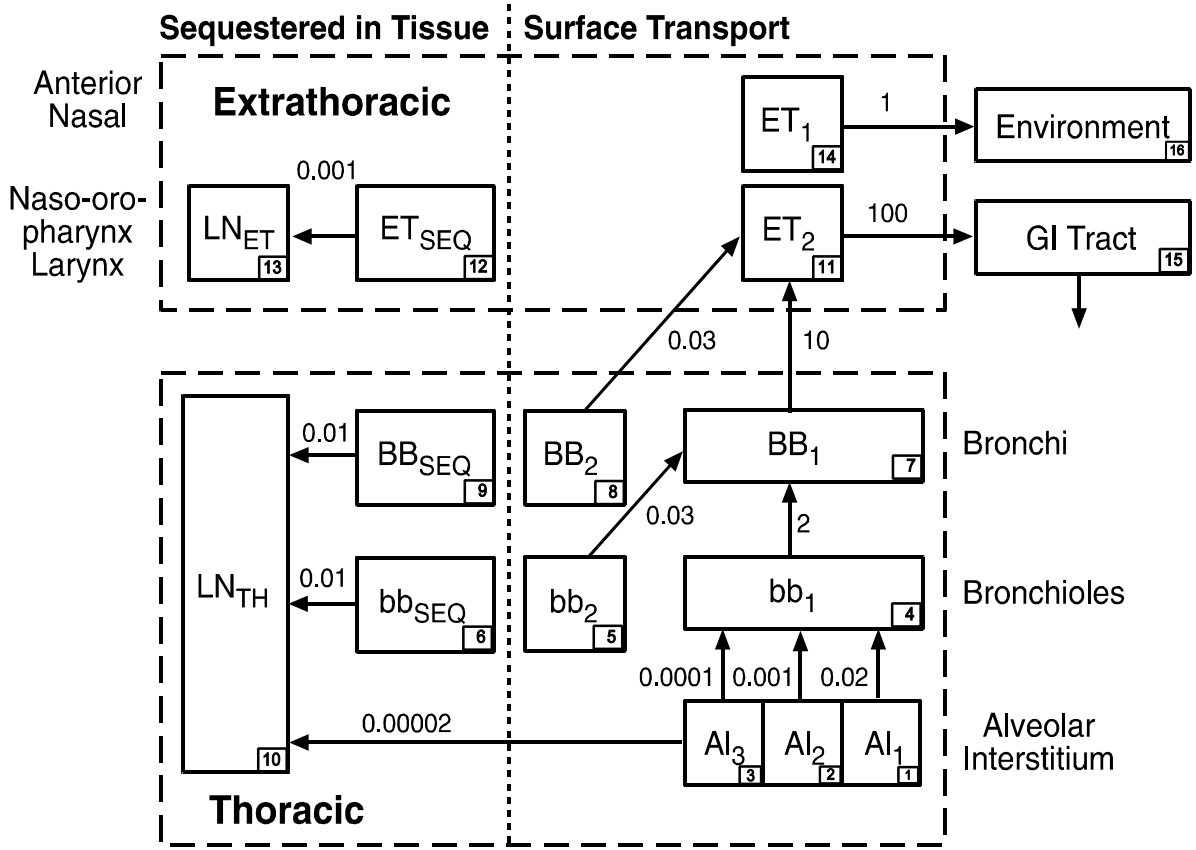
The ICRP (1994b, 1995) developed a Human Respiratory Tract Model for Radiological Protection, which contains respiratory tract deposition and clearance compartmental models for inhalation exposure that may be applied to particulate aerosols of americium compounds. The ICRP (1986, 1989) has a biokinetic model for human oral exposure that applies to americium. The National Council on Radiation Protection and Measurement (NCRP) has also developed a respiratory tract model for inhaled radionuclides (NCRP 1997). At this time, the NCRP recommends the use of the ICRP model for calculating exposures for radiation workers and the general public. Readers interested in this topic are referred to NCRP Report No. 125; *Deposition, Retention and Dosimetry of Inhaled Radioactive Substances* (NCRP 1997). In the appendix to the report, NCRP provides the animal testing clearance data and equations fitting the data that supported the development of the human mode for americium.

Human Respiratory Tract Model for Radiological Protection (ICRP 1994b)

Deposition. The ICRP (1994b) has developed a deposition model for behavior of aerosols and vapors in the respiratory tract. It was developed to estimate the fractions of radioactivity in breathing air that are deposited in each anatomical region of the respiratory tract. ICRP (1994b) provides inhalation dose coefficients that can be used to estimate radiation doses to organs and tissues throughout the body based on a unit intake of radioactive material. The model applies to three levels of particle solubility, a wide range of particle sizes (approximately 0.0005–100 μm in diameter), and parameter values that can be adjusted for various segments of the population (e.g., sex, age, and level of physical exertion). This model also allows one to evaluate the bounds of uncertainty in deposition estimates. Uncertainties arise from natural biological variability among individuals and the need to interpret some experimental evidence that remains inconclusive. It is applicable to particulate aerosols containing americium, but was developed for a wide variety of radionuclides and their chemical forms.

The ICRP deposition model estimates the fraction of inhaled material initially retained in each compartment (see Figure 3-2). The model was developed with five compartments: (1) the anterior nasal passages (ET_1); (2) all other extrathoracic airways (ET_2) (posterior nasal passages, the naso- and oropharynx, and the larynx); (3) the bronchi (BB); (4) the bronchioles (bb); and (5) the alveolar interstitium (AI). Particles deposited in each of the regions may be removed and redistributed either upward into the respiratory tree or to the lymphatic system and blood by different particle removal mechanisms.

Figure 3-2. Respiratory Tract Compartments* in Which Particles May be Deposited



*Compartment numbers shown in lower right corners are used to define clearance pathways. The clearance pathways as well as the compartment abbreviations are presented in Table 3-5.

Source: ICRP 1994b

For extrathoracic deposition of particles, the model uses measured airway diameters and experimental data, where deposition is related to particle size and airflow parameters, and scales deposition for women and children from adult male data. Similar to the extrathoracic region, experimental data served as the basis for lung (bronchi, bronchioles, and alveoli) aerosol transport and deposition. A theoretical model of gas transport and particle deposition was used to interpret data and to predict deposition for compartments and subpopulations other than adult males. Table 3-4 provides reference respiratory values for the general Caucasian population during various intensities of physical exertion.

Deposition of inhaled gases and vapors is modeled as a partitioning process that depends on the physiological parameters noted above as well as the solubility and reactivity of a compound in the respiratory tract (see Figure 3-3). The ICRP (1994b) model defines three categories of solubility and reactivity: SR-0, SR-1, and SR-2:

- Type SR-0 compounds include insoluble and nonreactive gases (e.g., inert gases such as H_2, He). These compounds do not significantly interact with the respiratory tract tissues, and essentially all compound inhaled is exhaled. Radiation doses from inhalation exposure of SR-0 compounds are assumed to result from the irradiation of the respiratory tract from the air spaces.

- Type SR-1 compounds include soluble or reactive gases and vapors which are expected to be taken up by the respiratory tract tissues and may deposit in any or all of the regions of the respiratory tract, depending on the dynamics of the airways and properties of the surface mucous and airway tissues, as well as the solubility and reactivity of the compound.

- Type SR-2 compounds include soluble and reactive gases and vapors which are completely retained in the extrathoracic regions of the respiratory tract. SR-2 compounds include sulfur dioxide (SO_2) and hydrogen fluoride (HF).

Respiratory Tract Clearance. This portion of the model identifies the principal clearance pathways within the respiratory tract. The model was developed to predict the retention of various radioactive materials. Figure 3-4 presents the compartmental model and is linked to the deposition model (see Figure 3-2) and to reference values presented in Table 3-5. This table provides clearance rates, expressed as a fraction per day and also as half-time (Part A), and deposition fractions (Part B) for each compartment for insoluble particles. ICRP (1994b) also developed modifying factors for some of the parameters, such as age, smoking, and disease status. Parameters of the clearance model are based on

Table 3-4. Reference Respiratory Values for a General Caucasian Population at Different Levels of Activity[a]

Activity:	Resting (sleeping)			Sitting awake			Light exercise			Heavy exercise		
Maximal workload (%):	8			12			32			64		
Breathing parameters:[b]	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})
Age Sex												
3 months	0.04	0.09	38	N/A	N/A	N/A	0.07	0.19	48	N/A	N/A	N/A
1 year	0.07	0.15	34	0.1	0.22	36	0.13	0.35	46	N/A	N/A	N/A
5 years	0.17	0.24	23	0.21	0.32	25	0.24	0.57	39	N/A	N/A	N/A
10 years Male:										0.841	2.22	44
Female:										0.667	1.84	46
Both:	0.3	0.31	17	0.33	0.38	19	0.58	1.12	32			
15 years Male:	0.500	0.42	14	0.533	0.48	15	1.0	1.38	23	1.352	2.92	36
Female:	0.417	0.35	14	0.417	0.40	16	0.903	1.30	24	1.127	2.57	38
Adult Male:	0.625	0.45	12	0.750	0.54	12	1.25	1.5	20	1.923	3.0	26
Female:	0.444	0.32	12	0.464	0.39	14	0.992	1.25	21	1.364	2.7	33

[a]See Annex B (ICRP 1994b) for data from which these reference values were derived.

[b]V_T = tidal volume; B = ventilation rate; f_R = respiration frequency

h = hour; L = liters; m = meters; min = minute; N/A = not applicable

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Table 3-5. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

Part A

Clearance Rates for Insoluble Particles				
Pathway	From	To	Rate (d^{-1})	Half-time[a]
$m_{1,4}$	AI_1	bb_1	0.02	35 days
$m_{2,4}$	AI_2	bb_1	0.001	700 days
$m_{3,4}$	AI_3	bb_1	0.0001	7,000 days
$m_{3,10}$	AI_3	LN_{TH}	0.00002	—
$m_{4,7}$	bb_1	BB_1	2	8 hours
$m_{5,7}$	bb_2	BB_1	0.03	23 days
$m_{6,10}$	bb_{seq}	LN_{TH}	0.01	70 days
$m_{7,11}$	BB_1	ET_2	10	100 minutes
$m_{8,11}$	BB_2	ET_2	0.03	23 days
$m_{9,10}$	BB_{seq}	LN_{TH}	0.01	70 days
$m_{11,15}$	ET_2	GI tract	100	10 minutes
$m_{12,13}$	ET_{seq}	LN_{ET}	0.001	700 days
$m_{14,16}$	ET_1	Environment	1	17 hours

See next page for Part B

Table 3-5. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract (*continued*)

Part B

Partition of deposit in each region between compartments[b]

Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment[c]
ET_2	ET_2	0.9995
	ET_{seq}	0.0005
BB	BB_1	0.993-f_s
	BB_2	f_s
	BB_{seq}	0.007
bb	bb_1	0.993-f_s
	bb_2	f_s
	bb_{seq}	0.007
AI	AI_1	0.3
	AI_2	0.6
	AI_3	0.1

[a]The half-times are approximate since the reference values are specified for the particle transport rates and are rounded in units of d^{-1}. A half-time is not given for the transport rate from AI_3 to LN_{TH}, since this rate was chosen to direct the required amount of material to the lymph nodes. The clearance half-time of compartment AI_3 is determined by the sum of the clearance rates from it.
[b]See paragraph 181, Chapter 5 (ICRP 1994) for default values used for relating f_s to d_{ae}.
[c]It is assumed that f_s is size-dependent. For modeling purposes, f_s is taken to be:

$$f_s = 0.5 \;\; for \; d_{ae} \le 2.5\sqrt{\rho / \chi} \;\; \mu m \;\; and$$

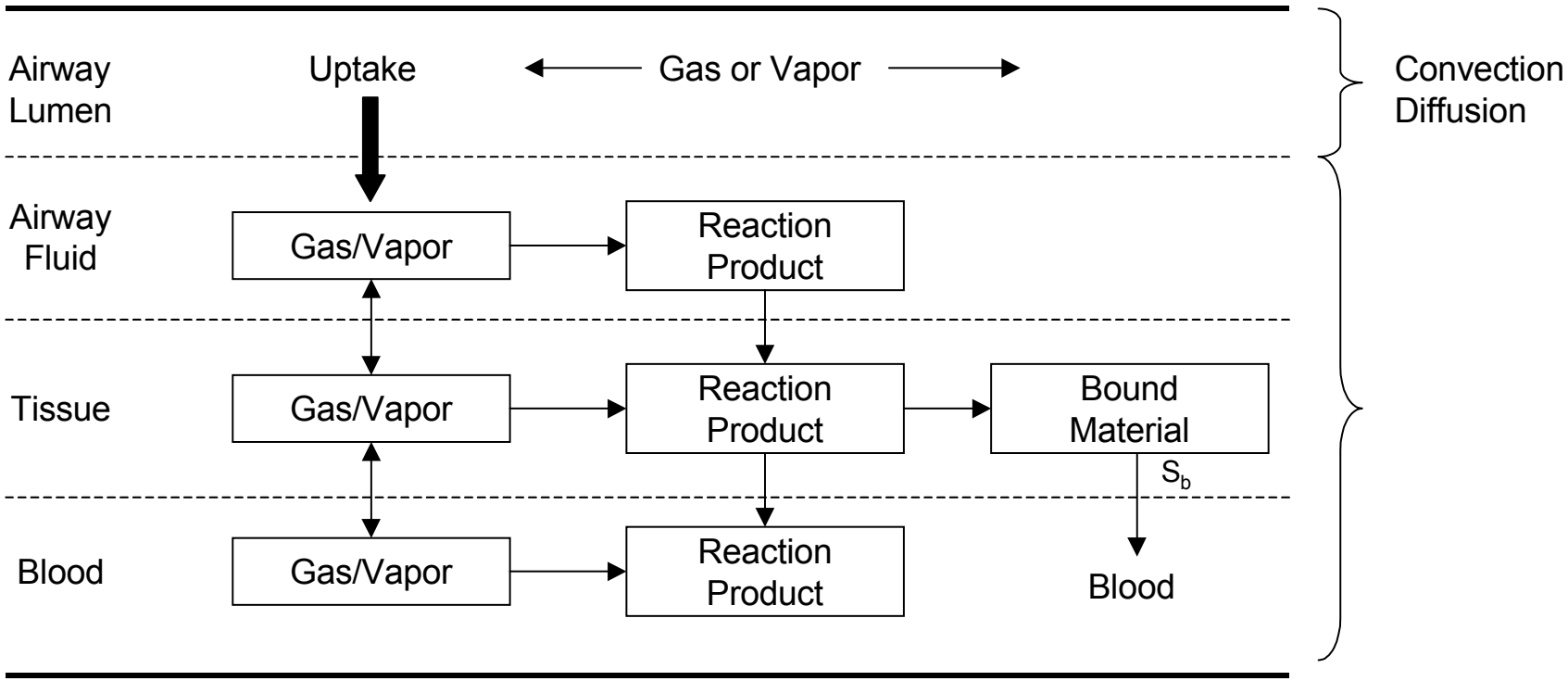
$$f_s = 0.5e^{0.63(d_{ae}\sqrt{\rho/\chi}-2.5)} \;\; for \; d_{ae} > 2.5\sqrt{\rho / \chi} \;\; \mu m$$

where
f_s = fraction subject to slow clearance
d_{ae} = aerodynamic particle diameter/(μm)
ρ = particle density (g/cm^3)
χ = particle shape factor

AI = alveolar-interstitial region; BB = bronchial region; bb = bronchiolar region; BB_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchial region; bb_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchiolar region; d = day(s); ET = extrathoracic region; ET_{seq} = compartment representing prolonged retention in airway tissue of small fraction of particles deposited in the nasal passages; LN_{ET} = lymphatics and lymph nodes that drain the extrathoracic region; LN_{TH} = lymphatics and lymph nodes that drain the thoracic region

Source: ICRP 1994

Figure 3-3. Reaction of Gases or Vapors at Various Levels of the Gas-Blood Interface



Source: ICRP 1994b

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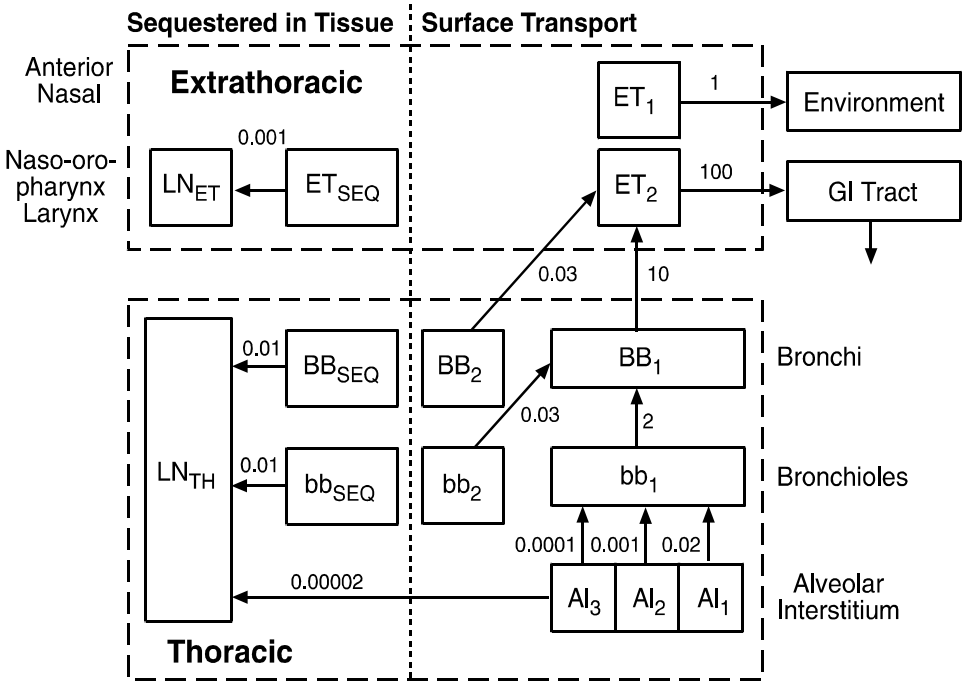
Figure 3-4. Compartment Model to Represent Time-Dependent Particle Transport in the Respiratory Tract



Source: ICRP 1994b

See Table 3-5 for abbreviations, rates, half-lives, and fractions by compartment.

human evidence for the most part, although particle retention in airway walls is based on experimental data from animal experiments.

The clearance of particles from the respiratory tract is a dynamic process. The rate of clearance generally changes with time from each region and by each route. Following deposition of large numbers of particles (acute exposure), transport rates change as particles are cleared from the various regions. Physical and chemical properties of deposited material determine the rate of dissolution and as particles dissolve, absorption rates tend to change over time. By creating a model with compartments of different clearance rates within each region (e.g., BB_1, BB_2, BB_{seq}), the ICRP model overcomes problems associated with time-dependent functions. Each compartment clears to other compartments by constant rates for each pathway.

Particle transport from all regions is toward both the lymph nodes and the pharynx, and a majority of deposited particles end up being swallowed. In the front part of the nasal passages (ET_1), nose blowing, sneezing, and wiping remove most of the deposited particles. Particles remain here for about a day. For particles with AMADs a few micrometers or greater, the ET_1 compartment is probably the largest deposition site. A majority of particles deposited at the back of the nasal passages and in the larynx (ET_2) are removed quickly by the fluids that cover the airways. In this region, particle clearance is completed within 15 minutes.

Ciliary action removes deposited particles from both the bronchi and bronchioles. Though it is generally thought that mucocilliary action rapidly transports most particles deposited here toward the pharynx, a fraction of these particles is cleared more slowly. Evidence for this is found in human studies. For humans, retention of particles deposited in the lungs (BB and bb) is apparently biphasic. The “slow” action of the cilia may remove as much as half of the bronchi- and bronchiole-deposited particles. In human bronchi and bronchiole regions, mucus moves more slowly, when it is closer to the alveoli. For the faster compartment, it has been estimated that it takes about 2 days for particles to travel from the bronchioles to the bronchi and 10 days from the bronchi to the pharynx. The second (slower) compartment is assumed to have approximately equal fractions deposited between BB_2 and bb_2 ,with both fractions having clearance half-times estimated at 20 days. Particle size is a primary determinant of the fraction deposited in this slow thoracic compartment. A small fraction of particles deposited in the BB and bb regions is retained in the airway wall for even longer periods (BB_{seq} and bb_{seq}).

If particles reach and become deposited in the alveoli, they tend to stay imbedded in the fluid on the alveolar surface or move into the lymph nodes. The one mechanism by which particles are physically resuspended and removed from the AI region is coughing. For modeling purposes, the AI region is divided into three subcompartments to represent different clearance rates, all of which are slow.

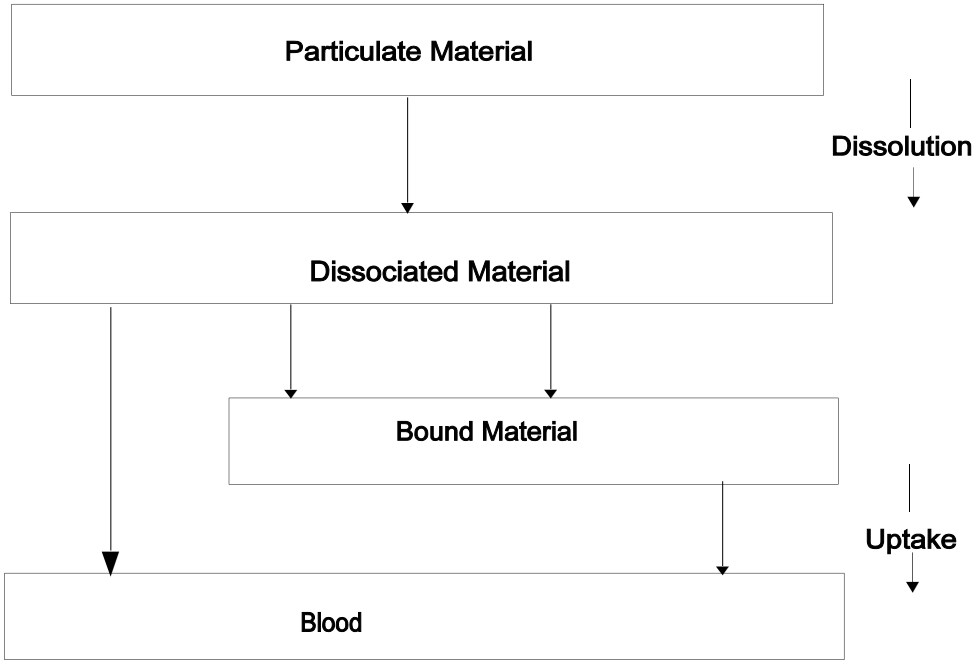
In the alveolar-interstitial region, human lung clearance has been measured. The ICRP model uses two half-times to represent clearance: about 30% of the particles have a 30-day half-time, and the remaining 70% are assigned a half-time of several hundred days. Over time, AI particle transport falls, and some compounds have been found in lungs 10–50 years after exposure.

Absorption into Blood. The ICRP model assumes that absorption into blood occurs at equivalent rates in all parts of the respiratory tract, except in the anterior nasal passages (ET_1), where no absorption occurs. It is essentially a 2-stage process, as shown in Figure 3-5. First, there is a dissociation (dissolution) of particles; then the dissolved molecules or ions diffuse across capillary walls and are taken up by the blood. Immediately following dissolution, rapid absorption is observed. For some elements, rapid absorption does not occur because of binding to respiratory-tract components. In the absence of specific data for specific compounds, the model uses the following default absorption rate values for those specific compounds that are classified as Types F (fast), M (medium), S (slow), and V (instantaneous):

- For Type F, there is rapid 100% absorption within 10 minutes of the material deposited in the BB, bb, and AI regions, and 50% of material deposited in ET_2. Thus, for nose breathing, there is rapid absorption of approximately 25% of the deposit in ET; for mouth breathing, the value is 50%.
- For Type M, about 70% of the deposit in AI reaches the blood eventually. There is rapid absorption of about 10% of the deposit in BB and bb, and 5% of material deposited in ET_2. Thus, there is rapid absorption of approximately 2.5% of the deposit in ET for nose breathing, and 5% for mouth breathing.
- For Type S, 0.1% is absorbed within 10 minutes and 99.9% is absorbed within 7,000 days, so there is little absorption from ET, BB, or bb, and about 10% of the deposit in AI reaches the blood eventually.
- For Type V, complete absorption (100%) is considered to occur instantaneously.

ICRP (1995) considers the experimental data on americium nitrate, chloride, citrate, and hydroxide to support classification of these compounds as either Type F or M. Americium oxides are classified as Type M. ICRP (1995) recommends assigning all americium aerosols to Type M in the absence of specific information supporting an alternative classification.

Figure 3-5. The Human Respiratory Tract Model: Absorption into Blood



Source: ICRP 1994b

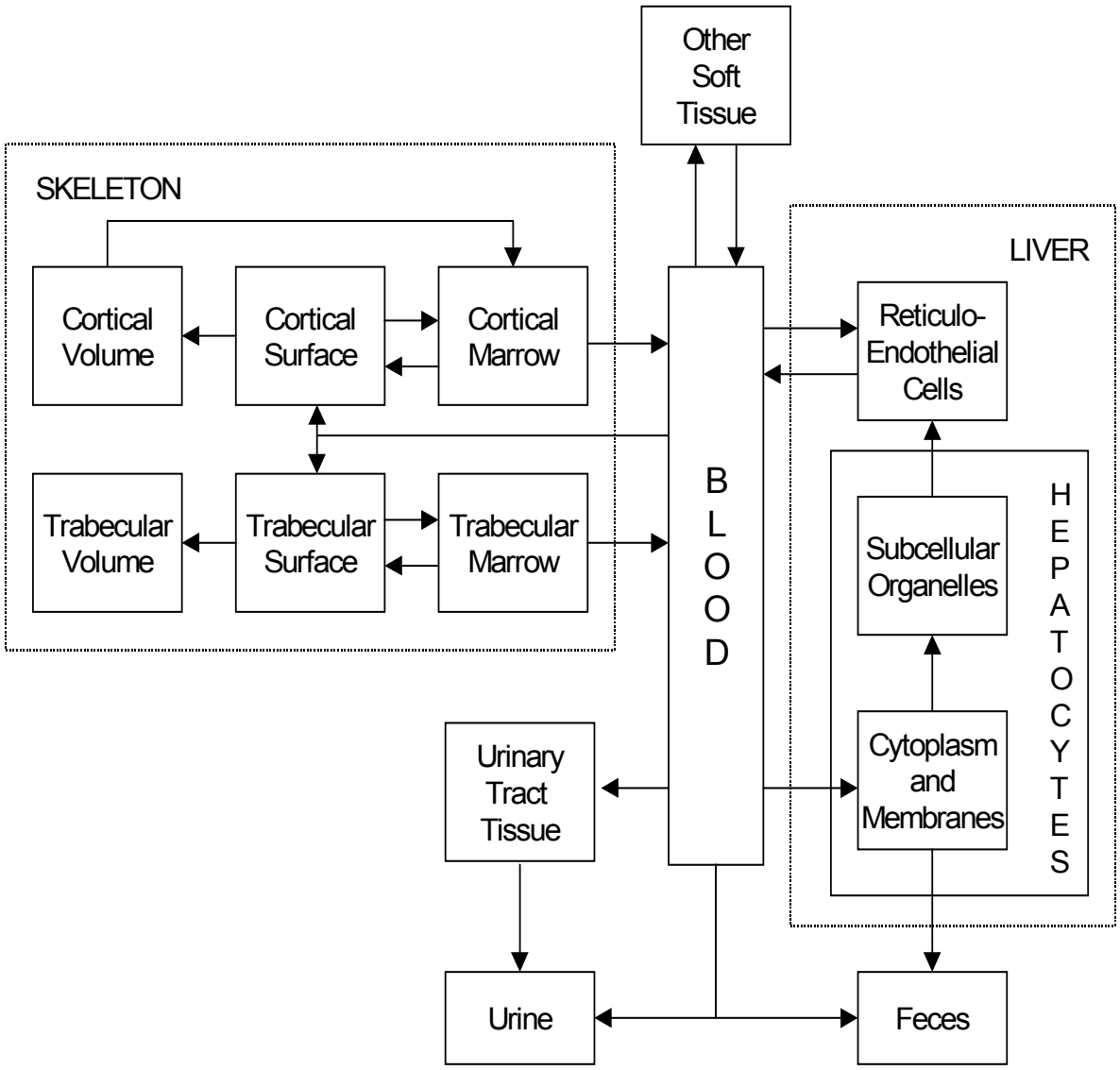
ICRP (1989) Americium Biokinetics Model

Description of the model.

ICRP (1989) developed a compartmental model of the kinetics of ingested americium in humans that is applicable to infants, children, adolescents, and adults. The model is a modification and expansion of a similar model for plutonium, described by Leggett (1984, 1985). The fraction of ingested americium that is absorbed (uptake to blood) is assumed to vary with age and have values of 0.01 in infants up to 12 months of age, and 0.001 from 12 months of age through adulthood (ICRP 1986). Absorbed americium enters the blood plasma where it distributes to the skeleton, liver, and other tissues (Figure 3-6). Excretion pathways included in the model are plasma to urine and liver to feces. Transfer rate coefficients between compartments are age-specific and, depending on the specific coefficient, values can change at ages 3 months, 1 year, 5 years, 10 years, 15 years, and adult (>15 years). The model assumes that in adults, 50% of the americium that enters the body and is not excreted is transferred to liver and 30% is transferred to the skeleton. In newborns, 70% is assumed to be transferred to the skeleton and 10% to the liver. Skeletal deposition is assumed to distribute into two pools: 50% goes to the trabecular bone surface and 50% to the cortical bone surface. A first order rate coefficient for elimination of americium from liver to plasma is assumed to be 0.0019 day^{-1} (half-time, 365 days).

Bone is divided into trabecular and cortical components, with each further divided into surface bone, bone volume, and bone cavity (marrow compartment). Deposition of americium is assumed to occur from plasma directly to bone surfaces, whereas elimination from bone occurs by transfer from the bone surface or volume to the marrow cavity, and then from the marrow cavity to plasma (Figure 3-6). Transfers of americium within the cortical or trabecular bone compartments are modeled based on assumptions about rates of bone formation and resorption, which are assumed to be vary with age, but are equal within a given age group (Leggett et al. 1982). Movement of americium to the marrow compartment is determined by the bone resorption rate, whereas, movement from the bone surface to the bone volume is assumed to occur by burial of surface deposits with new bone and is determined by the bone formation rate. During growth, bone formation and resorption are assumed to occur at different sites within bone; therefore, the rate of removal of americium from the bone surface is approximated by the sum of the bone resorption rate (represented in the model by the movement of americium to the marrow compartment) and the rate of bone formation which results in burial of surface deposits (represented by movement of americium from the bone surface to bone volume). In adults, the possibility of resorption and formation

Figure 3-6. ICRP (1989) Model of Americium Biokinetics



of bone occurring at the same site is assumed; therefore, only a portion of the bone formation rate results in burial of surface deposits and movement of americium from the bone surface to the bone volume.

Validation of the model.

The extent to which the ICRP model has been validated is not described in ICRP (1989).

Risk assessment.

The model has been used to establish the radiation dose (Sv) per unit of ingested ^{241}Am activity (Bq) for ages 3 months to 70 years (ICRP 1989).

Target tissues.

The model is designed to calculate the ^{241}Am intake that would produce the maximum allowed occupational radiation dose to all major organs, including the bone surfaces, bone marrow, and liver, but the conversion factors for other tissues and organs are published in the same tables.

Species extrapolation.

The model is based on both human and animal data. However, it is intended for applications to human dosimetry. Applications to other species would require consideration of species-specific adjustments in modal parameters.

Interroute extrapolation.

The model is designed to simulate oral exposures to americium. It can be applied to any other route of exposure for which the transfer rate to blood is available.

Leggett (1992) Americium Biokinetics Model

Description of the model.

Leggett (1992) proposed a model for the retention and excretion of americium in humans. The model is a modification of a similar model for plutonium (Leggett 1984, 1985) with the following changes (Figure 3-7): (1) the liver is represented as a single pool rather than as having distinct pools for hepatocytes and other cell types; (2) the gonads are represented by a separate compartment; (3) a kidney compartment is included, in place of the compartment for urinary tract tissue; (4) a compartment representing the urinary bladder contents is included; and (5) other soft tissues are represented as having slow, intermediate, and fast turnover pools.

The fraction of ingested americium that is absorbed (uptake from the small intestine to blood) is represented by age-specific functions that yield the following values: decreasing from 0.05 to 0.0005 during the first 12 months, 0.0005 from age 1 to 15 years, decreasing from 0.0005 to 0.0002 from age 15 to 18 years, and 0.0002 from age 18 years through adulthood. The model assumes that in adults, 62% of the americium that enters the body and is not excreted is transferred to liver and 38% is transferred to the skeleton (skeleton:liver deposition ratio, 3:5). In newborns, 88% is assumed to be transferred to the skeleton and 12% to the liver (skeleton:liver deposition ratio, 7:1). The half-time for elimination of americium from liver is assumed to be 365 days.

Leggett (1992) also proposed a respiratory tract model. Deposition of americium particles, depending on their size, are assumed to deposit in three compartments representing extrathoracic, fast-clearing thoracic, and slow-clearing thoracic regions of the respiratory tract (Figure 3-8).

Validation of the model.

Predictions based on the model have been compared to observed urinary or fecal excretion of americium in humans. Model predictions agreed reasonably well with the empirical observations (Leggett 1992).

Figure 3-7. Leggett (1992) Model of Americium Biokinetics

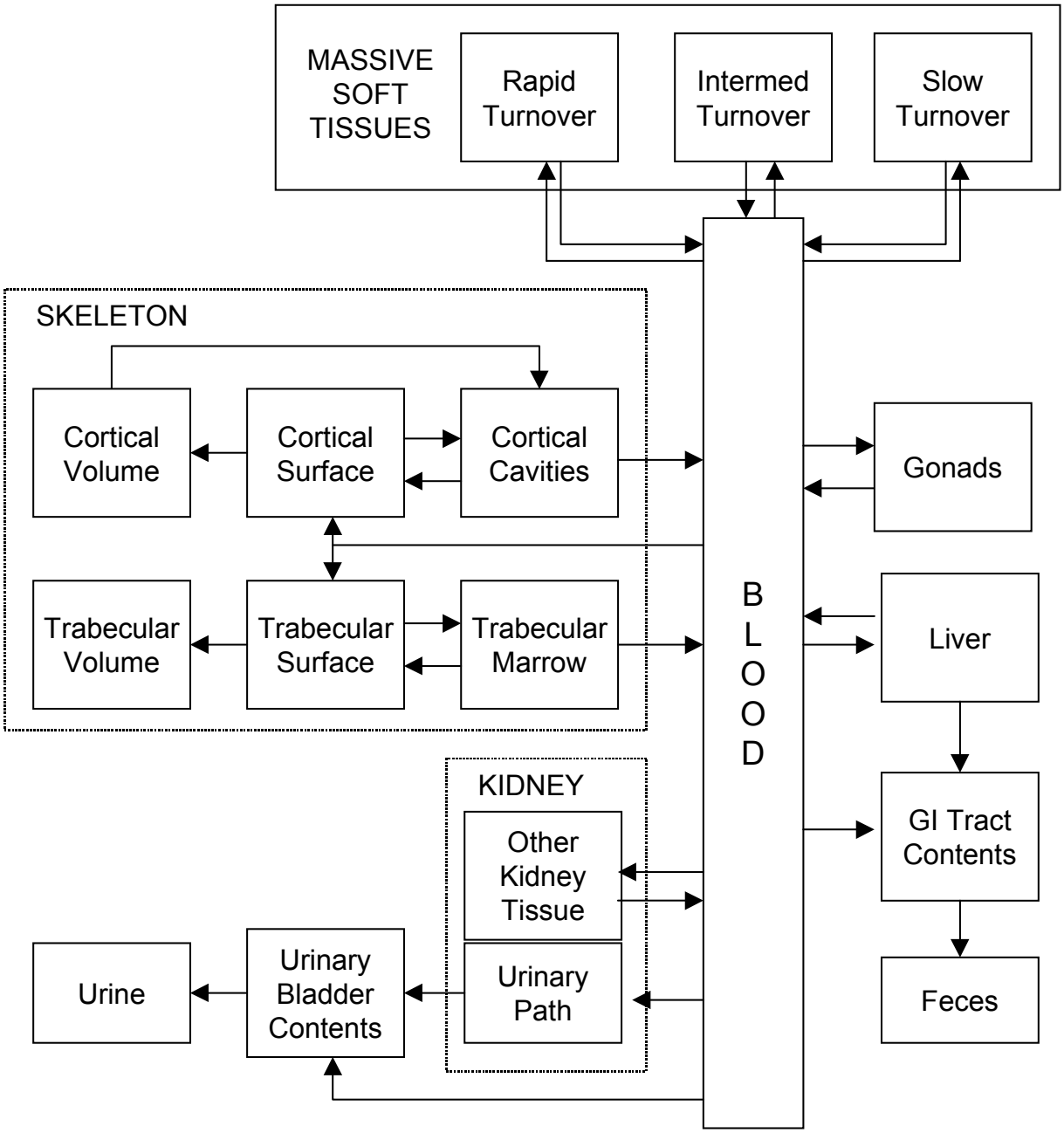
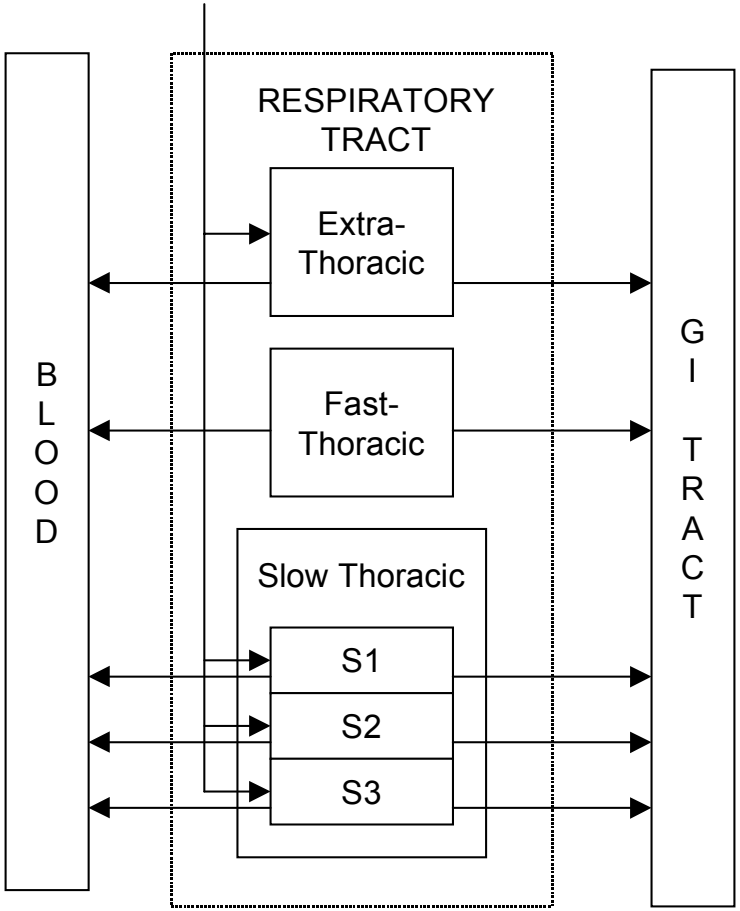


Figure 3-8. Leggett (1992) Model of Deposition and Retention of Americium in the Human Respiratory Tract



		AmO_2		Other Am	
Pool	To	$T_{1/2}$	f	$T_{1/2}$	f
Extra-thoracic	Blood	–	0.0	0.4 d	0.05
	GI Tract	0.4 d	1.0	0.4 d	0.95
Fast Thoracic	Blood	–	0.0	0.2 d	0.25
	GI Tract	0.2 d	1.0	0.2 d	0.75
Slow Thoracic					
S_1		(80%)		(45%)	
	Blood	11 d	0.90	15 h	0.80
	GI Tract	11 d	0.10	15 h	0.20
S_2		(17%)		(45%)	
	Blood	0.5 y	0.0	25 d	0.80
	GI Tract	0.5 y	1.0	25 d	0.20
S_3		(3%)		(10%)	
	Blood	3 y	0.20	500 d	0.20
	GI Tract	3 y	0.80	500 d	0.80

Risk assessment.

The Leggett (1992) model was developed to predict tissue doses and whole-body dose to people who may be exposed to americium. The model is considered an updated version of the ICRP (1989) model for americium, which has been used to establish risk-based limits of intake of ^{241}Am (ICRP 1989). The Leggett (1992) and ICRP (1989) models predict similar long-term average doses of americium to the liver and skeleton for an injection exposure and would be expected to predict similar radiation risks and risk-based intake limits (Leggett 1992). Descriptions of applications of the Leggett (1992) model in risk assessment have not been reported.

Target tissues.

The model is designed to calculate americium excretion and time courses for americium levels in the liver, skeleton, and gonads. This output could be used to predict radiation doses to these tissues.

Species extrapolation.

The model is designed for applications to human dosimetry and cannot be applied to other species without modification.

Interroute extrapolation.

The model is designed to simulate oral, inhalation, and parenteral (e.g., injection) exposures to americium and cannot be applied to other routes of exposure without modification.

USTUR (1994) Americium Biokinetics Model

Description of the model.

The USTUR proposed modifications to the ICRP americium model, based on post-mortem americium measurements in human exposure cases (Kathren 1994). The major modifications are: 1) the initial deposition fraction is assumed to be skeleton 45%, liver 25%, muscle 20%, other tissues 10%; 2) the half-times for elimination of americium were assumed to be 2.5 years in liver, 10 years in muscle, and 10 years in other tissues.

Validation of the model.

Predictions based on the model have been compared to observed post-mortem tissue americium levels in whole-body and tissue donations to the USTUR. Model predictions agreed reasonably well with the empirical observations (Kathren 1995; Kathren and Lynch 1996).

Risk assessment.

The USTUR (1994) model was developed to predict tissue doses and whole-body dose to people who may be exposed to americium. The model has been used to calculate Annual Limits of Intake (ALIs) for ^{241}Am, and yielded similar, but lower limits than those estimated using the ICRP model (1989).

Target tissues.

The model is designed to calculate americium excretion and time courses for americium levels in the liver, skeleton, and gonads. This output could be used to predict radiation doses to these tissues.

Species extrapolation.

The model is designed for application to human dosimetry and cannot be applied to other species without modification.

Interroute extrapolation.

The model is designed to simulate oral, inhalation, and parenteral (e.g., injection) exposures to americium and cannot be applied to other routes of exposure without modification.

Mewhinney and Griffith (1983) Americium Biokinetics Model

Description of the model.

Mewhinney and Griffith (1983) proposed a model for the absorption and tissue distribution of inhaled americium dioxide in humans. The model is a modification of a model of the beagle dog (Mewhinney and Griffith (1982), and formed part of the basis for the Leggett (1992) model. The model is similar to

other actinide models (ICRP 1989; Leggett 1992), but has several additional features in the respiratory tract portion of the model (Figure 3-9) including (1) dissolution of particles in the respiratory tract is simulated with rate functions, rather than zero or first-order rate constants, that include various parameters of particle size and surface area that affect dissolution rate (Mercer 1967); (2) the pulmonary region includes three pools; a pool of deposited particulate; a pool of dissolved americium which can be rapidly absorbed into the blood; and a pool of locally bound americium (transformed) which can be absorbed into blood or mechanically cleared to the tracheobronchial region; and (3) absorption from the pulmonary region of the respiratory tract includes a pathway for transfer to tracheobroncheal lymph nodes.

Excretion is assumed to be in feces, from either the intestine or from liver to the intestine, and from blood to urine (i.e., the kidney is represented as a distribution compartment). An absorption fraction of 0.0005 is assumed for americium in the small intestine

Validation of the model.

Predictions based on the model have been compared to observed time courses for lung, liver, and skeletal americium burden in dogs that inhaled americium oxide (Mewhinney and Griffith 1983). Data on lung retention for four humans who accidentally inhaled americium were also compared to model predictions. The empirical observations fell within predicted retention patterns for particle sizes (AMAD) 0.5 and 1.8 μm (Mewhinney and Griffith 1983).

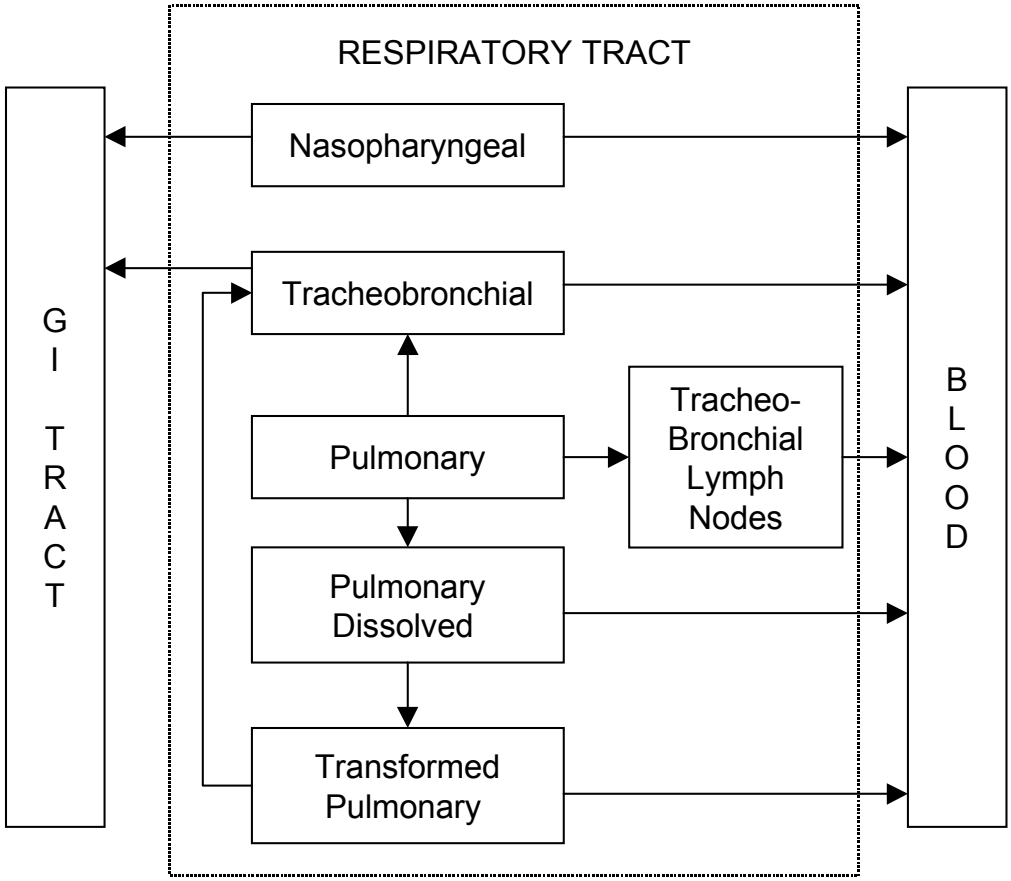
Risk assessment.

The Mewhinney and Griffith (1983) model was developed to predict lung retention and tissue distributions of americium in people who may be exposed to americium. Descriptions of applications of the model in risk assessment have not been reported.

Target tissues.

The model calculates americium burdens in lung, liver, skeleton, kidney, and body. This output could be used to predict radiation doses to these tissues.

Figure 3-9. Mewhinney and Griffith (1983) Model of Deposition and Retention of Americium in the Human Respiratory Tract



Species extrapolation.

The model is designed for applications to human dosimetry; however, it predicts reasonably well the retention of americium in beagle dogs as well (Mewhinney and Griffith 1982). The model cannot be applied to other species without modification.

Interroute extrapolation.

The model is designed to simulate inhalation exposures to americium; however, it could be applied to ingestion exposures since the model simulates the absorption of americium from the gastrointestinal tract. The model cannot be applied to other routes of exposure without modification.

Durbin and Schmidt (1985) Americium Biokinetics Model

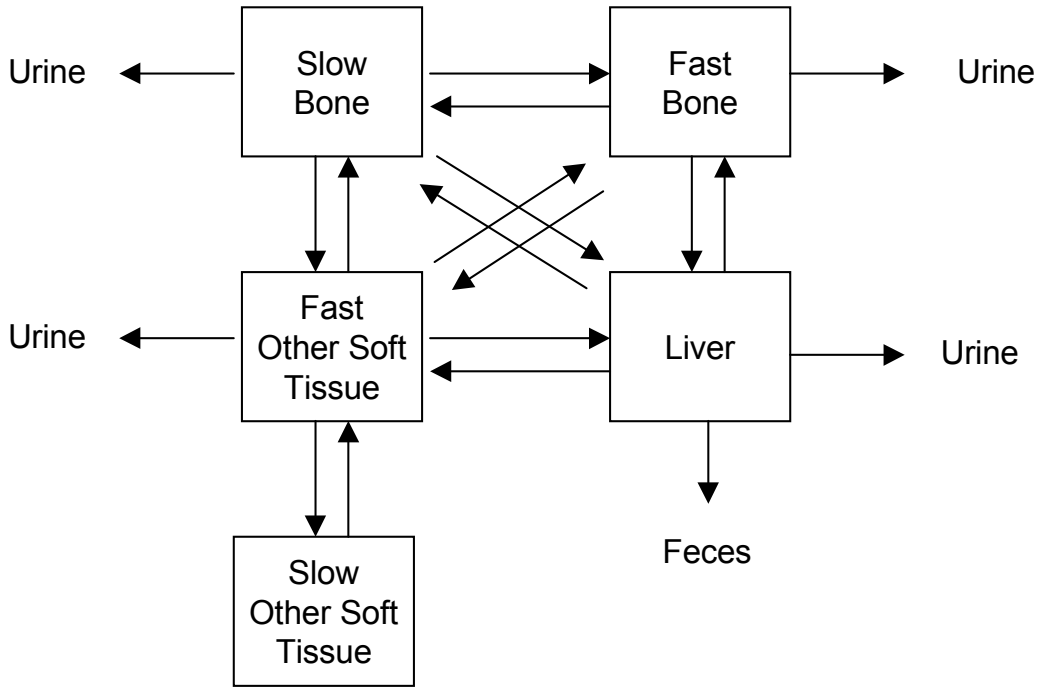
Description of the model.

Durbin and Schmidt (1985) proposed a model for tissue distribution and excretion of absorbed americium in humans. A unique feature of this model is that transfers from plasma to tissues are assumed to be instantaneous; therefore, a central plasma (and blood) compartment is not included in the model (see Figure 3-10). Tissue compartments included in the model are slow and fast turnover bone compartments, representing cortical and trabecular bone, respectively; liver; and slow and fast turnover for other soft tissue compartments. Excretion pathways include urine and feces. Urinary excretion is represented as a sum of the contributions from bone, liver, and other soft tissues. Fecal americium is assumed to be excreted from the liver.

Validation of the model.

Predicted skeletal americium concentrations were compared to values observed in a USTUR case and the predicted and observed values agreed reasonably well. However, the same USTUR case was used to derive values for model parameters and, therefore, agreement with the observations would be expected. No other validation results are described.

Figure 3-10. Durbin and Schmidt (1985) Model of Distribution and Excretion of Absorbed Americium in the Human



Risk assessment.

The model was developed to predict skeletal and soft tissue burdens of americium in people who may be exposed to americium. Comparisons of annual limits on intake derived from the model and the ICRP (1979) model are presented. Descriptions of applications of the model in risk assessment have not been reported.

Target tissues.

The model calculates americium burdens in liver and skeleton. This output could be used to predict radiation doses to these tissues.

Species extrapolation.

The model is designed for applications to human dosimetry. The model and cannot be applied to other species without modification.

Interroute extrapolation.

The model, as described in Durbin and Schmidt (1985), does not have an intake component, but it could be linked to either an oral or inhalation intake model to simulate the biokinetics of americium associated with such exposures.

3.5 MECHANISMS OF ACTION

3.5.1 Pharmacokinetic Mechanisms

Absorption. Airborne particulate aerosols of americium can be deposited in the respiratory tract when the aerosols are inhaled. Amounts and patterns of deposition of particulates in the respiratory tract are affected by the size of the inhaled particles, age-related factors, breathing route (e.g., nose breathing versus mouth breathing), airway geometry, and airstream velocity within the respiratory tract (Gehr 1994; James et al. 1994; Roy et al. 1994). In general, large particles (>2.5 μm) deposit in the nasopharyngeal region where high airstream velocities and airway geometry facilitate inertial impaction (Chan and Lippman 1980; James et al. 1994). In the tracheobronchial and alveolar regions, where airstream

velocities are lower, processes such as sedimentation and interception become important for deposition of smaller particles (<2.4 μm). Airflow velocity and airway geometry change with age, giving rise to age-related differences in particle deposition (James 1978; James et al. 1994; Phalen et al. 1985). Also, anatomical features affect the intake route (nose or mouth). Deposition in the various regions of the respiratory tract in children may be higher or lower than in adults depending on particle size; for submicron particles, fractional deposition in 2-year-old children has been estimated to be 1.5 times greater than in adults (Xu and Yu 1986). Absorption of deposited americium is influenced by particle size and solubility as well as the pattern of regional deposition within the respiratory tract. Larger particles (>2.5 μm) that are deposited in the ciliated airways (nasopharyngeal and tracheobronchial regions) can be transferred by mucociliary transport into the esophagus and swallowed. Particles deposited in the alveolar region can be absorbed after extracellular dissolution or ingestion by phagocytic cells. Americium-bearing pulmonary alveolar macrophages (PAMs) can migrate either to the airways where mucociliary transport to the esophagus can occur or they can migrate to tracheobronchial lymph nodes (Taya and Mewhinney 1992; Taya et al.1986, 1992). The relative contributions of these two pathways to americium absorption have not been quantified. Studies in dogs and monkeys have shown that americium is present in tracheobronchial lymph nodes within days after inhalation of americium dioxide (Mewhinney et al. 1982; Stanley et al. 1982; Thomas et al. 1972). PAMs isolated from dogs, monkeys, and rats demonstrated the presence of phagocytosed and dissolved americium dioxide particles (Taya et al. 1992).

The site and mechanism of absorption of ingested americium are not known. Based on studies of plutonium, it is likely that americium absorption occurs, at least to some extent, in the small intestine. Studies of the absorption of plutonium in preparations of *in situ* isolated segments of small intestine of immature miniature swine indicate that absorption of actinides can occur along the entire small intestine, with the highest rates of absorption occurring in the duodenum (Sullivan and Gorham 1982).

Distribution. Bone constitutes the largest fraction of the deposited body burden of americium in all mammalian species that have been studied. The mechanisms by which americium is taken up and retained in bone are only partially understood. The distribution of americium in bone initially is confined to bone surfaces, including endosteal and periosteal surfaces, and adjacent to vascular canals in cortical bone (Polig 1976; Priest et al. 1983, 1995; Schlenker et al. 1989). Deposition appears to be favored at sites of active bone resorption, and concentrations are highest in trabecular bone where there is a high surface to volume ratio (Kathren et al. 1987; Priest et al. 1983). Surface deposits can eventually appear in deeper regions of bone as a result of accretion of new bone and burial of older surface deposits (Polig

1976; Priest et al. 1983). The observation that americium does not distribute more uniformly in bone has been taken as evidence that it does not exchange readily with calcium or other minerals in the bone mineral matrix and that the initial interactions with bone may be with organic components of bone (Bruenger et al. 1973; Priest et al. 1983); however, experimental evidence for this is equivocal. While americium has been shown to bind to the glycoprotein fraction of bovine cortical bone, even in the presence of an excess mass of bone mineral (Chipperfield and Taylor 1972), it also binds to bone mineral and bone calcium hydroxyapatite (Guilmette et al. 1998).

The highest concentrations of americium in soft tissues occurs in the liver (Filipy and Kathren 1996). The mechanisms of accumulation and retention of americium in liver have not been characterized. In rats and hamsters, americium appears to initially and extensively associate with lysosomes (Gruner et al. 1981; Seidel et al. 1986; Sütterlin et al. 1984). Agents that disrupt microtubules and lysosomes formation and processing, such as colchicine and vinblastine, have been shown to decrease liver uptake of americium in rats (Seidel 1984, 1985). The effect is thought to involve disruption of hepatic microtubule formation, which is critical to the formation and intracellular processing of lysosomes, the initial site of accumulation of americium in the liver. The major bound form of americium in dog liver cytosol was shown to be a ferritin complex (Stover et al. 1970). Substantial species differences in liver retention of americium have been observed. Most notable is the Chinese hamster for which liver retention half-times of 3,400 days have been estimated, compared to 5–16 days in rat liver (Durbin 1973; McKay et al. 1972). Elimination of americium from liver is relatively slow in dogs (1–10 years), compared to rats and mice (Durbin 1973; Lindenbaum and Rosenthal 1972). Mechanisms involved in the species differences in retention of americium in the liver have not been elucidated, although it has been proposed that animals with shorter retention times might be capable of excreting lysosomes in the bile (Sütterlin et al. 1984). The relatively slow elimination of americium from the hamster liver does not appear to be related to the initial tissue distribution, as the degree of association of americium with the lysosomal fraction of livers from rats and hamsters is similar (Sütterlin et al. 1984). The placental transfer of ^{241}Am to the fetus has been studied in several animal models and in the *in situ* perfused guinea pig placenta (Sikov 1987). After injections of ^{241}Am in mice and rats, a small fraction (<0.1%) of the maternal dose is transferred to the fetus (Hisamatsu and Takizawa 1983; Sikov 1987; Stather et al. 1992; Van Den Heuvel 1992; Weiss et al. 1980). Placental concentrations generally exceed those of the fetus, and concentrations in the embryo/fetal membranes are higher than in the fetus (Sikov 1987). Mechanisms of transport across the placenta have not been elucidated.

Metabolism. As noted in Section 3.4.3, the metabolism of americium consists of binding interactions with proteins and probably complex formation with various inorganic anions such as carbonate and phosphate, and carboxylic acids such as citrate and lactate (Durbin 1973; Taylor 1973; Webb et al. 1998). Experiments have not been conducted to determine whether americium binds to metallothionein, but such binding is not likely.

Excretion. Although fecal excretion appears to be a major route of excretion of absorbed americium, the mechanisms by which americium is transferred to the feces have not been characterized. Injection studies in animals suggest a contribution of biliary excretion to the fecal excretion of absorbed americium (Durbin 1973). Similarly, the mechanisms by which americium is excreted in urine have not been elucidated. Renal plasma clearance has been measured in rats, dogs, and monkeys, and in all three species, the renal clearance was <10% of the glomerular filtration rate (Durbin 1973; Lloyd et al. 1970; Taylor et al. 1961; Turner and Taylor 1968). However, simultaneous measurements of the renal plasma clearance of ultrafilterable americium and glomerular filtration rate are not available; thus, the relative contributions of filtration, tubular secretion, and tubular reabsorption to urinary excretion of americium cannot be ascertained with the available information. Studies of human urine incubated with ^{241}Am or rat urine collected from rats that were exposed to americium indicate that the major form of americium in urine appears to be a low molecular weight complex (1,000–10,000 d) with citrate (Stradling et al. 1976). This would be consistent with citrate complexes occurring in plasma from which they could enter the urine by glomerular filtration.

3.5.2 Mechanisms of Toxicity

Americium toxicity results primarily from the damage done by the alpha particle emitted during radioactive decay. This alpha particle has very limited penetration in tissue, and hence, the cellular damage (including damage to genomic material) occurs only in the immediate vicinity of the sequestered americium.

3.5.3 Animal-to-Human Extrapolations

Significant interspecies differences are apparent regarding liver retention rates of absorbed americium (Durbin 1973; Griffith et al. 1983) (see Sections 3.4.2.4 and 3.5.1 for more detailed information). However, no data were located to indicate significant interspecies differences in health effects associated with exposure to americium.

3.6 ENDOCRINE DISRUPTION

Recently, attention has focused on the potential hazardous effects of certain chemicals on the endocrine system because of the ability of these chemicals to mimic or block endogenous hormones, or otherwise interfere with the normal function of the endocrine system. Chemicals with this type of activity are most commonly referred to as endocrine disruptors. Some scientists believe that chemicals with the ability to disrupt the endocrine system are a potential threat to the health of humans, aquatic animals, and wildlife. Others believe that endocrine disrupting chemicals do not pose a significant health risk, particularly in light of the fact that hormone mimics exist in the natural environment. Examples of natural hormone mimics are the isoflavinoid phytoestrogens (Adlercreutz 1995; Livingston 1978; Mayr et al. 1992). These compounds are derived from plants and are similar in structure and action as endogenous estrogen. While there is some controversy over the public health significance of endocrine disrupting chemicals, it is agreed that the potential exists for these compounds to affect the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, reproduction, development, and/or behavior (EPA 1997). As a result, endocrine disruptors may play a role in the disruption of sexual function, immune suppression, and neurobehavioral function. Endocrine disruption is also thought to be involved in the induction of breast, testicular, and prostate cancers, as well as endometriosis (Berger 1994; Giwercman et al. 1993; Hoel et al. 1992).

Significantly reduced thyroid weights and cellular changes were observed in adult beagle dogs administered single intravenous injections of ^{241}Am at activity levels ranging from approximately 0.3 to 2.75 µCi/kg (11 to 102 kBq/kg) (Taylor et al. 1993a). Lower serum thyroxine levels were noted in the peripheral blood of dogs in the two highest exposure groups. No other studies were located regarding endocrine disruptive effects resulting from exposure to americium.

3.7 CHILDREN'S SUSCEPTIBILITY

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans, when all biological systems will have fully developed. Potential effects on offspring resulting from exposures of parental germ cells are considered, as well as any indirect effects on the fetus and neonate resulting from maternal exposure during gestation and lactation. Relevant animal and *in vitro* models are also discussed.

Children are not small adults. They differ from adults in their exposures and may differ in their susceptibility to hazardous chemicals. Children's unique physiology and behavior can influence the extent of their exposure. Exposures of children are discussed in Section 6.6 Exposures of Children.

Children sometimes differ from adults in their susceptibility to hazardous chemicals, but whether there is a difference depends on the chemical (Guzelian et al. 1992; NRC 1993). Children may be more or less susceptible than adults to health effects, and the relationship may change with developmental age (Guzelian et al. 1992; NRC 1993). Vulnerability often depends on developmental stage. There are critical periods of structural and functional development during both prenatal and postnatal life and a particular structure or function will be most sensitive to disruption during its critical period(s). Damage may not be evident until a later stage of development. There are often differences in pharmacokinetics and metabolism between children and adults. For example, absorption may be different in neonates because of the immaturity of their gastrointestinal tract and their larger skin surface area in proportion to body weight (Morselli et al. 1980; NRC 1993); the gastrointestinal absorption of lead is greatest in infants and young children (Ziegler et al. 1978). Distribution of xenobiotics may be different; for example, infants have a larger proportion of their bodies as extracellular water and their brains and livers are proportionately larger (Altman and Dittmer 1974; Fomon 1966; Fomon et al. 1982; Owen and Brozek 1966; Widdowson and Dickerson 1964). The infant also has an immature blood-brain barrier (Adinolfi 1985; Johanson 1980) and probably an immature blood-testis barrier (Setchell and Waites 1975). Many xenobiotic metabolizing enzymes have distinctive developmental patterns. At various stages of growth and development, levels of particular enzymes may be higher or lower than those of adults, and sometimes unique enzymes may exist at particular developmental stages (Komori et al. 1990; Leeder and Kearns 1997; NRC 1993; Vieira et al. 1996). Whether differences in xenobiotic metabolism make the child more or less susceptible also depends on whether the relevant enzymes are involved in activation of the parent compound to its toxic form or in detoxification. There may also be differences in excretion, particularly in newborns who all have a low glomerular filtration rate and have not developed efficient tubular secretion and resorption capacities (Altman and Dittmer 1974; NRC 1993; West et al. 1948). Children and adults may differ in their capacity to repair damage from chemical insults. Children also have a longer remaining lifetime in which to express damage from chemicals; this potential is particularly relevant to cancer.

Certain characteristics of the developing human may increase exposure or susceptibility, whereas others may decrease susceptibility to the same chemical. For example, although infants breathe more air per kilogram of body weight than adults breathe, this difference might be somewhat counterbalanced by their

alveoli being less developed, which results in a disproportionately smaller surface area for alveolar absorption (NRC 1993).

No studies were located in which comparisons were made between the sensitivity of children and adults to the toxicity of americium. A study of bone cancer induction from injected ^{239}Pu or ^{226}Ra in dogs suggests that, on a per Gy basis, dogs exposed at 3 months of age were less sensitive than dogs exposed at 1.5 years of age (Lloyd et al. 1999). Both plutonium and radon are alpha-emitters that accumulate in bone, as does americium. No direct evidence was located to indicate that the pharmacokinetics of americium in children may be different from that in adults. Based on dosimetric considerations related to differences in the parameters of available models, as well as studies in animals, it seems likely that children may be more susceptible to americium toxicity than adults by virtue of age-related differences in pharmacokinetics. Differences in airway geometry and airflow rates between children and adults would be expected to result in higher fractional deposition of inhaled submicron particles in children than in adults (Xu and Yu 1986) (see Section 3.5.1). Thus, children who are exposed to americium aerosols may receive a higher dose to the lung and greater absorption than would similarly exposed adults (ICRP 1995).

Studies of experimental animals indicate that absorption of ingested americium may be as much as 200 times greater in neonates than in adults (Bomford and Harrison 1986; David and Harrison 1984; Sullivan et al. 1985) (see Section 3.4.1.2 and Table 3-2). Thus, infants exposed to levels of americium in food or water similar as adults, or who ingest similar amounts of americium subsequent to an inhalation exposure, may absorb more americium and acquire a higher internal burden of americium. The ICRP (1989, 1994b, 1995) assumes age-related differences in intestinal absorption of americium swallowed following clearance from the lungs. The fractional absorption value employed for a 3-month-old infant is a factor of 10 higher than that for an adult.

Absorption of americium is greater in iron deficient animals than in iron replete adult animals (Sullivan and Ruemmler 1988; Sullivan et al. 1986) (see Section 3.4.1.2). Concurrent oral exposure to Fe^{3+} and americium also appears to increase the absorption of ingested americium; the latter effect may result from redox reactions in the gastrointestinal tract catalyzed by Fe^{3+} (Sullivan et al. 1986). These differences are accounted for in the discussions and dosimetric/metabolic models of the ICRP (1989, 1993) and the NEA (1988).

As inherent in these ICRP (1989, 1993) models, deposition of americium in bone occurs predominantly at bone surfaces that are actively undergoing resorption; therefore, exchanges between bone and soft tissue

stores of americium would be expected to be more rapid during periods of active bone metabolism such as infancy and childhood, pregnancy, and menopause. Higher skeletal accumulation of americium has been observed in neonatal animals that received americium intravenously than in similarly exposed adult animals (Hollins et al. 1973; Schoeters et al. 1990; Stevens et al. 1977) and in young rats (3 months of age) compared to old rats (13 months of age) (Sontag 1983). This suggests the possibility that infants and children may accumulate higher concentrations of americium in bone than similarly exposed adults. Studies of other bone-seeking radionuclides provide further support for increased vulnerability during periods of rapid bone growth such as adolescence (Carnes et al. 1997; Lloyd et al. 1999). On the other hand, americium uptake into maternal bone of lactating rats was similar to that of nonlactating rats, while concurrent calcium uptake into bone was lower in lactating rats (Hollins and Durakovic 1972). Thus, active mobilization of bone mineral, *per se*, may not always promote release of americium from bone (see Section 3.4.2.1).

Information on the transplacental transfer of americium in humans is not available directly, but the information from experiments with americium and other actinides has been used to derive biokinetic models and perform dosimetric models for the human (NCRP 1998; NRC 1996; Sikov and Kelman 1989). Studies in animals that received parenteral injections of americium have shown that absorbed americium is transferred to the fetus (Hisamatsu and Takizawa 1983; Paquet et al. 1998; Sasser at al. 1986; Schoeters et al. 1990; Weiss et al. 1980) (see Section 3.4.2.1). Limited reports indicate that ^{241}Am may induce fetal death and teratogenic effects in rodents (Moskalev et al. 1969; Rommerein and Sikov 1986).

Information on the distribution of absorbed americium to mammary milk in humans is not available; however, maternal (oral or intravenous) exposures of animals, including cows and goats, have shown that transfer to milk occurs and that neonates can be exposed to americium during lactation (McClellan et al. 1962; Sasser et al. 1986; Sutton et al. 1979) (see Section 3.4.2.1). Thus, it is possible that children who breast feed from mothers who have been exposed to americium, or who ingest milk from cows or other livestock that have been exposed to americium, may also be exposed to americium.

Bone serves as a long-term reservoir of americium in the body. The kinetics of bone formation and remodeling appear to be important factors in the overall biokinetics of americium in experimental animals (ICRP 1989, 1993). Quantitative evidence for their effects in humans has not be characterized, but exchanges between bone and soft tissue stores of americium would be expected to be more rapid during periods of active bone metabolism such as infancy and childhood, pregnancy, and menopause (see

Section 3.4.2.1). Thus, it is possible that, in humans, some of the maternal bone americium stores may be transferred to the fetus during gestation and may be incorporated into fetal bone during the development of the fetal skeleton. However, the finding of similar americium uptake in maternal bone of lactating and nonlactating rats (Hollins and Durakovic 1972) may be an indication that active bone metabolism is not necessarily a major source of americium release from bone at this maternal stage.

3.8 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

Due to a nascent understanding of the use and interpretation of biomarkers, implementation of biomarkers as tools of exposure in the general population is very limited. A biomarker of exposure is a xenobiotic substance or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s), or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to americium are discussed in Section 3.8.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are not often substance specific. They also may not be directly

adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by americium are discussed in Section 3.8.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, a decrease in the biologically effective dose, or a target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 3.10 “Populations That Are Unusually Susceptible”.

3.8.1 Biomarkers Used to Identify or Quantify Exposure to Americium

Biomarkers or tests to identify or quantify exposure to americium are available only at a limited number of government or nuclear facilities. Americium is a radioactive element. Internalized americium can be quantified through the use of whole-body radiation counters that measure the gamma emissions specific to each isotope of americium (Graham and Kirkman 1983; Palmer and Rhoads 1989; Palmer et al. 1983). Americium radioactivity can be measured by radioassays of urine, feces, or tissue samples by gross alpha analysis, alpha spectrometry, gamma-ray spectroscopy, mass spectroscopy, and liquid scintillation techniques (Alvarez and Navarro 1996; Dacheux and Aupiais 1997; DOE 1997b; Guilmette 1986; McInroy et al. 1985). Americium radioactivity can be measured in the teeth of rats, where it accumulates in the dental pulp of developing teeth and eventually is incorporated into the mineralized dentin (Hammerström and Nilsson 1970).

3.8.2 Biomarkers Used to Characterize Effects Caused by Americium

Although chromosomal aberrations have been reported in lymphocytes following exposure to ^{241}Am (Bauchinger et al. 1997; Kelly and Dagle 1974), and these observations are consistent with the effects of exposure to radiation, these effects are not specific to radioactive americium or to ionizing radiation in general.

3.9 INTERACTIONS WITH OTHER CHEMICALS

Ethanol may enhance the fecal excretion of absorbed americium. In baboons that received repeated oral doses of ethanol (1 mL ethanol/kg body weight), 3–6 months after receiving americium citrate intravenously, fecal excretion was approximately 2.5 times that of baboons that received water in place of the ethanol dose (Cohen et al. 1978). Long-term ethanol consumption accelerated the elimination of americium from the livers of beagle dogs that had been administered single intravenous injections of ^{241}Am (Taylor et al. 1992). The risk of americium-induced liver tumors, however, was 2–3 times higher in the ethanol-treated dogs than in similarly ^{241}Am-injected dogs not consuming ethanol.

Colchicine (a drug used in treatment of gout) and vinblastine (a cancer chemotherapy agent) may decrease liver uptake of americium. In rats that received an intraperitoneal injection of either colchicine and vinblastine prior to an intravenous or intramuscular injection of americium citrate, liver uptake of americium was lower, relative to controls, and kidney and skeletal americium uptake were higher (Seidel 1984, 1985). The effect is thought to involve disruption of hepatic microtubule formation, which is critical to the formation and intracellular processing of lysosomes, the initial site of accumulation of americium in the liver.

3.10 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to americium than will most persons exposed to the same level of americium in the environment. Reasons may include genetic makeup, age, health and nutritional status, and exposure to other toxic substances (e.g., cigarette smoke). These parameters result in reduced detoxification or excretion of americium, or compromised function of organs affected by americium. Populations who are at greater risk due to their unusually high exposure to americium are discussed in Section 6.7, Populations With Potentially High Exposures.

As discussed in Section 3.7, theoretical pharmacokinetics consideration suggests that children may be at greater risk from the effects of exposure to americium. However, there are no human or animal studies to confirm this hypothesis. A study of bone cancer induction from injected ^{239}Pu or ^{226}Ra in dogs suggests that, on a per Gy basis, dogs exposed as young adults (1.5 years of age) were more susceptible than either juveniles exposed at 3 months of age or older adults (5 years of age) (Lloyd et al. 1999). Both plutonium and radon are alpha-emitters that accumulate in bone, as does alpha-emitting americium.

3.11 METHODS FOR REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to americium. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to americium. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice. The following texts provide specific information about treatment following exposures to ^{241}Am:

Ellenhorn MJ, Schonwald S, Ordog G, Wasserberger J, eds. 1997. Medical toxicology: Diagnosis and Treatment of Human Poisoning. 2nd Baltimore (Williams & Wilkins). 1682-1723.

Haddad LM, Shannon MW, Winchester JF, eds. 1998. Clinical Management of Poisoning and Drug Overdose. 3rd edition. Philadelphia (W.B. Saunders). 413-425.

Viccellio P, ed. 1998. Emergency Toxicology. 2nd edition. Philadelphia (Lippincott-Raven). 991-996.

3.11.1 Reducing Peak Absorption Following Exposure

Methods for reducing peak absorption of americium after inhalation or oral exposure have not been described. Topical applications of saline containing DTPA, tartaric acid, or citric acid (e.g., *Schubert's solution*) have been used to remove americium from the skin and wounds after accidental dermal exposures (Breitenstein 1983). These agents form stable, water soluble complexes with americium.

3.11.2 Reducing Body Burden

Calcium or zinc complexes of polycarboxylate compounds such as DTPA or ethylenediaminetetratacetic acid (EDTA) have been used as chelating agents to accelerate the urinary excretion of americium in humans who were accidentally exposed to americium (Breitenstein 1983; Doerfel and Oliveira 1989; Durbin 1973; Fasiska et al. 1971). Persistent chelation therapy was considered to have been a primary factor in the long-term survival of a man who had been accidently exposed to an otherwise likely lethal amount of ^{241}Am (Breitenstein and Palmer 1989; Filipy et al. 1995; Toohey and Kathren 1995). Immediate and continued DTPA treatment of rats that had been exposed to $^{241}AmO_2$ by inhalation resulted in the near-total blockage of ^{241}Am translocation to liver and bone (Guilmette and Muggenburg 1988). Continuous subcutaneous infusion of DTPA was more effective in blocking the uptake than were periodic intravenous injections. Dogs receiving DPTA treatments following single intravenous injections of ^{241}Am

at activity levels of approximately 0.03 μCi/kg (11 kBq/kg) exhibited greater longevity and lower rates of bone cancer than ^{241}Am-treated dogs not given subsequent DPTA treatment (Lloyd et al. 1998). DTPA and EDTA form relatively stable complexes with americium in the extracellular fluid that can be excreted in the urine (Durbin 1973; Taylor 1973). The calcium and zinc complexes are used to decrease the risk of calcium and zinc depletion. Both DTPA and EDTA appear to affect primarily the americium in soft tissues, which exchanges more rapidly with americium in plasma than does americium in bone. As a result, treatments with DTPA or EDTA may have only a marginal effect on bone americium levels (Durbin 1973). In a study in rats, DTPA treatment, administered beginning 1.5 hours following intravenously administered ^{241}Am citrate, decreased bone ^{241}Am levels in trabecular bone at locations where bone resorption and remodeling occurred, but had little effect on americium levels in cortical bone (Polig 1976). This effect may have resulted from accelerated excretion and, therefore, diminished redeposition of americium released during bone resorption.

3.11.3 Interfering with the Mechanism of Action for Toxic Effects

No data were located regarding reduction of the toxic effects of radioactive americium through interfering with mechanisms of action.

3.12 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of americium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of americium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

3.12.1 Existing Information on Health Effects of Americium

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to americium are summarized in Figure 3-11. The purpose of this figure is to illustrate the existing information concerning the health effects of americium. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not necessarily imply anything about the quality of the study or studies, nor should missing information in this figure be interpreted as a "data need". A data need, as defined in ATSDR's *Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles* (ATSDR 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

Information regarding health effects following exposure to americium is mainly limited to reports of acute exposure. Accidental occupational overexposure of a 64-year-old man resulted in signs of hematological and bone marrow damage. Animal studies indicate that overexposure to americium can result in compromised hematological and immunological systems, as well as degenerative changes in bone, liver, kidneys, and thyroid, and bone cancer in long-term surviving animals. Although most studies employed parenteral injection as the route of exposure, available inhalation data indicate similarity in targets of toxicity. Data regarding health effects related to oral or dermal exposure are lacking for both humans and animals. Levels of radioactivity from americium employed in animal studies much higher than those likely to be environmentally experienced by humans. Therefore, americium does not likely pose an immediate health concern to humans.

3.12.2 Identification of Data Needs

Acute-Duration Exposure. Human data regarding adverse health effects resulting from exposure to americium are limited to the accidental occupational overexposure (inhalation, dermal, subcutaneous) of a 64-year-old man in which persistent hematological and bone marrow changes were observed (Filipy et al. 1995; Priest et al. 1995). Overexposure of animals to americium by the inhalation exposure route resulted in compromised respiratory function and radiation pneumonitis (Buldakov et al. 1972; DOE 1978; Thomas et al. 1972), persistent depressed blood values (Buldakov et al. 1972; Thomas et al. 1972), degenerative changes in bone, liver and kidney lesions, and gross atrophy and fibrosis of the thyroid gland (Thomas et al. 1972). Following acute inhalation exposure to americium, 4 of 15 dogs surviving

Figure 3-11. Existing Information on Health Effects of Americium

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
				Systemic						
Inhalation				●					●	
Oral										
Dermal		●								
External										

Human

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
				Systemic						
Inhalation	●	●							●	
Oral										
Dermal										
External										

Animal

● Existing Studies

more than 1,000 days developed osteosarcomas (Gillett et al. 1985). With the exception of the respiratory effects following inhalation exposure, the other targets of toxicity were likewise identified in animals administered single parenteral injections of americium at relatively high levels of radioactivity (Carter et al. 1951; Dougherty 1970; Jee et al. 19985; Lloyd et al. 1970, 1994a, 1994b; Schoeters et al. 1991; Taylor et al. 1983, 1991, 1993a; Van Den Heuvel et al. 1995). Acute-duration inhalation and oral MRLs were not derived for radioactive americium due to a lack of human or animal data. To generate appropriate data for deriving acute-duration inhalation and oral MRLs for radioactive americium, at least one comprehensive acute inhalation study and one acute oral toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity. However, since americium is not found naturally, and is produced and used in small quantities, the risk of overexposure to americium in humans should be low. Thus, additional studies may not be presently needed.

Intermediate-Duration Exposure. A single report indicated the potential for cytogenetic damage in a radiation worker and his family members following exposure to an americium source used by the father for private experiments at home for several years. No additional human or animal data were located. Intermediate-duration inhalation and oral MRLs were not derived for americium due to the lack of human or animal data. To generate appropriate data for deriving intermediate-duration inhalation and oral MRLs for americium, at least one comprehensive intermediate-duration inhalation and one intermediate-duration oral toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity. However, since americium is not found naturally, and is produced and used in small quantities, the risk of overexposure to americium in humans should be low. Thus, additional studies may not be presently needed.

Chronic-Duration Exposure and Cancer. No data were located regarding chronic-duration exposure of humans or animals to americium. Chronic-duration inhalation and oral MRLs were not derived for americium due to the lack of human or animal data. To generate appropriate data for deriving chronic-duration inhalation and oral MRLs for americium, at least one comprehensive chronic-duration inhalation and one chronic-duration oral toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity. However, since americium is not found naturally, and is produced and used in small quantities, the risk of overexposure to americium in humans should be low. Thus, additional studies may not be presently needed.

Genotoxicity. Chromosomal aberrations have been reported in lymphocytes following exposure to ^{241}Am (Bauchinger et al. 1997; Kelly and Dagle 1974). A single animal study reported increased numbers of micronucleated and multinucleated pulmonary alveolar macrophages in lung lavage of mice that had been exposed to ^{241}Am by inhalation (Talbot et al. 1989). External and internal exposure to radioactivity is a genotoxicity concern. External exposure to gamma-emitting americium would be expected to result in genotoxic effects similar to those observed following external exposure to any other gamma-emitting radionuclide (see ATSDR 1999 for more information on ionizing radiation). Internalized americium could cause damage to nearby cellular components that might be encountered by high energy alpha particles released from americium. Additional genotoxicity studies could be designed to assess the potential for genotoxicity from internalized americium, with special emphasis on blood-forming cells.

Reproductive Toxicity. No reports were located regarding reproductive effects in humans or animals following inhalation, oral, or dermal exposure to americium. In a limited study, increased incidences of fetal death were observed following single intravenous injections of ^{241}Am, administered to female rats prior to mating (Moskalev et al. 1969). Concentrations of ^{241}Am were much higher in placental tissues than in fetuses, and the investigators indicated that death may have been the result of placental changes. Animal studies could be designed to assess the potential for the reproductive toxicity of americium.

Developmental Toxicity. Data concerning developmental effects related to exposure to americium are restricted to a single report of decreased fetal weight, increased fetal death, and a tendency toward increased incidences of fetuses with anomalous ribs following exposure of pregnant rats with single intravenous injections of ^{241}Am (Rommerein and Sikov 1986). Animal studies could be designed to further assess the potential for the developmental toxicity of americium.

Immunotoxicity. Information regarding the immunotoxicity of americium is limited to a report of lymphopenia in a man following accidental overexposure when a glass column containing ^{241}Am blew up in his face (Filipy et al. 1995) and reports of depressed white blood cell counts in dogs exposed to ^{241}Am by inhalation (Buldakov et al. 1972; Thomas et al. 1972) or intravenous injection (Dougherty 1970). Additional animal studies could be designed to assess dose-response relationships.

Neurotoxicity. No data were located regarding neurotoxicity in humans or animals following exposure to americium. Neurotoxic effects, noted in humans suffering from acute radiation syndrome due to ionizing radiation exposure, are well-characterized (see ATSDR 1999 for detailed information on the effects of ionizing radiation). External overexposure to any gamma-emitting source could result in

neurotoxic effects. However, it is extremely unlikely that an americium source capable of producing such an overexposure would be encountered. Studies concerning the potential of americium to induce neurotoxicity do not appear to be needed at present.

Epidemiological and Human Dosimetry Studies. Epidemiological studies of radiation dose typically involve estimates of exposure that are based on whole-body measurements of internally-deposited americium. A need remains for epidemiological data that can provide quantitative human dose-response information while supplying additional information on the health effects of exposure to ionizing radiation and americium; in particular, for cases of known internal exposure.

Biomarkers of Exposure and Effect.

Exposure. Americium may be detected in samples of urine, blood, feces, or body tissues. Due to the relatively long biological half-time of americium, short-term exposures cannot be readily distinguished from longer-term ones. No new biomarkers of exposure are needed at this time.

Effect. Chromosomal aberrations have been reported in lymphocytes following exposure to ^{241}Am (Bauchinger et al. 1997; Kelly and Dagle 1974). Additionally, high radiation doses from internally deposited americium can cause bone marrow changes and altered blood values (Filipy et al. 1995; Priest et al. 1995). However, none of these effects are specific to americium.

Absorption, Distribution, Metabolism, and Excretion. Inhaled americium can be absorbed and transferred to the blood (Edvardsson and Lindgren 1976; Fry 1976; Newton et al. 1983; Sanders 1974; Toohey and Essling 1980). Limited information indicates that americium is rather poorly absorbed via the gastrointestinal tract (Hunt 1998; Hunt et al. 1986a, 1986b). Once americium reaches the blood, it is distributed throughout the body, accumulating mainly in the skeleton, liver, and muscle (Filipy and Kathren 1996; Filipy et al. 1994; Kathren et al. 1988; McInroy et al. 1989). Animal studies indicate that americium can pass through the placental barrier of a pregnant mother to a developing fetus (Hisamatsu and Takizawa 1983; Paquet et al. 1998; Sasser et al. 1986; Schhoeters et al. 1990; Sikov 1987; DOE 1986; Van Den Heuvel et al. 1992; Weiss et al. 1980), and can also be distributed to the breast milk of lactating mothers (McClellan et al. 1962; Sasser et al. 1986; Sutton et al. 1979). The metabolism of americium consists of binding interactions with proteins and probably complex formation with various inorganic anions such as carbonate and phosphate, and carboxylic acids such as citrate and lactate (Durbin 1973; Taylor 1973; Webb et al. 1998). Americium is slowly eliminated from soft tissues and

bone, but may remain in the skeleton for much longer periods. Once absorbed into the general circulation, americium is excreted in both feces and urine (Cohen and Wrenn 1973; Durbin 1973; Guilmette et al. 1980; Stather et al. 1979a). Additional quantitative toxicokinetic studies could be designed to further assess absorption following inhalation, oral, and dermal exposure, as well as age-related differences in the toxicokinetics of americium. However, the predominant data need for americium, and other sources of internal radiation exposure, is the verification (through biokinetic studies) and refinement of the models that describe internal distribution from which the radiation dose derives.

Comparative Toxicokinetics. Toxicokinetic properties of americium are generally similar in humans and animals. Inhaled americium, in relatively soluble forms, is readily absorbed in the respiratory system of humans and animals. Ingested americium is not absorbed to any great extent in humans or animals. Distribution patterns are generally similar; americium is found in highest concentrations in the skeleton, liver, and muscle of humans and animals. Although studies were not located regarding transfer of americium from mother to developing fetus, studies in animals suggest that americium could be incorporated by a fetus following inhalation exposure of the pregnant mother. Additional comparative toxicokinetic studies do not appear to be presently needed.

Methods for Reducing Toxic Effects. Methods for reducing peak absorption of americium after inhalation or oral exposure have not been described. Topical applications of saline containing DTPA, tartaric acid, or citric acid (e.g., *Schubert's solution*) have been used to remove americium from the skin and wounds after accidental dermal exposures. These agents form stable, water soluble complexes with americium. Calcium or zinc complexes of polycarboxylate compounds such as DTPA or ethylenediaminetetratacetic acid (EDTA) have been used as chelating agents to accelerate the urinary excretion of americium. Americium toxicity results primarily from the damage done by the alpha particle emitted during radioactive decay. This alpha particle has very limited penetration in tissue, and hence, the cellular damage (including damage to genomic material) occurs only in the immediate vicinity of the sequestered americium. There are no known methods for interfering with the process of radioactive decay.

Children's Susceptibility. No studies were located in which comparisons were made between the sensitivity of children and adults to the toxicity of americium. Animal studies indicate that juvenile dogs are less susceptible than adults to americium-induced bone cancer (Lloyd et al. 1999). No direct evidence was located to indicate that the pharmacokinetics of americium in children may be different from that in

adults. Based on dosimetric considerations related to differences in the parameters of available models, as well as studies in animals, it seems likely that children may be more susceptible to americium toxicity than are adults by virtue of age-related differences in pharmacokinetics. Absorption of ingested americium may be as much as 200 times greater in neonatal animals than in adults. (Bomford and Harrison 1986; David and Harrison 1984; Sullivan et al. 1985).

As inherent in ICRP (1989, 1993) models, deposition of americium in bone occurs predominantly at bone surfaces that are actively undergoing resorption; therefore, exchanges between bone and soft tissue stores of americium would be expected to be more rapid during periods of active bone metabolism such as infancy and childhood, pregnancy, and menopause. Higher skeletal accumulation of americium has been observed in neonatal animals that received americium intravenously than in similarly exposed adult animals (Hollins et al. 1973; Schoeters et al. 1990; Stevens et al. 1977) and in young rats (3 months of age) compared to old rats (13 months of age) (Sontag 1983). This suggests the possibility that infants and children may accumulate higher concentrations of americium in bone than similarly exposed adults. Studies of other bone-seeking radionuclides provide further support for increased vulnerability during periods of rapid bone growth such as adolescence (Carnes et al. 1997; Lloyd et al. 1999).

Information on the transplacental transfer of americium in humans is not available. Studies in animals that received parenteral injections of americium have shown that absorbed americium is transferred to the fetus (Hisamatsu and Takizawa 1983; Paquet et al. 1998; Sasser at al. 1986; Schoeters et al. 1990; Weiss et al. 1980).

Information on the distribution of absorbed americium to mammary milk in humans is not available; however, maternal (oral or intravenous) exposures of animals, including cows and goats, have shown that transfer to milk occurs and that neonates can be exposed to americium during lactation (McClellan et al. 1962; Sasser et al. 1986; Sutton et al. 1979).

Exchanges between bone and soft tissue stores of americium would be expected to be more rapid during periods of active bone metabolism such as infancy and childhood, pregnancy, and menopause. Thus, it is possible that, in humans, some of the maternal bone americium stores may be transferred to the fetus during gestation and may be incorporated into fetal bone during the development of the fetal skeleton.

Child health data needs relating to exposure are discussed in 6.8.1 Identification of Data Needs: Exposures of Children.

3.12.3 Ongoing Studies

No ongoing studies were identified in the Federal Research in Progress database (FEDRIP 2000).

4. CHEMICAL, PHYSICAL, AND RADIOLOGICAL INFORMATION

4.1 CHEMICAL IDENTITY

Americium is a man-made actinide element (atomic number 95) and has no stable isotopes. It was discovered by Glen Seaborg, Leon Morgan, and Ralph James in 1944 and isolated by B.B. Cunningham as the isotope ^{241}Am in $Am(OH)_3$ in the fall of 1945; it was named after the Americas (Seaborg 1991; Seaborg and Loveland 1990). Actinides are the 15 elements, all of whose isotopes are radioactive starting with actinium, atomic number 89 and extending to lawrencium, atomic number 103. Of the 15 americium isotopes and isomers currently identified, the longest-lived is ^{243}Am. ^{241}Am is of particular concern as it is formed by the action of neutrons on ^{239}Pu or uranium 238 (^{238}U) and is produced in nuclear reactors and nuclear explosions. ^{239}Pu and ^{238}U are transformed into ^{241}Pu which decays into ^{241}Am. ^{243}Am is produced by the absorption of neutrons by ^{241}Am. ^{242}Am is not a major isotope and is not available in macroscopic quantities.

4.2 PHYSICAL, CHEMICAL, AND RADIOLOGICAL PROPERTIES

The Chemical Abstract Service (CAS) registry numbers, decay modes, half-lives, and specific activities of the four principal americium isotopes and isomers, ^{241}Am,^{242m} Am, ^{242} Am, and ^{243}Am, are presented in Table 4-1. ^{241}Am and ^{243}Am decay by alpha emission forming neptunium (atomic number 93), ^{237}Np ($t_{½}$=2.14x10^6 years) and ^{239}Np ($t_{½}$=2.355 days) as the products. ^{239}Np subsequently decays to ^{239}Pu ($t_{½}$=2.411x10^4 years). The decay of ^{241}Am to ^{237}Np is accompanied by a predominant gamma ray photon of 59.54 keV. This photon is actually a L-shell x-ray but is invariably referred to as a gamma ray. ^{242m}Am is an isomer (long-lived excited state of the nucleus) of ^{242}Am and has a half-life of 141 years; 95.5% of ^{242m}Am undergoes an isomeric transition to ^{242}Am (half-life 16.02 hours) with the emission of a 48.6 keV gamma ray and 0.5% decays by alpha emission to ^{238}Np. ^{242}Am primarily (87%) undergoes beta decay to cerium (atomic number 96) 242 ($t_{½}$=162.8 days) which decays to ^{238}Pu ($t_{½}$=87.74 years) and then to ^{234}U ($t_{½}$=2.45x10^5 years); 17% undergoes electron capture to ^{242}Pu ($t_{½}$=3.76x10^5 years). ^{241}Am has a high specific activity of 3.428 Ci/g, emitting ~7x10^9 alpha particles/mg/minute. ^{243}Am has a specific alpha activity about 17 times lower than ^{241}Am and is therefore more attractive for chemical investigations of the element (ICRP 1983; Lide 1998; Seaborg 1991).

The known oxidation states of americium are +2, +3, +4, +5, and +6. However, the stable oxidation states are +3 and +4; the common oxidation state is +3, in which state, the behavior of americium and

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Table 4-1. Principal Americium Isotopes

Isotope[a]	CAS Registry No.	Decay mode/% (product)	Decay mode energy(MeV)	Particle energy: Energy (MeV)	Particle energy: Intensity (percent)	Half-life (years[b])	Specific activity[c] Ci/g	Gamma energy (keV)
^{241}Am	14596-10-2	α ($^{237}_{93}$Np)	5.637	5.4431 5.4857 5.3884 Others	12.8% 85.2% 1.4% <1%	432.2	3.43	26.34 33.192 59.536
^{242m}Am	13981-54-9	I.T./95.5% (^{242}Am)	0.048			141	10.5	48.63
		α /0.5%($^{238}_{93}$Np)	5.62	5.141 5.2070	0.026% 0.4%			86.48 109.44 163.04
^{242}Am	13981-54-9	β^-/83% ($^{242}_{96}$Cm)	0.665	0.63	46%	16.02 hr	808,000	42.2
		EC/17% ($^{242}_{94}$Pu)	0.750	0.67	37%			44.53
^{243}Am	14993-75-0	α ($^{239}_{93}$Np)	5.438	5.1798 5.2343 5.2766 Others	1.1% 11% 88% <1%	7,370	0.199	43.54 74.67 86.57 117.70 141.97

[a]An m after the atomic mass indicates one of multiple isomers of a given atomic mass.
[b]Unless otherwise specified
[c]LANL 1999

Source: DOE 1997b; ICRP 1983; LBL 2000; Lide 1998

other actinides are similar to those of the lanthanides. The trivalent state is the only state of importance in biological systems. The +2 oxidation state is very unstable and has only been produced in solid compounds. The stability of the americium oxidation states higher than +3 are less than those of uranium, neptunium and plutonium (Cotton and Wilkinson 1980; Nenot and Stather 1979; Seaborg 1991). Am^{+3} hydrolyzes and forms weak complexes with serum proteins and other ligands. The physical and chemical properties of americium and selected americium compounds are shown in Table 4-2. Properties of some americium ions are shown in Table 4-3.

Table 4-2. Physical and Chemical Properties of Americium and Selected Americium Compounds

	Value			
Property	Americium	Americium dioxide	Americium oxide	Americium chloride
Atomic/molecular weight[a]	243	275	534	349
Chemical formula	Am	AmO_2	Am_2O_3	$AmCl_3$
Synonyms	No data	No data	No data	No data
Chemical Abstracts Service Registry No.	7440-35-9	12005-67-3	12254-64-7	13464-46-5
Color	Silvery	Black	Tan	Pink
Physical form	Solid metal	Cubic crystals	Hexagonal crystals	Hexagonal crystals
Odor	No data	No data	No data	No data
Melting point, EC	1,176	>1,000 (decomposes)	No data	500
Boiling point, EC	2,011	Not relevant	No data	No data
Autoignition temperature	No data	Not relevant	Not relevant	Not relevant
Solubility:				
Water	Insoluble	No data	No data	Soluble
Other solvents	Soluble in acid	Soluble in acid	Soluble in acid	No data
Density, g/cm^3	12	11.68	11.77	5.87
Partition coefficients	No data	No data	No data	No data
Vapor pressure	No data	No data	No data	No data
Henry's law constant	Not relevant	Not relevant	Not relevant	Not relevant
Refractive index	No data	No data	No data	No data
Flashpoint	Not relevant	Not relevant	Not relevant	Not relevant
Flammability limits	Not relevant	Not relevant	Not relevant	Not relevant

Table 4-2. Physical and Chemical Properties of Americium and Selected Americium Compounds *(continued)*

Property	Value		
	Americium fluoride	Americium nitrate	Americium citrate
Atomic/molecular weight[a]	300	429	432
Chemical formula	AmF_3	$Am(NO_3)_3$	$AmC_6H_5O_7$
Synonyms	No data	Americium trinitrate	No data
Chemical Abstracts Service Registry No.	13708-80-0	25933-53-3	11078-88-9
Color	Pink	No data	No data
Physical form	Hexagonal crystals	No data	No data
Odor	No data	No data	No data
Melting point, EC	1,393	No data	No data
Boiling point, EC	No data	No data	No data
Autoignition temperature	Not relevant	Not relevant	Not relevant
Solubility:			
Water	Insoluble	No data	No data
Other solvents	No data	No data	No data
Density, g/cm^3	9.53	No data	No data
Partition coefficients	No data	No data	No data
Vapor pressure	No data	No data	No data
Henry's law constant	Not relevant	Not relevant	Not relevant
Refractive index	No data	No data	No data
Flashpoint	Not relevant	Not relevant	Not relevant
Flammability limits	Not relevant	Not relevant	Not relevant

Source: Chemical Abstract Service 2000; Lide 1994, 1998
[a]Calculated for ^{243}Am

Table 4-3. Properties of Some Americium Ions

Ion	Color	Stability
Am^{3+}	Pink or yellow	Stable; difficult to oxidize
Am^{4+}	Color unknown	Occurs in solution only as complex fluoride and carbonate ions
AmO_2^{+}	Yellow	Disproportionates in strong acid; reduces fairly rapidly under action of own alpha emissions (^{241}Am) at low acidification
AmO_2^{2+}	Rum colored	Easily reduced; reduces fairly rapidly under the action of its own alpha radiation (^{241}Am)

Source: Seaborg 1991

DRAFT FOR PUBLIC COMMENT

5. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

5.1 PRODUCTION

Americium does not occur in nature. The only isotope produced for commercial use is ^{241}Am. No production figures were available, although the amount of ^{241}Am available is believed to be several kilograms a year. Similarly, the amount of ^{243}Am available ranges from 10 to 100 g (Seaborg and Loveland 1990).

^{241}Am is produced when ^{239}Pu is exposed to neutrons, such as may occur in nuclear reactors. (^{239}Pu, it should be noted, is produced when uranium 238 [^{238}U], is exposed to neutrons.) The reaction sequence involves the successive absorption of neutrons and emission of gamma rays, written as (n,γ) and the emission of a beta particle, β^-.

$$^{239}_{94}\mathrm{Pu} + n \;\vee\; ^{240}_{94}\mathrm{Pu} + \gamma$$

$$^{240}_{94}\mathrm{Pu} + n \;\vee\; ^{241}_{94}\mathrm{Pu} + \gamma$$

$$^{241}_{94}\mathrm{Pu} \;\vee\; ^{241}_{95}\mathrm{Am} + \beta^-$$

The relative activity of americium isotopes for a typical pressurized-water reactor (PWR) fuel assembly are 1,700, 11, and 13 Ci for ^{241}Am, ^{242}Am, and ^{243}Am (DOE 1999). The respective activity ratios for a typical boiling water reactor (BWR) are 680, 4.6, and 4.9 Ci. There are 78 PWR and 41 BWR reactors in the United States, several of which have ceased operation. Total projected inventories of these three radionuclides for all reactors are 2.3×10^8, 1.4×10^6, and 1.7×10^6 Ci, respectively. The post irradiation americium content of typical PWR and BWR reactor fuel assemblies are 600 g (0.09%) and 220 g (0.07%), respectively.

The ratio of plutonium isotopes to ^{241}Am is often reported in monitoring studies as it is an important tool in dose assessment by enabling a determination of plutonium concentrations. ^{243}Am is produced directly by the capture of two neutrons by ^{241}Am. The parent of ^{241}Am is ^{241}Pu, which constitutes about 12% of the 1% content of a typical spent fuel rod from a nuclear reactor, has a half-life of 14 years. Separation of ^{241}Am from its parent ^{241}Pu and the other isotopes present can be effected by precipitation, ion exchange, or solvent extraction.

Americium metal has been obtained by heating americium oxide, Am_2O_3, with lanthanum at 1,200 EC; americium, which is more volatile than other actinides, volatilizes and can readily be separated from other actinides. AmO_2 can be obtained by igniting most trivalent americium compounds (Budavari 1996; Cotton and Wilkinson 1980; UIC 1997).

5.2 IMPORT/EXPORT

No information is available on the importation of americium into the United States or its export.

5.3 USE

^{241}Am has a long half-life and a predominant gamma ray energy of 59.5 keV, which makes it useful for a wide range of industrial gauging applications and for diagnosing certain thyroid disorders (Seaborg 1991). The most common application of americium is in ionization smoke detectors, and most of the several kilograms of americium produced each year is used for this purpose. The use of ^{241}Am in these devices has increased rapidly since 1972–1973. A typical household smoke detector contains 0.9 microcuries (μCi) of ^{241}Am, which is much lower than the activity in earlier ^{241}Am smoke detector or radium 226 (^{226}Ra) smoke detectors used in the past. In 1989, the average activity per unit was 3 μCi (Scientific Applications 1979). One gram of americium dioxide, AmO_2, provides enough active material for more than 5,000 smoke detectors. In the 1980s, annual sales of smoke detectors approached 12 million units. A mixture of ^{241}Am and beryllium known as ^{241}Am-Be is used as a neutron source in non-destructive testing of machinery and equipment, and as a thickness gauge in the glass industry (UIC 1997). ^{241}Am is used worldwide to measure the rate of production by oil wells (Seaborg 1991). No commercial uses for other americium isotopes were found. However, ^{243}Am can be used as a target material for the production of transcurium elements in high neutron flux reactors (Budavari 1996). ^{243}Am is also the preferred isotope for investigating the chemical properties of americium because of its lower specific activity than ^{241}Am.

5.4 DISPOSAL

Radioactive wastes are categorized according to their origin, the type of waste present, and their level of activity. The first distinction is between defense waste and commercial waste. The former was generated during and after World War II principally at the DOE's facilities at Hanford, Washington; Savannah River, South Carolina,; and Idaho Falls, Idaho where plutonium and other isotopes were separated from

production reactor spent fuel or nuclear-powered naval vessels. Commercial waste is produced by nuclear power plants, except for the long defunct commercial reprocessing facility at West Valley, New York. Nuclear waste is also classified as transuranic waste (TRU), high-level waste (HLW), and low-level waste (LLW). LLW is further differentiated into three classes, A, B, and C, according to increasing level of activity. A fourth category, commercial greater-than-class-C LLW (listed in Tables 1 and 2 of 10 CFR 61.55 for long and short half-life radionuclides, respectively) are not generally suitable for near-surface disposal. This could include operating and decommissioning waste from nuclear power plant and sealed radioisotope sources. The final disposition for this waste is not known. If LLW also contains hazardous material (i.e., that which is toxic, corrosive, inflammable, or explosive), it is termed mixed waste. Mine tailings from uranium mining is still another category of radioactive waste, but this category does not concern americium (DOE 1999; Murray 1994).

TRU are those containing isotopes, like ^{241}Am and ^{243}Am, that follow uranium in the periodic table and whose half-lives are >20 years. If their level of activity was more than 100 nanocuries of alpha-emitters per gram of waste material (up from 10 nanocuries/g in 1982), the waste could be disposed of by shallow burial. Otherwise, the waste had to be placed in retrievable storage for eventual transfer to a permanent repository. TRUs generally have low levels of radioactivity, generate very little heat, and can be handled by ordinary means without remote control (Eisenbud 1987; Murray 1994).

HLW includes spent fuel that is contained in fuel rods that have been used in a nuclear reactor. These may contain transuranic elements. After removal, these rods are placed in pools adjacent to the commercial nuclear power plants and DOE facilities where they were produced. It was originally intended that the fuel rods remain in these pools for only about 6 months to allow for a reduction in radioactivity and temperature, and then be transferred to a reprocessing or storage facility. There is no commercial reprocessing facility or permanent disposal facility for HLW operating in the United States. The Nuclear Regulatory Commission (NRC) has issued standards for the disposal of HLW (10 CFR 60), and the DOE is pursuing the establishment of a HLW facility. Efforts to establish a HLW facility, which began over 2 decades ago, have experienced many delays. A facility for the permanent disposal of HLW is not projected to be in operation before the year 2010 (Eisenbud 1987; Murray 1994).

LLW is officially defined as radioactive waste other than those previously defined. These wastes come from reactors, and institutions such as hospitals, universities, and research centers. Most LLW contains very little radioactivity and contains essentially no transuranic elements. It requires little or no shielding or special handling, and may be disposed of by shallow burial. However, some LLW contains sufficient

quantities of radioactivity as to require special treatment. Although U.S. NRC regulations for LLW disposal (10 CFR 61) permit shallow land burial, many states have enacted more stringent regulations (Eisenbud 1987; Murray 1994). The EPA is developing cleanup regulations as well as general environmental standards for LLW disposal that would apply to DOE facilities (EPA 2001d). The standards will facilitate planning and reduce costs for clean-up and disposal. There are currently 23 DOE and commercial LLW disposal sites in the United States (EPA 2001d). While some LLW facilities are closed, they are continuously monitored to detect releases of radioactivity into the environment. The Manifest Information Management System (MIMS), maintained by the Idaho National Environmental and Engineering Laboratory (INEEL), contains information on low-level radioactive waste shipments received at commercial low-level radioactive waste disposal facilities at Barnwell, South Carolina (January 1, 1986–present), Beaty, Nevada (April 1, 1986–December 31, 1992), Richland, Washington (January 1, 1986–present), and Envirocare, Utah (January 1, 1998–December 31, 1999). In 1999, 3.67 Ci (136 GBq) of ^{241}Am and <0.01 Ci (<0.4 GBq) of ^{243}Am LLW was received at these facilities from academic, industrial, government, and utility generators throughout the United States (INEEL 2000).

At present, DOE stores most of its spent fuel at three primary locations: the Hanford site, in Washington, the INEEL, in Idaho, and the Savannah River Site, in South Carolina. Some spent fuel is also stored at the dry storage facility at Fort St. Vrain in Colorado. Much smaller amounts of spent nuclear fuel stored at other sites were to be shipped to the three prime sites for storage and preparation for ultimate disposal (DOE 1999). The DOE National Spent Fuel Program maintains a spent nuclear fuel data base that lists the total volume, mass, and metric tons of heavy metal (MTHM) of 16 DOE categories of spent nuclear fuel stored in each of the three locations. The categories having the highest ^{241}Am activities per spent nuclear fuel canister (decayed to 2030) are ‘uranium metal’, ‘mixed oxides’, and ‘plutonium/uranium carbide, nongraphite.’ The ^{241}Am solid waste stored on the Hanford site in 1998 included 2.3 Ci (85 GBq) as LLW and 11 Ci (410 GBq) as TRU (Hanford 1999). Between 1945 and 1970, the Farallon Islands Nuclear Waste Dump Site (FINWDS), approximately 30 miles west of San Francisco, received at least 500 TBq (14,000 Ci) of reportedly low-level nuclear waste, concrete-encapsulated in at least 47,500 55-gallon 16-gauge steel drums (life-expectancy ~30 year in sea water) (Suchanek et al. 1996). These were deposited at three sites 100, 900, and 1,800 m deep.

Smoke detectors containing americium that are inoperable, damaged, or no longer needed should be returned to the supplier for disposal.

6. POTENTIAL FOR HUMAN EXPOSURE

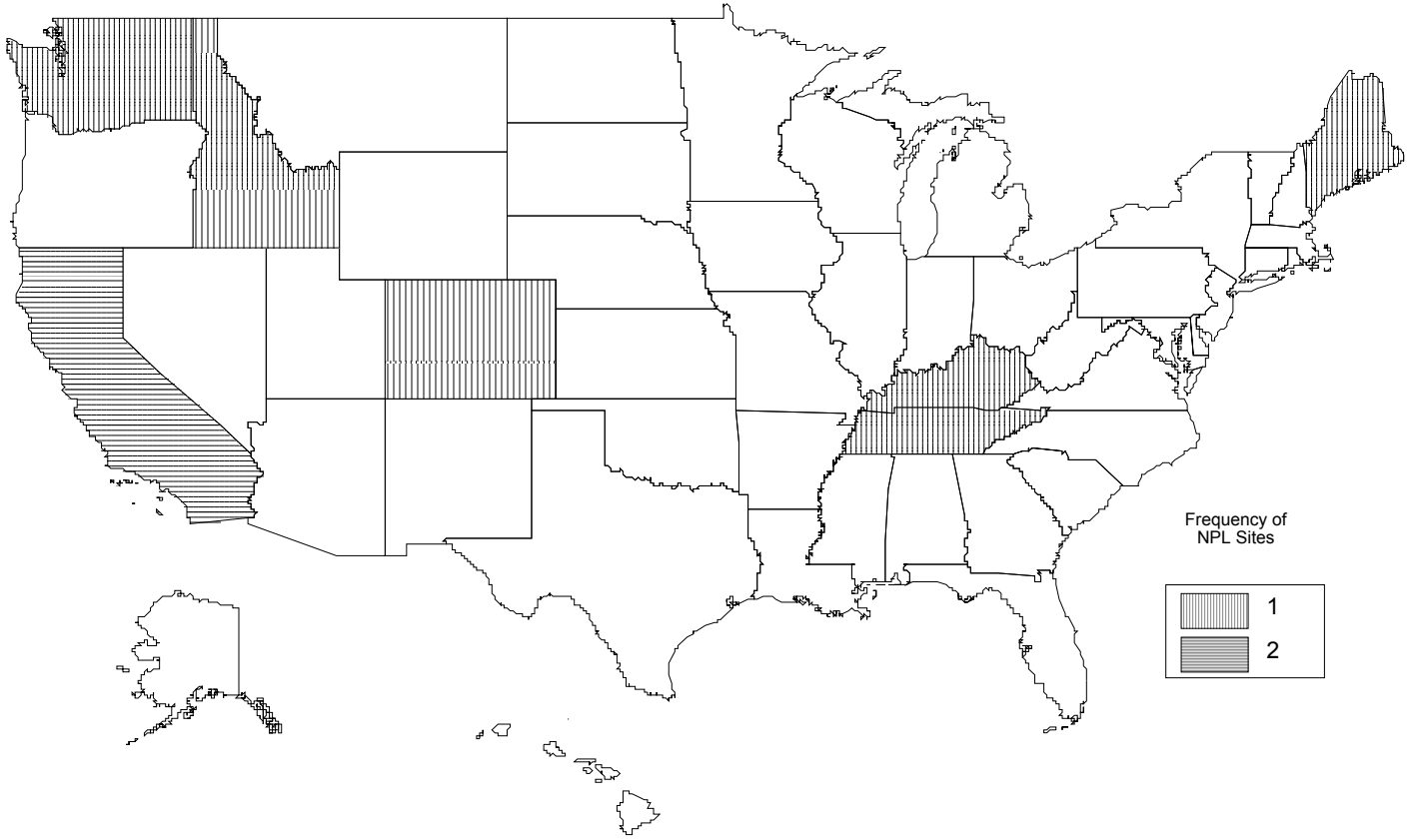
6.1 OVERVIEW

Americium has been identified in at least 8 of the 1,585 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2001). However, the number of sites evaluated for americium is not known. The frequency of these sites can be seen in Figure 6-1.

Americium is a man-made element. There are basically two sources of ^{241}Am into the environment: fallout from nuclear explosions and releases from nuclear reactors and reprocessing plants. Additionally, there are accidental releases such as the Chernobyl accident, the burn-up of a satellite powered by the aerospace reactor SNAP 9A, and the crash of a B-52 aircraft carrying nuclear weapons in Thule, Greenland. Today, ^{241}Am along with ^{90}Sr, ^{137}Cs, ^{238}Pu, ^{239}Pu, and ^{240}Pu are the isotopes that produce the majority of radiation associated with nuclear fallout (DOE 1997a). Very low levels of ^{241}Am may be found in environmental media worldwide from atmospheric nuclear weapons test; these levels are referred to as fallout background levels. In nuclear reactors, ^{241}Am has been detected in primary coolant water, stack aerosols, and waste water (Rosner et al. 1978). Because ^{241}Am is produced from the decay of ^{241}Pu (half-life 14.4 years), releases of ^{241}Pu will result in an ingrowth of ^{241}Am; clearly, releases of ^{241}Pu and ^{241}Am must be considered collectively. The maximum quantity of ^{241}Am following an accidental release of ^{241}Pu occurs in 70–80 years (EPA 1976). Consequently, the impact of ^{241}Am from atmospheric nuclear weapons testing will reach its peak in about the year 2035 and then levels will decline according to its rate of decay. ^{241}Am is also used commercially as an ionization source in smoke detectors and it may be released into the environment from its production, use, and disposal. ^{241}Am is the only americium isotope to be used commercially.

Americium released to the atmosphere will be associated with particulate matter and will be deposited on land or surface water by dry deposition or wet deposition (Essien et al. 1985). Dry deposition results from gravitational settling and impaction on surfaces, and wet deposition returns americium to earth in precipitation. Radionuclides resulting from atmospheric weapons tests are often injected into the lower stratosphere, while other atmospheric releases are into the troposphere. The residence time of particles in the atmosphere will depend on the altitude, latitude, season, and hemisphere because of atmospheric

Figure 6-1. Frequency of NPL Sites with Americium Contamination



Derived from HazDat 2001

circulatory patterns. For example, residence half-lives of particles in the lower stratosphere range from 3–12 months in polar regions and 8–24 months in equatorial regions, with removal being highest in spring and lowest in fall. Half-lives in the troposphere are about 1 month. Larger particles will remain inthe atmosphere for shorter periods of time and are deposited closer to their emission source while smaller particles remain in the air for longer periods of time and are deposited farther from their source. In the absence of actual measurements, the deposition of ^{241}Am in a region can be estimated by using the relationship between air concentration and deposition quantities for other isotopes that are attached to particulate matter in air. Using this methodology, the annual deposition of ^{241}Am in New York was estimated to have reached a maximum of 0.010 mCi/km^2 (0.37 MBq/km^2) in 1964 (Bennett 1979). The cumulative ^{241}Am deposition in the year 2000 is estimated to be 0.058 mCi/km^2 (2.1 MBq/km^2). If the ingrowth from ^{241}Pu decay is included, the cumulative ^{241}Am deposited by the year 2000 would be 0.80 mCi/km^2 (30 MBq/km^2). Americium may also be injected into the atmosphere from water surface by the action of bubbles scavenging the water column and wave action (McKay et al. 1994a; Walker et al. 1986). Similarly, ^{241}Am-contaminated soil may be released into air by wind action.

Americium released to water in effluent, runoff, or atmospheric deposition adsorbs to particulate matter and is rapidly depleted from the water column and deposited in surface sediment (Murray and Avogadro 1979). Adsorption is very high with distribution coefficients between the particulate-associated phase and the dissolved phase in sediment and water of the order of 10^5–10^6 (Davis et al. 1997; NRC 1981; Pattenden and McKay 1994). Aquatic organisms can bioaccumulate americium via intake from the water or by ingesting sediment or organisms lower on the food scale. In crustacean zooplankton, crustaceans, and molluscs, americium is accumulated from water by passive absorption onto exoskeletons (Fisher et al. 1983). Ingested in suspended diatoms or other food, americium largely passes through the gut and is eliminated. Biogenic debris from these animals, such as fecal pellets and discarded molts, facilitates vertical transport of ^{241}Am into the depths and sediment.

If land deposition occurs, americium will sorb to the soil, leach into the subsurface soil, and redistribute by the action of wind, water, or biotic transport. Small mammals inhabiting areas containing radioactive contamination or radioactive waste sites may become contaminated with americium by consuming contaminated soil or plants and disturb ^{241}Am-contaminated soil through their burrowing and excavating activities. In addition, small mammals may be consumed by animals higher in the food chain such as hawks and coyotes which would add to the dispersal of americium from disposal areas. Since fallout ^{241}Am is accompanied by its precursor, ^{241}Pu, which also leaches into the ground, the amount of ^{241}Am at a depth is a function of the leaching of both the americium and plutonium as well as the decay of these

isotopes. Fallout ^{241}Am is predominantly retained to the upper few cm of soil where it is associated with organic matter and bound to manganese and iron oxides (Bennett 1976; Bunzl et al. 1995; Vyas and Mistry 1980).

Americium may be taken up from the soil by plants and translocated to other parts of the plants. ^{241}Am-contaminated dust may also be deposed on the above ground parts of the plant. Uptake of ^{241}Am from soil is low (Bennett 1979; EPA 1979; Nisbet and Shaw 1994; Romney et al. 1981; Schultz et al. 1976; Zach 1985). Uptake from soil is a function of the chemical species, soil chemistry, plant species, and environmental conditions. Concentration ratios of ^{241}Am in plants to that in soil range over about 2 orders of magnitude depending on crop and soil type. Uptake is higher under acidic conditions and lower in soil with high fractions of organic matter that play an important role in complexing americium, making it less available for uptake. Translocation of transuranic elements taken up through the roots into seeds and fruit is generally very low (Bennett 1979; Schreckhise and Cline 1980). Concentration ratios in seed, grain, and fruit range from 10^{-6} to 10^{-4}, about a factor of 10 lower than in foliage. Animals can accumulate americium by eating contaminated plants as well as ingesting soil, or other animals and breathing air. Biological uptake in aquatic organisms may depend on factors such as temperature, time after intake, season of the year, and water quality. The bioconcentration of ^{241}Am in fish is low, especially to the edible parts of the fish (DOE 1996). In a study performed in a nuclear waste pond where the levels of ^{241}Am were about 3 orders of magnitude above background levels, concentrations in fish filet were rarely >10 times that of controls (Emery et al. 1981). In marine organisms consumed by humans, uptake is generally highest in mussels where the target organs are mainly the digestive gland, gill, and exoskeleton (Chassard-Bouchaud 1996; Fisher et al. 1996; Hamilton and Clifton 1980). Transfer coefficients have been reported for many of these processes. Available evidence suggests that americium is not biomagnified up the food chain to humans (Bulman 1978; Jaakkola et al. 1977).

Exposure of the general population to ^{241}Am via air, water, soil, and food are generally very low; these ‘background’ levels are a result of fallout from past atmospheric nuclear weapons tests. Since 1973, ^{241}Am air concentrations have been less than 1 aCi/m^3 (0.037 μBq/m^3) and are continuing to decline (Bennett 1979). Levels around nuclear power plants are indistinguishable from fallout background (EPRI 1981). ^{241}Am levels in surface seawater of the North Sea and North Atlantic Ocean have stayed around 10 Bq/m^3 (270 pCi/m^3) between 1976 and 1988 (Pattenden and McKay 1994), but may be considerably higher near discharges from nuclear facilities. ^{241}Am in soil resulting from cumulative deposition and ingrowth in the New York region should reach a maximum of 0.88 mCi/km^2 (33 MBq/km^2) in the year 2035. In the Food and Drug Administration (FDA) Total Diet Studies in 1983–1986 and 1987–1992, the

concentrations of radionuclides were sufficiently low as to require no specific action or simple monitoring.

The general population may be exposed to elevated levels of ^{241}Am from nuclear accidents or proximity to hazardous waste sites where this radionuclide is present. Exposure is generally through the inhalation and ingestion of dust from these sites. Workers involved in producing ionization smoke detectors or other devices containing ^{241}Am and workers at nuclear reactors or Department of Energy (DOE) facilities may also be exposed to higher levels of ^{241}Am. Americium is usually encountered in the environment and most occupational situations in combination with plutonium and curium; exposure to americium alone is unlikely. Elevated levels of americium have been identified in at least 8 of the 1,585 current or former EPA National Priorities List (NPL) hazardous waste sites (HazDat 2001). However, the number of sites evaluated for americium is not known. The distribution of these sites within the United States is shown in Figure 6-1.

6.2 RELEASES TO THE ENVIRONMENT

Throughout this chapter, the units used to express concentration or intake of ^{241}Am are the same units reported by the authors, which may be followed by converted units in parenthesis. Concentrations of ^{241}Am are generally expressed in terms of activity, either in the conventional unit, the curie (Ci) or the SI unit, the becquerel (Bq), where 1 Ci=3.7x10^{10} Bq=0.037 TBq or 1 Bq=2.7x10^{-11} Ci=27 pCi. Tables are provided in Section 1.2 to facilitate conversion. Activities may be converted into mass units using the specific activity, μCi/g. It should be remembered that since ^{241}Pu (half-life 14 years) decays into ^{241}Am, over time, inventories of ^{241}Am will be augmented by releases of ^{241}Pu. The ratio of americium to plutonium isotopes is often used to characterize the nature of a radionuclide source.

The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

6.2.1 Air

Americium has been identified in 2 air samples collected from 1,585 current or former NPL hazardous waste sites where it was detected in some environmental media (HazDat 2001).

^{241}Am is released into the atmosphere primarily by plutonium production reactors or in nuclear accidents and nuclear weapons testing. The ^{241}Am released is augmented by decay of ^{241}Pu released during these events. The amount of ^{241}Am released is often estimated by the amount deposited on earth. In 1982, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) estimated that the integrated deposition density of ^{241}Am in the north temperate zone of the earth was 25 Bq/m^2 (Suchanek et al. 1996). Since then the effective deposition of ^{241}Am has grown by 1 Bq/m^2 per year because of the radioactive decay of 730 Bq/m^2 of deposited ^{241}Pu (to approximately 45 Bq/m^2).

In atmospheric nuclear weapons tests, radionuclides are often injected into the stratosphere. The amount of ^{241}Am present in the atmosphere from nuclear weapons testing depends on the amount of unfissioned ^{241}Am and ^{241}Pu injected into the atmosphere and the transport and residence time of both the ^{241}Am and its precursor in the various atmospheric compartments (e.g., stratosphere and troposphere). For a particular weapon's test, this will depend on such factors as the amount of plutonium and americium in the weapon, the detonation yield, the altitude and latitude of the detonation, and the season. High yield U.S. tests were characterized by very high ratios of ^{241}Pu/^{239,240}Pu and these would produce relatively large quantities of ^{241}Am (Roos et al. 1994). At this time, the legacy of past atmosphere nuclear weapons tests is a fairly uniform low level radiation background. The peak global activity of ^{241}Am resulting from nuclear weapons testing between 1951 and 1978 and receiving global distribution is estimated to be 130 kCi (4,800 TBq), taking into account the simultaneous production of ^{241}Am from ^{241}Pu and it's decay (Bennett 1979).

The reactor accident at Chernobyl in April 1986 released radionuclides into the atmosphere, mostly between April 26 and May 6. Estimates of quantities released are based on observations of deposition within 30 km of the reactor. Releases in this area were predominantly highly irradiated fuel particles. It is estimated that the discharge of ^{241}Pu through May 6, 1986 was 5,200 TBq (140 kCi), which amounted to 3% of the reactor content of this radionuclide (Askbrant et al. 1996; Pattenden and McKay 1994). The material was released mainly in the lower troposphere.

Subsequent to the primary events releasing radionuclides into the atmosphere, radionuclides can enter the atmosphere from resuspension of soil particles and forest fires (Lujaniene et al. 1999). Resuspension studies performed with ^{241}Am, indicate that the activity to mass ratio is greatest for particles <45 μm in diameter (Cooper et al. 1994). Therefore, the fine resuspended soil released into the air would have a higher concentration of ^{241}Am than the remaining surface soil. This enhancement factor for resuspended soil in the inhalable fraction (<7 μm) ranged from 5.4 to 32.5 at six sites at Taranaki, a weapons testing

range at Maralinga, South Australia (Cooper et al. 1994). Other potential sources of release is in windblown soil from low-level waste (LLW) disposal sites. While low-level radioactive waste is required to be buried in a manner protective of near-term release, biotic transport by plants and small mammals may transport americium to the soil surface where it is accessible to wind. In order to predict the impact of biotic transport, a model was applied to six composite waste streams containing isotopic concentrations for the western United States prepared by the NRC to which current and past LLW disposal conditions applied. The containers and wastes were assumed to decompose with a 35-year half-life and active biotic transport was assumed to occur for 100 years following site closure and before institutional control of the site was lost. After 100 years, the activity at the soil surface (assumed to be uniformly mixed in the upper 0.5 m of the entire burial ground) was estimated to be 8.7×10^3 and 2.8×10^5 pCi/m^2 for ^{243}Am and ^{241}Am, respectively (Kennedy et al. 1985).

^{241}Am is released to the atmosphere at DOE facilities. Air sampling data were used to estimate effluent release from the Savannah River Site (SRS) from the plant's start-up in 1954 through 1989 (DOE 1991). From this monitoring, it was estimated that 5.49 mCi (200 MBq) of ^{241}Am was released to the atmosphere between 1977 and 1989. The SRS was a major U.S. nuclear production facility and included five nuclear reactors, a fuel and target fabrication plant, a naval fuel materials facility, two chemical separation plants, a heavy water production plant, and a laboratory. SRS reported releasing approximately 5.2 mCi (190 MBq) of ^{241}Am into the atmosphere (DOE 1996). Americium has been identified in air at Rocky Flats at the NPL site and in stack emissions at Hanford, Washington (HazDat 2001). The stack emissions, measured in 1991, averaged 13 MBq/L (350 μCi/L). In 1994, atmospheric discharges of ^{241}Am from four areas at Hanford National Laboratory ranged from 0.067 to 66 μCi (2.5 to 2,400 kBq) (PNL 2000).

6.2.2 Water

Americium has been identified in 25 groundwater samples but was not detected in any surface water samples collected from 1,585 NPL hazardous waste sites, where it was detected in some environmental media (HazDat 2001).

Americium is released into surface water primarily from plutonium production reactors, nuclear fuel reprocessing facilities, or in nuclear accidents. It may also be released from radioactive waste storage facilities. Since ^{241}Pu decays into ^{241}Am, ^{241}Am is also released as a result of ^{241}Pu releases. Water sampling data were used to estimate effluent releases from the SRS from the plant's start up in 1954 through 1989 (DOE 1991). From this monitoring, it was estimated that 290 mCi (11 GBq) of ^{241}Am

was released into seepage basins between 1977 and 1989. However, no ^{241}Am was released directly into streams.

Radionuclides have been released since 1952 at Sellafield, United Kingdom on the Irish Sea by the British Nuclear Fuels Limited (BNFL) reprocessing plant and, before that, by two plutonium-producing nuclear reactors. In 1988, the estimated environmental inventory of ^{241}Am from all previous operations totaled 859 TBq (23.2 kCi), consisting of 537 TBq (14.5 kCi) actually released plus 322 TBq (8.7 kCi) produced from the decay of ^{241}Pu that was also released. Annual discharges of ^{241}Am from Sellafield ranged from 4.5 to 120 TBq/year (120 to 3,200 Ci) between 1964 and 1989, while those for ^{241}Pu ranged from 1 to 2,800 TBq/year (27 to 76,000 Ci) between 1952 and 1989; maximum annual discharges occurred in 1973 for ^{241}Pu and 1974 for ^{241}Am (Pattenden and McKay 1994). The annual liquid effluent discharges from a typical U.K. Magnox Station contained 34.3–121 Bq/L (0.926–3.27 nCi) of ^{241}Am for the years 1991–1995 (Ware et al. 1998). These discharges are from the final monitoring and delay tank. (Magnox, an alloy of magnesium oxide and aluminum, has been used as a protective covering or cladding material for fuel elements in British nuclear reactors. The name "Magnox" is widely used to refer to the early gas cooled because it provides a simple way to uniquely identify the group.)

^{241}Am has been detected in groundwater samples at two of eight hazardous waste sites where americium has been identified in some environmental component, but was not detected in any surface water samples (HazDat 2001). It was identified in surface water at Oak Ridge, Tennessee and Rocky Flats, Colorado and in groundwater at Hanford, Washington and Rocky Flats NPL sites (HazDat 2001). The distribution of Superfund NPL sites is shown in Figure 6-1.

6.2.3 Soil

Americium has been identified in 6 soil samples but was not detected in any sediment samples collected from 1,585 NPL hazardous waste sites, where it was detected in some environmental media (HazDat 2001).

As a manufactured element, americium is not naturally present in rocks and soils. Contamination of the soil can occur either from deposition of americium or precursor plutonium originally discharged into the atmosphere, or from waste products discharged directly into or on the ground. Except for the reentry into the atmosphere of the SNAP 9A satellite and the French atmospheric tests in the Mururoa Atoll between 1966 and 1974 that occurred in the southern hemisphere, the main events resulting in the deposition of

artificial radionuclides occurred in the northern hemisphere. As a consequence, there is a progressive decline in the levels of artificial radioactivity from northern to southern latitudes. Recent studies of the multi-isotopic radioactive content in ice layers in Antarctica have determined that 0.21 Bq/m^2 (5.7 pCi/m^2) of ^{241}Am was deposited in fallout between 1969 and 1993 (Baeza et al. 1999). Therefore, the mean annual fallout in Antarctica is about 8.3 mBq/m^2 (0.22 pCi/m^2).

Low level waste from commercial facilities is buried on site. The Nuclear Regulatory Commission (NRC) has projected the activities and volumes of low level radioactive waste from all sources buried at commercial sites to the year 2000 using information from the Idaho National Environmental and Engineering Laboratory (INEEL) waste retrieval project and assuming that the waste disposal practices then used would continue into the future. The 20-year decayed ^{241}Am and ^{243}Am concentrations were estimated to be 380 and 230 pCi/m^3 (14 and 8.5 Bq/m^3) , respectively (Kennedy et al. 1985).

The largest releases of ^{241}Am to soil in the United States is at DOE facilities. The ^{241}Am inventory in surface soil at the Nevada Test Site (NTS) estimated as part of the DOE's Radionuclide Inventory and Distribution Program between 1981 and 1986 was 150 Ci (5,600 GBq) (DOE 1991b). The highest concentration of ^{241}Am was found in the Yucca Flats area. The United States began testing nuclear weapons at the NTS in January 1951. Since then, NTS has become the primary site for testing nuclear weapons and studying the effect of these weapons on structures and military equipment; >600 nuclear explosions have taken place at this site. Americium has been identified in subsurface soil at the INEEL, Idaho; Lawrence Livermore Laboratory, California; Rocky Flats, Colorado; Hanford, Washington; and Treasure Island Naval Station, California NPL sites (HazDat 2001). At Rocky Flats, ^{241}Am contamination in soil was largely formed from ^{241}Pu in the storage tanks or in the ground (Litaor et al. 1996). Waste oil stored in the tanks was from processing weapons-grade plutonium, which contained 0.58% ^{241}Pu and $<10^{-4}$% ^{241}Am. Between 1978 and 1981, 48 and 179 kCi (1,800 and 6,600 TBq) of ^{241}Am and ^{241}Pu, respectively, were disposed in the Subsurface Disposal Area of INEEL (Arthur and Janke 1986). No estimates are available of the quantities of these isotopes disposed of between 1952 and 1971 at the Radioactive Waste Disposal Complex of INEEL. ^{241}Am is present at plutonium facilities and at plutonium reclamation facilities where ^{241}Am is routinely separated from plutonium. The Hanford plant was the site of a 1976 column explosion, which resulted in extensive contamination and worker exposure (PNL 2000).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

Atmospheric Transport. In the atmosphere, americium is associated with particulate matter and therefore, the transport of americium in air will be governed by that of its host particles (Bennett 1979). Radionuclides resulting from atmospheric weapons tests are often injected into the lower stratosphere, while other atmospheric releases are into the troposphere. The residence time of particles in the atmosphere will depend on the altitude, latitude, season, and hemisphere because of atmospheric circulatory patterns. For example, residence half-lives of particles in the lower stratosphere range from 3 to 12 months in polar regions and from 8 to 24 months in equatorial regions, with removal being highest in spring and lowest in fall. Half-lives in the troposphere are about 1 month. Larger particles will remain in the atmosphere for short periods of time and are deposited close to their emission source while small particles remain in the air for longer periods of time and are deposited further from their source. There are three deposition mechanisms: rain-out caused by droplet formation on aerosols within clouds, wash-out by falling precipitation, and dry deposition of particles onto the ground. Rain-out is the most important deposition process for americium, wash-out occurs relatively slowly being dependent on the solubility of the pollutant in rainwater, and dry deposition makes an important contribution in areas of low rainfall. Therefore, deposition of ^{241}Am-containing particulate matter will depend on location, season, and meteorological conditions. In the absence of actual measurements, the deposition of ^{241}Am in a region can be estimated by using the relationship between air concentration and deposition quantities for other isotopes that are attached to particulate matter in air. In this way, using the average annual air concentration to annual deposition rate for strontium 90 (^{90}Sr), the deposition rate of ^{241}Am in the New York area was estimated to reach a maximum of 10 μCi/km^2 (37 kBq/km^2) in 1964 and decline thereafter to 0.06 μCi/km^2 (2.2 kBq/km^2) by 1981 (Bennett 1979). This would be expected to result in cumulative ^{241}Am deposit of 58 μCi/km^2 (2.1 MBq/km^2) in the New York region in 2000, assuming no additional atmospheric nuclear testing after 1976. If the ingrowth from ^{241}Pu decay is included, the cumulative ^{241}Am deposit in the year 2000 would be 800 μCi/km^2 (30 MBq/km^2).

Sea-air Transport. Sea-air transport may occur as a result of bubble scavenging in the water column coupled with their ejection into the air when bubbles burst on the water surface. Laboratory and field studies of this process showed respective ^{241}Am enrichments in aerosols of 92–580 and 20–40 over that in the bulk seawater (Belot et al. 1982; McKay et al. 1994a; Walker et al. 1986). In extensive field studies, Mackay et al. (1994) found clear evidence of localized temporal and spatial variability in the enrichment

of americium in sea spray over that in adjacent surf-zone seawater. While the enrichment is typically between 50 and 80, it ranges from 4 to 400. The aerosols produced by the sea spray are deposited nearby. Coastal strips in Cumbria, England have elevated levels of ^{241}Am from sea spray (Pattenden and McKay 1994; Walker et al. 1986). The water in this region of the Irish Sea receives effluent from the BNFL Sellafeld reprocessing plant. Studies of coastal soils in Northern Scotland are also believed to obtain their ^{241}Am contamination from the sea, albeit by a different mechanism. The activity level decreases with distance from the sea and is indistinguishable from background more than a few hundred meters inland. The actinide levels in these soils are in excess of those due to past nuclear explosions. The influence of sea spray was ruled out because of the much lower actinide concentrations in the Scottish water compared with that in the Irish Sea. In Northern Scotland, a stable foam or spume is formed along the coast that collects in rocky inlets. It is thought that this spume, that is rich in particulate matter and actinides, builds up in inlets, both overflowing onto the surrounding land and blowing inland on onshore wind (Pattenden and McKay 1994).

Leaching in Soil. After being deposited on soil, ^{241}Am may then leach into the soil. Since fallout of ^{241}Am is accompanied by its precursor ^{241}Pu, which also leaches into the ground, the amount of ^{241}Am at a depth is a function of the leaching of both the americium and plutonium as well as the decay of these isotopes. A depth profile of fallout americium was performed on samples at a representative site in North Eastham, Massachusetts in 1972 (see Table 6-1). Sixty-two percent of the activity resided in the upper 2 cm of soil and an additional 21% in the 2–4 cm depth (Bennett 1976). A comparison of modeling and experimental results indicates that americium is slightly less mobile than plutonium in this soil. Leaching studies of surface-deposited ^{241}Am in three Indian soils of widely differing characteristics, namely laterite, medium black, and alluvial, were conducted in soil columns using simulated rain corresponding to the mean annual precipitation (Vyas and Mistry 1980). It was found that 98% of the americium was retained in upper 2 cm of soil and amending the soil with organic matter only slightly reduced its mobility. The greatest retention was obtained with the medium black and lateritite soils where over 99% of the ^{241}Am was found in the upper 1 cm. In 1990, Bunzl et al. (1994) investigated the residence time, and consequently the migration rates, of fallout ^{241}Am in a typical undisturbed grassland soil (Alfisol) in Germany. Since ^{241}Am is present from its own deposition and also due to ingrowth from ^{241}Pu, the residence time of plutonium also had to be determined and a compartment model had to be employed. The residence half-time in a particular zone and mean migration rates of americium at various soil depths were (depth, residence half-time, mean migration rate): 0–2 cm, 8 years, 0.3 cm/year; 2–5 cm, 8 years, 0.4 cm/year; 5–10 cm, 10 years, 0.50 cm/year; 10–15 cm, 10 years, 0.5 cm/year; 15–20 cm, 4 years, 1 cm/year; and 20–30 cm, 9 years, 1 cm/year. The migration rates were similar to, but somewhat greater

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Table 6-1. Concentrations of ^{241}Am in Soil and Sediment

Site/sample type (year)	Depth, cm	Number of samples	Concentration	Units	Type	Reference
Bikini Atoll (not specified)[a], U.S. Nuclear Test Site from 1946 to 1958, including BRAVO explosion on 3/1/54						Robison et al. 1997b
Interior of island	0–5	157	0.26 (0.30)	Bq/g d.w.	Median (mean)	
	5–10	151	0.19 (0.27)	Bq/g d.w.	Median (mean)	
	10–15	127	0.081 (0.18)	Bq/g d.w.	Median (mean)	
	15–25	80	0.026 (0.11)	Bq/g d.w.	Median (mean)	
	25–40	59	0.012 (0.051)	Bq/g d.w.	Median (mean)	
	40–60	23	0.017 (0.035)	Bq/g d.w.	Median (mean)	
	0–40	53	0.11 (0.14)	Bq/g d.w.	Median (mean)	
Village area	0–5	63	0.11 (0.22)	Bq/g d.w.	Median (mean)	
	5–10	62	0.13 (0.20)	Bq/g d.w.	Median (mean)	
	10–15	63	0.12 (0.19)	Bq/g d.w.	Median (mean)	
	15–25	59	0.064 (0.15)	Bq/g d.w.	Median (mean)	
	25–40	52	0.059 (0.13)	Bq/g d.w.	Median (mean)	
	40–60	20	0.012 (0.11)	Bq/g d.w.	Median (mean)	
	0–40	51	0.13 (0.17)	Bq/g d.w.	Median (mean)	
Northern Marshall Islands Radiological Survey (NMIRS)[b], site of U.S. atmospheric weapons testing from 1946 to 1958, including BRAVO explosion on 3/1/54						Robison et al. 1997a
16 Atolls/islands	0–5	182	0.49–569	Bq/g d.w.	Range of means	
	5–10	182	0.26–435	Bq/g d.w.	Range of means	
	10–15	182	0.47–207	Bq/g d.w.	Range of means	

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Table 6-1. Concentrations of ^{241}Am in Soil and Sediment (*continued*)

Site/sample type (year)	Depth, cm	Number of samples	Concentration	Units	Type	Reference
	15–25	182	0.15–48	Bq/g d.w.	Range of means	
	25–40	182	0.038–21	Bq/g d.w.	Range of means	
Nuclear Weapons Testing Range, Maralinga, South Australia, site of 12 hydronuclear experiments at Taranaki in 1960, 1961, and 1963 (Vixen B Trials) in which 22.2 kg of plutonium was dispersed, having been ejected 2,500 feet vertically into the atmosphere.						Cooper et al. 1994
Taranaki, 5 sites	0–1	1	0.227–4.40	Bq/g d.w.	Range	
	1–2	1	0.026–0.799	Bq/g d.w.	Range	
	2–3	1	0.005–0.099	Bq/g d.w.	Range	
	3–4	1	0.009–0.022	Bq/g d.w.	Range	
	4–5	1	0.004–0.019	Bq/g d.w.	Range	
Lake Michigan, site 7 km from Grandhaven, Michigan, water depth 67 m (cores and sediment trap at 60 m)						Alberts et al. 1989
Core	0–1	NS	651±118	µBq/g d.w.	Mean±SD	
	1–2	NS	1,147±111	µBq/g d.w.	Mean±SD	
	2–3	NS	1,427±181	µBq/g d.w.	Mean±SD	
	3–4	NS	1,110±148	µBq/g d.w.	Mean±SD	
	4–5	NS	363±67	µBq/g d.w.	Mean±SD	
Suspended sediment, 60 m		NS	555±107	µBq/g d.w.	Mean±SD	
Cruise of RV Scotia, Orkney Islands, and northeast Scottish coast						Pattenden and McKay 1994
Bottom sediment	Surface	NS	0.44–21	mBq/g d.w.	Range	

Table 6-1. Concentrations of ^{241}Am in Soil and Sediment (*continued*)

Site/sample type (year)	Depth, cm	Number of samples	Concentration	Units	Type	Reference
Savannah River Estuary sediment[d]						DOE 1980
Tidal freshwater	0–5	NS	426±150	µBq/g d.w.	Mean±SD	
	5–15	NS	140±70	µBq/g d.w.	Mean±SD	
	15–30	NS	200±70	µBq/g d.w.	Mean±SD	
	30–50	NS	74±30	µBq/g d.w.	Mean±SD	
	50–70	NS	8.5±8.5	µBq/g d.w.	Mean±SD	
Mouth of estuary	0–5	NS	411±63	µBq/g d.w.	Mean±SD	
	5–15	NS	110±85	µBq/g d.w.	Mean±SD	
	15–25	NS	67±20	µBq/g d.w.	Mean±SD	
Soil in northeastern United States from 1970 to 1974, undisturbed soil unless otherwise indicated[e]						Bennett 1979
Brookhaven, New York	0–5	1	16[c]	mBq/km^2		
North Eastham, Massachusetts	0–30	1	17[c]	mBq/km^2		
	0–30	2	18[c]	mBq/km^2	Mean	
North Eastham, Massachusetts-cultivated soil	0–25	5	19[c]	mBq/km^2	Mean	
North Eastham, Massachusetts, background fallout[e]						Bennett 1979
	0–2	NS	8.1±2	mBq/km^2	Mean±SD	
	2–4	NS	2.7±0.3	mBq/km^2	Mean±SD	
	4–6	NS	1.1±0.2	mBq/km^2	Mean±SD	

Table 6-1. Concentrations of ^{241}Am in Soil and Sediment (*continued*)

Site/sample type (year)	Depth, cm	Number of samples	Concentration	Units	Type	Reference
	6–8	NS	0.59±0.1	mBq/km^2	Mean±SD	
	8–10	NS	0.1±0.07	mBq/km^2	Mean±SD	
	10–12	NS	0.1±0.07	mBq/km^2	Mean±SD	
	12–14	NS	0.1±0.04	mBq/km^2	Mean±SD	
	0–30	NS	19±2	mBq/km^2	Mean±SD	

[a]Continuous monitoring program 1975–1993; decay corrected to 1999
[b]Decay corrected to 1996
[c]Decay corrected to sampling date
[d]Concentration units converted from fCi/g
[e]Concentration units converted from mCi/km^2

d.w. = dry weight; NMIRS = Northern Marshall Islands Radiological Survey; NS = not specified; SD = standard deviation

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than, those of plutonium. After a period of 30 years, the ^{241}Am concentration on the surface layer was significantly diminished as a result of the migration. Stability constants for Am(III) indicate strong complexation with soil organic matter and this could explain its increased mobility with depth. In another study, Bunzl et al. (1995) investigated the association of ^{241}Am from global fallout with various soil components in six successive layers of undisturbed grassland in Germany from 0 to 30 cm using a sequential extraction procedure. In this way, the fractions that were readily exchangeable, bound to iron and manganese oxides, bound to organic matter, and bound to mineral (residual fraction) were identified. The association of a chemical with soil components controls their availability for plant uptake and transport. The americium was mainly deposited on the soil surface 30–40 years ago as a result of atmospheric nuclear weapon tests. The total deposition (sum over all layers) of ^{241}Am was 18±2 Bq/m^2 (490±50 pCi/m^2). On a Bq/kg basis, the data shows that the highest activity was contained in the 2–5 cm layer; slightly less was present in the 5–10 cm layer; activity declined markedly below 10 cm. On a Bq/m^2-cm thickness basis, the maximum activity was in the 5–10 cm layer. The attachment of americium to soil component varied considerably with soil layer. The largest amount of ^{241}Am was attached to organic matter, 18–74%, depending on the soil layer. A substantial fraction, 12–64%, was bound to oxides. In the 5–10 cm layer, the ^{241}Am in the readily exchangeable fraction was a minimum, 1.5%, while that attached to soil minerals was a maximum, 68%. In contrast, very little ^{241}Am (<0.5%) was bound to mineral in the surface (0–2 cm) layer and the deepest soil layers (15–20 and 20–30 cm). Compared with plutonium, much more americium is contained in the exchangeable fraction, a result which has been ascribed to americium's greater hydrolyzability. Americium has also been shown to be largely associated with the high molecular weight organic factions of dissolved organic matter in the soil solution of two grassland soils, a soddy podzolic soil and a peat soil in the vicinity of the nuclear reactor at Chernobyl. The distribution coefficients for ^{241}Am in these soils are (soil type (depth), K_d in mL/g): soddy podzolic—sod layer (0–2 cm), 1,220; soddy podzolic—mineral layer (2–5 cm), 810; peat (0–2 cm), 2,760; peat (2–5 cm), 4,550 (Bunzl et al. 1998). While it was similarly shown that the concentration of ^{241}Am was 2–3 times higher in organic matter than in whole sediment from Lake Michigan, organic matter was a very minor constituent of the sediment (<0.5%), so organic matter was associated with a smaller percentage of americium despite its higher concentration (Alberts et al. 1989). The bulk of the ^{241}Am in Lake Michigan was found in the hydrous oxides fraction of both the sediment core samples and suspended particulate matter.

Sorption to Sediment and Particles in Water. When discharged into water, americium rapidly sorbs onto particulate matter in the water column, settles, and adsorbs to bottom sediment (Murray and Avogadro 1979). The distribution coefficient, K_d, of americium between the particulate-associated phase and the dissolved phase is 2.0×10^6 (Pattenden and McKay 1994). The sediment-water distribution coefficient, K_d, was also 2.0×10^6 for intertidal sediment from the Ravenglass estuary near Sellafield, United Kingdom in agreement with other investigators (Davis et al. 1997). Equilibrium was established in 30 minutes. Adsorption is greatest for fine-grained particles. A large fraction of the heavier sediment particles will settle out quickly and remain close to the source. Finer particles (and soluble species) will remain suspended for longer periods and be dispersed by currents and tides. The distribution in sediment will be site specific, being largely determined by hydrology and the nature of the underlying sediment. Being a particle-reactive nuclide, like plutonium, americium's distribution in sediment after release will mirror that of plutonium, although its loss from the water column appears to be more rapid. The distribution of americium released from the British Nuclear Fuels Limited (BNFL) reprocessing plant at Sellafield, United Kingdom in the Irish Sea changed very little between 1978 and 1988 (McCartney et al. 1994). Americium in the effluent was entirely as Am(III). In their study of actinide activity in waters entering the northern North Sea, Murray et al. (1978) noted that the activity level of ^{241}Am relative to ^{238,239,240}Pu near the northwest coast of Scotland was very low, 0.06. Assuming that the contamination was derived from discharges into the Irish Sea 2–2.5 years earlier and 300–500 km to the south where Am-to-Pu alpha activity ratios varied from 0.9 to 2.56; there was a decrease of about 1.7 in the ratio from the point of discharge in spite of the increase in ^{241}Am due to the decay of ^{241}Pu. Similar results have been observed during the transport of actinides discharged from the nuclear fuel reprocessing plant at La Hague, France and in the case of fallout into Mediterranean waters (Murray et al. 1979). These observations have been ascribed to the americium's greater adsorption to suspended particulate matter which then settle out; americium consequently disappears more rapidly from surface water than plutonium. Support for this conclusion was found in another study where the percentage activity associated with suspended materials was 36±8 and 80±7 for plutonium and americium, respectively, <10 km from the point of discharge. Therefore, twice as much americium was associated with suspended matter than plutonium. At a distance of 100 km from the point of discharge, this fraction was still >50%. Core profiles of sediment in the northeast Irish field reveal major subsurface peaks of Sellafield-derived ^{241}Am in both the solid phase and interstitial water (Malcolm et al. 1990). These peaks were not related to the distribution of organic carbon or iron or magnesium oxides in the sediment or composition of the interstitial water, but rather resembled the discharge of ^{241}Am and plutonium. Davis et al. (1997) investigated how various environmental factors influenced K_d. The presence of CO_2 gas had a marked effect, increasing the amount of americium leached from sediment by a factor of 40. The increase is attributable to the presence of stable americium-

carbonate complexes in seawater and, to a lesser extent, the accompanying decrease in pH to 5.5 as a result of purging with CO_2. Experiments on the effect of pH on K_d suggested that desorption of americium from sediment at low salinity is due to low pH rather than to a drop in ionic strength. Therefore, the effect of low salinity and low pH in an estuary at low tide and when fresh water is backed up by the incoming tide is the remobilization of ^{241}Am. Resorption to sediment entering the estuary from rivers will result in the redistribution of activity.

The disposal of large quantities of radionuclides in Arctic seas have raised concern about the behavior of these chemicals in these waters. Fisher et al. (1999) found that the mean (range) K_d of americium in Kara Sea sediment was $1x10^6$ ($1x10^5$–$3x10^6$) mL/g which is in accord with that for temperate coastal sediment, $2x10^6$ ($1x10^5$–$2x10^7$) mL/g. The mean K_d values for americium in two areas in the Kara Sea, an arctic bay where radionuclides have been dumped, were $4.3x10^4$ and $35x10^4$ for ambient water column samples and $57x10^4$ and $16x10^4$ for bottom sediment samples (Carroll and Harms 1999; Carroll et al. 1999). The mean sediment water distribution function recommended by the International Atomic Energy Agency (IAEA) is $2x10^6$ (range $2x10^5$–$2x10^7$). Therefore use of the IAEA value in risk assessment modeling would underestimate the americium concentration in sea water. Carroll et al. (1999) stressed that for particle-reactive elements like americium, it is important to use site specific information for K_d.

Extensive adsorption determinations of ^{241}Am were obtained in freshwater, estuarine, and marine sediment-water systems (NRC 1981). The K_d values for sorption range from 10^4 to 10^6 mL/g, accounting for upward of 95% removal of americium to the solid phase. There were no apparent trends in K_d with salinity. There was an increase in K_d with increasing pH that is typical of hydrolyzable metal ions. The K_d declined with increasing sediment concentration. The mean sediment-water distribution coefficients obtained were (sample type, K_d standard deviation (SD)$x10^{-4}$): Lake Michigan—three sites, 54.8 (37.3); Clinch River, Tennessee, 73.3 (36.4); Hudson River estuary—three sites, 6.76 (2.43)–29.6 (8.8); Lake Washington, Washington, 18.0 (4.3); Lake Nitinat, Washington—anoxic marine, 25.4 (3.1); Washington Continental Shelf, 10.3 (1.8); Sinclair Inlet, Puget Sound, Washington, 45.4 (12.0); Saanich Inlet, British Columbia—anoxic fjord, 37.1 (8.4); Skagit Bay, Puget Sound, Washington—estuary—two sites, 26.8 (7.4), and 38.0 (10.6). Sediment-water distribution coefficients for ^{241}Am in various environments obtained from the literature include (site, K_d in mL/g): Lake Michigan, $>50x10^4$; Hanford, U-Pond, $1.9x10^4$ and $8.7x10^4$; and Rocky Flats, Pond A, $0.65x10^4$ (NRC 1981). Laboratory measurements yielded K_d values of 5,000 and 400 in Dutch subsoil and river sand, respectively (NRC 1981). Despite its high absorptivity, americium has migrated into groundwater and traveled underground at several national laboratories; the mobile forms were colloids and anionic complexes (McCarthy et al. 1998a, 1998b;

Penrose et al. 1990; Toran 1994). Artinger et al. (1998) found that increasing concentrations of humic substances reduced americium adsorption on sandy aquifer material, confirming the involvement of colloid-sorbed americium in its subsurface transport. The leachability of radionuclides in waste was assessed in soil column experiments in Carjo loam soil (B horizon) collected in the vicinity of the low level radioactive burial site at Los Alamos. In the case of ^{241}Am, 90% was retained in the silica sand layer placed on top of the soil column, 5% was in the upper 5 cm of the column, and the remainder was distributed at greater depths. In batch experiments, 40 and 49% of soluble ^{241}Am in waste, 0.3% of the total ^{241}Am present, sorbed to Puyo sandy loam and Carjo loam, respectively (Fowler et al. 1981). While americium should strongly adsorb to silicates and mineral oxides of the highly weathered shale saprolite in the unlined disposal trenches used for transuranic waste (TRU) at Oak Ridge National Laboratory, rapid migration in groundwater with little retardation over distances of 80 meters has been observed (McCarthy et al. 1998a, 1998b). This occurred when seasonal fluctuations in the water table permitted groundwater to contact TRU in the trenches. As a result of laboratory studies, the mobilization and transport of the americium has been demonstrated to result from complexation of the actinide by natural organic matter. Storm events aid the mobilization by providing the physical connection between the TRU and groundwater and increasing the amount of organic matter in the soil solution and groundwater. In the absence of natural organic matter, the adsorption coefficient on the site-specific saprolite was 8,900. A similar situation was documented in Los Alamos where nuclear wastes infiltrated a small aquifer. While the movement of the ^{241}Am was predicted to be limited to a few meters, the actinide was detectable in monitoring wells as far as 3,390 meters downgradient from the discharge (Penrose et al. 1990). Further investigation indicated that the mobile americium was tightly bound to colloidal material between 25 and 450 nm in dimension. The distributions of ^{241}Am activity with colloid particle size (<450 nm) in the well water were (particle size, percent): 25–450 nm, 28%; 5–25 nm, 0.75%; 2–5 nm, 26%; and <2 nm (filtrate), 43%. The unbound americium fraction appeared to be a stable anionic complex of unknown composition. At other sites at Los Alamos, contaminated plumes traveled 30 meters in <30 years and 40 meters in <20 years (Nyhan et al. 1985; Toran 1994). Migration appeared to be related to the water content of the tuff (a rock composed of the finer kinds of volcanic detritus usually fused together by heat) bed underlying the disposal site, and not simply to fractures in the rock (Nyhan et al. 1985). Investigation of the possible mobility of ^{241}Am in soils around Rock Flats contaminated by leaking drums under simulated conditions of extreme rain events found that 90% of ^{241}Am activity resided in the upper 12 cm of soil, below which it rapidly decreased; ^{241}Am activity in interstitial water never exceeded 0.4 Bq/L (11 pCi/L) at the deepest sampling layers even in a 100 year rain event (Litaor et al. 1996). However, evidence was found, in line with other investigators, that actinides may move to greater depths in macropores possibly formed by decaying root channels or other biological processes.

The behavior of colloidal americium was studied in an oligotrophic soft water lake used as the source of cooling water for a nuclear power plant in Wales (Orlanditi et al. 1990). ^{241}Am concentrations in the dissolved fraction (#450 nm) ranged from 5.8 to 18.2 fCi/L (210 to 673 μBq/L). Most of the americium was distributed in two size ranges, filterable particles ($450 nm) that contained ~37% of the activity and medium-sized colloids (5–100 nm) that contained ~54%; only a small fraction (~7%) of americium was 'dissolved' or in true solution, size <1.5 nm (3,000 molecular weight [MW]), where the americium would occur as free ions. The K_d of americium to filterable particles ($450 nm), the minimum sized particle that would settle by gravity, was found to be 470,000; the K_d of americium to colloidal particles was estimated to be about a factor of 20 higher. Lower K_d values obtained for ^{241}Am in dialysis experiments, which restrict the size of particles, than those obtained in shaking sorption experiments is a further indication that chemical species affects sorption (NRC 1981).

Distribution of ^{241}Am in a dialysis system containing sediment, phytoplankton, and detrital matter established that a substantial amount of americium accumulated in all three phases both in fresh and marine waters (NRC 1981). The adsorption process was not reversible and the longer the americium was adsorbed, the more difficult the chemical was to desorb. Appreciable amounts of americium have been shown to adsorb to bacterial cells such as those found in the Waste Isolation Pilot Plant in New Mexico (Francis et al. 1998). There is a potential that americium attached to biocolloids may facilitate its transport from the waste site.

Biotic Transport. Biotic transport can be defined as the actions of plants and animals that result in the transport of a radioactive material or other substance from a waste site to locations where it can enter pathways that may result in exposure to humans. Small mammals are ubiquitous and inhabit areas containing radioactive contamination or radioactive waste sites. Mammals inhabiting these areas may become contaminated with americium by consuming contaminated soil or plants and disturb americium-contaminated soil through their burrowing and excavating activities. These animals may therefore affect the distribution of americium within the waste site or transport americium to previously uncontaminated areas. In addition, small mammals may be consumed by animals higher in the food chain such as hawks and coyotes, which would add to the dispersal of americium from disposal areas. However, results of studies of numerous species of wildlife that inhabit the waste disposal area at INEEL indicate that wildlife do not contribute significantly to the spread of radionuclides to the surrounding environment (Arthur and Janke 1986). What may be more significant is the contribution of biotic intrusion on the soil covering the waste areas and the effect of these animals on moisture infiltration rates. A study was conducted of the dispersal of radioactive isotopes by deer mice at the Subsurface Disposal Area (SDA) at INEEL in

southwest Idaho (Arthur et al. 1987). These animals, which make up 69% of the small mammal biomass at the SDA, contained significantly higher levels of ^{241}Am than in control areas, the isotope accumulating primarily in the pelt and lung tissue, but also the gastrointestinal tract. Of the 22.8 μCi radionuclide inventory estimated to be contained in deer mice inhabiting the SDA during a 1-year period, about 8.4 μCi was estimated to have been transported off site, of which about 1% was the transuranic radionuclides ^{238}Pu, $^{239+240}$Pu, and ^{241}Am. These transuranic radionuclides also make up about 1% of the 28.8 μCi inventory present in deer mice defecation. It was estimated that during 1978–1979, deer mice excavated 12,450 kg of contaminated soil to the surface which could be further dispersed by wind. Evidence of burrowing activity by earthworms and ants were also found at Rocky Flats, Colorado and other sites (Litaor et al. 1994). Models have been developed to estimate the amount of radioactivity transported to the surface as a result of burrowing activity by animals and plant uptake at a generic LLW disposal site in an arid region (Kennedy et al. 1985). It was found that for ^{243}Am, plants were about 200 times more effective at transporting waste to the surface than biota. These results indicate that long-term biotic transport may affect the integrity of land burial, redistributing radionuclides such that they become a source of human exposure. Biotic transport will be affected by the oxygenation of soil, as waterlogged and anaerobic soils are less likely to be disturbed by earthworms and other burrowing animals (Livens and Hursthouse 1993).

Uptake by Plants. The uptake of chemicals by plants and their ingestion and bioaccumulation by animals is one mechanism by which a chemical present in soil, air, and water enters into the food chain of humans. Plants may become contaminated with americium as a result of uptake via the root and deposition on the upper part of the plant. Direct deposition occurs during the deposition period, but surface contamination may also occur from resuspension of or direct contact with contaminated soil. Surface deposits may be reduced by weathering or by washing or processing, thereby reducing the amount of americium consumed by humans. Some studies indicate that foliar uptake and subsequent translocation, which is affected by rainfall and size of the deposited particles, may rival the soil-root pathway in magnitude (Cataldo et al. 1980). Translocation of transuranic elements taken up through the roots into seeds and fruit is generally very low (Bennett 1979). Uptake in seed is significantly lower than for other above ground plant parts, and legumes accumulate about 10 times more than grasses (Schreckhise and Cline 1980).

Plant uptake from soil depends on factors such as the form and availability of the chemical, soil chemistry, plant species, and environmental conditions. Various studies indicate that ^{241}Am uptake by plants is on the order of 10^{-2}–10^{-6}, about an order of magnitude greater than ^{239}Pu under the same

conditions (Bennett 1979; EPA 1979; Romney et al. 1981; Schultz et al. 1976; Zach 1985). Uptake is somewhat greater under acid conditions and greater to the leaves than to the grain or fruit. Plant uptake experiments with potatoes, peas, and corn were performed using 'fallout background' soil in North Eastham, Massachusetts. The ^{241}Am in the plant was compared with the average activity in the upper 30 cm of dried soil, 0.0033 Bq/g. The concentration ratios (CRs) (activity per unit wet weight of plant/activity per unit dry weight of soil), also referred to as the transfer coefficient, were 1.7×10^{-3} for husked corn plus cob, 1.0×10^{-3} for peeled potatoes, and $<1.6 \times 10^{-3}$ for shelled peas (Bennett 1979). Flours that were milled from crops of wheat, rye, barley, and oats grown on German soil where radionuclides resulted exclusively from global fallout contained #5.2, 11, 31, and 70 μBq/kg (1.9 fCi/kg) of ^{241}Am, respectively (Bunzl and Kracke 1987). The respective soil to plant transfer factors were #0.86×10^{-4}, 1.5×10^{-4}, 4.7×10^{-4}, and 37×10^{-4}. The concentration of ^{241}Am in the bran was a factor of 10–38 higher than in the flour for wheat, rye, and oats. Romney et al. (1981) studied ^{241}Am uptake in crops of peas, soybeans, tomatoes, and wheat grown in seven soils representative of major food production areas of the United States. The CRs in the foliage ranged from 10^{-5} to 10^{-2} and the CR in the seed, grain, and fruit parts ranged from 10^{-6} to 10^{-4}, about a factor of 10 lower than in the foliage. Uptake varied among the difference soils by a factor of about 100. However, Schulz et al. (1976) obtained less of a variation in uptake of ^{241}Am by wheat in three California soils. The CRs were 6.3×10^{-6}, 16.4×10^{-6}, and 6.1×10^{-6} in Aiken (a slightly acidic forest soil), Yolo (an agricultural soil), and Panoche (a calcareous soil) soil, respectively. The CRs for $^{239, 240}$Pu were approximately the same. Nisbet and Shaw (1994) obtained CRs ranging from 0.04×10^{-4} to 4×10^{-4} for ^{241}Am for carrots, cabbage, and barley grown in artificially-contaminated peat, loam, and sand soils; the CRs were in the order carrot > cabbage > barley. The CRs therefore ranged over 2 orders of magnitude with crop and soil type. Uptake was lowest in peat soil where the organic matter present plays an important role in complexing the ions making them less available for uptake. The CR remained unchanged for plants grown $3 years after contamination of the soil. The soil-plant transfer appears to be related to the concentration of ^{241}Am in the soil solution, rather than the bulk soil. While Livens et al. (1994) obtained plant-soil CRs for ^{241}Am in the range of 10^{-5}–10^{-6}, the plant-soil solution CR obtained was much higher, 210–640 L/kg (Bennett 1979). Amendation of soil with lime and organic matter decreases uptake (Adriano 1979; Hoyt and Adriano 1979). This is thought to reduce the solubility or increase americium binding, thereby reducing its availability.

The uptake of radionuclides into plants from soil at contaminated sites at DOE facilities and elsewhere has been the impetus for several studies. Such concerns led to experiments conducted in May 1996 at the Los Alamos National Laboratory (LANL), New Mexico using stream channel sediment from Los Alamos

Canyon (LAC). This stream received various amounts of untreated radioactive waste in the 1940s (Fresquez et al. 1998b). Pinto beans, sweet corn, and zucchini were grown in this contaminated soil as well as in soil from seven regional background locations (RBG) between 32 and 96 km around the LANL The radionuclide content of this latter soil is reflective of global fallout. The ^{241}Am levels in LAC and RBG soils were 0.708 and 0.005 pCi/g d.w.(dry weight), respectively. The mean ^{241}Am concentrations (dry weight basis) in edible tissue of crops grown on LAC soil and RBG soil were (crop, LAC level [10^{-5} pCi/g], RBG level [10^{-5} pCi/g]): beans, 21.5, 7.0; corn, 27.8, 4.1; and zucchini, 182.5, 27.1. The CRs (activity of ^{241}Am in dry plant tissue to that in dry soil) in the edible LAC crop tissues were (crop, concentration ratio): LAC beans, 3.04×10^{-4}; LAC corn, 3.93×10^{-4}; and LAC zucchini, 25.78×10^{-4}. These ratios are much lower than those in composite nonedible crop tissue which ranged from 48×10^{-4} to 740×10^{-4}. A garden plot was established on a portion of a drained contaminated pond at the Savannah River (SRS) site having a mean ^{241}Am concentration of 1.60±0.59 Bq/kg in soil (0–15 cm). Geometric mean concentration ratios of ^{241}Am (Bq/kg dry plant/Bq/kg dry soil) in the various crops were: bush beans, 0.0072; corn husk, 0.0016; corn kernel, 0.0011; turnip green, 0.0528; and turnip tuber, 0.0053 (Whicker et al. 1999). Uptake for americium was similar to that of curium, but was much higher than for plutonium, thorium, and uranium. Based on the conditions of the study, it was thought that root uptake was the primary mechanism of plant contamination. The concentration ratios for americium are about an order of magnitude higher than a similar study in Tennessee. Since it has been demonstrated that liming decreased uptake, the high uptakes in the SRS study have been ascribed to the acidity of the soil (pH 3.8–4.5 vs. 7–7.5). SRS served as a primary source of reactor-produced nuclear materials, primarily plutonium and tritium, for nearly 4 decades. The CR for ^{241}Am in barley grown on contaminated soil at the Nevada Test Site was of the order of 10^{-4} for vegetative growth and 25–100 times lower for the grain (Schulz et al. 1976). Similar CRs (3×10^{-4}–7×10^{-4}) were obtained for green vegetables and root vegetables on land reclaimed from the sea that was contaminated with Sellafield discharges (Green and Wilkins 1995); an earlier study with potatoes, obtained CRs about an order of magnitude less (Popplewell et al. 1984).

Bioconcentration in Aquatic Organisms. Transuranic elements are generally associated with the gut or external surfaces of aquatic organisms. Bioconcentration factors (BCFs) for ^{241}Am measured at a pond at Savannah River, Georgia for fish and other plants and animals are found in Table 6-2. A study was performed at a nuclear waste pond at Hanford where the sediment concentration of ^{241}Am was about 150 pCi/g (5.5 Bq/g), approximately 3 orders of magnitude above background levels, to ascertain the maximum concentration of the actinide that would accumulate in the whole fish and fish fillet (Emery et al. 1981). Both the bluegill and largemouth bass were studied. The concentration of ^{241}Am in the water

Table 6-2. Bioconcentration Factors for ^{241}Am

	Bioconcentration factor[a]			
Species	Value	Mean	Number of samples	Site
Large-mouth bass, muscle	2,500		1	SRS
Fish muscle		50		Literature value[b]
Fish muscle		25		NRC[c]
Bullhead catfish, bone	4,200		1	SRS
Macroinvertebrates, larvae	78,000–240,0000		2	SRS
Macrophyte, rooted vascular	1,400–21,000		3	SRS
Macrophyte, floating vascular	75,000		1	SRS
Turtle, muscle	5,600		1	SRS
American coot, muscle	650		1	SRS
Bahia grass	0.03–0.12	0.067	6	SRS
	2.3×10^{-7}–0.005			Literature value[b]
		2.5×10^{-4}		NRC[c]
Pine tree leaves	0.0052–0.021	0.013	2	SRS
	0.0001–0.1			Literature value[b]

[a]Bioconcentration factor is concentration in organism dry weight/concentration in medium (i.e., water, soil as appropriate).
[b]Values obtained from various literature sources cited in DOE 1996
[c]NRC values cited in DOE 1996

NRC = Nuclear Regulatory Commission; SRS = Savannah River Site

Source: DOE 1996

was about 0.2 fCi/mL (7 μBq/mL). The results indicate that both short- and long-term uptake of americium were low, that uptake was similar for short-term, 5 days, and long-term, 430 days, experiments and that direct sediment-to-fish transfer was the primary route for americium uptake. In both species of fish, there were only a few cases where fillet concentrations were >10 times those in controls. The maximum concentration of ^{241}Am obtained in bass and bluegill were 0.030 and 0.028 pCi/g (1.1 and 1.0 mBq/mL) dry weight in fillet and 0.067 and 2.0 pCi/g (2.5 and 74 mBq/mL) in bass.

In marine organisms, the target organs and tissues of americium bioaccumulation are mainly the digestive gland, gill, and exoskeleton (Chassard-Bouchaud 1996). Uptake in mussels appears to be primarily from seawater rather than ingested sediment (Hamilton and Clifton 1980). Americium taken up by mussels from water was primarily associated with the shell, while that obtained from food was bound to soft tissue (Fisher et al. 1996). Absorption efficiencies from ingested food in mussels was also critical in determining bioaccumulation. Clifton et al. (1983) studied the depuration profiles in mussel populations chronically exposed to radioactive waste discharges from the BNFL reprocessing plant at Sellafield, Cumbria, into the northeast Irish Sea. Apart from clearance from the digestive tract (half-life 0.9 hours), the half-time of ^{241}Am is 303 days. Essentially all of the ^{241}Am taken up by a euphausiid, a shrimp-like zooplankton, was by passive adsorption onto exoskeletons, so that negligible americium was retained by the animal after molting (Fisher et al. 1983). Wet weight concentration factors were of the order of 100 after a week of exposure with the BCFs varying linearly with the surface area-to-weight ratio of the organism. Americium was also taken up by feeding on suspensions of diatoms, but there was negligible assimilation and most americium passed through the gut and was defecated.

Uptake and retention of americium by benthic organisms is quite variable (Fowler and Carvalho 1985). In general, filter feeders such as tunicates can clear particles containing ^{241}Am from seawater by filtration through the granchial basket and accumulate small amounts in internal tissue. Mucous feeding guilds of suspension-feeders like crinoids do not efficiently trap americium. Uptake here appears to occur by complexation or adsorption of dissolved americium to the body wall. Echinoderms and some large crustaceans assimilated americium with their ingested prey, although large differences in half-times and assimilation efficiencies in different groups suggest different feeding-digestion physiologies. Uptake in benthic marine isopods is to a great extent by surface adsorption to the exoskeleton and to a lesser extent with the gut, digestive gland, muscle, and haemolymph (Carvalho and Fowler 1985). Half-times in the long-lived compartment were 261 days. Elimination from the internal tissue was more rapid than from the exoskeleton. The fraction assimilated into tissue from food was <5%. The transfer of ^{241}Am from

marine sediments contaminated by nuclear tests to benthic polychaets was <1% (dry weight) (Hamilton et al. 1991).

The biological uptake in aquatic organisms may depend on factors such as temperature, time after intake, season of the year, and water quality. In the brittle star (*Ophiothrix fragilis*), a species that dominates Arctic benthic communities, the initial uptake was 3 times greater at 12 EC than at 2 EC, but depuration was not temperature dependent (Hutchins et al. 1996a). After 14 days, the BCF was 48 at 2 EC and 53 at 12 EC. In the sea star (*Asteruas forbesi*), another Arctic organism, temperature greatly enhanced the retention of americium ingested in food (Hutchins et al. 1996b). The biological half-time of americium ingested in food was 31 days at 12 EC and essentially infinite at 2 EC. Experiments performed on the freshwater snail (*Lymnaea stagnalis*) found higher ^{241}Am uptakes from mesotrophic pond water at acid pH 5.0 than at pH 8.0 (Thiels et al. 1984). In summer, the BCF in the freshwater snail was more than twice as high in acidic as in alkaline media. In addition, uptake was a function of season, being greater in August than in December. This has been ascribed to changes in metabolic rate with changing photoperiods. Uptake of ^{241}Am by two freshwater benthic invertebrates, *Hyallella* and *Tubifex* from water was two orders of magnitude higher than from detritus or sand, suggesting that soluble ^{241}Am is the principal route of uptake and that desorption from particulate matter must occur before the substance is available for uptake (Sibley and Stohr 1990). No uptake was observed from labeled National Bureau of Standards (NBS) standard sediment.

Mahon (1982) measured BCFs of 1,576 and 459 in algae and plankton, respectively. Horikoshi et al. (1981) determined BCFs in several species of bacteria that ranged from 2,794 to 354,000. However, bioconcentration by the bacteria represented adsorption onto the cell surfaces of the bacteria rather than true biological uptake.

Biomagnification in the Food Chain. The available evidence suggests that the transport of americium up the food chain to man is slight (Bulman 1978). Americium in the food chain lichen-reindeer-man was studied by determining the concentrations ^{241}Am in lichen and reindeer liver in Finnish Lapland (Jaakkola et al. 1977). During 1963–1974, ^{241}Am in lichen ranged from 1 to 40 pCi/kg dry weight. Levels in six reindeer livers in 1974–1976 contained 0.3–5.4 pCi/kg fresh weight.

6.3.2 Transformation and Degradation

Americium isotopes are transformed by radioactive decay. However, the half-lives of the principal americium isotopes, ^{241}Am and ^{243}Am, are very long, 432 and 7,370 years, respectively, and there is only a small amount of transformation over a human lifetime. ^{241}Am is formed by the decay of ^{241}Pu (half-life 14.4 years) and this can lead to a significant transformation of that isotope to ^{241}Am in humans, especially for ^{241}Pu that is fixed in the bone.

6.3.2.1 Air

There is limited information available regarding the abiotic transformation of americium in the atmosphere. Oxidation is the most common reaction that occurs in the atmosphere. This would not be expected for americium compounds, which are generally present in the +3 oxidation date under environmental conditions.

6.3.2.2 Water

The principal abiotic processes affecting americium in water is the precipitation and complex formation. In natural waters, americium solubility is limited by the formation of hydroxyl-carbonate ($AmOHCO_3$) precipitates. Solubility is unaffected by redox condition. Increased solubility at higher temperatures may be relevant in the environment of radionuclide repositories. In environmental waters, americium occurs in the +3 oxidation state; oxidation-reduction reactions are not significant (Toran 1994).

6.3.2.3 Sediment and Soil

Americium will occur in soil in the trivalent state. The transformations that may occur would involve complexation with inorganic and organic ligands (see Section 6.3.1) and precipitation reactions with anions and other substances present in the soil solution. The ^{241}Am occurring as an ingrowth progeny of ^{241}Pu and trapped in a plutonium matrix will exhibit solubility and biokinetic characteristics of the plutonium, rather than americium.

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

In 1973, the EPA established The Environmental Radiation Monitoring System (ERAMS), a nationwide network, for obtaining data in environmental samples. ERAMS consists of a network of sampling stations that provide air, surface and drinking water, and milk samples that the EPA uses to obtain environmental concentrations of radioactive material. The objective of this system is to identify trends in the accumulation of long-lived radionuclides in the environment (Virginia Department of Health 2000). Sampling locations for ERAMS are located near primary population centers to provide optimal population coverage. In the most recent ERAMS report covering July–September 1997, no monitoring levels appeared for americium (EPA 1997c).

6.4.1 Air

The annual average ^{241}Am concentration in surface air in the mid-latitudes resulting from nuclear testing between 1951 and 1978 was estimated using the atmospheric transport model and the amount of ^{241}Am that would have been produced as a result of the testing. The results show a rapid increase in ^{241}Am surface air concentrations, which reached a maximum of 23 aCi/m^3 (0.85 μBq/m^3) in 1963–1964. Since 1973, levels have been <1 aCi/m^3 (0.037 μBq/m^3) and are continuing to decline (Bennett 1979). Measurement of these low 1973 levels would have required exceedingly long sampling periods at common sampling rates. Integral concentration over the entire fallout period is estimated to be 0.12 fCi-year/m^3 (4.4 μBq-year/m^3). Confirmation for these concentration estimates come from two sources: comparison with plutonium fallout and cumulative deposits of ^{241}Am activity in soil, which is a reflection of integral air concentrations.

The Electric Power Research Institute (EPRI 1981) conducted a survey of transuranic radionuclides in the terrestrial environs of nuclear power plants in the United States in 1978–1979. The plants included two pressurized water reactors (PWRs) and two BWRs that were of modern design and had been in operation at least 3 years. The ^{241}Am air concentrations around all of the power plants were extremely low and indistinguishable from fallout background

Selected radionuclide levels in air were measured at Bragin, 55 km north of Chernobyl from April 28, 1986, 2 days after the start of releases from the Chernobyl accident, for 18 days until May 15. Mean activity concentrations of ^{241}Am during this period were 40 μBq/m^3 (1.1 fCi/m^3) while the mean total radionuclide activity was 20 Bq/m^3 (Knatko et al. 1993). Debris from the Chernobyl accident arrived in

Denmark 1.5 days after the explosion. Air samples collected at Roskilde, Denmark on April 27–28, contained a mean air concentration of ^{241}Am of 5.2 μBq/m^3 (0.14 fCi/m^3). In May 1986, the mean concentration was 11 μBq/m^3 (0.30 fCi/m^3) (Aarkrog 1988). Whereas debris from nuclear weapons testing is injected into the stratosphere, debris from Chernobyl was injected into the troposphere. As the mean residence time in the troposphere is 20–40 days, it would appear that the fallout would have decreased to very low levels by the end of 1986. However, from the levels of other radioactive elements, this was not the case. Sequential extraction studies were performed on aerosols collected in Lithuania after dust storms in September 1992 carried radioactive aerosols to the region from contaminated areas of the Ukraine and Belarus. The fraction distribution of ^{241}Am in the aerosol samples was approximately (fraction, percent): organically-bound, 18%; oxide-bound, 10%; acid-soluble, 36%; and residual, 32% (Lujaniene et al. 1999). Very little americium was found in the more readily extractable 'exchangeable and water soluble' and 'specifically adsorbed' fractions.

The accident at the Three Mile Island nuclear reactor in March 1979 resulted in contamination of the containment and auxiliary buildings. An aerosol sample from the auxiliary building obtained by filtering about 1.4x10^9 cm^3 of air through a fiber glass filter for 8 days contained an estimated total transuranic actinide radioactivity of 13 Bq (350 pCi), of which ^{241}Am was the major contributor (Kanapilly et al. 1983).

Air monitoring of ^{241}Am was started at Hanford in 1993 in order to estimate regional background concentrations of the radionuclide before large-scale remediation work commenced (PNL 2000). Maximum and mean onsite levels of ^{241}Am were 0.50 and 0.46 aCi/m^3 (0.019 and 0.017 μBq/m^3) in 1994 compared with 0.90 and 0.41 aCi/m^3 (0.033 and 0.015 μBq/m^3) in 1993. Concentrations were not measurable at perimeter sites or at distant community-operated surveillance stations. In an attempt to obtain background ^{241}Am concentrations around the Waste Isolation Pilot Plant (WIPP) in southeastern New Mexico, a plant developed for demonstrating the safe disposal of transuranic waste, high volume samples were obtained continuously over a 14-day period between February 23 and May 6, 1996 1 km from the facility. Samplers concurrently obtained total suspended solids (TSP) and particles >10 μm (PM_{10}) at a 5 meter level elevation and PM_{10} at a 2 meter level. The activities of ^{241}Am were 8±2, 6±2, and 11±1 nBq/m^3 (0.2±0.05, 0.16±0.054, and 0.31±0.03 aCi/m^3) in the PM_{10}-2 m, PM_{10}-5 m, and TSP-5 m samples, respectively (Lee et al. 1998). Approximately 50% of the ^{241}Am activity concentration was in the PM_{10} fraction. Using values for the ^{239,240}Pu concentrations measured at the site and those obtained in the early 1980s and estimated mean residence times for Pu in the troposphere and stratosphere, it is likely that the observed ^{241}Am levels reflect resuspension of dust contamination at the

WIPP site and not background ^{241}Am levels. Present background levels result from past atmospheric nuclear testing and would be fairly uniform throughout the northern hemisphere.

6.4.2 Water

^{241}Am may enter surface water from nuclear power plants sited on the shores. A region of possible concern is the Great Lakes where 35 nuclear facilities are located in the lakes basin. A sample of water from Lake Ontario collected in 1985 and filtered through a 0.45 μm Millipore filter contained 0.3 mBq ^{241}Am/L (8 fCi/L) (Platford and Joshi 1986). A few water samples from the Savannah River, which received runoff and discharges from the DOE Savannah River Plant, contained 0.05 fCi ^{241}Am/L (2 μBq/L), about the same levels as other American rivers (DOE 1980).

In some surface waters that have been contaminated by waste discharge, the concentrations of americium may be higher than background levels. For example, at times, measurable levels of ^{241}Am have been detected in the creek where waste water is discharged from Argonne National Laboratory's treatment plant. However, this creek is not used for drinking water and no ^{241}Am has been detected in the Des Plains River into which it empties (ANL 1999). The 1995–1996 levels of ^{241}Am in rivers and lakes in the exclusion zone of the Chernobyl Nuclear Power Plant ranged from 20 to 200 μBq/L (0.5–5 fCi/L) with 70% of the radioactivity being contained in the particulate phase (Matsunaga et al 1998). The particles appear to have originated from the erosion of surface soil in the area.

The concentration of ^{241}Am in filtered sea water is very variable. Some ^{241}Am levels (filtered through a 0.22 μm filter) are (site, date, and concentration): Arctic ocean, 7/81, 1.77 μBq/L (0.048 fCi/L); English Chanel, 4/85, 4.96 μBq/L (0.13 fCi/L); and Eastern Irish Sea, 4/84, 4,840 μBq/L (131 fCi/L) (Lovett et al. 1990). Filtered seawater in the North Channel of the Irish Sea was about 200 and 120 mBq/m^3 in 1979 and 1986, respectively (Pattenden and McKay 1994). Because of the higher sediment load, near shore water generally has a larger fraction of americium bound to particles, and therefore, seawater from the northern coast of Scotland (which is impacted by the United Kingdom Atomic Energy Agency (UKAEA) Technology Dounreay Establishment, a nuclear research and development facility at Dounreay, Scotland) shows a large variation in near-shore concentrations (see Table 6-3). Levels were much higher in the immediate vicinity of Dounreay than elsewhere along the coast. The 1984 level was unusually high because the monthly discharge from the plant before the sampling was 40 times the mean monthly level. Time series measurements on seaweeds and winkles show that the americium levels peaked in the early 1980s and then declined by 1989, levels were the lowest they had been in 12 years. In comparison,

Table 6-3. Concentrations of ^{241}Am in Water

Site/sample type	Year	Number of samples	Concentration (mBq/m^3)	Concentration (pCi/m^3)	Type
Dounreay, Scotland (north coast) near U.K. AEA Technology Dounreay Establishment R&D (nuclear reactor, reprocessing)					
Filtered seawater (0.45 µm)	1984	13	6,058±9,912	164±268	Mean±SD
	1985	16	36±18	0.97±0.49	Mean±SD
	1986	14	47±62	1.3±1.7	Mean±SD
	1987	30	15±17	0.41±0.46	Mean±SD
Unfiltered seawater	1986	14	110±147	3.0±4.0	Mean±SD
	1987	30	35±65	0.95±1.8	Mean±SD
Scotland mainland and some island coasts (near shore)					
Filtered seawater (13 sites)	1982	NR	1.9–140	0.051–3.78	Range
Cruise of RV Scotia, Orkney Islands, and northeast Scottish coast					
Near surface unfiltered seawater	1982	NR	6–270	0.2–7.3	Range
30–40 m unfiltered seawater		NR	220–650	5.9–17.6	Range

NR = not reported; R&D = Research and Development; SD = standard deviation; U.K. AEA = United Kingdom Atomic Energy Agency

Source: Pattenden and McKay 1994

offshore ^{241}Am levels in surface seawater of the North Sea and North Atlantic Ocean has remained relatively constant, ranging from around 10 Bq/m^3 (270 pCi/m^3) between 1976 and 1988 (Pattenden and McKay 1994). Nearshore levels of ^{241}Am were much greater than offshore concentrations, and the activity in the dissolved and particle-associated fractions were broadly similar.

The concentration of ^{241}Am in New York City tap water in 1974 was estimated to be 0.03 fCi/L (1 μBq/L) based on the average ^{241}Am/^{239,240}Pu ratio of 0.10 obtained in 1975–1976 and the 1974 ^{239,240}Pu concentration (Bennett 1979).

6.4.3 Soil

Soil may become contaminated with ^{241}Am from the deposition of the element released to the atmosphere from nuclear weapons' testing and nuclear reactors, or secondarily, from the resuspension of contaminated soil. ^{241}Am levels may then increase due to ingrowth from the decay of ^{241}Pu (half-life 14.4 years). Soil surveillance provides us with baseline information with which unplanned releases can be compared, as well as long-term contamination trends. It also provides us with information about direct exposure to humans (inhalation of dust, ingestion of dirt) as well as potential sources of exposure (contamination of groundwater, uptake into plants and animals). The concentration of ^{241}Am in soil samples from the northeastern United States from 1970 to 1974 is shown in Table 6-1. The areal activity at the sampling date ranged from 0.37 to 0.49 mCi/km^2 (14 to 18 MBq/km^2) which compares well with that calculated from estimated values obtained from releases from nuclear weapons testing combined with an atmospheric transport model (Bennett 1979). ^{241}Am in soil resulting from cumulative deposition in the New York region was estimated as being fairly constant since about 1969 at about 0.060 mCi/km^2 (2 MBq/km^2), whereas ^{241}Pu peaked at about 20.1 mCi/km^2 (744 MBq/km^2) in 1965. The decay of ^{241}Pu adds to the cumulative deposits of ^{241}Am in soil, bringing the total ^{241}Am to about 0.8 mCi/km^2 (30 MBq/km^2). The ^{241}Am inventory should reach a maximum of 0.88 mCi/km^2 (33 MBq/km^2) in 2035. The radioactive decay of ^{241}Am will then exceed its ingrowth, and the cumulative deposits of ^{241}Am will then slowly be depleted.

Nuclear Power Plants. EPRI (1981) conducted a survey of transuranic radionuclides in the terrestrial environs of nuclear power plant in 1978–1979. The plants included two PWRs and two BWRs that were of modern design and had been in operation at least 3 years. The ^{241}Am levels in soil samples collected around all of the power plants were indistinguishable from fallout background.

A survey of Big Trees Park was conducted in 1998 after soil samples taken in the park in 1993 and 1995 revealed higher-than-expected concentrations of plutonium (LLNL 1999). Big Trees Park is a public park in the city of Livermore, California located about one-half mile from Lawrence Livermore National Laboratory (LLNL). The pattern of ^{241}Am distribution was the same as that for plutonium with levels significantly higher inside ornamental tree wells than outside the wells or at depths. Activity levels were well below EPA's residential preliminary remediation goal of 2.5 pCi/g (93 mBq/g) which may have necessitated cleanup. The source of the contamination was most likely sewage sludge from LLNL that was applied to the ornamental trees in the park.

Great Britain. In 1993, the Ministry of Agriculture, Fisheries, and Food (MAFF) in the United Kingdom systematically surveyed the concentrations of ^{241}Am in soil and grass samples near 18 nuclear sites in England and Wales (Sanchez et al. 1996). ^{241}Am was detected in most soil samples; the range was 0.03–0.40 Bq/kg (0.8–11 pCi/kg) soil; three samples were below the detection limit. Sanchez et al. (1998) measured the ^{241}Am activity in root mat samples from tide washed pastures in 17 estuaries spanning the eastern seaboard of the Irish Sea from Solway in northwest England to David's Head in southern Wales. Fourteen of the pastures had detectable levels of ^{241}Am with median levels ranging from 64 to 5,570 Bq/kg (1.7 to 150 nCi/kg) dry weight and individual samples ranging from 14 to 10,230 Bq/kg (0.38 to 276 nCi/kg) dry weight. ^{241}Am accumulations in soils (to 10–15 cm depth) near Greeny Geo, Scotland ranged from 2,000 to 11,400 Bq/m^2 (54 to 308 nCi/m^2) (Pattenden and McKay 1994). This coastal area is contaminated by foam or spume that forms in seawater in rocky inlets and blows over the land. The seawater in the area receives discharges from the BNFL Sellafield facility. Sediment from two Harbors on the Isle of Mann, which is impacted by the reprocessing plant at Sellafield in the United Kingdom, contained ^{241}Am ranging from <2.0 to 8.1 Bq/kg (<50 to 220 pCi/kg) dry weight during 1990–1993 (McKenna and Longworth 1995). Nearshore sediment core samples taken at several sites off the northern coast of Scotland (see Table 6-1) had fairly constant ^{241}Am levels with depth showing that mixing had occurred to a depth of 74 cm (Pattenden and McKay 1994).

LANL. Soil surveillance is conducted at national laboratories to monitor the levels of radionuclides and ascertain their possible migration. Data from such a program at the LANL in 1974–1996 was evaluated to ascertain whether there were changes in radionuclide concentrations during that period and whether changes could be ascribed to air emissions and fugitive dust (Fresquez et al. 1998a). Surface samples (0–5 cm) were obtained on-site and from perimeter and background sites. Americium levels at LANL sites were significantly higher than at background sites. The mean 1974–1996 concentrations of ^{241}Am in soil at LANL and background sites were 0.011±0.006 and 0.006±0.002 pCi/g (0.41±0.22 and

0.22±0.07 mBq/g) dry weight, respectively. Sediment 2.1 km downstream of a National Pollutant Discharge Elimination System (NPDES) outfall in Mortandad Canyon, Los Alamos County, New Mexico in 1994 contained high levels, 8.93 pCi/g (330 Bq/g) dry weight of ^{241}Am. However, the ^{241}Am concentrations in sediment upstream of the outfall and 4.0 km downstream below a sediment trap were below upper limits of regional background levels, 0.023 pCi/g (0.85 Bq/g) dry weight. (Bennett et al. 1996).

Rocky Flats. Rocky Flats (RF) near Golden, Colorado was contaminated with ^{241}Am as a result of past waste storage practices, such as leaking drums of contaminated oil (Litaor et al. 1994). Sampling was performed in four regions of expected actinide activity (26 pits) east of RF. Activity decreased with increasing distance east of the former storage site and was predominantly confined to the upper 12 inches of soil. The mean ^{241}Am activity in the upper 3 inches of soil in the four regions were 1,653, 47.4, 2.9, and 3.7 Bq/kg (44.6, 1.28, 0.078, and 0.100 nCi/kg). In the region with the highest activity, the mean concentrations were 1,653, 1,063, 758, 595, 122, and 2.6 Bq/kg (44.6, 28.7, 20.5, 16.1, 3.3, and 0.070 nCi/kg) at depths of 0–3, 3–6, 6–9, 9–12, 12–18, and 36–48 inches, respectively.

INEEL. ^{241}Am contamination occurred outside the SDA to a distance of 2,500 meters at the INEEL (Markham et al. 1978). Maximum concentrations of ^{241}Am, 2,048 nCi/m^2 (75.8 kBq/m^2) in the 0–4" surface layer, near the perimeter of the SDA were thought to be due to flooding and to localized drainage of water, while low concentrations away from the SDA perimeter are a result of wind transport. Soil sampled at 118 plots around RF contained ^{241}Am ranging from 0.18 to 9,990 Bq/kg (0.0049–270 nCi/kg) with a mean and SD of 321 and 1,143 Bq/kg (8.67 and 30.9 nCi/kg), respectively (Litaor 1995). The distribution pattern reflects wind dispersion consistent with the prevailing winds at RF.

Superfund NPL Sites. ^{241}Americium has been detected in soil samples at one of eight hazardous waste sites where americium has been identified in some environmental compartment (HazDat 2001). It was not found in sediment at any of these sites. The distribution of Superfund NPL sites is shown in Figure 6-1.

Chernobyl. ^{241}Am, in five samples of surface soil collected 1.5–15 km from the Chernobyl accident in July 1987, was predominantly in the 0.005–0.25 mm size fraction of soil, which comprised 65% of the mass (Berezhnoi et al. 1991).

Bikini Atoll. Surface soil collected at 20 sites on Bikini Atoll in 1972 contained 1.2–45 pCi/g of ^{241}Am (Nevissi et al. 1976). This level will increase for several decades as a result of ingrowth from ^{241}Pu before it begins to decrease. Bikini Atoll was the site of 23 nuclear detonations between 1946 and 1958.

Alaska. The National Oceanic and Atmospheric Administration (NOAA) conducted a study in the Beaufort Sea in northwestern Alaska during the summer of 1993 to ascertain the levels of radionuclide activity in the U.S. Arctic (DOE 1997). ^{241}Am was present in sediment at detectable levels at 8 of the 13 sites sampled, ranging from 0.06 to 0.12 Bq/kg (1.6 to 3.2 pCi/kg). From the average ratio of ^{241}Am:$^{239+240}$Pu, it was concluded that the americium originated from global fallout. In 1976, sediment at several sites were sampled in Lakes Erie and Ontario. ^{241}Am was detected at only one site in Lake Erie, a site influenced by the Nuclear Fuels Reprocessing plant at West Valley, New York (U.S. Energy Research and Development Administration 1977). Levels of ^{241}Am increased with depth with concentrations of 3.4, 7.9, 11.1, and 11.8 Bq/kg (92, 210, 300, and 319 pCi/kg) dry weight at 0–2, 2–4, 4–6, and 6–8 cm, respectively.

6.4.4 Other Environmental Media

Concentrations of ^{241}Am have been determined in various foods (Table 6-4) and biota (Table 6-5), some of which may be eaten by humans. Levels in food items and animals include those from diet studies as well as from areas contaminated by nuclear discharges and nuclear waste. In a studies at DOE facilities and in the vicinity of the BNFL reprocessing plant at Sellafield and the Ravenglass Estuary, where pastures are subject to tidal inundations with sea water receiving discharges from the plant, results clearly show the effect on animals from feeding on contaminated pasture land (Arthur and Janke 1986; Bradford and Curtis 1984; DOE 1979c; Janke and Arthur 1985).

As part of the U.S. Mussel Watch program, monthly samples of mussels were collected from Bodega Head, California and Narragansett Bay, Rhode Island in 1977 and 1978 (Goldberg 1986). The mean concentration of ^{241}Am in samples ranged from 4.2 to 11.6 Bq/kg dry weight at Bodega Head and from 0.2 to 0.7 Bq/kg dry weight at Narragansett Bay. No seasonal pattern was evident. The ratio of ^{241}Am to $^{239+240}$Pu ranged from 1.3 to 5.3 on the west coast site and from 0.2 to 1.0 on the east coast. A surveillance program of U.S. coastal waters involving somewhat over 100 sites was another component of the Mussel Watch program. Both oysters and mussels were collected from the three major coastlines of the United States in 1976, 1977, and 1978 and the flesh was analyzed (EPA 1984). The results are included in Table 6-5. The levels of ^{241}Am were higher on the west coast which was ascribed to seasonal upwelling

Table 6-4. Average Concentrations of ^{241}Am in Food

Local Food	Concentration[a] mBq/kg	Concentration[a] fCi/kg	Reference
Bikini Atoll[b], U.S. Nuclear Test Site from 1946 to 1958, including BRAVO explosion on 3/1/54			Robison et al. 1997b
Reef fish	6.5	180	
Tuna	1.3	35	
Mahi mahi	1.3	35	
Marine crabs	26	700	
Lobster	26	700	
Clams	460	12,000	
Coconut crabs	28	760	
Land crabs	28	760	
Octopus	6.5	180	
Turtle	6.5	180	
Chicken, muscle	6.0	160	
Pork, muscle	6.0	160	
Eggs, chicken	6.0	160	
Pandanus fruit (n=69)	3.8	100	
Breadfruit (n=41)	1.2	32	
Coconut juice, drinking (n=747)	8.5	230	
Coconut meat, drinking (n=812)	3.6	97	

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Table 6-4. Average Concentrations of ^{241}Am in Food (*continued*)

Local Food	Concentration[a] mBq/kg	Concentration[a] fCi/kg	Reference
Copra meat (n=188)	1.1	30	
Papaya (n=93)	0.36	9.7	
Squash (n=53)	3.0	81	
Pumpkin	3.0	81	
Banana (n=39)	0.36	9.7	
Citrus	0.36	9.7	
Rain water	0.0037	0.10	
Well water	0.044	1.2	
Northern Marshall Islands Radiological Survey (NMIRS)[f], fallout from U.S. atmospheric weapons testing from 1946 to 1958, including BRAVO explosion on 3/1/54			Robison et al. 1997a
Reef fish, muscle; 11 atolls/islands	<0.23–1.4	<6.2–38	
Pelagic fish, muscle; 9 atolls/islands	<0.14–0.27	<3.8–7.3	
Clams, soft parts; 10 atolls/islands	<1.4–46	<38–1,200	
Pork; 6 atolls/islands	<0.11–2.8	<3.0–76	
Chicken; 4 atolls/islands	1.2–4.1	32–110	
Coconut crab; 2 atolls/islands	4.3–28	120–760	
Drinking coconut meat; 5 atolls/islands	<1.3	<35	
Copra meat; 10 atolls/islands	<0.42–7.4	<11–200	

Table 6-4. Average Concentrations of ^{241}Am in Food (*continued*)

Local Food	Concentration[a] mBq/kg	Concentration[a] fCi/kg	Reference
Pandanus; 12 atolls/islands	<0.23–8.5	<6.2–230	
Breadfruit; 7 atolls/islands	0.26–8.2	7.0–220	
Total Diet Study – New York, 1974[d]			Bennett 1979
Shellfish	0.48±0.15	13±4	
Whole grain products	0.074±0.044	2.0±1.2	
Bakery products	0.063±0.02	1.7±0.6	
Fresh vegetables	0.048±0.015	1.3±0.4	
Fresh fruit	0.041±0.015	1.1±0.4	
Root vegetables	0.030±0.007	0.80±0.19	
Dry beans	0.022±0.012	0.60±0.34	
Poultry	0.019±0.011	0.52±0.31	
Potatoes (peeled)	0.013±0.002	0.36±0.06	
Flour	0.013±0.007	0.36±0.18	
Fruit juice	0.011±0.006	0.29±0.16	
Meat	0.010±0.006	0.28±0.17	
Rice	0.010±0.012	0.27±0.34	
Milk	0.0093±0.0004	0.25±0.01	
Fresh fish	0.0089±0.0030	0.24±0.08	

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Table 6-4. Average Concentrations of ^{241}Am in Food (*continued*)

Local Food	Concentration[a] mBq/kg	Concentration[a] fCi/kg	Reference
Eggs	0.0059±0.002	0.16±0.06	
Macaroni	0.0059±0.009	0.16±0.25	
Canned fruit	0.0026±0.0033	0.07±0.09	
Near Sellafield, England, 1994			Fulker et al. 1998
Honey	.0007	0.02	
Blackberry	.043	1.2	
Pheasant	.029	0.78	
Mushrooms	.0065	0.18	

[a]Concentrations on a wet or fresh weight basis. Concentrations originally expressed in mBq/kg unless otherwise indicated.
[b]Terrestrial food is from a continuous monitoring program in 1975–1993; marine food is from a 1988 report.
[c]Decay correction to 1996
[d]Concentrations originally expressed as pCi/kg

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Table 6-5. Concentrations of ^{241}Am in Fauna

Species	Sample	Location	Concentration	Units	Type	Reference
Fish						
Farallon Islands Nuclear Waste Disposal Site (FINWDS) and Point Arena (reference site), 1986–1987						Suchanek et al. 1996
Dover sole	Flesh	FINWDS (n=3) Point Arena (n=6)	1,630±2,360 126±88	mBq/kg w.w.	Mean±SD	
Sablefish	Flesh	FINWDS (n=1) Point Arena (n=5)	2,850 1,000±1,400	mBq/kg w.w.	Mean±SD	
Thornyheads	Flesh	FINWDS (n=2) Point Arena (n=3)	186±35 299±157	mBq/kg w.w.	Mean±SD	
Vicinity of Los Alamos National Laboratory, 1997, Bottom-feeding fish (n=10/site)						Fresquez et al. 1999
Fish (two sites)	Muscle+bone	Reservoirs	1.7 (19.8)	10^{-5} pCi/g d.w.	Mean (SD)	
Fish (four sites)	Muscle+bone	Rio Grande	84.1 (54.6)	10^{-5} pCi/g d.w.	Mean (SD)	
Fish (five sites)	Viscera	Rio Grande	31.2–198.0	10^{-5} pCi/g d.w.	Range of means	
Fish from Irish ports possibly impacted by Sellafield, 1988–1997						Ryan et al. 1999
Plaice	Edible portion		0.4–144	mBq/kg w.w.	Range of means	
Whiting	Edible portion		0.42–2.8	mBq/kg w.w.	Range of means	
Cod	Edible portion		0.62–26	mBq/kg w.w.	Range of means	
Crustacea						
Fish ports possibly impacted by Sellafield, 1988–1997						Ryan et al. 1999
Prawns	Edible portion		12–138	mBq/kg w.w.	Range of means	

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Table 6-5. Concentrations of ^{241}Am in Fauna (*continued*)

Species	Sample	Location	Concentration	Units	Type	Reference
Bivalves						
Farallon Islands Nuclear Waste Dump Site (FINWDS) and Point Arena (reference site), 1986–1987						Suchanek et al. 1996
Mussels	Flesh	FINWDS (n=1)	6	mBq/kg w.w.	Mean	
Mussels	Flesh	Point Arena (n=2)	126±88	Bq/kg d.w.	Mean±SD	
U.S. Mussels Watch, 1976–1978 (East Coast: Maine-Virginia; East/Gulf Coast: Virginia-Texas; West Coast: Washington-California)						EPA 1984
Mussels and oysters	Flesh	East coast	0.43–0.65 (0.03–1.7)	Bq/kg d.w.	Range of means (range)	
Mussels and oysters	Flesh	East/Gulf coasts	0.32–0.60 (0.03–1.95)	Bq/kg d.w.	Range of means (range)	
Mussels and oysters	Flesh	West coast	0.49 (0.14–18.5)	Bq/kg d.w.	Range of means (range)	
Carlinford, Ireland (port city), impact from Sellafield Reprocessing Plant, 1991–1997						Ryan et al. 1999
Mussels	Edible portion		<11–109 (69)	mBq/kg w.w.	Range of means (median)	
Oysters	Edible portion		14–27	mBq/kg w.w.	Range of means	
Irish Sea, site impacted by Windscale Reprocessing Plant and reference sites, 1977						EPA 1984
Mussels	Flesh	Isle of Man	550 (55)	Bq/kg d.w.	Mean (SD)	
Mussels	Flesh	Ravenglass estuary	63.9×10^3 (6,390)	Bq/kg d.w.	Mean (SD)	
Mussels (two sites)	Flesh	Ebro estuary, Spain	0.52–0.88	Bq/kg d.w.	Range of means	

Table 6-5. Concentrations of ^{241}Am in Fauna (*continued*)

Species	Sample	Location	Concentration	Units	Type	Reference
Sea Mammals						
North Rona and Isle of May, Scotland - Possible impact from Sellafield and Chernobyl, 1987						Anderson et al. 1990
Grey seals (n=8)	Milk		<0.2–<0.3	Bq/L	Range	
Grey seals (n=2)	Liver		<0.3	Bq/kg f.w.	Range	
Grey seals (n=3)	Muscle		<0.3	Bq/kg f.w.	Range	
Mammals						
Halla, Finland, reindeer graze on lichen impacted by Chernobyl fallout						Paatero and Jaakkola 1998
Reindeer	Meat		0.014 (0.003)	Bq/kg d.w.	Mean (SD)	
Reindeer	Liver		0.013 (0.004)	Bq/kg d.w.	Mean (SD)	
Reindeer	Kidney		0.0043 (0.0010)	Bq/kg d.w.	Mean (SD)	
Nevada Test Site: Area 13 (outer compound) and near Area 13, but outside the area, 1973–1976						DOE 1979
Cattle (11 animals)	Muscle	Area 13	0.020–0.17	pCi/kg w.w.	Range	
Cattle (4 animals)	Muscle	Near Areas 13	<0.05–<0.11	pCi/kg w.w.	Range	
Fox (adult)	Muscle	Area 13	25.0	pCi/kg w.w.		
Fox (adult)	Muscle	Area 13	17.0	pCi/kg w.w.		
Coyote	Muscle	Area 13	4.7	pCi/kg w.w.		
Vicinity of Sellafield and the Ravenglass Estuary, England, 1977–1982						Bradford and Curtis 1984
Cattle	Liver	Sellafield (n=1)	0.23	Bq/kg f.w.		
		Ravenglass (n=3)	0.037–0.48		Range	
		Control (n=2)	0.003, 0.005			

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Table 6-5. Concentrations of ^{241}Am in Fauna (*continued*)

Species	Sample	Location	Concentration	Units	Type	Reference
Cattle	Meat	Ravenglass (n=2) Control (n=1)	0.002, 0.012 0.008	Bq/kg f.w.		
Cattle	Lung	Ravenglass (n=3) Control (n=2)	0.05–0.085 0.003, 0.002	Bq/kg f.w.	Range	
Cattle	Bone	Ravenglass (n=3) Control (n=2)	0.25–0.29 0.013, 0.018	Bq/kg f.w.	Range	
Sheep	Meat	Ravenglass (n=3) Control (n=1)	0.040–0.064 0.0038	Bq/kg f.w.	Range	
Sheep	Liver	Ravenglass (n=3) Control (n=2)	2.6–6.6 0.006–0.0012	Bq/kg f.w.	Range	
Sheep	Lung	Ravenglass (n=3) Control (n=1)	0.018–0.24 0.017	Bq/kg f.w.	Range	
Sheep	Bone	Ravenglass (n=3) Control (n=1)	0.79–1.7 0.006	Bq/kg f.w.	Range	
Idaho National Engineering Laboratory - Subsurface Disposal Area (SDA) and control area, 1978–1979						Arthur and Janke 1986; Janke and Arthur 1985
Coyote	Feces	SDA (n=24) Control (n=12)	0.4±0.9 0.003±0.002	pCi/kg w.w.	Mean±SD	
Cottontail rabbit	Carcass	SDA (n=10) Control (n=5)	0.010±0.009 0.001±0.001	pCi/kg w.w.	Mean±SD	
Cottontail rabbit	Hide	SDA (n=10) Control (n=5)	0.029±0.046 0.002±0.001	pCi/kg w.w.	Mean±SD	
Deer mice	Carcass	SDA (n=21) Control (n=5)	0.010±0.02 0.003±0.002	pCi/kg w.w.	Mean±SD	

Table 6-5. Concentrations of ^{241}Am in Fauna (*continued*)

Species	Sample	Location	Concentration	Units	Type	Reference
Deer mice	Hide	SDA (n=21) Control (n=5)	0.27±0.83 0.039±0.050	pCi/kg w.w.	Mean±SD	
Invertebrates						
Idaho National Engineering Laboratory, Subsurface Disposal Area (SDA) and control area, 1978–1979						Arthur and Janke 1986
Invertebrates	Composite	SDA (n=3) Control (n=1)	0.022±0.020 <0.004	pCi/kg w.w.	Mean±SD	

d.w. = dry weight; FINWDS = Farallon Islands Nuclear Waste Dump Site; f.w. = fat weight basis; n = number of samples; SD = standard deviation; SDA = subsurface disposal area; w.w. = wet weight

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of the ^{241}Pu and ^{241}Am-rich mid-depth north Pacific Ocean waters (affected by nuclear weapons test fallout), rather than from releases from the low level nuclear waste site off the Farallon Islands. Another factor is the association of ^{241}Am with fine particles and the greater exposure of west coast bivalves to open ocean fine particles than east coast bivalves. Paradoxically, americium does not show the same seasonal fluctuations associated with upwelling as does plutonium and this may be due to americium's greater adsorptivity, and associated removal from the water column, or its slower uptake in the bivalves. The NOAA conducted a study in the Beaufort Sea in northwestern Alaska during the summer of 1993 to ascertain the levels of radionuclide activity in the U.S. Arctic (DOE 1997a). As part of this study, ^{241}Am was determined in ashed samples of a variety of biota, including fish, invertebrates, crustaceans, whales, seal, and ducks, most of which was collected in the coastal waters off Barrows, Alaska and analyzed by high-resolution gamma spectroscopy. ^{241}Am was not detectable in any of the samples (detection limits #0.6–#80 mBq/kg [<20–<2,000 fCi/kg]).

Fish and mussels were sampled near the Farallon Islands Nuclear Waste Dump Site (FINWDS), approximately 30 miles west of San Francisco and a reference site (see Table 6-5) (Suchanek et al. 1996). While many of the drums have been breached or are nearing their expected lifetime, no statistical difference was found in the levels of ^{241}Am in the FINWDS fish and those from the reference site. A simple explanation is the fact that the fish species tested are quite mobile. However, ^{241}Am levels found in fish samples, 1.0 and 1.5 Bq/kg (27 and 41 pCi/kg), were higher than comparable fish muscle reported from relatively contaminated sites such as the Pacific Testing Grounds in Micronesia and in the Irish Sea near the Windscale Nuclear Plant at Sellafield, none of which exceeded 0.74 Bq/kg (20 pCi/kg). As part of the Environmental Surveillance Program (at LANL), bottom-feeding fish were collected from reservoirs upstream and downstream from LANL in September 1997, as well as from five sites on the Rio Grande River starting at one site about 1 mile upstream of any stream crossing LANL land and analyzed for ^{241}Am and other radionuclides (Fresquez et al. 1999). The concentration of ^{241}Am in muscle + bone in fish collected in the reservoirs was significantly lower than that collected in the river (see Table 6-5). Mussel samples collected from two sites in the Irish Sea where the Windscale nuclear reprocessing plant is located show the influence of low levels of radionuclides from the nuclear fuel cycle (Table 6-5). The ^{241}Am found in the mussels were orders of magnitude higher than those on the U.S. coast. The Isle of Mann site is in the middle of the Irish Sea about 50 km from Windscale and the Ravenglass estuary site is 16 km to the south of the plant. The ^{241}Am input into the Irish Sea from fallout is dwarfed by the contribution by the Sellafield reprocessing facility in England. Observations from surveys of ^{241}Am in edible parts of fish and shellfish sampled from commercial landings at major Irish shipping ports between 1988 and 1997 (Table 6-5) indicate that activity levels in fish landed at northeast Irish ports have

remained low, generally a fraction of a mBq/kg wet weight, while those in mussel, prawns, and oysters are much higher and have fluctuated in activity (up to 180 mBq/kg wet weight in prawns) over time (Ryan et al. 1999).

Fifteen cattle grazing for 1,064 days on a site at the Nellis Bombing and Gunnery Range, Nevada, where 16 years previously a nuclear device was explosively destroyed, had ^{241}Am levels ranging from 0.74 to 590 mBq/kg (0.02 to 16 pCi/kg) wet weight, 4.4 mBq/kg (0.10 pCi/kg) geometric mean in muscle (Gilbert et al. 1989). Levels in liver and kidney were much higher, 72 and 11 mBq/kg (1.9 and 0.30 pCi/kg), geometric mean, respectively.

In 1993, the MAFF, United Kingdom systematically surveyed the concentrations of ^{241}Am in grass samples near 18 nuclear sites in England and Wales (Sanchez et al. 1996). ^{241}Am levels in vegetation samples were <0.10 Bq/kg (<2.7 pCi/kg). Sanchez et al. (1998) measured the ^{241}Am activity in vegetation from tide-washed pastures in 17 estuaries spanning the eastern seaboard of the Irish Sea from Solway in northwest England to David's Head in southern Wales. In 11 of the pastures, the median level of ^{241}Am in vegetation ranged from 14 to 575 Bq/kg (0.38 to 15.5 nCi/kg) dry weight; the others were <1 Bq/kg (<30 pCi/kg) dry weight. Individual samples in the 11 pastures ranged from 13 to 1,260 Bq/kg (0.35–34 nCi/kg) dry weight.

Sludge from Stickney, Illinois that was used as an organic fertilizer contained 3.3 and 1.5 fCi/g (14 and 575 Bq/kg) dry weight of ^{241}Am in 1974 and 1975, respectively. The source of the americium was thought to be runoff from the metropolitan area. In Texas, ^{241}Am was present in sludge samples during 1983–1986 from a facility that received sanitary waste from the Gulf Nuclear, Inc. facility. Two incidents of contaminated sludge from companies producing foils for smoke detectors occurred in New York State (NRC 1979). One of the receiving facilities that incinerated its sludge had 180–750 pCi/g (6.7–28 Bq/g) of ^{241}Am in dried incinerator ash between April 17, and May 7, 1984, after which it dropped to 13 pCi/g (0.48 Bq/g), corresponding to a sludge concentration of 5.2 pCi/g (0.19 Bq/g). During the latter half of 1984 and 1985, the ^{241}Am concentrations in sludge were 3.5 and 2.5 pCi/g (130 and 93 mBq/g), respectively. In the second incident, ^{241}Am in sludge was as high as 120 pCi/g (4,400 mBq/g) in 1984, but dropped to an average of 24.8 pCi/g (920 mBq/g) in the latter half of 1986. This facility landfilled its sludge.

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Americium in the environment is transferred to man by inhalation and ingestion of food and, in the case of children, ingestion of soil. Much of the ^{241}Am so taken is found in the skeleton and liver. In addition to the ^{241}Am taken up, additional ^{241}Am is produced by the decay of ^{241}Pu that has been inhaled or ingested and absorbed. No data on the body burden of ^{241}Am in the general population was found. However, the body burden from fallout was estimated from the concentration of ^{241}Am in air and estimates of intake by ingestion. The modeling study indicated that most of the ^{241}Am body burden, 89% in 1978, was due to ingrowth from ^{241}Pu. The model also estimated that the contribution of ingestion to the ^{241}Am body burden was <1% of that due to inhalation. The total skeletal burden for 1970–1974 in New York was estimated from ashed skeletal samples to be 0.27 pCi (Bennett 1979).

Ash from a 1974 ^{90}Sr total diet study in New York consisting of representational foods from 19 separate categories was analyzed for ^{241}Am to ascertain the dietary intake of the radionuclide from fallout (Bennett 1979). It was estimated that over 95% of the ^{241}Am transfer to food was from cumulative deposits in soil, rather than direct deposition. The highest concentrations of ^{241}Am in food occurred in shellfish followed by grain products and fresh fruits and vegetables. The lowest concentrations were in meat, milk, eggs, fish, and processed foods. The same patterns of concentrations were obtained for ^{239,240}Pu. The activity levels of ^{241}Am in foods is shown in Table 6-4. Using dietary consumption estimates and the activity levels in the food items and tap water, the ^{241}Am ingestion intake for a New Yorker was 0.40±0.05 pCi/year (15±2 mBq/year) in 1974. The concentration of ^{241}Am in tap water was estimated at 30±3 aCi/kg (1±0.1 μBq/kg) with 1,200 kg of water being consumed in a year. The food samples obtained in 1974 occurred at a time when direct deposition from fallout was very low and the concentration of ^{241}Am in food was largely a result of uptake from soil derived from cumulative deposits. Direct deposition from fallout was greatest in 1963–1964. Additionally, direct deposition is a much less significant means of plants acquiring activity than root uptake. The ^{241}Am intake in diet from fallout will continue to increase due to ingrowth from ^{241}Pu until the cumulative deposit reaches its projected maximum of 0.88 mCi/km^2 (33 MBq/km^2) in the year 2035, at which time, the dietary intake of ^{241}Am will have reached a maximum of 0.7 pCi/year (26 mBq/year) .

As part of its Radionuclides in Food Program, the FDA determined concentrations of radionuclides in their Total Diet Study, as well as food originating from the vicinity of nuclear reactors including raw vegetables, food crops (primarily fruits), fish, and milk. While not specifically analyzed for ^{241}Am, concentrations of radionuclides in both of these surveys in 1983–1986 and 1987–1992 were sufficiently

low as to require no specific action or simple monitoring. Imported foods were extensively monitored starting in May 1986 for contamination resulting from the Chernobyl accident. While ^{241}Am was identified in samples of imported food that probably originated from Chernobyl, the vast majority of samples were below FDA's level of concern with only one oregano and three cheese shipments being detained (Cunningham et al. 1989, 1994).

A study in the Ravenglass estuary where tidal action inundates pasture land with water that receives discharges from the BNF reprocessing plan in Sellafield, it was concluded that consumption of a diet consisting of locally produced food would result in dose levels far below recommended limits (Bradford and Curtis 1984). Sanchez et al. (1998) measured the ^{241}Am activity in vegetation from tide washed pastures in 17 estuaries spanning the eastern seaboard of the Irish Sea from Solway in northwest England to David's Head in southern Wales. From the mean and median ^{241}Am activity in grass and appropriately reduced transfer factors, they predicted the activity in animal products, namely milk, beef, beef liver, lamb, and lamb liver. From this, they estimated the committed effective dose for a 1-year-old, 10-year-old, and adult from eating these foods. The internal dose from ingesting a representative vegetable, potatoes, was estimated assuming that the land above the high water mark could be reclaimed for growing vegetables. The results are shown in Table 6-6.

Using transfer factors derived for uptake into plants and animals, Friberg and Vesanen (1999) have listed critical activity levels for americium and other nuclides deposited on soil and pasture vegetation from a nuclear accident required to exceed the action level for foodstuffs recommended by the IAEA. Experiments have been conducted to assess uptake of ^{241}Am and other radionuclides from fallout or nuclear facilities in plants.

The consumption of fish exposed to actinide contamination is a potential source of ^{241}Am exposure. This potential source of exposure has been evaluated by measuring the concentration of ^{241}Am in bass and bluegill living in a nuclear waste pond on the Hanford site where the levels of ^{241}Am in sediments are approximately 3 orders of magnitude above background levels. Assuming that actinide behavior in the body is similar to that defined by the International Commission on Radiation Protection (ICRP) for Standard Man, the maximum levels occurring in fish fillets after 1 year in the waste pond (see Table 6-5) would not be sufficient to produce a significant radiation dose to a human eating 1 pound of fish fillets every day for 70 years (Emery et al. 1981). Similarly, the consumption of fish from the Farallon Islands Nuclear Waste Dump Site off of San Francisco is expected to add ~1.5% to the typical annual dose to the

Table 6-6. Estimated Effective ^{241}Am Dose from Food Raised on Land Impacted by Tidal Estuaries along the Irish Sea

Food item	Transfer factors		^{241}Am concentration[a]		Committed effective dose (μSv) from ^{241}Am[b]		
	Mean	Median	Mean	Median	Adult	10-year old	1-year old
Milk	1.0×10^{-7}	1.0×10^{-7}	6.3×10^{-4}	2.5×10^{-4}	0.01	0.02	0.03
Beef	1.0×10^{-5}	1.0×10^{-5}	0.06	0.02	0.2	0.2	0.07
Beef liver	2.0×10^{-3}	2.0×10^{-3}	12.61	4.94	14.6	8.3	4.9
Lamb	4.0×10^{-5}	4.0×10^{-5}	0.03	0.01	0.05	0.03	0.01
Lamb Liver	3.0×10^{-3}	3.0×10^{-3}	2.18	0.86	2.5	1.4	0.8
Potatoes					3.3	3.1	1.2

Source: Sanchez et al. 1998.

[a]Food concentrations were estimated from measured ^{241}Am levels in grass (485 Bq/kg dry weight mean, 190 Bq/kg dry weight median), by applying grass-to-food transfer factors. Units are Bq/L for milk and Bq/kg fresh weight for other food items.
[b]The average consumption rates for the animal products were obtained from Byrom et al. (1995) and the dose coefficients were taken from SEPA (2000).

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whole body effective dose equivalent value to pooled radionuclide concentrations committed to age 70 (Suchanek et al. 1996). This increase is derived largely from the ingestion of ^{241}Am.

Lichens are very efficient collectors of deposited radionuclides and since caribou and reindeer depend on lichen for food during the winter season, the lichen-reindeer/caribou-man pathway can lead to elevated exposures to indigenous Inuit and Sami populations who eat large quantities of meat and organs from caribou and reindeer. ^{241}Am has been deposited in arctic regions as a component of global fallout from atmospheric nuclear weapons testing primarily during the 1950s and early 1960s (China from global atmospheric nuclear weapons testing as well as fallout from the Chernobyl accident in April 1986. Paatero and Jaakkola (1998) established that americium was transferred from lichen to reindeer via the gastrointestinal tract and not inhalation, and obtained a gastrointestinal-tract absorption coefficient of 0.00075 for ^{241}Am. This gastrointestinal-tract absorption coefficient is similar to that estimated for humans (0.0006—0.0008, See Section 3.3.1.2). Concentrations of ^{241}Am in reindeer resulting from Chernobyl fallout appear in Table 6-5. Global fallout of plutonium, which correlates with that of americium, was considerably higher than Chernobyl fallout, ca. 70 Bq/m^2 (2 pCi/m^2) compared to 1.5 Bq/m^2 (0.04 pCi/m^2) (Paatero and Jaakkola 1998). Based on ^{241}Am concentrations in reindeer in Finnish Lapland, estimated ^{241}Am intake is 2 pCi/year (74 mBq/year) for men and 1 pCi/year (37 mBq/year) for women (Jaakkola et al. 1977).

A modeling study for estimating exposure to man from dumping of radioactive waste in the Arctic Seas concluded that ingestion of molluscs was the dominant pathway for ^{241}Am, and that ^{241}Am along with ^{239}Pu would dominate the collective dose at longer time scales (Nielsen et al. 1997). Another modeling study dealing with the effect of long-term release of ^{241}Am into an estuarine coastal sea ecosystem found that the greatest intake by man is from eating marine food items such as seaweed, molluscs, crustaceans, and fish (Murray and Avogadro 1979). Another important route of exposure is the inhalation of contaminated airborne coastal sediment. Ingestion of sediment by children and the contamination of crops by airborne sediment was estimated to be of minor importance. Radiation from contaminated mud flats and direct exposure from water were considered to be of little consequence. A survey of ^{241}Am and plutonium in fish and shellfish in Ireland between 1988 and 1997 estimated the mean doses from ^{241}Am for the typical and heavy Irish consumer were 0.024 and 0.104 µSv/year (2.4 and 10.4 µrem/year), respectively (Ryan et al. 1999). Americium contributes somewhat over a quarter of the combined plutonium-americium dose. In 1988, this combined dose was roughly 10% of that which the typical Irish seafood consumer obtained from ^{137}Cs; in 1997, this percentage had increased to 48%.

Both the general population and workers may be exposed to ^{241}Am from the production, distribution, and use of ionization chamber smoke detectors. The NRC investigated exposure relating to this device. Their 1979 report concluded that the sum of doses to the population resulting from production, distribution, use, and disposal of 14 million smoke detectors containing 3 μCi (100 kBq) of ^{241}Am each (over 3 times that presently used) that were distributed in 1978 would result in a collective total body dose of 1,100 person-rem (11 person-Sv)—much lower than that which could potentially result in one cancer death (NRC 1979). Exposure to ^{241}Am could result from improper disposal of smoke detectors. The detector could be broken and then disposed of in a municipal landfill or incinerated. The ^{241}Am from the broken detector could be consumed by a child.

Occupational exposure to ^{241}Am may occur to workers manufacturing the ^{241}Am-containing foil used in ionization-type smoke detectors or assembling the device. Two manufacturers of the ^{241}Am foils were surveyed in 1977 (NRC 1979). One reported a cumulative external dose of 0.8 person-rem per curie (0.2 person-pSv/Bq) of ^{241}Am processed; 150 workers were involved and 16.6 curies of ^{241}Am processed. The maximum dose for an individual was approximately 800 mrem. The second manufacturer reported 0.2 person-rem per curie (0.05 person-pSv/Bq) of ^{241}Am processes. It should be noted that manufacturers of ionization chamber smoke detectors must be licensed by the NRC and certain work practices are required to minimize exposure. Workers wear shoe covers, lab coats, and gloves and wipe tests are performed weekly. During the manufacture of the smoke detector units, one manufacturer reported the yearly external doses among badged workers in 1978: 40% had no measurable dose, 40% had <100 mrem (<1 mSv), 15% had doses between 250 and 500 mrem (2.5 and 5.0 mSv), 4% had doses between 500 and 750 mrem (5.0 and 7.5 mSv), and one individual had a dose between 750 and 1,000 mrem (7.5 and 10 mSv). The total occupational dose in the ionization chamber smoke detector industry in 1978 was estimated to range from 15 to 48 person-rem. The external doses to people involved in the distribution and retail sale of smoke detectors would be much less. The hand dose to installers is estimated to be 0.6 μrem (6 nSv) per unit installed. An individual having one detector in the hallway and one in the bedroom would have an annual dose of 9.3 μrem (93 nSv).

Workers at plutonium reprocessing facilities, nuclear reactors, transuranium and low level waste storage facilities, or those engaged in the production or processing of ^{243}Am or ^{241}Am may be occupationally exposed to americium. In addition, workers at sites where nuclear testing was conducted may also be exposed to americium. Workers in nuclear power stations may be exposed to airborne radionuclides. The NRC publishes annual reports on occupational radiation exposure at NRC licensed facilities. Monthly cumulative 24-hour air filter samples were obtained from five different working areas of a BWR in India

during normal operation and during refueling. The average air concentrations during normal operation were (work area, concentration in mBq/m^3 (fCi/m^3)): reactor building—103 feet elevation, 0.95 (26); reactor building—143 feet elevation, 0.22 (5.9); reactor building—200 feet elevation, 0.25 (6.8); turbine building—133 feet elevation, 0.07 (1.9); and radwaste building—2.50 (68) (Hedge and Bhat 1983). During refueling, the respective concentrations were: 1.64, 1.33, 0.78, 0.19, and 6.34 mBq/m^3 (44.3, 35.9, 21, 5.1, and 171 fCi/m^3). The derived air concentration recommended by the ICRP for occupational exposure is 80.0 mBq/m^3 (2,200 fCi/m^3). In 1997, the French radiation protection office conducted monitoring (24-hour urine analysis/whole body activity measurements) of workers in the non-nuclear energy field (i.e., nuclear medicine, research laboratories, and non-nuclear industries) to ascertain the occupational intake of radionuclides (De Vathaire et al. 1998). ^{241}Am was not detected in samples from any of the 37 workers who worked with the isotope.

The U.S. Transuranium and Uranium Registries (USTUR) includes The National Human Radiobiology Tissue Repository that contains post-mortem tissue from persons (primarily workers) with documented intakes of uranium or transuranic elements, such as americium. USTUR publishes annual reports detailing cases of accidental exposure and tissue levels of the different radioactive isotopes. These reports and other information is available over the internet at http://www.tricity.wsu.edu/ustur/. Exposure is generally by inhalation, but entry into the body may also occur through wounds. Americium is largely retained in the skeleton and liver, but after long term exposure, significant amounts of ^{241}Am are found in the muscle, respiratory tract, and other soft tissue (Filipy et al. 1994; McInroy et al. 1989). In 25 USTUR cases involving the registry of individuals that typically had $20 years of employment in facilities where ^{241}Am was processed and who were likely to have had chronic low-level inhalation exposure to the actinide, the ^{241}Am organ content of the liver and skeleton ranged from 0.01 to 554 Bq (0.26 Bq, median) (0.3 to 15,000 pCi, 7.0 pCi, median) and 0.04 to 6,623 Bq (0.99 Bq, median) (1–179,000 pCi, 27 pCi, median), respectively (Kathren et al. 1988). Seventy-five percent of the combined activity in the liver and skeleton resided in the skeleton (see Section 3.4).

6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in 3.6 Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a

larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

Specific information on the exposure of children to americium is not available. As for adults in the general population, small exposures to ^{241}Am occur from normal ingestion of food and drinking water and inhaling air. While the potential for americium exposure is greater for children who consume foods grown in areas with elevated levels of americium in the soil, the uptake of americium into plants and animals is very low. Children may be exposed to a substance brought into the home on parents' work clothes, skin, hair, tools, or other objects from the workplace; however, with one exception, no instances of americium exposure in children by this route was found in the literature. In this case, a man who did some experiments using an americium source brought home a secondary source and stored it in a dresser drawer (Kelly and Dagle 1974). No information was located on americium levels in amniotic fluid, meconium, cord blood, neonatal blood, or breast milk, or other potential measurements relating to fetal or childhood body burdens.

^{241}Am may also find its way into municipal landfills because of improper disposal of smoke detectors or rubble of buildings containing smoke detectors. Smoke detectors may be broken, exposing the ^{241}Am ionization source. Young children might get access to the ^{241}Am and ingest it. At hazardous waste sites, americium that is found in excess of fallout background levels is most likely to be in soil and presents a special hazard for young children. Hand-to-mouth activity and inadvertent or intentional (pica) eating of contaminated dirt will result in oral exposure to americium.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Occupational exposures and general population exposures to potentially high levels of ^{241}Am and other radionuclides might occur to people occupying land like the southern Solway and Dee marshes, areas tidally inundated by water contaminated by the BNFL nuclear fuel reprocessing plant at Sellafield, United Kingdom plant in England (Rose et al. 1996). Modeling studies of human populations exposed to ^{241}Am in such coastal environments show that the highest levels of ^{241}Am would occur in sediment and marine organisms, especially molluscs and seaweed (Murray and Avogadro 1979). These human populations

include farm workers and turf cutters and recreational users such as birdwatchers, hikers, fishermen, and bird shooters, as well as those who consume contaminated marsh produce, molluscs, and seaweed. Exposure routes would be ingestion and inhalation of dust. Even so, the dose rates that would be received by these pathways are very low (Murray and Avogadro 1979).

Populations with potentially high exposure to ^{241}Am from commercial devices such as ionization chamber smoke detectors might include production workers and firemen attending to fires in production facilities, warehouses where a large number of sources or devices are housed, or homes where a small number of the devices might be used.

6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of americium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of americium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.8.1 Identification of Data Needs

Physical, Chemical, and Radiological Properties. Data on the physical and chemical properties of americium and americium compounds are available in the literature (Cotton and Wilkinson 1980; Lide 1994, 1998; Nenot and Stather 1980; Seaborg 1991). Data are also available on the radiological properties of americium isotopes (ICRP 1983; Lide 1998).

Production, Import/Export, Use, Release, and Disposal. Data regarding the past and present production and import/export volumes for ^{241}Am and ^{243}Am are not available. The limited commercial use of ^{241}Am provides a good estimate of probable production volumes. The uses of americium are known (Budavari 1996; NRC 1979; Seaborg 1991; Uranium Information Center 1997). However, information on the production, import/export volumes, and the amounts of ^{241}Am used in various devices would be useful for estimating exposure. Estimates of the amounts of ^{241}Am released as a result of nuclear tests and accidents are available (Bennett 1979; Suchanek et al. 1996). The disposal of americium is governed by the NRC regulations (10 CFR 61). The Manifest Information Management System (MIMS), maintained by the INEEL, contains information on low-level radioactive waste shipments received at commercial low-level radioactive waste disposal facilities (INEEL 2000). The DOE stores most of its spent fuel at three primary locations: the Hanford site, Washington, the INEEL, Idaho, and the Savannah River Site, South Carolina. The DOE National Spent Fuel Program maintains a database of spent nuclear fuel stored in each of the three locations (DOE 1999). Information concerning the americium content of civilian nuclear power plants and naval nuclear propulsion plant spent fuel now in storage and awaiting disposition would be useful for estimating the amount of americium stored in the country.

Environmental Fate. The environmental fate of americium has been extensively studied in relation to its introduction into the Irish Sea from the BNFL nuclear fuel reprocessing plant at Sellafield, United Kingdom (Belot et al. 1982; Bennett 1976; Bunzl et al. 1994, 1995; Malcolm et al. 1990; McCartney et al. 1994; McKay et al. 1994a; Murray et al. 1978, 1979; Pattenden and McKay 1994; Walker et al. 1986).

Other studies have investigated transport in soil and at DOE waste disposal sites (Fowler et al. 1981; McCarthy et al. 1998a, 1998b; Penrose et al. 1990). As a result of these studies, the environmental fate of americium is reasonably well understood.

Bioavailability from Environmental Media. The absorption and distribution of americium as a result of inhalation and ingestion exposures have been discussed in Sections 3.3.1 and 3.3.2. EPA lists identical uptake factors for inhaled and ingested americium (and all the other transuranics other than plutonium) regardless of compound solubility, indicating that the knowledge base for americium is not sufficiently developed to quantify the differences that are recognized for most other elements.

Food Chain Bioaccumulation. Information about americium the levels of americium in aquatic and terrestrial organisms and its bioaccumulation in these organisms is available (Fresquez et al. 1999; DOE 1996; Suchanek et al. 1996). Data are also available on the uptake of americium in plants (Bennett 1979; Cataldo et al. 1980; EPA 1979; Romney et al. 1981; Schreckhise and Cline 1980; Schulz et al. 1976; Zach 1985) and levels in food (Bennett 1979; Cunningham et al. 1989, 1994; Robison et al. 1997a, 1997b). These data indicate that americium does not biomagnify in the food chain (Bennett 1979; Bulman 1978).

Exposure Levels in Environmental Media. Reliable monitoring data for the levels of americium in contaminated media at hazardous waste sites are needed so that the information obtained on levels of americium in the environment can be used in combination with the known body burden of americium to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Information is available on the levels of ^{241}Am in soil and sediment in areas affected only by global fallout, at DOE installations and other nuclear facilities, as well as sites of nuclear explosions and accidents (Alberts et al. 1989; Bennett 1979; Cooper et al. 1994; DOE 1980; Pattenden and McKay 1994; Robison et al. 1997a, 1997b; Sanchez et al. 1996). ^{241}Am levels in soil around nuclear power plants in the United States were indistinguishable from fallout background (EPRI 1981).

For FDA's Radionuclides in Food Program, the FDA has determined concentrations of radionuclide activity in their Total Diet Study. Levels of ^{241}Am in both the 1983–1986 and 1987–1992 studies were sufficiently low as to require no specific action or simple monitoring (Cunningham et al. 1989, 1994). The FDA (1998) has published derived intervention levels for ^{241}Am in food at which action should be considered. Dietary values are 2.0 Bq/kg for 3-month-old children, increasing to 17 Bq/kg for 1-year-old children, and then decreasing to 8.8 Bq/kg for adults. Since 1973, background fallout levels of ^{241}Am in air have been <1 aCi/m^3 (0.037 μBq/m^3) and are continuing to decline (Bennett 1979). Levels in tap water are sufficiently low that levels must be estimated (Bennett 1979).

Exposure Levels in Humans. Although some data on the levels of americium in human tissues exposed to natural background levels (food, water, and air) are available, few measurements have been made on the americium content in human tissues. The principal source of information about occupationally exposed individuals is the U.S. Transuranium and Uranium Registries (USTUR) Tissue Program and database, established to document levels and distribution of uranium and transuranium

isotopes in human tissues for occupationally exposed workers (USTUR 1999). Several major database files are available. The Radiochemical file contains information about radiochemical analysis of tissue donations from occupationally exposed individuals. The Health Physics file contains bioassay and other health physics data. These two databases are regularly updated. The Medical file contains abstracted personal, medical, and clinical data; the Pathology file contains autopsy and pathology information; and the Skeletal Estimate file contains estimated actinide concentrations for unanalyzed half skeletons from donors (USTUR 1999). The DOE has developed the Comprehensive Epidemiologic Data Resource (CEDR) Program to provide public access to health and exposure data concerning DOE installations. In addition, studies relating to populations residing near DOE installations, and other studies of radiation exposures and health effects, such as atomic bomb survivors, are included in CEDR (CEDR 2000).

Exposures of Children. Children will be exposed to americium in the same manner as adults in the general population (i.e., ingestion of food and water and inhalation of air). Americium is potentially found at hazardous waste sites at elevated levels. Since children may have oral exposure to soil through hand-to-mouth activity, bioavailability studies of americium in soil via the oral route may be useful to assess the risk of this type of exposure.

Child health data needs relating to susceptibility are discussed in 3.12.2 Identification of Data Needs: Children's Susceptibility.

Exposure Registries. No registries of people exposed to americium has been located.

6.8.2 Ongoing Studies

The Federal Research in Progress database lists ongoing studies about environmental effects of americium (FEDRIP 2000). These are shown in Table 6-7. The International Science and Technology Center (ISTC), headquartered in Moscow, which began operations on March 3, 1994, also is active in research concerning transuranic elements.

Table 6-7. Ongoing Studies on Environmental Effects of Americium

Investigator	Affiliation	Study	Sponsor
Beasley, TM	Oregon State University, College of Oceanography, Corvallis, Oregon	This project will investigate the cycling of transuranic radionuclides in the Columbia River estuary and the North Pacific Ocean and the biokinetic behavior of transuranic radionuclides in selected freshwater and marine organisms.	DOE
Cleveland, JM	Department of the Interior, U.S. Geological Survey	Determine the speciation (oxidation-state distribution, solubility, hydrolysis, and complex formation) of plutonium and other transuranium elements in groundwaters so as to more accurately predict the long-term mobilization and transport of these long-lived radionuclides from nuclear waste repositories.	DOI, U.S. Geological Survey
Heit, M	Environmental Measurements Laboratory, Environmental Studies Division	The fallout of nuclear debris from both atmospheric weapons tests and accidental atmospheric releases is measured to determine the mechanisms of transport and deposition and to verify and correct fallout models. These data are used to identify the global distribution radionuclides and estimate a continuous global budget and inventory. Soil samples are taken in selected geographical areas where the accumulated fallout deposit is uncertain to study the lateral and vertical distribution of radionuclides with the aim of correlating body burdens with intake amounts and to follow the long-term behavior of these anthropogenic radionuclides in the environment.	DOE Energy Research

DOE = Department of Energy; DOI = Department of Interior

Source: FEDRIP 2000

7. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, measuring, and/or monitoring americium, its metabolites, and other biomarkers of exposure and effect to americium. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

7.1 BIOLOGICAL MATERIALS

Entry of americium into the human body can be gained through ingestion, inhalation, or penetration through skin. The quantities of americium within the body can be assessed through the use of bioassays that are comprised of *in vivo* measurements and/or *in vitro* measurements. *In vivo* measurements can be obtained through techniques that directly quantify internally deposited americium (using, e.g., a whole body counter). Conversely, *in vitro* measurements provide an estimate of internally deposited americium, utilizing techniques that measure americium in body fluids, feces, or tissue. Examples of these analytical techniques are given in NCRP Report No. 87 (1987) and are also listed in Table 7-1.

7.1.1 Internal Americium Measurements

In vivo measurement techniques are the most direct and widely used approach for assessing the burden of many radioisotopes, including americium, within the body. The *in vivo* measurement of americium within the body is performed with various radiation detectors and associated electronic devices that are collectively known as whole body counters. These radiation detectors commonly utilize sodium iodide (NaI), phoswich (NaI and CsI sandwich), and hyperpure germanium to detect the 59.5 keV gamma-ray that ^{241}Am promptly emits in 35.9% of its alpha decays to ^{237}Np (DOE 1979b; Palmer et al. 1983). Because the attenuation half thickness for the 59.5 keV gamma-ray is 3.5 cm for soft tissues and 1.3 cm for bone, ^{241}Am that has been deposited into specific organs or tissues, such as the lungs, liver, bones, or lymph nodes, can be detected and quantified using whole body counting techniques that appropriately

Table 7-1. Analytical Methods for Determining Americium in Biological Samples

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Urine	None	Gamma-ray spectrometry (phoswich detector)	0.08 nCi/200 cm^3	No data	Ide et al. 1985
Urine	Co-precipitation with oxalate	α-Spectrometry	0.08 pCi/80 cm^3	No data	Ide et al. 1985
Urine	None	Gamma-ray spectrometry	0.04 pCi/cm^3	95% at 0.04 pCi/cm^3	Guilmette 1986
Urine	Sample wet ashed, treated with HNO_3 and H_2O_2	α-Liquid scintillation	0.7 pCi/125 cm^3	95% at 0.01–1,000 nCi	Guilmette and Bay 1981
Urine	Sample wet ashed, purified by solvent extraction	Liquid scintillation	14 pCi/100 cm^3	96% at 20,000 dpm spike	Ham et al. 1977
Urine	Sample with ^{241}Am spike co-precipitated with $CaHPO_4$ then with oxalate, purification by diglycol succinate column	α-Liquid scintillation	0.02 pCi/L	96% at 20 dpm spike	Hafez and Hafez 1992
Urine	Sample cleaned-up by co-precipitation, treated with HNO_3 and H_2O_2, wet ashed	Biphasic liquid scintillation	1 pCi/200 cm^3	84%	Bomben et al. 1994
Urine	Spiked sample clean-up by co-precipitation, purified by TRU-spec column and electrodeposition	α-Spectrometry	0.016 pCi/800 cm^3	95% at 0.1–100 pCi/sample	Goldstein et al. 1997

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Table 7-1. Analytical Methods for Determining Americium in Biological Samples (*continued*)

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Soft tissue	Sample wet ashed, spiked with ^{243}Am, purified by anion exchange, solvent extraction, and electrodeposition	α-Spectrometry	No data	98%	McInroy et al. 1985
Soft tissue	Spiked sample wet ashed, treated with HNO_3/H_2O_2, purified by A-CU column, anion exchange, TRU-spec column, and electrodeposition	α-Spectrometry	No data	53%	Qu et al. 1998
Soft tissue	Sample wet ashed, purified by solvent extraction	Liquid scintillation	1.3 pCi/100 cm^3	96% at 20,000 dpm spike	Ham et al. 1977
Soft tissue	Sample wet ashed, purified by solvent extraction	α-Liquid scintillation	0.7 pCi/g	99% at 3,000 dpm spike	Guilmette and Bay 1981
Bone	Sample wet ashed, purified by solvent extraction	α-Liquid scintillation	0.7 pCi/g	99% at 3,000 dpm spike	Guilmette and Bay 1981
Bone	Sample wet ashed, spiked with ^{243}Am, and purified by anion exchange resin column, solvent extraction, and electrodeposition	α-Spectrometry	No data	98%	McInroy et al. 1985
Feces	Sample wet ashed, purified by solvent extraction	Liquid scintillation	13 pCi/g	91% at 20,000 dpm spike	Ham et al. 1977

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Table 7-1. Analytical Methods for Determining Americium in Biological Samples (*continued*)

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Feces	Sample wet ashed, purified by solvent extraction	α-Liquid scintillation	1.3 pCi/sample	96% at 3,000 dpm spike	Guilmette and Bay 1981
Feces	None	Phoswich detector	0.02–0.09 nCi/200g	No data	Kramer et al. 1989
Teeth	Sample dissolved in HNO_3, purified by TRU-spec column and electrodeposition	α-Spectrometry	2.7 fCi/sample	98%	Culot et al. 1997
Whole organs and tissues	Animal placed, backbone down in lucite box and positioned 33 cm from NaI(Tl) crystal. Livers and other tissues were counted between two NaI(Tl) crystals.	Gamma-ray spectrometry	No data	No data	Lloyd et al. 1970

TRU = transuranic

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account for internal shielding (Graham and Kirkman 1983; Palmer and Rhoads 1989; Palmer et al. 1983). Many configurations of the whole body counter have been utilized, ranging from the more common chest detectors to linear whole body scanners that can be utilized to assess the regional distributions of ^{241}Am over the entire length of the body (Palmer et al. 1983; Toohey and Essling 1980). Where appropriate, shielding of the room that houses the whole body counter and/or the detector is often used to increase the detection sensitivity of the equipment by minimizing background radiation. Also, *in vitro* measurements of americium (see Section 7.1.2) are often used in conjunction with whole body counting when monitoring individuals working with americium.

Calibration of whole body counters is achieved through the use of tissue-equivalent phantoms that are constructed to mimic the shape and density of the anatomical structure (e.g., the human torso), using tissue equivalent materials such as polystyrene or epoxies. In some phantoms, a human rib cage is added to account for the attenuation of gamma-rays by bone in the whole body counts (DOE 1979b). Americium standards are inserted or molded into the phantom at locations where this isotope is expected to accumulate, such as in the lung, liver, or bone. Comparisons of the activity obtained from the phantom to the known activity of the americium standards are used to determine the efficiency of the counting technique and, thus, provide the basis for calibrating the technique. Calibration of whole body counts can be further refined by obtaining actual anatomical measurements of the individual to be scanned. For example, chest wall measurements using ultrasound techniques are used to account for the variability in attenuation that results from individual differences in the chest wall thickness and improving the calibration of chest counts of americium (DOE 1979b). Another approach to refining the calibration of whole body counters is the comparison of external measurements to the actual americium content in organs and bone of cadavers (Palmer et al. 1985). These refinements in calibration phantoms can lead to a more accurate assessment of the total body or organ burden of americium.

The NRC requires that the occupational intake of americium isotopes not exceed certain specified Annual Limits on Intake (ALIs) for the inhalation and oral routes of exposure. For ^{241}Am and ^{243}Am, the oral ingestion ALI is 0.8 μCi and the inhalation ALI is 0.006 μCi, both of which are based on the deterministic dose limit to the bone surface (NRC 2000).

In assessing initial exposure, whole body counting techniques measure amounts of americium that have been deposited within organs or tissues and have not been excreted. In cases of accidental ingestion, some of the americium may have been excreted in the urine or through the feces, before exposure is assessed. In particular, soluble forms of americium are thought to be readily excreted through the urine.

Long-term assessment of americium burden within an individual can be complicated by the mobilization of americium from the original site of deposition (e.g., the lung) to other sites within the body, such as the liver or bone (Fry 1976). This can lead to overestimates of the overall total body burden of americium depending on which regions the americium counts are obtained (Kathren et al. 1988). Additionally, the retention (biological half-life) of americium within the body can vary greatly between individuals (Fry 1976). Also, direct comparisons of americium body burdens and clearance rates between laboratories can be complicated by the differing whole body measurement techniques, calibration methods, and methods used to account for normal background radiation counts that are utilized within the different laboratories (DOE 1979b). These limitations are largely resolved by performing periodic internal monitoring and adjusting the model parameters to account for the individual's actual distributed retention.

7.1.2 External Radiation Measurements

In vitro analyses of americium are routinely performed in situations where *in vivo* analyses cannot be obtained or in support of an *in vivo* monitoring program. Urine is the preferred sample for *in vitro* analyses of americium, although other sample types, such as feces, tissue, bone, or blood, can also be used on a more limited basis. Urine provides for an analysis of soluble or transportable americium, fecal analysis can be used to measure ingestion or clearance of americium, and tissue is used to assess whole or regional body burdens of americium (Guilmette and Bay 1981; Ide 1986; Ide et al. 1985; McInroy et al. 1985).

Urinalysis for americium can be performed on the basis of total either mass or total activity. There are a number of methods that have the selectivity and/or sensitivity to measure americium in biological matrices including spectrophotometry, fluorometry, mass spectrometry (MS), and radioassays (Dacheux 1998; Hafez and Hafez 1992; Poupard and Jouniaux 1990; Thouvenout et al. 1993). Of these methods, radioassays (e.g., gross alpha, analysis alpha spectroscopy, gamma-ray spectroscopy, liquid scintillation techniques) are preferred because of their ease of use, detection sensitivity, and rapidity of analysis (Alvarez and Navarro 1996; Dacheux and Aupiais 1997; Guilmette 1986). These methods typically involve a preliminary concentration step and wet/dry ashing of the sample that is often followed by an oxidation of the radionuclides in the sample residue. To remove the possible interferences of other alpha-emitters, ion exchange, co-precipitation, and adsorption techniques are applied to the purified sample before alpha or liquid scintillation techniques are applied (see Table 7-1). Radioassays can also be applied to the measurement of americium in fecal, tissue, and bone samples using methods that are similar to those described for urinalysis, except for some additional purification and extraction steps that

are required to remove interfering materials such as iron or excess mass (Guilmette and Bay 1981; Hafez and Hafez 1992; Ham et al. 1977; Qu et al. 1998).

Of the radioassays that are commonly used to quantify americium, α-spectroscopy is used when isotopic analyses of americium must be conducted (e.g., ^{241}Am and ^{243}Am). ^{243}Am is often added as a tracer to estimate the efficiency of the sample preparation method when quantifying ^{241}Am in biological matrices. The α-spectroscopy technique differentiates between the two americium isotopes based on the difference in the energies of the alpha particles that are emitted from ^{241}Am and ^{243}Am, and then quantifies the amounts present in the sample considering the activity that each contributes, its detection efficiency, and the frequency with which the individual alpha particles are emitted. Mass spectrometric techniques are also capable of isotopic quantification of americium (Dacheux and Aupiais 1998; Poupard and Jouniaux 1990). These techniques are more rapid than the α-spectroscopy detection method, but the cost has been much higher. However, the cost of mass spectrometers has decreased in recent years, making the cost of analyzing samples by mass spectroscopy and α-spectroscopy more comparable. Higher sensitivity can also be achieved with α-spectroscopy by resorting to long sampling times, sometimes referred to as time-averaging.

Accuracy of *in vivo* and *in vitro* measurements of americium is determined through the use of standard, certified radioactive sources with known concentrations of americium. The primary source of certified americium standards is the National Institute of Standards and Technology (NIST). Standard solutions are available for ^{241}Am (SRM 4322, 40 Bq/g [1.1 nCi/g]) and ^{243}Am (SRM 4332, 40 Bq/g [1.1 nCi/g]). Standard Reference Materials for human lung (SRM 4351) and human liver (SRM 4352) are also available from NIST.

7.2 ENVIRONMENTAL SAMPLES

There are two common approaches for measuring americium in the environment. Americium can either be measured directly in the field (*in situ*) using portable survey instruments or samples can be procured from the field and returned to the laboratory for quantification of americium.

7.2.1 Field Measurements of Americium

In situ measurement techniques are extremely useful for the rapid characterization of radionuclide contamination in the environment, such as soils, sediments, and vegetation, or when monitoring personnel

for exposure to radionuclides. Information regarding field measurement methods, minimum detectable concentrations, and soil-to-plant concentration factors is available for various radionuclides, including americium (MARSSIM 2000; NRC 1992, 1998b). The measurement of radionuclides in the environment is conducted with portable survey instruments that are equipped with α-scintillators or gamma-ray spectrometers. However, the use of gamma-spectrometers in field survey equipment is preferred for measuring americium in the field because the low-energy gamma-ray photon that is emitted during the decay of ^{241}Am is more penetrating and measurements are less affected by the presence of vegetation and surrounding soil than is the alpha particle that is also emitted (Byrne and Komosa 1993). This provides the advantage for assessing the level of americium both on and below the surface (e.g., up to 3-cm depth in some soils). These gamma-ray spectrometers are equipped with either a thin sodium iodide or a high purity germanium detector that is able to distinguish the 59.5 keV gamma-ray emitted from ^{241}Am from most environmental gamma-rays emitted from other radionuclides (Fong and Alvarez 1997). Another advantage of these spectrometers is the ability to discriminate the 59.5 keV photons of ^{241}Am from the much lower energy photons emitted from the ^{239}Pu and ^{240}Pu that are usually associated with americium. Minimum detectable activities (MDAs) of 0.4 Bq/g (10 pCi/g) for ^{241}Am are routinely achieved with MDAs as low as 0.04 Bq/g (1 pCi/g) obtained with longer counting times (~30 days).

One of the limitations of the portable field survey instruments in the measurement of americium is that their quantitative accuracy depends on how well the lateral and vertical distribution of americium in the soil compares with the calibration parameters used. These methods can provide a rapid assessment of americium levels on or below surfaces in a particular environment; however, laboratory-based analyses of samples procured from these environmental surfaces must be performed in order to ensure accurate quantification of americium (and other radionuclides). This is due, in part, to the strong self absorption of the 59.5 keV gamma-ray by environmental media, such as soil. Consequently, the uncertainty in the depth distribution of americium and the density of the environmental media may contribute to a >30% error in the field survey measurements. Currently, refinements in calibration strategies are being developed to improve both the precision and accuracy (10%) of gamma-ray spectroscopy measurements of americium within contaminated soils (Fong and Alvarez 1997).

7.2.2 Laboratory Analysis of Environmental Samples

Analytical methods for quantifying americium in environmental samples are summarized in Table 7-2. The methods that are commonly used in the analysis of americium based on activity are gross α analysis, α-spectrometry and gamma-ray spectrometry. MS detection techniques are used to measure the mass of americium in environmental samples. (The mass-activity conversion factor for ^{241}Am is 0.29 μCi/μg or 3.43 μg/μCi [Harvey et al. 1993]).

The analysis of americium in air is based on the quantification of americium within particulates that become trapped on cellulose or glass fiber filters after a calibrated amount of air is pulled through the filters. A common method of analysis for americium on a glass fiber filter is a rather complex procedure involving many solvent extraction and column purification steps, followed by electrodeposition and α-spectroscopy. The extensive purification is required to prevent impurities within the sample from absorbing or reducing the energy of emitted alpha particles, termed self-absorption. Alpha-emitting contaminants must also be removed (e.g., ^{238}Pu) from the samples to prevent these materials from interfering with the α-spectrometry measurements of ^{241}Am and ^{243}Am (ASTM 1997; Lovette et al. 1990). Initially, the filter media is dissolved with HNO_3 and H_2O_2, the residue wet ashed with HNO_3, and then purified using anion exchange chromatography, solvent extraction with 50% bis-(2-ethylhexyl) phosphoric acid (HDEHP) in toluene, another passage of the sample through an anion exchange column, and then oxidation of the sample with HNO_3 and H_2O_2. Because ^{243}Am is commonly used as an internal standard when quantifying ^{241}Am, α-spectrometry is utilized to resolve and quantify the americium isotopes in the purified filter sample. Preparation of the purified filter sample for α-spectrometry requires electrodeposition of the americium from a sulfate solution onto a stainless steel or platinum disc from which alpha counts are obtained during the α-spectrometry analysis (DOE 1997b). The accuracy of this method of analysis for americium can vary between 85 and 102% and the minimum detectable activity (MDA) often ranges between 0.032 and 0.023 pCi/sample (Goldstein et al. 1997).

For the analysis of americium in water, there is a broad array of sample preparation and detection methodologies that are available (see Table 7-2). Many of the common and standardized analytical methodologies typically include the minimization of sample volume, purification through co-precipitation, anion exchange column chromatography, and solvent extraction techniques followed by radiochemical detection of americium in the purified sample. Gross alpha analysis or liquid scintillation are common detection techniques that are utilized to quantify americium in these methods. However, if lower detection sensitivity or isotopic determination is required, then α-spectrometry is the preferred

Table 7-2. Analytical Methods for Determining Americium in Environmental Samples

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Air	Sample collection on cellulose filter, dry ashed, solvent extracted	Biphasic liquid scintillation	1 pCi	95%	Bomben et al. 1994
Air	Filter wet ashed in HNO_3/HF, purified with cation and anion exchange columns and electrodeposition	α-Spectroscopy	No data	No data	Knab 1979
Air	Cellulose filter dry ashed, dissolved in HNO_3/HF, H_2O_2/$HClO_4$, purified with anion exchange, TRU-spec columns followed by electrodeposition.	α-Spectroscopy	0.023 pCi/sample	102%	Goldstein et al. 1997
Water	Sample fusion with pyrosulfate, precipitated with barium sulfate	Scintillation counter	No data	99.5%	Sill and Williams 1969
Water	Wet ashed, purified by solvent extraction	Biphasic liquid scintillation	1 pCi/sample	95%	Bomben et al. 1994
Water	Treated with HNO_3/H_2O_2, HF/HCl, anion exchange, TRU-spec column, electrodeposition	α-Spectroscopy	0.026 pCi/L	101%	Goldstein et al. 1997
Water	Solvent extracted	PERALS	0.007 pCi/L	104%	Dacheux and Aupiais 1998

Table 7-2. Analytical Methods for Determining Americium in Environmental Samples (*continued*)

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Sea water	Co-precipitation with iron hydroxide, purified by anion exchange, co-precipitation with $BiPO_4$, cation exchange, electrodeposition	α-Spectroscopy	No data	64–79%	Lovette et al. 1990
Sediments	Sample fusion with KF and pyrosulfate, coprecipitate with $BaSO_4$	Scintillation counter	No data	No data	Sill and Williams 1969
Sediments	Sample leached with HNO_3/HF, filtered, purified by KL-HDEHP resin columns, solvent extracted, and electrodeposition	α-Spectroscopy	No data	95–99%	Guogang et al. 1998
Sediments	None	Gamma-ray spectroscopy	0.02–0.06 pCi/g	108–118%	Joshi 1989
Soil	Sample fusion with KF and pyrosulfate, coprecipitate with $BaSO_4$	Scintillation counter	No data	No data	Sill and Williams 1969
Soil	Wet ash in HNO_3/HF, purified with cation and anion exchange columns, electrodeposition	α-Spectroscopy	No data	No data	Knab 1979

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Table 7-2. Analytical Methods for Determining Americium in Environmental Samples (*continued*)

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Soil	Dry ash, digest in HNO_3/HCl, anion exchange, Ca-oxalate and Fe $(OH)_2$ coprecipitation, anion exchange, electrodeposition	α-Spectroscopy	27 pCi/g	75–92%	Sanchez and Singleton 1996
Soil	Sample leached with HNO_3/HF, filtered, purified by KL-HDEHP resin columns, solvent extracted, and electrodeposition	α-Spectroscopy	No data	95–99%	Guogang et al. 1998
Soil	None	Gamma-ray spectroscopy	0.02–0.06 pCi/g	108–118%	Joshi 1989
Vegetation (grasses)	Ashed, HNO_3/HF, precipitation with oxalate and La, anion exchange, solvent extraction	α-Spectroscopy	0.011 pCi/g	No data	Bunzl and Kracke 1990
Vegetation	Ashed, digested with HNO_3-H_2O_2, oxalate and Fe precipitations, anion exchange, solvent extraction, electrodeposition	α-Spectroscopy	0.3 fCi/g	73–109%	Cooper et al. 1993
Vegetation	Ashed, digested with HNO_3-HCl, anion exchange, Ca-oxalate and Fe precipitations, anion exchange, electrodeposition	α-Spectroscopy	27 pCi/g	75–92%	Sanchez and Singleton 1996

Table 7-2. Analytical Methods for Determining Americium in Environmental Samples (*continued*)

Sample matrix	Sample preparation	Analytical method	Detection limit	Accuracy	Reference
Lichen, moss	Ashed, leached with HCl, Microthene-TNOA and KL-HDEHP column extractions, solvent extraction, electrodeposition	α-Spectroscopy	0.9 fCi/g	No data	Jia et al. 1997
Biota	Ashed, digested with HNO_3-H_2O_2, oxalate, and Fe precipitations, anion exchange, solvent extraction, electrodeposition	α-Spectroscopy	0.3 fCi/g	98–100% 480% (shrimp)	Cooper et al. 1993

KL-HDEHP = 50% di(2-ethylhexyl) phosphoric acid, 60-100 mesh resin; PERALS = Photon/electron rejecting alpha liquid scintillation; TNOA = tri-*n*-octylamine; TRU = transuranic

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method to quantify ^{241}Am, especially when ^{243}Am is used as an internal standard in the radiochemical assay (Dacheux and Aupiais 1997; DOE 1997b; Goldstein et al. 1997; Harvey et al. 1993; Sill and Williams 1969). These detection methods can provide measurements of total activity of americium within a sample, especially when appropriate steps have been taken to purify the sample of interfering materials or minimize the influence of other radionuclides on radiochemical activity (Dazhu et al. 1991). The presence of alpha, beta, and/or gamma emitting radionuclides in the sample can contribute to the counts measured in radiochemical detection methods and, thus, affect the accuracy of the assay for determining the quantity of americium within a sample.

There are methods available to quantify the total mass of americium in environmental samples. Mass spectrometric methods provide total mass measurements of americium isotopes (Dacheux and Aupiais 1997, 1998; Halverson 1984; Harvey et al. 1993) however, these detection methods have not gained the same popularity as is found for the radiochemical detection methods. This may relate to the higher purchase price of a MS system, the increased knowledge required to operate the equipment, and the selection by EPA of α-spectrometry for use in its standard analytical methods. Fluorimetric methods, which are commonly used to determine the total mass of uranium and curium in environmental samples, have limited utility to quantify americium, due to the low quantum yield of fluorescence for americium (Thouvenout et al. 1993).

Several rapid radiochemical analysis techniques have been developed that require either no or minimal sample preparation that can, under optimized conditions, provide the required selectivity and sensitivity to quantify americium in environmental samples, such as soils and sediments (Byrne and Komosa 1993; Cutshall et al. 1983; Guilmette 1986; Joshi 1989). These techniques utilize a phoswich-based low energy photon detector that detects the gamma-rays emitted from ^{241}Am in a neat or ashed environmental or biological sample. On average, these methods have a minimum significant measurable activity (MSMA) of approximately 1 pCi/sample. To achieve this low MSMA, the counting efficiency of the detector must be standardized against the sample size and composition in order to assure the desired accuracy of the assay (Cutshall et al. 1983; Joshi 1989). However, one limitation of the assay is the need to know the isotopic composition of the sample since the x-rays that accompany the decay of other radionuclides may also be counted by the phoswich detector.

Several methods have been described in the Multi-Agency Radiation Survey and Site Investigation Manual (MARSSIM 2000) for the survey and investigation of sites contaminated with radioactive materials. At the high end of the survey instruments, costing over $1,000,000 in 1995, is an inductively

coupled plasma mass spectrometer (ICP-MS) with a laser added to the front end to vaporize small portions of a surface to be analyze (laser ablation), thereby avoiding physical sample collection.

The quantity of americium in soil, sediments, vegetation, and biota is determined using methods similar to those described above. For example, in a standardized method developed by Department of Energy (DOE) (DOE 1997b) soil samples are dissolved with a series of acid treatments (e.g., HNO_3, HF) and initially purified through co-precipitation using calcium oxalate followed by co-precipitation with an Fe carrier. A final purification of the sample is achieved by passing the sample through a series of anion exchange columns (e.g., HNO_3, HCl, and NH_4 SCN), followed by electrodeposition of the americium (chloride form) onto a platinum disc in preparation for α-spectrometry analysis.

In another standardized method developed for the analysis of americium in soil (ASTM 1997), a different approach is taken towards purifying the dissolved soil sample that relies on a series of co-precipitations and solvent extractions to prepare the soil sample for α-spectrometry analysis. After the soil has been dissolved, the sample is initially purified by co-precipitation with barium sulfate followed by solvent extraction of the redissolved precipitate with 15% HDEHP in n-hexane. The extracted trivalent actinides and lanthanides are stripped from the organic phase using nitric acid containing sodium bromate. The subsequent solution containing the trivalent actinides and lanthanides is extracted again with 15% HDEHP in n-hexane to remove plutonium, thorium, and tetravalent curium. The aqueous phase is further purified through co-precipitation with a lanthanum carrier to isolate the rare earth fluorides followed by a treatment using silver nitrate and ammonium persulfate/ammonium fluoride to precipitate all remaining rare earth fluorides, except for hexavalent americium fluoride. The hexavalent americium is reduced back to the trivalent state using hydrogen peroxide and then reprecipitated with a neodymium carrier in preparation for α-spectrometry analysis. Both the DOE and the American Society for Testing and Materials (ASTM) methods of analysis provide good precision (<6% standard derivation) with no statistically significant bias (at the 5% level) observed. Analysis of americium in sediments, vegetation, and biota can also be performed using variations of the abovementioned methods or other methods, as exemplified in Table 7-2.

The detection limits, accuracy, and precision of any analytical methodology, as well as the composition of the sample medium, are important parameters in determining the appropriateness of a method to quantify a specific analyte at the desired level of sensitivity within a particular matrix. The lower limit of detection (LLD) has been adopted to refer to the intrinsic detection capability of a measurement procedure (sampling through data reduction and reporting) to aid in determining which method is best

suited for the required sample quantification (NRC 1984). Several factors influence the LLD, including background counting rates, size or concentration of sample, detector sensitivity, recovery of desired analyte during sample isolation and purification, level of interfering contaminants, and, particularly, counting time. Because of these variables, the LLDs between laboratories, utilizing the same or similar measurement procedures, will vary.

The accuracy of a measurement technique in determining the quantity of a particular analyte in environmental samples is greatly dependent on the reliability of the calibrating technique. Thus, the availability of standard, certified radiation sources with known concentrations of americium are required in order to ensure the reliability of the calibration methods and accuracy of americium measurements in environmental samples. The primary source of certified americium standards is the NIST. Standard solutions of ^{241}Am (SRM 4322, 40 Bq/g [1.1 nCi/g]) and ^{243}Am (SRM 4332, 40 Bq/g [1.1 nCi/g]) are available. Standard reference materials are also available from NIST and International Atomic Energy Agency (IAEA) for a number of environmental matrices; for example, soils and sediments (Rocky Flats Soil [SRM 4353], river sediment [SRM 4350B], and Peruvian soil [SRM 4355], sediments [IAEA 367, IAEA 135]).

7.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of americium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of americium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

7.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Analytical methods with satisfactory sensitivity and precision are available to determine the levels of americium in human tissues and body fluids. However, improved methods are needed to assess the biological effects of americium in tissues.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods with the required sensitivity and accuracy are available for quantification of americium, both total and isotopic, in environmental matrices (see Table 7-2). Knowledge of the levels of americium in various environmental media, along with the appropriate modeling (see Chapters 3 and 5), can be used to evaluate potential human exposures through inhalation and ingestion pathways.

Whether in the environment or in the human body, americium will undergo radioactive decay to form a series of radioactive nuclides that end in a stable isotope of lead (for ^{243}Am) or bismuth (for ^{241}Am) (see Chapter 4). However, more sensitive analytical methods are needed for accurately measuring very low levels of these radionuclides. Practically speaking, since ^{239}Pu (for ^{243}Am) and ^{237}Np (for ^{241}Am) have such extremely long half-lives, 2.41×10^4 and 2.14×10^6 years, respectively, few decay products need to be considered.

7.3.2 Ongoing Studies

In the Federal Research in Progress (FEDRIP) lists an ongoing effort by G. Entine of Radiation Monitoring Devices, Inc., in Watertown, Massachusetts, which proposes development of a new type of larger, high resolution, high efficiency germanium detector in order to provide effective detection, localization, as well as quantification of the radionuclides inhaled by workers. The proposed effort is expected to lead to a simpler, higher performance, and more economical germanium detector system for lung screening. This effort is supported by the National Heart, Lung, and Blood Institute (FEDRIP 2000).

Table 7-3. Ongoing Studies on Analytical Methods for Americium

Investigator	Affiliation	Subject	Sponsor
Entine, G	Radiation Monitoring Devices, Inc., Watertown, Massachusetts	The goal of the proposed effort is to develop a new type of larger, high resolution, high efficiency germanium detector in order to provide effective detection, localization, as well as quantification of the radionuclides inhaled by workers. The proposed effort is expected to lead to a simpler, higher performance, and more economical germanium detector system for lung screening.	National Heart, Lung, and Blood Institute

Source: FEDRIP 2000

8. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding americium in air, water, and other media are summarized in Table 8-1.

The Nuclear Regulatory Commission (NRC) maintains a database of information regarding licensees authorized to possess americium isotopes (^{241}Am through ^{244}Am) within the 18 non-Agreement States (states not committed to self-regulation). This database includes more than 2,000 records of licensees and site possession limits in the microcurie to curie range. The isotope ^{241}Am accounts for most of the licenses.

No inhalation or oral MRLs were derived for americium or americium compounds.

The EPA classifies all radionuclides, including americium, as Group A (known human) carcinogens (EPA 1997b). Lifetime excess total cancer risk per unit intake or exposure for ingestion, inhalation, and external exposure to ^{241}Am and ^{243}Am are included in Table 8-1. The EPA has not derived reference concentrations (RfCs) or reference doses (RfDs) for americium (IRIS 2001).

Table 8-1. Regulations and Guidelines Applicable to Americium

Agency	Description	Information	Reference
INTERNATIONAL Guidelines:			
IARC		No data	
NATIONAL Regulations and Guidelines:			
a. Air			
ACGIH		No data	
EPA	Concentration levels for environmental compliance		EPA 2001a 40CFR61, Appendix E
	^{241}Am	1.9×10^{-15} Ci/m^3	
	^{242}Am	1.5×10^{-11} Ci/m^3	
	^{243}Am	1.8×10^{-15} Ci/m^3	
NIOSH	REL (TWA)	No data	
NRC	Occupational values—inhalation	ALI(µCi) DAC(µCi/mL)	NRC 2001a 10CFR20, Appendix B
	^{241}Am	6×10^{-3} 3×10^{-12}	
	^{242}Am	8×10^{1} 4×10^{-8}	
	^{243}Am	6×10^{-3} 3×10^{-12}	
	Effluent concentrations—air		NRC 2001a 10CFR20, Appendix B
	^{241}Am	2×10^{-14} µCi/mL	
	^{242}Am	1×10^{-10} µCi/mL	
	^{243}Am	2×10^{-14} µCi/mL	
OSHA	PEL (8-hour TWA)	No data	
b. Water			
NRC	Effluent concentrations—water		NRC 2001a 10CFR20, Appendix B
	^{241}Am	2×10^{-8} µCi/mL	
	^{242}Am	5×10^{-5} µCi/mL	
	^{243}Am	2×10^{-8} µCi/mL	
	Releases to sewers—monthly average concentration		NRC 2001a 10CFR20, Appendix B
	^{241}Am	2×10^{-7} µCi/mL	
	^{242}Am	5×10^{-4} µCi/mL	
	^{243}Am	2×10^{-7} µCi/mL	
c. Food			
FDA	Protective action goals and derived intervention levels by age group (Bq/kg)		FDA 1998
	^{241}Am (bone surface)		
	PAGs (mSv)	50	
	3-months	2.0	
	1-year	17	
	5-years	13	
	10-years	11	
	15-years	9.1	
	Adult	8.8	

Table 8-1. Regulations and Guidelines Applicable to Americium *(continued)*

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
FDA	Protective action goals and derived intervention levels by age group (Bq/kg)		FDA 1998
	^{241}Am		
	PAGs (mSv)	5	
	3-months	3.3	
	1-year	27	
	5-years	25	
	10-years	24	
	15-years	21	
	Adult	20	
	Sources of radiation used for inspection of food; sealed units producing radiation—^{241}Am	# 2.2 million electron volts	FDA 2000 21CFR179.21
d. Other			
DOE	Radiation standards—inhaled air DAC for lung retention		DOE 2000 10CFR835, Appendix A
	^{241}Am	$2x10^{-12}$	
	^{242}Am	$3x10^{-8}$	
	^{243}Am	$2x10^{-12}$	
DOT	Activity values for radionuclides		DOT 2001 40CFR173.435
	^{241}Am, ^{242}Am, and ^{243}Am		
	A_1	$5.41x10^{1}$ Ci	
	A_2	$5.41x10^{-3}$ Ci	
EPA	Annual possession quantities for environmental compliance		EPA 2001a 40CFR61, Appendix E
	^{241}Am		
	Gaseous form	$2.3x10^{-6}$ Ci/year	
	Liquid/powder forms	$2.3x10^{-3}$ Ci/year	
	Solid form	$2.3x10^{0}$ Ci/year	
	^{242}Am		
	Gaseous form	$1.8x10^{-2}$ Ci/year	
	Liquid/powder forms	$1.8x10^{1}$ Ci/year	
	Solid form	$1.8x10^{4}$ Ci/year	
	^{243}Am		
	Gaseous form	$2.3x10^{-6}$ Ci/year	
	Liquid/powder forms	$2.3x10^{-3}$ Ci/year	
	Solid form	$2.3x10^{0}$ Ci/year	
	Radioactive waste—release limits for containment requirements[a]		EPA 2001b 40CFR191, Appendix A
	^{241}Am or ^{243}Am	$1x10^{2}$ Ci	
	Reportable quantity		EPA 2001c 40CFR302.4, Appendix B
	^{241}Am	$1x10^{-2}$ Ci	
	^{242}Am	$1x10^{2}$ Ci	
	^{243}Am	$1x10^{-2}$ Ci	

Table 8-1. Regulations and Guidelines Applicable to Americium *(continued)*

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
EPA	Carcinogenicity—slope factors[b]		EPA 1997
	Ingestion—lifetime excess total cancer risk/pCi		
	^{241}Am	3.28×10^{-10}	
	^{243}Am	3.27×10^{-10}	
	Inhalation—lifetime excess total cancer risk/pCi		
	^{241}Am	3.85×10^{-8}	
	^{243}Am	3.82×10^{-8}	
	External exposure—risk/year per pCi/g in soil		
	^{241}Am	4.59×10^{-9}	
	^{243}Am	2.43×10^{-8}	
NRC	Activity values for radionuclides		NRC 2001b 10CFR71, Table A-1
	^{241}Am, ^{242}Am, and ^{243}Am		
	A_1	5.41×10^{1} Ci	
	A_2	5.41×10^{-3} Ci	
	Specific gravity		
	^{241}Am	3.4 Ci/g	
	^{242}Am	1.0×10^{1} Ci/g	
	^{243}Am	2.0×10^{-1} Ci/g	
	Byproduct material for *in vitro* clinical or laboratory testing—^{241}Am	#0.005 µCi	NRC 2001c 10CFR31.11
	Calibration or reference sources—shall not possess at any one time, at any one location of storage or use of ^{241}Am	#5 µCi	NRC 2001d 10CFR31.8
	Exemption for low-level materials—contains only Americium in special form	aggregate radioactivity # 20 Ci	NRC 2001e 10CFR71.10
	Export of byproduct material	#1 Ci/shipment or #100 Ci/year	NRC 2001f 10CFR110.23
	General applicability to domestic licensing of byproduct material —quantity of licensed material requiring labeling		NRC 2001g 10CFR30, Appendix B
	^{241}Am	1×10^{-2} µCi	

Table 8-1. Regulations and Guidelines Applicable to Americium *(continued)*

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
NRC	Ionizing radiation measuring instruments containing, for purposes of internal calibration or standardization, one or more sources of byproduct material—exempt quantities of ^{241}Am	0.05 µCi	NRC 2001g 10CFR30.15(a)(9)
	Occupational values—oral ingestion (ALI)		NRC 2001a 10CFR20, Appendix B
	^{241}Am	$8x10^{-1}$ µCi	
	^{242}Am	$4x10^{3}$ µCi	
	^{243}Am	$8x10^{-1}$ µCi	
	Quantity of radioactive material requiring need for an emergency plan for responding to a release—^{241}Am, ^{242}Am, and ^{243}Am		NRC 2001h 10CFR30.72, Schedule C
	Release fraction	0.001%	
	Quantity	2 Ci	
	Standards for protection against radiation—quantity of licensed material requiring labeling		NRC 2001i 10CFR20, Appendix C
	^{241}Am	$1x10^{-3}$ µCi	
	^{242}Am	$1x10^{1}$ µCi	
	^{243}Am	$1x10^{-3}$ µCi	
STATE			
a. Air			
Illinois	Concentrations in air above natural background		BNA 2001
	^{241}Am	S $2x10^{-13}$ µCi/mL	
		I $4x10^{-12}$ µCi/mL	
	^{242m}Am	S $2x10^{-13}$ µCi/mL	
		I $9x10^{-12}$ µCi/mL	
	^{242}Am	S $1x10^{-9}$ µCi/mL	
		I $2x10^{-9}$ µCi/mL	
	^{243}Am	S $2x10^{-13}$ µCi/mL	
		I $4x10^{-12}$ µCi/mL	
	^{244}Am	S $1x10^{-7}$ µCi/mL	
		I $8x10^{-7}$ µCi/mL	

Table 8-1. Regulations and Guidelines Applicable to Americium *(continued)*

Agency	Description	Information	Reference
STATE *(cont.)*			
New Jersey	Maximum permissible average concentrations in air Occupational (40-hour week)		BNA 2001
	^{241}Am	S $6x10^{-12}$ µCi/mL I $1x10^{-10}$ µCi/mL	
	^{242m}Am	S $6x10^{-12}$ µCi/mL I $3x10^{-10}$ µCi/mL	
	^{242}Am	S $4x10^{-8}$ µCi/mL I $5x10^{-8}$ µCi/mL	
	^{243}Am	S $6x10^{-12}$ µCi/mL I $1x10^{-10}$ µCi/mL	
	^{244}Am	S $4x10^{-6}$ µCi/mL I $2x10^{-5}$ µCi/mL	
New Jersey	Maximum permissible average concentrations in air Non-occupational		BNA 2001
	^{241}Am	S $2x10^{-13}$ µCi/mL I $4x10^{-12}$ µCi/mL	
	^{242m}Am	S $2x10^{-13}$ µCi/mL I $9x10^{-12}$ µCi/mL	
	^{242}Am	S $1x10^{-9}$ µCi/mL I $2x10^{-9}$ µCi/mL	
	^{243}Am	S $2x10^{-13}$ µCi/mL I $4x10^{-12}$ µCi/mL	
	^{244}Am	S $1x10^{-7}$ µCi/mL I $8x10^{-7}$ µCi/mL	
b. Water			
Colorado	Standards applicable to surface waters—^{241}Am	0.15 pCi/L	BNA 2001
	Groundwater levels of radioactive materials shall not exceed this amount—Americium	0.15 pCi/L	CO Dept of Public Health and Environ 1999

Table 8-1. Regulations and Guidelines Applicable to Americium *(continued)*

Agency	Description	Information	Reference
STATE *(cont.)*			
New Jersey	Maximum permissible average concentrations in water		BNA 2001
	Occupational (40-hour week)		
	^{241}Am	S $1x10^{-4}$ µCi/mL	
		I $8x10^{-4}$ µCi/mL	
	^{242m}Am	S $1x10^{-4}$ µCi/mL	
		I $3x10^{-3}$ µCi/mL	
	^{242}Am	S $4x10^{-3}$ µCi/mL	
		I $4x10^{-3}$ µCi/mL	
	^{243}Am	S $1x10^{-4}$ µCi/mL	
		I $8x10^{-4}$ µCi/mL	
	^{244}Am	S $1x10^{-1}$ µCi/mL	
		I $1x10^{-1}$ µCi/mL	
	Non-occupational		
	^{241}Am	S $4x10^{-6}$ µCi/mL	
		I $2x10^{-5}$ µCi/mL	
	^{242m}Am	S $4x10^{-6}$ µCi/mL	
		I $9x10^{-5}$ µCi/mL	
	^{242}Am	S $1x10^{-4}$ µCi/mL	
		I $1x10^{-4}$ µCi/mL	
	^{243}Am	S $4x10^{-6}$ µCi/mL	
		I $3x10^{-5}$ µCi/mL	
	^{244}Am	S $5x10^{-3}$ µCi/mL	
		I $5x10^{-3}$ µCi/mL	
c. Food		No data	
d. Other			
Arkansas	Determination of A_1 and A_2 quantities		BNA 2001
	^{241}Am		
	A1	8 Ci	
	A2	0.008 Ci	
	Specific gravity	3.2 Ci/g	
	^{243}Am		
	A1	8 Ci	
	A2	0.008 Ci	
	Specific gravity	$1.9x10^{-1}$ Ci/g	
	Standards for protection against radiation—^{241}Am	0.01 µCi	BNA 2001
California	Ionizing radiation measuring instruments containing, for purposes of internal calibration or standardization, one or more source of radioactive material—^{241}Am is considered an exempt quantity	<0.05 µCi	BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Americium *(continued)*

Agency	Description	Information	Reference
STATE *(cont.)*			
Colorado	Determination of A_1 and A_2		BNA 2001
	^{241}Am, ^{242m}Am, and ^{243}Am		
	A1	54.1 Ci	
	A2	5.41×10^{-3} Ci	
Florida	Quantity of radioactive material requiring need for an emergency plan for responding to a release—^{241}Am, ^{242}Am, and ^{243}Am		BNA 2001
	Release fraction	0.001%	
	Quantity	2 Ci	
Georgia	Packages transported between locations within the U.S. which contain only Am or Pu in special form with an aggregate radioactivity	not to exceed 20 Ci	BNA 2001
Kansas	Determination of A_1 and A_2 quantities		BNA 2001
	^{241}Am		
	A1	8 Ci	
	A2	0.008 Ci	
	Specific gravity	3.2 Ci/g	
	^{243}Am		
	A1	8 Ci	
	A2	0.008 Ci	
	Specific gravity	1.9×10^{-1} Ci/g	
Mississippi	Packages transported between locations within the U.S. which contain only Am or Pu in special form with an aggregate radioactivity	not to exceed 20 Ci	BNA 2001
Nevada	Quantities of radioactive material for signs, labels, and signals	0.01 μCi	BNA 2001

[a]Release limit per 1,000 metric tons of heavy metal (MTHM) or other unit of waste.
[b]Radionuclide slope factors are calculated by EPA's Office of Radiation and Indoor Air (ORIA) to assist HEAST users with risk-related evaluations and decision-making at various stages of the remediation process. Ingestion and inhalation slope factors are central estimates in a linear model of the age-averaged, lifetime attributable radiation cancer incidence (fatal and nonfatal cancer) risk per unit of activity inhaled or ingested, expressed as risk/picocurie (pCi). External exposure slope factors are central estimates of the lifetime attributable radiation cancer incidence risk for each year of exposure to external radiation from photon-emitting radionuclides distributed uniformly in a thick layer of soil, and are expressed as risk/year per pCi/gram of soil.

ACGIH = American Conference of Governmental Industrial Hygienists; ALI = annual limits on intake; BNA = Bureau of National Affairs; CFR = Code of Federal Regulations; DAC = derived air concentration; DOE = Department of Energy; DOT = Department of Transportation; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; I = insoluble; IARC = International Agency for Research on Cancer; mSv = millisievert; NIOSH = National Institute for Occupational Safety and Health; NRC = Nuclear Regulatory Commission; OSHA = Occupational Safety and Health Administration; PAGs = protective action goals; PEL = permissible exposure limit; S = soluble; REL = recommended exposure limit; TWA = time-weighted average

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10. GLOSSARY

Absorbed Dose, Chemical—The amount of a substance that is either absorbed into the body or placed in contact with the skin. For oral or inhalation routes, this is normally the product of the intake quantity and the uptake fraction divided by the body weight and, if appropriate, the time, expressed as mg/kg for a single intake or mg/kg/day for multiple intakes. For dermal exposure, this is the amount of material applied to the skin, and is normally divided by the body mass and expressed as mg/kg.

Absorbed Dose, Radiation—The mean energy imparted to the irradiated medium, per unit mass, by ionizing radiation. Units: rad (rad), gray (Gy).

Absorbed Fraction—A term used in internal dosimetry. It is that fraction of the photon energy (emitted within a specified volume of material) which is absorbed by the volume. The absorbed fraction depends on the source distribution, the photon energy, and the size, shape and composition of the volume.

Absorption—The process by which a chemical penetrates the exchange boundaries of an organism after contact, or the process by which radiation imparts some or all of its energy to any material through which it passes.

Absorption Coefficient—Fractional absorption of the energy of an unscattered beam of x- or gamma-radiation per unit thickness (linear absorption coefficient), per unit mass (mass absorption coefficient), or per atom (atomic absorption coefficient) of absorber, due to transfer of energy to the absorber. The total absorption coefficient is the sum of individual energy absorption processes (see Compton Effect, Photoelectric Effect, and Pair Production).

Absorption Coefficient, Linear—A factor expressing the fraction of a beam of x- or gamma radiation absorbed in a unit thickness of material. In the expression $I=I_o e^{-\mu x}$, I_o is the initial intensity, I the intensity of the beam after passage through a thickness of the material x, and μ is the linear absorption coefficient.

Absorption Coefficient, Mass—The linear absorption coefficient per cm divided by the density of the absorber in grams per cubic centimeter. It is frequently expressed as μ/ρ, where μ is the linear absorption coefficient and ρ the absorber density.

Absorption Ratio, Differential—Ratio of concentration of a nuclide in a given organ or tissue to the concentration that would be obtained if the same administered quantity of this nuclide were uniformly distributed throughout the body.

Activation—The process of making a material radioactive by bombardment with neutrons or protons.

Activity—The number of radioactive nuclear transformations occurring in a material per unit time (see Curie, Becquerel). The term for activity per unit mass is specific activity.

Activity Median Aerodynamic Diameter (AMAD)—The diameter of a unit-density sphere with the same terminal settling velocity in air as that of the aerosol particle whose activity is the median for the entire size distribution of the aerosol.

Acute Exposure, Chemical—Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Acute Exposure, Radiation—The absorption of a relatively large amount of radiation (or intake of a radioactive material) over a short period of time.

Acute Radiation Syndrome—The symptoms which taken together characterize a person suffering from the effects of intense radiation. The effects occur within hours or days.

Ad libitum—Available in excess and freely accessible.

Adsorption Coefficient (K_{oc})—The ratio of the amount of a chemical adsorbed per unit surface area or per unit weight of organic carbon of a specific particle size in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d)—See Distribution Coefficient

Alpha Particle—A positively charged particle ejected spontaneously from the nuclei of some radioactive elements. It is identical to a helium nucleus, i.e., 2 neutrons and two protons, with a mass number of 4 and an electrostatic charge of +2.

Alpha Track—The track of ionized atoms (pattern of ionization) left in a medium by an alpha particle that has traveled through the medium.

Annihilation (Positron-Electron)—An interaction between a positive and a negative electron in which they both disappear; their rest mass, being converted into electromagnetic radiation (called annihilation radiation) with two 0.51 MeV gamma photons emitted at an angle of 180E to each other.

Atom—The smallest particle of an element that cannot be divided or broken up by chemical means. It consists of a central core called the *nucleus*, which contains *protons* and *neutrons* and an outer shell of *electrons*.

Atomic Mass (u)—The mass of a neutral atom of a nuclide, usually expressed in terms of "atomic mass units." The "atomic mass unit" is one-twelfth the mass of one neutral atom of carbon-12; equivalent to 1.6604×10^{-24} g.

Atomic Number—The number of protons in the nucleus of an atom. The "effective atomic number" is calculated from the composition and atomic numbers of a compound or mixture. An element of this atomic number would interact with photons in the same way as the compound or mixture. (Symbol: Z).

Atomic Mass Number—See Mass Number.

Atomic Weight—The weighted mean of the masses of the neutral isotopes of an element expressed in atomic mass units.

Attenuation—A process by which a beam from a source of radiation is reduced in intensity by absorption and scattering when passing through some material.

Attenuation Coefficient—The fractional reduction in the intensity of a beam of radiation as it passes through an absorbing medium. It may be expressed as reduction per unit distance, per unit mass thickness, or per atom, and is called the linear, mass, or atomic attenuation coefficient, respectively.

Auger Effect—The emission of an electron from the extranuclear portion of an excited atom when the atom undergoes a transition to a less excited state.

Background Radiation—The amount of radiation to which a member of the general population is exposed from natural sources, such as terrestrial radiation from naturally occurring radionuclides in the soil, cosmic radiation originating from outer space, and naturally occurring radionuclides deposited in the human body.

Becquerel (Bq)—International System of Units unit of activity and equals that quantity of radioactive material in which one transformation (disintegration) occurs per second (see Units).

Beta Particle—An electron that is emitted from the nucleus of an atom during one type of radioactive transformation. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 or -1. Beta particles with +1 charges are called positrons (symbolized β^+), and beta particles with -1 charges are called negatrons (symbolized β^-).

Biological Half-time—The time required for a biological system, such as that of a human, to eliminate by natural process half of the amount of a substance (such as a chemical substance, either stable or radioactive) that has entered it.

Bioconcentration Factor (BCF)—The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biologic Effectiveness of Radiation—See Relative Biological Effectiveness.

Biomarkers—Broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Body Burden, Chemical—The total amount of a chemical found in an animal or human body.

Body Burden, Radioactivity—The amount of radioactive material found in an animal or human body.

Bone Seeker—Any compound or ion which migrates in the body and preferentially deposits into bone.

Branching—The occurrence of two or more modes by which a radionuclide can undergo radioactive decay. For example, ^{214}Bi can undergo alpha or beta minus decay, ^{64}Cu can undergo beta minus, beta plus, or electron capture decay. An individual atom of a nuclide exhibiting branching disintegrates by one mode only. The fraction disintegrating by a particular mode is the "branching fraction" for that mode. The "branching ratio" is the ratio of two specified branching fractions (also called multiple disintegration).

Bremsstrahlung—X rays that are produced when a charged particle accelerates (speeds up, slows down, or changes direction) in the strong field of a nucleus.

Buildup Factor—The ratio of the radiation intensity, including both primary and scattered radiation, to the intensity of the primary (unscattered) radiation.

Cancer Effect Level (CEL)—The lowest dose of chemical or radiation in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Capture, Electron—A mode of radioactive decay involving the capture of an orbital electron by its nucleus. Capture from a particular electron shell, e.g., K or L shells, is designated as "K-electron capture" or "L-electron capture."

Capture, K-Electron—Electron capture from the K shell by the nucleus of the atom. Also loosely used to designate any orbital electron capture process.

Carcinogen—A chemical or radiation that is capable of inducing cancer.

Carcinoma—Malignant neoplasm composed of epithelial cells, regardless of their derivation.

Case-Control Study—A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report—Describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Cataract—A clouding of the crystalline lens of the eye which obstructs the passage of light.

Ceiling Value—A concentration of a substance that should not be exceeded, even temporarily.

Charged Particle—A nuclear particle, atom, or molecule carrying a positive or negative charge.

Chronic Exposure—Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study—A type of epidemiological study of a specific group or groups of people who have had a common insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Collective dose—The sum of the individual doses received in a given period of time by a specified population from exposure to a specified source of radiation. Collective dose is expressed in units such as man-rem and person-sievert.

Compton Effect—An attenuation process observed for x- or gamma radiation in which an incident photon interacts with an orbital electron of an atom to produce a recoil electron and a scattered photon whose energy is less than the incident photon.

Containment—The confinement of a chemical or radioactive substance in such a way that it is prevented from being dispersed from its container or into the environment, or is released only at a specified rate.

Contamination—Deposition of a stable or radioactive substance in any place where it is not desired.

Cosmic Rays—High-energy particulate and electromagnetic radiations which originate outside the earth's atmosphere.

Count (Radiation Measurements)—The external indication of a radiation-measuring device designed to enumerate ionizing events. It refers to a single detected event. The term "count rate" refers to the total number registered in a given period of time. The term is sometimes erroneously used to designate a disintegration, ionizing event, or voltage pulse.

Counter, Gas-flow Proportional (GPC)—β-particles are detected by ionization of the counter gas which results in an electrical impulse at an anode wire. If a sufficient amount of radiostrontium is present and the ionization efficiency is calibrated, the quantity of radiostrontium can be determined.

Counter, Geiger-Mueller (GM counter)—Highly sensitive, gas-filled radiation-measuring device to detect (count) individual photons or particulate radiation.

Counter, Scintillation—The combination of phosphor, photomultiplier tube, and associated circuits for counting light emissions produced in the phosphors by ionizing radiation. Scintillation counters generally are more sensitive than GM counters for gamma radiation.

Counting, Cerenkov—Relatively energetic β-particles pass through a transparent medium of high refractive index and a highly-directional, bluish-white light ("Cerenkov" light) is emitted. This light is detected using liquid scintillation counting equipment.

Cross-sectional Study—A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Curie (Ci)—A unit of radioactivity. One curie equals that quantity of radioactive material in which there are 3.7×10^{10} nuclear transformations per second. The activity of 1 gram of radium is approximately 1 Ci.

> **Attocurie (aCi)**—One-thousandth of a femtocurie (3.7×10^{-8} disintegrations per second).
> **Femtocurie (fCi)**—One-billionth of a microcurie (3.7×10^{-5} disintegrations per second).
> **Megacurie (MCi)**—One million curies (3.7×10^{16} disintegrations per sec).
> **Microcurie (μCi)**—One-millionth of a curie (3.7×10^{4} disintegrations per sec).
> **Millicurie (mCi)**—One-thousandth of a curie (3.7×10^{7} disintegrations per sec).
> **Nanocurie (nCi)**—One-billionth of a curie (3.7×10^{1} disintegrations per sec).
> **Picocurie (pCi)**—One-millionth of a microcurie (3.7×10^{-2} disintegrations per second.

Daughter Products—See Progeny and Decay Product

Decay, Radioactive—Transformation of the nucleus of an unstable nuclide by spontaneous emission of charged particles and/or photons (see Disintegration).

Decay Chain or Decay Series—A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus, the parent, decays into a daughter or progeny nucleus that differs from the first by whatever particles were emitted during the decay. If further decays take place, the subsequent nuclei are also usually called daughters or progeny. Sometimes, to distinguish the sequence, the daughter of the first daughter is called the granddaughter, etc.

Decay Constant (λ)—The fraction of the number of atoms of a radioactive nuclide which decay in unit time (see Disintegration Constant).

Decay Product, Daughter Product, Progeny—A new nuclide formed as a result of radioactive decay. A nuclide resulting from the radioactive transformation of a radionuclide, formed either directly or as the result of successive transformations in a radioactive series. A decay product (daughter product or progeny) may be either radioactive or stable.

Delta Ray—Electron removed from an atom during the process of ionization (also called secondary electron). Delta rays cause a track of ionizations along their path.

Depleted uranium (DU)—Uranium having a percentage of ^{235}U smaller than the 0.7% found in natural uranium. It is obtained as a by-product from ^{235}U enrichment.

Developmental Toxicity—The occurrence of adverse effects on the developing organism that may result from exposure to a chemical or radiation prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Disintegration Constant—Synonymous with decay constant. The fraction of the number of atoms of a radioactive material that decays per unit time (see Decay Constant.)

Disintegration, Nuclear—A spontaneous nuclear transformation (radioactivity) characterized by the emission of energy and/or mass from the nucleus. When large numbers of nuclei are involved, the process is characterized by a definite half-life (see Transformation, Nuclear).

Distribution coefficient (K_d)—Describes the distribution of a chemical between the solid and aqueous phase at thermodynamic equilibrium, is given as follows:

$$K_d = \frac{[C]_s}{[C]_w}\ , \text{ Units = (L solution)/(kg solid)},$$

where $[C]_s$ is the concentration of the chemical associated with the solid phase in units of (mg)/(kg solid), and $[C]_w$ is the concentration of the chemical in the aqueous phase in units of (mg)/(L solution).
As the magnitude of K_d decreases, the potential mobility of the chemical to groundwater systems increases and vice versa .

Dose—A general term denoting the quantity of a substance, radiation, or energy absorbed. For special purposes it must be appropriately qualified. If unqualified, it refers to radiation absorbed dose.

> **Absorbed Dose**—The energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad equals 100 ergs per gram. In SI units, the absorbed dose is the gray which is 1 J/kg (see Rad).
>
> **Cumulative Dose (Radiation)**—The total dose resulting from repeated or continuous exposures to radiation.
>
> **Dose Assessment**—An estimate of the radiation dose to an individual or a population group usually by means of predictive modeling techniques, sometimes supplemented by the results of measurement.
>
> **Dose Equivalent (DE)**—A quantity used in radiation safety practice to account for the relative biological effectiveness of the several types of radiation. It expresses all radiations on a common scale for calculating the effective absorbed dose. It is defined as the product of the absorbed dose in

rad and certain modifying factors. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

Dose, Radiation—The amount of energy imparted to matter by ionizing radiation per unit mass of the matter, usually expressed as the unit rad, or in SI units, the gray. 100 rad ' 1 gray (Gy) (see Absorbed Dose).

Maximum Permissible Dose Equivalent (MPD)—The greatest dose equivalent that a person or specified part thereof shall be allowed to receive in a given period of time.

Median Lethal Dose (MLD)—Dose of radiation required to kill, within a specified period (usually 30 days), 50% of the individuals in a large group of animals or organisms. Also called the LD_{50}, or $LD_{50/30}$ if for 30 days..

Threshold Dose—The minimum absorbed dose that will produce a detectable degree of any given effect.

Tissue Dose—Absorbed dose received by tissue in the region of interest, expressed in rad (see Dose, Gray, and Rad).

Dose, Fractionation—A method of administering therapeutic radiation in which relatively small doses are given daily or at longer intervals.

Dose, Protraction—A method of administering therapeutic radiation by delivering it continuously over a relatively long period at a low dose rate.

Dose Rate—Absorbed dose delivered per unit time.

Dose-Response Relationship—The quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Dosimetry—Quantification of radiation doses to cells, tissues, organs, individuals or populations resulting from specified exposures.

Early Effects (of radiation exposure)—Effects that appear within 60 days of an acute exposure.

Electron—A stable elementary particle having an electric charge equal to $\pm 1.60210 \times 10^{-19}$ C (Coulombs) and a rest mass equal to 9.1091×10^{-31} kg. A positron is a positively charged "electron" (see Positron).

Electron Volt—A unit of energy equivalent to the energy gained by an electron in passing through a potential difference of one volt. Larger multiple units of the electron volt are frequently used: keV for thousand or kilo electron volts; MeV for million or mega electron volts (eV). 1 eV=1.6×10^{-12} erg.

Embryotoxicity and Fetotoxicity—Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and *in utero* death.

Energy—Capacity for doing work. "Potential energy" is the energy inherent in a mass because of its spatial relation to other masses. "Kinetic energy" is the energy possessed by a mass because of its motion (SI unit: joules):

Binding Energy (Electron)—The amount of energy that must be expended to remove an electron from an atom.

Binding Energy (Nuclear)—The energy represented by the difference in mass between the sum of the component parts and the actual mass of the nucleus. It represents the amount of energy that must be expended to break a nucleus into its component neutrons and protons.

Excitation Energy—The energy required to change a system from its ground state to an excited state. Each different excited state has a different excitation energy.

Ionizing Energy—The energy required to knock an electron out of an atom. The average energy lost by electrons or beta particles in producing an ion pair in air or in soft tissue is about 34 eV.

Radiant Energy—The energy of electromagnetic radiation, such as radio waves, visible light, x and gamma rays.

Enriched Material—Material in which the relative amount of one or more isotopes of a constituent has been increased.

Enriched Uranium—Uranium in which the abundance of the ^{235}U isotope is increased above normal.

Enrichment, Isotopic—An isotopic separation process by which the relative abundances of the isotopes of a given element are altered, thus producing a form of the element that has been enriched in one or more isotopes and depleted in others. In uranium enrichment, the percentage of uranium-235 in natural uranium can be increased from 0.7% to >90% in a gaseous diffusion process based on the different thermal velocities of the constituents of natural uranium (^{234}U, ^{235}U, ^{238}U) in the molecular form UF_6.

EPA Health Advisory—An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology—Refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Equilibrium, Radioactive—In a radioactive series, the state which prevails when the ratios between the activities of two or more successive members of the series remains constant.

Secular Equilibrium—If a parent element has a very much longer half-life than the daughters (so there is not appreciable change in its amount in the time interval required for later products to attain equilibrium) then, after equilibrium is reached, equal numbers of atoms of all members of the series disintegrate in unit time. This condition is never exactly attained, but is essentially established in such a case as ^{226}Ra and its transformation series to stable ^{206}Pb. The half-life of ^{226}Ra is about 1,600 years; of ^{222}Rn, approximately 3.82 days, and of each of the subsequent members, a few minutes. After about a month, essentially the equilibrium amount of radon is present; then (and for a long time) all members of the series disintegrate the same number of atoms per unit time. At this time, the activity of the daughter is equal to the activity of the parent.

Transient Equilibrium—If the half-life of the parent is short enough so the quantity present decreases appreciably during the period under consideration, but is still longer than that of successive members of the series, a stage of equilibrium will be reached after which all members of

the series decrease in activity exponentially with the period of the parent. At this time, the ratio of the parent activity to the daughter activity is constant.

Equilibrium, Electron—The condition in a radiation field where the energy of the electrons entering a volume equals the energy of the electrons leaving that volume.

Equilibrium Fraction (F)—In radon-radon daughter equilibrium, the parents and daughters have equal radioactivity, that is, as many decay into a specific nuclide as decay out. However, if fresh radon is continually entering a volume of air or if daughters are lost by processes other than radioactive decay, e.g., plate out or migration out of the volume, a disequilibrium develops. The equilibrium fraction is a measure of the degree of equilibrium/disequilibrium. The equilibrium fraction is used to estimate working levels based on measurement of radon only. For radon, 1 working-level concentration is defined at 100 pCi of radon in equilibrium with its 4 successive progeny in 1 liter of air. Thus, 100 pCi/L radon at 50% equilibrium is 0.5 WL.

Excitation—The addition of energy to a system, thereby transferring it from its ground state to an excited state. Excitation of a nucleus, an atom, or a molecule can result from absorption of photons or from inelastic collisions with other particles. The excited state of an atom is an unstable or metastable state and will return to ground state by radiation of the excess energy.

Exposure (Chemical)—Contact of an organism with a chemical or physical agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, gut) and available for absorption.

Exposure (Radiation)—Being exposed to ionizing radiation or to a radioactive material. A measure of the ionization produced in air by x or gamma radiation; the sum of the electric charges on all ions of one sign produced in air when all electrons liberated by photons in a volume of air are completely stopped in air (dQ), divided by the mass of the air in the volume (dm). The unit of exposure in air is the roentgen, or coulomb per kilogram (SI units). One roentgen is equal to 2.58×10^{-4} coulomb per kilogram (C/kg).

Fission, Nuclear—A nuclear transformation characterized by the splitting of a nucleus into at least two other nuclei and several neutrons, and is accompanied by the release of a relatively large amount of energy.

Gamma Ray, Penetrating—Short wavelength electromagnetic radiation of nuclear origin.

Genetic Effect of Radiation—Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells. Genetic effects have not been observed in any human population exposed at any dose level.

Genotoxicity—A specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Gray (Gy)—SI unit of absorbed dose, 1 J/kg. One gray equals 100 rad (see Units).

Half-life, Radioactive—Time required for a radioactive substance to lose 50% of its activity by decay. Each radio-nuclide has a unique physical half-life. Known also as physical half-time and symbolized as T_r or T_{rad}.

Half-life, Effective—See Half-Time, Effective.

Half-time, Biological—Time required for an organ, tissue, or the whole body to eliminate one-half of any absorbed substance by regular processes of elimination. This is the same for both stable and radioactive isotopes of a particular element, and is sometimes referred to as half-time, symbolized as t_{biol} or T_b.

Half-time, Effective—Time required for a radioactive element in an organ, tissue, or the whole body to be diminished 50% as a result of the combined action of radioactive decay and biological elimination, symbolized as T_e or T_{eff}.

$$\textit{Effective Half\&time} = \frac{\textit{Biological half\&time x Radioactive half\&life}}{\textit{Biological half\&time \% Radioactive half\&life}}$$

Immediately Dangerous to Life or Health (IDLH)—The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Immunologic Toxicity—The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

Immunological Effects—Functional changes in the immune response.

In Vitro—Isolated from the living organism and artificially maintained, as in a test tube. Literally, “in glass.”

In Vivo—Occurring within the living organism. Literally, “in life.”

Intensity—Amount of energy per unit time passing through a unit area perpendicular to the line of propagation at the point in question.

Intermediate Exposure—Exposure to a chemical for a duration of 15–364 days, as specified in the Toxicological Profiles.

Internal Conversion—Process in which a gamma ray knocks an electron out of the same atom from which the gamma ray was emitted. The ratio of the number of internal conversion electrons to the number of gamma quanta emitted in the de-excitation of the nucleus is called the "conversion ratio."

Ion—Atomic particle, atom or chemical radical bearing a net electrical charge, either negative or positive.

Ion Pair—Two particles of opposite charge, usually referring to the electron and positive atomic or molecular residue resulting after the interaction of ionizing radiation with the orbital electrons of atoms.

Ionization—The process by which a neutral atom or molecule acquires a positive or negative charge.

> **Primary Ionization**—(1) In collision theory: the ionization produced by the primary particles as contrasted to the "total ionization" which includes the "secondary ionization" produced by delta rays. (2) In counter tubes: the total ionization produced by incident radiation without gas amplification.
>
> **Specific Ionization**—Number of ion pairs per unit length of path of ionizing radiation in a medium; e.g., per centimeter of air or per micrometer of tissue.

Total Ionization—The total electric charge of one sign on the ions produced by radiation in the process of losing its kinetic energy. For a given gas, the total ionization is closely proportional to the initial ionization and is nearly independent of the nature of the ionizing radiation. It is frequently used as a measure of absorption of radiation energy.

Ionization Density—Number of ion pairs per unit volume.

Ionization Path (Track)—The trail of ion pairs produced by an ionizing particle in its passage through matter.

Ionizing Radiation—Any radiation capable of knocking electrons out of atoms and producing ions. Examples: alpha, beta, gamma and x rays, and neutrons.

Isobars—Nuclides having the same mass number but different atomic numbers.

Isomers—Nuclides having the same number of neutrons and protons but capable of existing, for a measurable time, in different quantum states with different energies and radioactive properties. Commonly the isomer of higher energy decays to one with lower energy by the process of isomeric transition.

Isotopes—Nuclides having the same number of protons in their nuclei, and hence the same atomic number, but differing in the number of neutrons, and therefore in the mass number. Identical chemical properties exist in isotopes of a particular element. The term should not be used as a synonym for nuclide because isotopes refer specifically to different nuclei of the same element.

Stable Isotope—A nonradioactive isotope of an element.

Kerma (k)—A measure of the kinetic energy transferred from gamma rays or neutrons to a unit mass of absorbing medium in the initial collision between the radiation and the absorber atoms. The SI unit is J/kg. The special name of this unit is the rad (traditional system of units) or Gray (SI).

Joule—The S.I. unit for work and energy. It is equal to the work done by raising a mass of one newton through a distance of one meter (J = Nm), which corresponds to about 0.7 ft-pound.

Labeled Compound—A compound containing one or more radioactive atoms intentionally added to its structure. By observations of radioactivity or isotopic composition, this compound or its fragments may be followed through physical, chemical, or biological processes.

Late Effects (of radiation exposure)—Effects which appear 60 days or more following an acute exposure.

$LD_{50/30}$—The dose of a chemical or radiation expected to cause 50% mortality in those exposed within 30 days. For radiation, this is about 350 rad (3.5 gray) received by humans over a short period of time.

Lethal Concentration$_{(LO)}$ (LC_{LO})—The lowest concentration of a chemical in air that has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC_{50})—A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population within a specified time, usually 30 days.

Lethal Dose$_{(L0)}$ (LD_{L0})—The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals within a specified time, usually 30 days.

Lethal Dose$_{(50)}$ (LD$_{50}$)—The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT$_{50}$)—A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Linear Energy Transfer (LET)—A measure of the energy that a charged particle transfers to a material per unit path length.

> **Low-LET**—Energy transfer characteristic of light charged particles such as electrons produced by x and gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus.
>
> **High-LET**—Energy transfer characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.
>
> **Average LET**—The energy of a charged particle divided by the length of the path over which it deposits all its energy in a material.

Lowest-Observed-Adverse-Effect Level (LOAEL)—The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lung Clearance Class (fast, F; medium, M; slow, S)—A classification scheme for inhaled material according to its rate of clearance from the pulmonary region of the lungs to the blood and the gastrointestinal tract.

Lymphoreticular Effects—Represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations—Permanent structural changes that may adversely affect survival, development, or function.

Mass Numbers (A)—The number of nucleons (protons and neutrons) in the nucleus of an atom.

Minimal Risk Level—An estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Morbidity—State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mutagen—A substance that causes changes (mutations) in the genetic material in a cell. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy—The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity—The occurrence of adverse effects on the nervous system following exposure to a substance.

Neutrino (v)—A neutral particle of infinitesimally small rest mass emitted during beta plus or beta minus decay. This particle accounts for conservation of energy in beta plus and beta minus decays. It plays no role in damage from radiation.

No-Observed-Adverse-Effect Level (NOAEL)—The dose of a substance at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Nuclear Reactor—A power plant that heats water by using nuclear reactions instead of burning coal, oil, or natural gas. All of these sources of energy simply heat water and use the steam which is produced to turn turbines that make electricity or propel a ship.

Nucleon—Common name for a constituent particle of the nucleus. Applied to a proton or neutron.

Nuclide—A species of atom characterized by the constitution of its nucleus. The nuclear constitution is specified by the number of protons (Z), number of neutrons (N), and energy content; or, alternatively, by the atomic number (Z), mass number A ' (N+Z), and atomic mass. To be regarded as a distinct nuclide, the atom must be capable of existing for a measurable time. Thus, nuclear isomers are separate nuclides, whereas promptly decaying excited nuclear states and unstable intermediates in nuclear reactions are not so considered.

Octanol-Water Partition Coefficient (K_{ow})—The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Odds Ratio (OR)—A means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Pair Production—An absorption process for x- and gamma radiation in which the incident photon is absorbed in the vicinity of the nucleus of the absorbing atom, with subsequent production of an electron and positron pair (see annihilation). This reaction can only occur for incident photon energies exceeding 1.02 MeV.

Parent—A radionuclide which, upon disintegration, yields a new nuclide, either directly or as a later member of a radioactive series.

Permissible Exposure Limit (PEL)—A maximum allowable atmospheric level of a substance in workplace air averaged over an 8-hour shift.

Pharmacokinetics—The science of quantitatively predicting the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Pharmacokinetic Model—A set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based

and physiologically-based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereas the physiologically-based model compartments represent real anatomic regions of the body.

Physiologically Based Pharmacodynamic (PBPD) Model—A type of physiologically-based dose-response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model—A model comprising a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also utilize biochemical information such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Photon—A quantum of electromagnetic energy (E) whose value is the product of its frequency (ν) in hertz and Planck's constant (h). The equation is: $E = h\nu$.

Photoelectric Effect—An attenuation process observed for x and gamma radiation in which an incident photon interacts with a tightly bound inner orbital electron of an atom delivering all of its energy to knock the electron out of the atom. The incident photon disappears in the process.

Positron—A positively charged electron.

Potential, Ionization—The energy expressed as electron volts (eV) necessary to separate one electron from an atom, resulting in the formation of an ion pair.

Power, Stopping—A measure of the ability of a material to absorb energy from an ionizing particle passing through it; the greater the stopping power, the greater the energy absorbing ability (see Linear Energy Transfer).

Progeny—The decay product or products resulting after a radioactive decay or a series of radioactive decays. The progeny can also be radioactive, and the chain continues until a stable nuclide is formed.

Proton—Elementary nuclear particle with a positive electric charge equal numerically to the charge of the electron and a rest mass of 1.007 mass units.

Quality—A term describing the distribution of the energy deposited by a particle along its track; radiations that produce different densities of ionization per unit intensity are said to have different "qualities."

Quality Factor (Q)—The linear-energy-transfer-dependent factor by which absorbed doses are multiplied to obtain (for radiation protection purposes) a quantity that expresses - on a common scale for all ionizing radiation - the approximate biological effectiveness of the absorbed dose.

Rad—The unit of absorbed dose equal to 100 ergs per gram, or 0.01 joule per kilogram (0.01 Gy) in any medium (see Absorbed Dose).

Radiation—The emission and propagation of energy through space or through a material medium in the form of waves (e.g., the emission and propagation of electromagnetic waves, or of sound and elastic waves). The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation commonly is classified according to frequency, as microwaves, infrared, visible (light), ultraviolet, and x and gamma rays (see Photon.) and, by extension, corpuscular emission, such as alpha and beta radiation, neutrons, or rays of mixed or unknown type, as cosmic radiation.

Radiation, Annihilation—Photons produced when an electron and a positron unite and cease to exist. The annihilation of a positron-electron pair results in the production of two photons, each of 0.51 MeV energy.

Radiation, Background—See Background Radiation.

Radiation, Characteristic (Discrete)—Radiation originating from an excited atom after removal of an electron from an atom. The wavelength of the emitted radiation is specific, depending only on the element and particular energy levels involved.

Radiation, External—Radiation from a source outside the body.

Radiation, Internal—Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

Radiation, Ionizing—Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter (see Radiation).

Radiation, Monoenergetic—Radiation of a given type in which all particles or photons originate with and have the same energy.

Radiation, Scattered—Radiation which during its passage through a substance, has been deviated in direction. It may also have been modified by a decrease in energy.

Radiation, Secondary—A particle or ray that is produced when the primary radiation interacts with a material, and which has sufficient energy to produce its own ionization, such as bremsstrahlung or electrons knocked from atomic orbitals with enough energy to then produce ionization (see Delta Rays).

Radioactive Material—Material containing radioactive atoms.

Radioactivity—Spontaneous nuclear transformations that result in the formation of new elements. These transformations are accomplished by emission of alpha or beta particles from the nucleus or by the capture of an orbital electron. Each of these reactions may or may not be accompanied by a gamma photon.

Radioactivity, Artificial—Man-made radioactivity produced by particle bombardment or nuclear fission, as opposed to naturally occurring radioactivity.

Radioactivity, Induced—Radioactivity produced in a substance after bombardment with neutrons or other particles. The resulting activity is "natural radioactivity" if formed by nuclear reactions occurring in nature and "artificial radioactivity" if the reactions are caused by man.

Radioactivity, Natural—The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

Radioisotope—An unstable or radioactive isotope of an element that decays or disintegrates spontaneously, emitting radiation. Approximately 5,000 natural and artificial radioisotopes have been identified.

Radionuclide—Any radioactive isotope of any element.

Radiosensitivity—Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are currently used in a comparative sense, rather than in an absolute one.

Reference Dose (RfD)—An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to non-threshold effects such as cancer.

Relative Biological Effectiveness (RBE)—The RBE is a factor used to compare the biological effectiveness of absorbed radiation doses (i.e., rad) due to different types of ionizing radiation. More specifically, it is the experimentally determined ratio of an absorbed dose of a radiation in question to the absorbed dose of a reference radiation (typically ^{60}Co gamma rays or 200 keV x rays) required to produce an identical biological effect in a particular experimental organism or tissue (see Quality Factor).

Rem—A unit of dose equivalent that is used in the regulatory, administrative, and engineering design aspects of radiation safety practice. The dose equivalent in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor (1 rem is equal to 0.01 sievert).

Rep—Roentgen equivalent, physical: A former unit of ionizing radiation equivalent to the dosage of 93 ergs absorbed per gram of tissue (93 erg/gm=0.93 rad).

Reportable Quantity (RQ)—The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity—The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Roentgen (R)—A unit of exposure (in air) to ionizing radiation. It is the amount of x or gamma rays required to produce ions carrying 1 electrostatic unit of electrical charge in 1 cubic centimeter of dry air under standard conditions. Named after William Roentgen, a German scientist who discovered x rays in 1895.

Retrospective Study—A type of cohort study based on a group of persons known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to causal factors that can be ascertained from existing records and/or examining survivors of the cohort.

Self-Absorption—Absorption of radiation (emitted by radioactive atoms) by the material in which the atoms are located; in particular, the absorption of radiation within a sample being assayed.

Short-Term Exposure Limit (STEL)—The maximum concentration to which workers can be exposed for up to 15 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily TLV-TWA may not be exceeded.

SI Units—The International System of Units as defined by the General Conference of Weights and Measures in 1960. These units are generally based on the meter/kilogram/second units, with special quantities for radiation including the becquerel, gray, and sievert.

Sickness, Acute Radiation (Syndrome)—The complex symptoms and signs characterizing the condition resulting from excessive exposure of the whole body (or large part) to ionizing radiation. The earliest of these symptoms are nausea, fatigue, vomiting, and diarrhea, and may be followed by loss of hair (epilation), hemorrhage, inflammation of the mouth and throat, and general loss of energy. In severe cases, where the radiation dose is relatively high (over several hundred rad or several gray), death may occur within two to four weeks. Those who survive six weeks after exposure of a single high dose of radiation may generally be expected to recover.

Sievert (Sv)—The SI unit of any of the quantities expressed as dose equivalent. The dose equivalent in sieverts is equal to the absorbed dose, in gray, multiplied by the quality factor (1 sievert equals 100 rem).

Specific-activity—Radioactivity per unit mass of material containing a radionuclide, expressed, for example, as Ci/gram or Bq/gram.

Specific Energy—The actual energy per unit mass deposited per unit volume in a small target, such as the cell or cell nucleus, as the result of one or more energy-depositing events. This is a stochastic quantity as opposed to the average value over a large number of instance (i.e., the absorbed dose).

Standard Mortality Ratio (SMR)—A ratio of the observed number of deaths and the expected number of deaths in a specific standard population.

Stopping Power—The average rate of energy loss of a charged particle per unit thickness of a material or per unit mass of material traversed.

Surface-seeking Radionuclide—A bone-seeking internal emitter that is deposited and remains on the bone surface for a long period of time, although it may eventually diffuse into the bone mineral. This contrasts with a volume seeker, which deposits more uniformly throughout the bone volume.

Target Organ Toxicity—This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Target Theory (Hit Theory)—A theory explaining some biological effects of radiation on the basis that ionization, occurring in a discrete volume (the target) within the cell, directly causes a lesion which subsequently results in a physiological response to the damage at that location. One, two, or more "hits" (ionizing events within the target) may be necessary to elicit the response.

Teratogen—A chemical that causes birth defects.

Threshold Limit Value (TLV)—The maximum concentration of a substance to which most workers can be exposed without adverse effect. TLV is a term used exclusively by the ACGIH. Other terms used to express the same concept are the MAC (Maximum Allowable Concentration) and PEL (Permissible Exposure Limits).

Time-Weighted Average (TWA)—An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose (TD_{50})—A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic—The study of the absorption, distribution and elimination of toxic compounds in the living organism.

Toxicosis —A diseased condition resulting from poisoning.

Transformation, Nuclear—The process by which a nuclide is transformed into a different nuclide by absorbing or emitting particulate or electromagnetic radiation.

Transition, Isomeric—The process by which a nuclide decays to an isomeric nuclide (i.e., one of the same mass number and atomic number) of lower quantum energy. Isomeric transitions (often abbreviated I.T.) proceed by gamma ray and/or internal conversion electron emission.

Tritium—The hydrogen isotope with one proton and two neutrons in the nucleus (Symbol: ^{3}H). It is radioactive and has a physical half-life of 12.3 years.

Unattached Fraction—That fraction of the radon daughters, usually ^{218}Po and ^{214}Po, which has not yet attached to a dust particle or to water vapor. As a free atom, it has a high probability of being exhaled and not retained within the lung. It is the attached fraction which is primarily retained.

Uncertainty Factor (UF)—A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

Units, Radiological—

Units	Equivalents
Becquerel* (Bq)	1 disintegration per second = 2.7×10^{-11} Ci
Curie (Ci)	3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq
Gray* (Gy)	1 J/kg = 100 rad
Rad (rad)	100 erg/g = 0.01 Gy
Rem (rem)	0.01 sievert
Sievert* (Sv)	100 rem

*International Units, designated (SI)

Working Level (WL)—Any combination of short-lived radon daughters in 1 liter of air that will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy.

Working Level Month (WLM)—A unit of exposure to radon daughters corresponding to the product of the radon daughter concentration in Working Level (WL) and the exposure time in nominal months (1 nominal month = 170 hours). Inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours results in an exposure of 1 WLM.

X rays—Penetrating electromagnetic radiations whose wave lengths are very much shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. X rays (called characteristic x rays) are also produced when an orbital electron falls from a high energy level to a low energy level.

Zero-Threshold Linear Hypothesis—The assumption that a dose-response curve derived from data in the high dose and high dose-rate ranges may be extrapolated through the low dose and low dose range to zero, implying that, theoretically, any amount of radiation will cause some damage.

APPENDIX A
ATSDR MINIMAL RISK LEVEL AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as a hundredfold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agencywide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, Mailstop E-29, Atlanta, Georgia 30333.

APPENDIX B

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Relevance to Public Health

This chapter provides an overview of the nature, manufacture, uses, general population exposures, and health effects of the substance under review. This overview is followed by a discussion of the most critical health effects. The discussion of health effects is based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The chapter covers end points in the same order they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this chapter. If data are located in the scientific literature, a table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal risk levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Chapter 3 Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, we have derived minimal risk levels (MRLs) for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not meant to support regulatory action; but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2, "Relevance to Public Health," contains basic information known about the substance. Other sections such as Chapter 3 Section 3.9, "Interactions with Other Substances," and Section 3.10, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses for lifetime exposure (RfDs).

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (3-1, 3-2, and 3-3) and figures (3-1 and 3-2) are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, minimal risk levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse- Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (LOAELs), or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 3-1 and Figure 3-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 3-1

(1) Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not therefore have all five of the tables and figures. In the case of radionuclide profiles, there are separate LSE tables, by route of exposure, for toxicity of stable isotopes and radioactive isotopes; for radioactive isotopes, there may be an LSE table for a fourth route, external exposure.

(2) Exposure Period Three exposure periods - acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more) are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3) Health Effect The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4) Key to Figure Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the 2 "18r" data points in Figure 3-1).

(5) Species The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.4, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6) Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to 1,1,2,2-tetrachloroethane via inhalation for 6 hours per day, 5 days per week, for 3 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.

(7) System This column further defines the systemic effects. These systems include: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, 1 systemic effect (respiratory) was investigated.

(8) NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "b").

(9) LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(10) Reference The complete reference citation is given in Chapter 9 of the profile.

(11) CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(12) Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Figure 3-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure concentrations for particular exposure periods.

(13) Exposure Period The same exposure periods appear as in the LSE table. In this example, health effects observed within the intermediate and chronic exposure periods are illustrated.

(14) Health Effect These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL In this example, 18r NOAEL is the critical end point for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates to a NOAEL for the test species-rat. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17) CEL Key number 38r is 1 of 3 studies for which Cancer Effect Levels were derived. The diamond symbol refers to a Cancer Effect Level for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

(19) Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 6

Table 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 6 INTERMEDIATE EXPOSURE							
	5	6	7	8	9		10
3 6 Systemic	9	9	9	9	9		9
4 6 18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
CHRONIC EXPOSURE							
						11	
Cancer						9	
38	Rat	18 mo 5 d/wk 7 hr/d				20 (CEL, multiple organs)	Wong et al. 1982
39	Rat	89–104 wk 5 d/wk 6 hr/d				10 (CEL, lung tumors, nasal tumors)	NTP 1982
40	Mouse	79–103 wk 5 d/wk 6 hr/d				10 (CEL, lung tumors, hemangiosarcomas)	NTP 1982

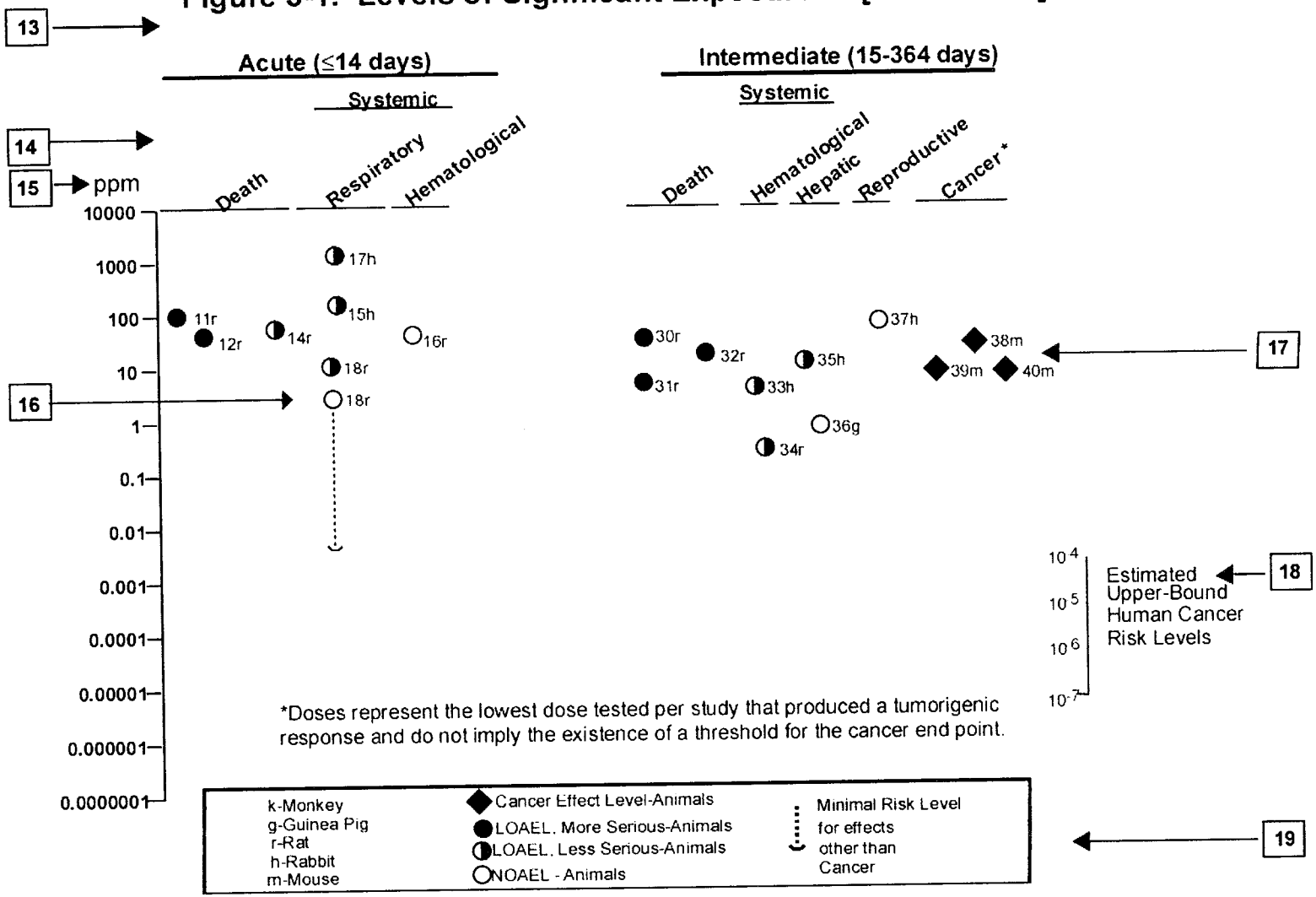
[a] The number corresponds to entries in Figure 3-1.

12 6 [b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5 x 10^{-3} ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

DRAFT FOR PUBLIC COMMENT

SAMPLE

Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation



DRAFT FOR PUBLIC COMMENT

APPENDIX C

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADI	Acceptable Daily Intake
ADME	Absorption, Distribution, Metabolism, and Excretion
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	Best Available Technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BSC	Board of Scientific Counselors
C	Centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	Cancer Effect Level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CNS	central nervous system
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
d	day
Derm	dermal
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense
DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation
DOT/UN/	Department of Transportation/United Nations/
NA/IMCO	North America/International Maritime Dangerous Goods Code
DWEL	Drinking Water Exposure Level
ECD	electron capture detection
ECG/EKG	electrocardiogram
EEG	electroencephalogram

EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
ft	foot
FR	*Federal Register*
g	gram
GC	gas chromatography
Gd	gestational day
gen	generation
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
hr	hour
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
in	inch
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration, low
LC_{50}	lethal concentration, 50% kill
LD_{Lo}	lethal dose, low
LD_{50}	lethal dose, 50% kill
LT_{50}	lethal time, 50% kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
m	meter
MA	*trans,trans*-muconic acid
MAL	Maximum Allowable Level
mCi	millicurie
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
mg	milligram
min	minute
mL	milliliter
mm	millimeter
mm Hg	millimeters of mercury

mmol	millimole
mo	month
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCI	National Cancer Institute
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NFPA	National Fire Protection Association
ng	nanogram
NLM	National Library of Medicine
nm	nanometer
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council; Nuclear Regulatory Commission
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OSHA	Occupational Safety and Health Administration
OSW	Office of Solid Waste, EPA
OTS	Office of Toxic Substances
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA
PAH	Polycyclic Aromatic Hydrocarbon
PBPD	Physiologically Based Pharmacodynamic
PBPK	Physiologically Based Pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
PID	photo ionization detector
pg	picogram
pmol	picomole
PHS	Public Health Service

PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	Pretreatment Standards for New Sources
REL	recommended exposure level/limit
RfC	Reference Concentration
RfD	Reference Dose
RNA	ribonucleic acid
RTECS	Registry of Toxic Effects of Chemical Substances
RQ	Reportable Quantity
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
sec	second
SIC	Standard Industrial Classification
SIM	selected ion monitoring
SMCL	Secondary Maximum Contaminant Level
SMR	standard mortality ratio
SNARL	Suggested No Adverse Response Level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	Total Organic Compound
TPQ	Threshold Planning Quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TRI	Toxics Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
VOC	Volatile Organic Compound
yr	year
WHO	World Health Organization
wk	week
$>$	greater than
$\geq$	greater than or equal to
$=$	equal to
$<$	less than
$\leq$	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
µm	micrometer
µg	microgram
q_1^*	cancer slope factor
–	negative
+	positive

(+) weakly positive result
(–) weakly negative result

APPENDIX D

OVERVIEW OF BASIC RADIATION PHYSICS, CHEMISTRY AND BIOLOGY

Understanding the basic concepts in radiation physics, chemistry, and biology is important to the evaluation and interpretation of radiation-induced adverse health effects and to the derivation of radiation protection principles. This appendix presents a brief overview of the areas of radiation physics, chemistry, and biology and is based to a large extent on the reviews of Mettler and Moseley (1985), Hobbs and McClellan (1986), Eichholz (1982), Hendee (1973), Cember (1996), and Early et al. (1979).

D.1 RADIONUCLIDES AND RADIOACTIVITY

The substances we call elements are composed of atoms. Atoms in turn are made up of neutrons, protons and electrons: neutrons and protons in the nucleus and electrons in a cloud of orbits around the nucleus. Nuclide is the general term referring to any nucleus along with its orbital electrons. The nuclide is characterized by the composition of its nucleus and hence by the number of protons and neutrons in the nucleus. All atoms of an element have the same number of protons (this is given by the atomic number) but may have different numbers of neutrons (this is reflected by the atomic mass numbers or atomic weight of the element). Atoms with different atomic mass but the same atomic numbers are referred to as isotopes of an element.

The numerical combination of protons and neutrons in most nuclides is such that the nucleus is quantum mechanically stable and the atom is said to be stable, i.e., not radioactive; however, if there are too few or too many neutrons, the nucleus is unstable and the atom is said to be radioactive. Unstable nuclides undergo radioactive transformation, a process in which a neutron or proton converts into the other and a beta particle is emitted, or else an alpha particle is emitted. Each type of decay is typically accompanied by the emission of gamma rays. These unstable atoms are called radionuclides; their emissions are called ionizing radiation; and the whole property is called radioactivity. Transformation or decay results in the formation of new nuclides some of which may themselves be radionuclides, while others are stable nuclides. This series of transformations is called the decay chain of the radionuclide. The first radionuclide in the chain is called the parent; the subsequent products of the transformation are called progeny, daughters, or decay products.

In general there are two classifications of radioactivity and radionuclides: natural and artificial (man-made). Naturally-occurring radioactive material (NORM) exists in nature and no additional energy is necessary to place them in an unstable state. Natural radioactivity is the property of some naturally occurring, usually heavy elements, that are heavier than lead. Radionuclides, such as radium and uranium, primarily emit alpha particles. Some lighter elements such as carbon-14 and tritium (hydrogen-3) primarily emit beta particles as they transform to a more stable atom. Natural radioactive atoms heavier than lead cannot attain a stable nucleus heavier than lead. Everyone is exposed to background radiation from naturally-occurring radionuclides throughout life. This background radiation is the major source of radiation exposure to man and arises from several sources. The natural background exposures are frequently used as a standard of comparison for exposures to various artificial sources of ionizing radiation.

Artificial radioactive atoms are produced either as a by-product of fission of uranium or plutonium atoms in a nuclear reactor or by bombarding stable atoms with particles, such as neutrons or protons, directed at the stable atoms with high velocity. These artificially produced radioactive elements usually decay by emission of particles, such as positive or negative beta particles and one or more high energy photons (gamma rays). Unstable (radioactive) atoms of any element can be produced.

Both naturally occurring and artificial radioisotopes find application in medicine, industrial products, and consumer products. Some specific radioisotopes, called fall-out, are still found in the environment as a result of nuclear weapons use or testing.

D.2 RADIOACTIVE DECAY

D.2.1 Principles of Radioactive Decay

The stability of an atom is the result of the balance of the forces of the various components of the nucleus. An atom that is unstable (radionuclide) will release energy (decay) in various ways and transform to stable atoms or to other radioactive species called daughters, often with the release of ionizing radiation. If there are either too many or too few neutrons for a given number of protons, the resulting nucleus may undergo transformation. For some elements, a chain of daughter decay products may be produced until stable atoms are formed. Radionuclides can be characterized by the type and energy of the radiation emitted, the rate of decay, and the mode of decay. The mode of decay indicates how a parent compound undergoes transformation. Radiations considered here are primarily of nuclear origin, i.e., they arise from nuclear excitation, usually caused by the capture of charged or uncharged nucleons by a nucleus, or by the radioactive decay or transformation of an unstable nuclide. The type of radiation may be categorized as charged or uncharged particles, protons, and fission products) or electromagnetic radiation (gamma rays and x rays). Table D-1 summarizes the basic characteristics of the more common types of radiation encountered.

D.2.2 Half-Life and Activity

For any given radionuclide, the rate of decay is a first-order process that is constant, regardless of the radioactive atoms present and is characteristic for each radionuclide. The process of decay is a series of random events; temperature, pressure, or chemical combinations do not effect the rate of decay. While it may not be possible to predict exactly which atom is going to undergo transformation at any given time, it is possible to predict, on average, the fraction of the radioactive atoms that will transform during any interval of time.

The *activity* is a measure of the quantity of radioactive material. For these radioactive materials it is customary to describe the activity as the number of disintegrations (transformations) per unit time. The unit of activity is the curie (Ci), which was originally related to the activity of one gram of radium, but is now defined as that quantity of radioactive material in which there are:

> 1 curie (Ci) = 3.7×10^{10} disintegrations (transformations)/second (dps) or 2.22×10^{12} disintegrations (transformations)/minute (dpm).

The SI unit of activity is the becquerel (Bq); 1 Bq = that quantity of radioactive material in which there is 1 transformation/second. Since activity is proportional to the number of atoms of the radioactive material, the quantity of any radioactive material is usually expressed in curies, regardless of its purity or concentration. The transformation of radioactive nuclei is a random process, and the number of transformations is directly proportional to the number of radioactive atoms present. For any pure radioactive substance, the rate of decay is usually described by its radiological half-life, T_R, i.e., the time it takes for a specified source material to decay to half its initial activity. The specific activity is an indirect measure of the rate of decay, and is defined as the activity per unit mass or per unit volume. The higher the specific activity of a radioisotope, the faster it is decaying.

The activity of a radionuclide at time t may be calculated by:

$$A = A_o e^{-0.693t/Trad}$$

where A is the activity in dps or curies or becquerels, A_o is the activity at time zero, t is the time at which measured, and T_{rad} is the radiological half-life of the radionuclide (T_{rad} and t must be in the same units of time). The time when the activity of a sample of radioactivity becomes one-half its original value is the radioactive half-life and is expressed in any suitable unit of time.

Table D-1. Characteristics of Nuclear Radiations

Radiation	Rest mass[a]	Charge	Typical energy range	Path length[b]		Comments
				Air	Solid	
Alpha (α)	4.00 amu	+2	4–10 MeV	5–10 cm	25–80 µm	Identical to ionized He nucleus
Negatron (β^-)	5.48×10^{-4} amu; 0.51 MeV	–1	0–4 Mev	0–10 m	0–1 cm	Identical to electron
Positron (β^+)	5.48×10^{-4} amu; 0.51 Mev	+1	0-4 Mev	0–10 m	0–1 cm	Identical to electron except for sign of charge
Neutron	1.0086 amu; 939.55 MeV	0	0–15 MeV	b	b	Free half-life: 16 min
X ray (e.m. photon)	–	0	5 keV–100 keV	b	b	Photon from transition of an electron between atomic orbits
Gamma (p) (e.m. photon)	–	0	10 keV–3 MeV	b	b	Photon from nuclear transformation

[a] The rest mass (in amu) has an energy equivalent in MeV that is obtained using the equation $E=mc^2$, where 1 amu = 932 MeV.
[b] Path lengths are not applicable to x- and gamma rays since their intensities decrease exponentially; path lengths in solid tissue are variable, depending on particle energy, electron density of material, and other factors.

amu = atomic mass unit; e.m. = electromagnetic; MeV = MegaElectron Volts

The specific activity is a measure of activity, and is fined as the activity per unit mass or per unit volume. This activity is usually expressed in curies per gram and may be calculated by

$$\text{curies/gram} = 1.3 \times 10^8 / (T_{rad})\ (\text{atomic weight}) \quad \text{or}$$

$$[3.577 \times 10^5 \times \text{mass(g)}] / [T_{rad} \times \text{atomic weight}]$$

where T_{rad} is the radiological half-life in days.

In the case of radioactive materials contained in living organisms, an additional consideration is made for the reduction in observed activity due to regular processes of elimination of the respective chemical or biochemical substance from the organism. This introduces a rate constant called the biological half-life (T_{biol}) which is the time required for biological processes to eliminate one-half of the activity. This time is virtually the same for both stable and radioactive isotopes of any given element.

Under such conditions the time required for a radioactive element to be halved as a result of the combined action of radioactive decay and biological elimination is the effective clearance half-time:

$$T_{eff} = (T_{biol} \times T_{rad}) / (T_{biol} + T_{rad}).$$

Table D-2 presents representative effective half-lives of particular interest.

Table D-2. Half-Lives of Some Radionuclides in Adult Body Organs

		Half-life[a]		
Radionuclide	Critical organ	Physical	Biological	Effective
Uranium-238	Kidney	4,460,000,000 y	4 d	4 d
Hydrogen-3[b] (Tritium)	Whole body	12.3 y	10 d	10 d
Iodine-131	Thyroid	8 d	80 d	7.3 d
Strontium-90	Bone	28 y	50 y	18 y
Plutonium-239	Bone surface	24,400 y	50 y	50 y
	Lung	24,400 y	500 d	500 d
Cobalt-60	Whole body	5.3 y	99.5 d	95 d
Iron-55	Spleen	2.7 y	600 d	388 d
Iron-59	Spleen	45.1 d	600 d	42 d
Manganese-54	Liver	303 d	25 d	23 d
Cesium-137	Whole body	30 y	70 d	70 d

[a]d = days, y = years
[b]Mixed in body water as tritiated water

D.2.3 Interaction of Radiation with Matter

Both ionizing and nonionizing radiation will interact with materials; that is, radiation will lose kinetic energy to any solid, liquid or gas through which it passes by a variety of mechanisms. The transfer of energy to a medium by either electromagnetic or particulate radiation may be sufficient to cause formation of ions. This process is called ionization. Compared to other types of radiation that may be absorbed, such as ultraviolet radiation, ionizing radiation deposits a relatively large amount of energy into a small volume.

The method by which incident radiation interacts with the medium to cause ionization may be direct or indirect. Electromagnetic radiations (x rays and gamma photons) are indirectly ionizing; that is, they give up their energy in various interactions with cellular molecules, and the energy is then utilized to produce a fast-moving charged particle such as an electron. It is the electron that then may react with a target molecule. This particle is called a “primary ionizing particle. Charged particles, in contrast, strike the tissue or medium and directly react with target molecules, such as oxygen or water. These particulate radiations are directly ionizing radiations. Examples of directly ionizing particles include alpha and beta particles. Indirectly ionizing radiations are always more penetrating than directly ionizing particulate radiations.

Mass, charge, and velocity of a particle all affect the rate at which ionization occurs. The higher the charge of the particle and the lower the velocity, the greater the propensity to cause ionization. Heavy, highly charged particles, such as alpha particles, lose energy rapidly with distance and, therefore, do not penetrate deeply. The result of these interaction processes is a gradual slowing down of any incident particle until it is brought to rest or "stopped" at the end of its range.

D.2.4 Characteristics of Emitted Radiation

D.2.4.1 Alpha Emission. In alpha emission, an alpha particle consisting of two protons and two neutrons is emitted with a resulting decrease in the atomic mass number by four and reduction of the atomic number of two, thereby changing the parent to a different element. The alpha particle is identical to a helium nucleus consisting of two neutrons and two protons. It results from the radioactive decay of some heavy elements such as uranium, plutonium, radium, thorium, and radon. All alpha particles emitted by a given radioisotope have the same energy. Most of the alpha particles that are likely to be found have energies in the range of about 4 to 8 MeV, depending on the isotope from which they came.

The alpha particle has an electrical charge of +2. Because of this double positive charge and their size, alpha particles have great ionizing power and, thus, lose their kinetic energy quickly. This results in very little penetrating power. In fact, an alpha particle cannot penetrate a sheet of paper. The range of an alpha particle (the distance the charged particle travels from the point of origin to its resting point) is about 4 cm in air, which decreases considerably to a few micrometers in tissue. These properties cause alpha emitters to be hazardous only if there is internal contamination (i.e., if the radionuclide is inside the body).

D.2.4.2 Beta Emission. A beta particle (&) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (&-) or a positively charged electron, termed a positron (&+). Although the precise definition of "beta emission" refers to both &- and &+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the &+ particle.

D.2.4.2.1 Beta Negative Emission. Beta particle (&-) emission is another process by which a radionuclide, with a neutron excess achieves stability. Beta particle emission decreases the number of neutrons by one and increases the number of protons by one, while the atomic mass number remains unchanged.[1] This transformation results in the formation of a different element. The energy spectrum of beta particle emission ranges from a certain maximum down to zero with the mean energy of the spectrum being about one-third of the maximum. The range in tissue is much less. Beta negative emitting radionuclides can cause injury to the skin and superficial body tissues, but mostly present an internal contamination hazard.

D.2.4.2.2 Positron Emission. In cases in which there are too many protons in the nucleus, positron emission may occur. In this case a proton may be thought of as being converted into a neutron, and a positron (&+) is emitted.[1] This increases the number of neutrons by one, decreases the number of protons by one, and again leaves the atomic mass number unchanged. The gamma radiation resulting from the annihilation (see glossary) of the positron makes all positron emitting isotopes more of an external radiation hazard than pure & emitters of equal energy.

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(1)Neutrinos also accompany negative beta particles and positron emissions.

D.2.4.2.3 Gamma Emission. Radioactive decay by alpha, beta, or positron emission, or electron capture often leaves some of the energy resulting from these changes in the nucleus. As a result, the nucleus is raised to an excited level. None of these excited nuclei can remain in this high-energy state. Nuclei release this energy returning to ground state or to the lowest possible stable energy level. The energy released is in the form of gamma radiation (high energy photons) and has an energy equal to the change in the energy state of the nucleus. Gamma and x rays behave similarly but differ in their origin; gamma emissions originate in the nucleus while x rays originate in the orbital electron structure or from rapidly changing the velocity of an electron (e.g., as occurs when shielding high energy beta particles or stopping the electron beam in an x ray tube).

D.3 ESTIMATION OF ENERGY DEPOSITION IN HUMAN TISSUES

Two forms of potential radiation exposures can result: internal and external. The term exposure denotes physical interaction of the radiation emitted from the radioactive material with cells and tissues of the human body. An exposure can be "acute" or "chronic" depending on how long an individual or organ is exposed to the radiation. Internal exposures occur when radionuclides, which have entered the body (e.g., through the inhalation, ingestion, or dermal pathways), undergo radioactive decay resulting in the deposition of energy to internal organs. External exposures occur when radiation enters the body directly from sources located outside the body, such as radiation emitters from radionuclides on ground surfaces, dissolved in water, or dispersed in the air. In general, external exposures are from material emitting gamma radiation, which readily penetrate the skin and internal organs. Beta and alpha radiation from external sources are far less penetrating and deposit their energy primarily on the skin's outer layer. Consequently, their contribution to the absorbed dose of the total body dose, compared to that deposited by gamma rays, may be negligible.

Characterizing the radiation dose to persons as a result of exposure to radiation is a complex issue. It is difficult to: (1) measure internally the amount of energy actually transferred to an organic material and to correlate any observed effects with this energy deposition; and (2) account for and predict secondary processes, such as collision effects or biologically triggered effects, that are an indirect consequence of the primary interaction event.

D.3.1 Dose/Exposure Units

D.3.1.1 Roentgen. The roentgen (R) is a unit of x or gamma-ray exposure and is a measured by the amount of ionization caused in air by gamma or x radiation. One roentgen produces 2.58×10^{-4} coulomb per kilogram of air. In the case of gamma radiation, over the commonly encountered range of photon energy, the energy deposition in tissue for a dose of 1 R is about 0.0096 joules (J) /kg of tissue.

D.3.1.2 Absorbed Dose and Absorbed Dose Rate. The absorbed dose is defined as the energy imparted by the incident radiation to a unit mass of the tissue or organ. The unit of absorbed dose is the rad; 1 rad = 100 erg/gram = 0.01 J/kg in any medium. An exposure of 1 R results in a dose to soft tissue of approximately 0.01 J/kg. The SI unit is the gray which is equivalent to 100 rad or 1 J/kg. Internal and external exposures from radiation sources are not usually instantaneous but are distributed over extended periods of time. The resulting rate of change of the absorbed dose to a small volume of mass is referred to as the absorbed dose rate in units of rad/unit time.

D.3.1.3 Working Levels and Working Level Months. Working level (WL) is a measure of the atmospheric concentration of radon and its short-lived progeny. One WL is defined as any combination of short-lived radon daughters (through polonium-214), per liter of air, that will result in the emission of 1.3×10^5 MeV of alpha energy. An activity concentration of 100 pCi radon-222/L of air, in equilibrium with its daughters, corresponds approximately to a potential alpha-energy concentration of 1 WL. The WL unit can also be used for thoron daughters. In this case, 1.3×10^5 MeV of alpha energy (1 WL) is released by the thoron daughters in equilibrium with 7.5 pCi thoron/L. The potential alpha energy exposure of miners is commonly expressed in the unit Working Level Month (WLM). One WLM corresponds to exposure to a concentration of 1 WL for the reference period of 170 hours, or more generally

WLM = concentration (WL) x exposure time (months) (one "month" = 170 working hours).

D.3.2 Dosimetry Models

Dosimetry models are used to estimate the dose from internally deposited to radioactive substances. The models for internal dosimetry consider the amount of radionuclides entering the body, the factors affecting their movement or transport through the body, distribution and retention of radionuclides in the body, and the energy deposited in organs and tissues from the radiation that is emitted during spontaneous decay processes. The dose pattern for radioactive materials in the body may be strongly influenced by the route of entry of the material. For industrial workers, inhalation of radioactive particles with pulmonary deposition and puncture wounds with subcutaneous deposition have been the most frequent. The general population has been exposed via ingestion and inhalation of low levels of naturally occurring radionuclides as well as radionuclides from nuclear weapons testing.

The models for external dosimetry consider only the photon doses to organs of individuals who are immersed in air or are exposed to a contaminated object.

D.3.2.1 Ingestion. Ingestion of radioactive materials is most likely to occur from contaminated foodstuffs or water or eventual ingestion of inhaled compounds initially deposited in the lung. Ingestion of radioactive material may result in toxic effects as a result of either absorption of the radionuclide or irradiation of the gastrointestinal tract during passage through the tract, or a combination of both. The fraction of a radioactive material absorbed from the gastrointestinal tract is variable, depending on the specific element, the physical and chemical form of the material ingested, and the diet, as well as some other metabolic and physiological factors. The absorption of some elements is influenced by age, usually with higher absorption in the very young.

D.3.2.2 Inhalation. The inhalation route of exposure has long been recognized as being a major portal of entry for both nonradioactive and radioactive materials. The deposition of particles within the lung is largely dependent upon the size of the particles being inhaled. After the particle is deposited, the retention will depend upon the physical and chemical properties of the dust and the physiological status of the lung. The retention of the particle in the lung depends on the location of deposition, in addition to the physical and chemical properties of the particles. The converse of pulmonary retention is pulmonary clearance. There are three distinct mechanisms of clearance which operate simultaneously. Ciliary clearance acts only in the upper respiratory tract. The second and third mechanisms act mainly in the deep respiratory tract. These are phagocytosis and absorption. Phagocytosis is the engulfing of foreign bodies by alveolar macrophages and their subsequent removal either up the ciliary "escalator" or by entrance into the lymphatic system. Some inhaled soluble particles are absorbed into the blood and translocated to other organs and tissues.

D.3.3 Internal Emitters

An internal emitter is a radionuclide that is inside the body. The absorbed dose from internally deposited radioisotopes depends on the energy absorbed per unit tissue by the irradiated tissue. For a radioisotope distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the isotope. An infinitely large medium may be approximated by a tissue mass whose dimensions exceed the range of the particle. All alpha and most beta radiation will be absorbed in the organ (or tissue) of reference. Gamma-emitting isotope emissions are penetrating radiation, and a substantial fraction of gamma energy may be absorbed in tissue. The dose to an organ or tissue is a function of the effective retention half-time, the energy released in the tissue, the amount of radioactivity initially introduced, and the mass of the organ or tissue.

D.4 BIOLOGICAL EFFECTS OF RADIATION

When biological material is exposed to ionizing radiation, a chain of cellular events occurs as the ionizing particle passes through the biological material. A number of theories have been proposed to describe the interaction of radiation with biologically important molecules in cells and to explain the resulting damage to biological systems from those interactions. Many factors may modify the response of a living organism to a given dose of radiation. Factors related to the exposure include the dose rate, the energy of the radiation, and the temporal pattern of the exposure. Biological considerations include factors such as species, age, sex, and the portion of the body exposed. Several excellent reviews of the biological effects of radiation have been published, and the reader is referred to these for a more in-depth discussion (Brodsky 1996; Hobbs and McClellan 1986; ICRP 1984; Mettler and Moseley 1985; Rubin and Casarett 1968).

D.4.1 Radiation Effects at the Cellular Level

According to Mettler and Moseley (1985), at acute doses up to 10 rad (100 mGy), single strand breaks in DNA may be produced. These single strand breaks may be repaired rapidly. With doses in the range of 50–500 rad (0.5–5 Gy), irreparable double-stranded DNA breaks are likely, resulting in cellular reproductive death after one or more divisions of the irradiated parent cell. At large doses of radiation, usually greater than 500 rad (5 Gy), direct cell death before division (interphase death) may occur from the direct interaction of free-radicals with essentially cellular macromolecules. Morphological changes at the cellular level, the severity of which are dose-dependent, may also be observed.

The sensitivity of various cell types varies. According to the Bergonie-Tribondeau law, the sensitivity of cell lines is directly proportional to their mitotic rate and inversely proportional to the degree of differentiation (Mettler and Moseley 1985). Rubin and Casarett (1968) devised a classification system that categorized cells according to type, function, and mitotic activity. The categories range from the most sensitive type, "vegetative intermitotic cells, " found in the stem cells of the bone marrow and the gastrointestinal tract, to the least sensitive cell type, "fixed postmitotic cells," found in striated muscles or long-lived neural tissues.

Cellular changes may result in cell death, which if extensive, may produce irreversible damage to an organ or tissue or may result in the death of the individual. If the cell recovers, altered metabolism and function may still occur, which may be repaired or may result in the manifestation of clinical symptoms. These changes may also be expressed at a later time as tumors or cellular mutations, which may result in abnormal tissue.

D.4.2 Radiation Effects at the Organ Level

In most organs and tissues the injury and the underlying mechanism for that injury are complex and may involve a combination of events. The extent and severity of this tissue injury are dependent upon the radiosensitivity of the various cell types in that organ system. Rubin and Casarett (1968) describe and schematically display the events following radiation in several organ system types. These include: a rapid renewal system, such as the gastrointestinal mucosa; a slow renewal system, such as the pulmonary epithelium; and a nonrenewal system, such as neural or muscle tissue. In the rapid renewal system, organ injury results from the direct destruction of highly radiosensitive cells, such as the stem cells in the bone marrow. Injury may also result from constriction of the microcirculation and from edema and inflammation of the basement membrane, designated as the histohematic barrier (HHB), which may progress to fibrosis. In slow renewal and nonrenewal systems, the radiation may have little effect on the parenchymal cells, but ultimate parenchymal atrophy and death over several months result from HHB fibrosis and occlusion of the microcirculation.

D.4.3 Low Level Radiation Effects

Cancer is the major latent harmful effect produced by ionizing radiation and the one that most people exposed to radiation are concerned about. The ability of alpha, beta, and gamma radiation to produce cancer in virtually every tissue and organ in laboratory animals has been well-demonstrated. The development of cancer is not an immediate effect. In humans, radiation-induced leukemia has the shortest latent period at 2 years, while other radiation induced cancers have latent periods >20 years. The mechanism by which cancer is induced in living cells is complex and is a topic of intense study. Exposure to ionizing radiation can produce cancer at any site within the body; however, some sites appear to be more common than others, such as the breast, lung, stomach, and thyroid.

DNA is a major target molecule during exposure to ionizing radiation. Other macromolecules, such as lipids and proteins, are also at risk of damage when exposed to ionizing radiation. The genotoxicity of ionizing radiation is an area of intense study, as damage to the DNA is ultimately responsible for many of the adverse toxicological effects ascribed to ionizing radiation, including cancer. Damage to genetic material is basic to developmental or teratogenic effects, as well. However, for effects other than cancer, there is little evidence of human effects at low levels of exposure.

D.5 UNITS IN RADIATION PROTECTION AND REGULATION

D.5.1 Dose Equivalent and Dose Equivalent Rate

Dose equivalent or rem is a special radiation protection quantity that is used, for administrative and radiation safety purposes only, to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. The ICRU has defined the dose equivalent, H, as the product of the absorbed dose, D, and the quality factor, Q, at the point of interest in biological tissue. This relationship is expressed as $H = D \times Q$. The dose equivalent concept is applicable only to doses that are not great enough to produce biomedical effects.

The quality factor is a dimensionless quantity that depends in part on the stopping power for charged particles, and it accounts for the differences in biological effectiveness found among the types of radiation. Originally relative biological effectiveness (RBE) was used rather than Q to define the quantity, rem, which was of use in risk assessment. The generally accepted values for quality factors for various radiation types are provided in Table D-3. The dose equivalent rate is the time rate of change of the dose equivalent to organs and tissues and is expressed as rem/unit time or sievert/unit time.

Table D-3. Quality Factors (Q) and Absorbed Dose Equivalencies

Type of radiation	Quality factor (Q)	Absorbed dose equal to a unit dose equivalent*
X, gamma, or beta radiation	1	1
Alpha particles, multiple-charged particles, fission fragments and heavy particles of unknown charge	20	0.05
Neutrons of unknown energy	10	0.1
High-energy protons	10	0.1

*Absorbed dose in rad equal to 1 rem or the absorbed dose in gray equal to 1 sievert.

Source: USNRC. 1999. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C.

D.5.2 Relative Biological Effectiveness

RBE is used to denote the experimentally determined ratio of the absorbed dose from one radiation type to the absorbed dose of a reference radiation required to produce an identical biologic effect under the same conditions. Gamma rays from cobalt-60 and 200–250 keV x-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor used in calculations of dose equivalents for radiation safety purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). RBE applies only to a specific biological end point, in a specific exposure, under specific conditions to a specific species. There are no generally accepted values of RBE.

D.5.3 Effective Dose Equivalent and Effective Dose Equivalent Rate

The absorbed dose is usually defined as the mean absorbed dose within an organ or tissue. This represents a simplification of the actual problem. Normally when an individual ingests or inhales a radionuclide or is exposed to external radiation that enters the body (gamma), the dose is not uniform throughout the whole body. The simplifying assumption is that the detriment will be the same whether the body is uniformly or non-uniformly irradiated. In an attempt to compare detriment from absorbed dose of a limited portion of the body with the detriment from total body dose, the ICRP (1977) has derived a concept of effective dose equivalent. The effective dose equivalent, H_E, is

$$H_E = \text{(the sum of) } W_t H_t$$

where H_t is the dose equivalent in the tissue, W_t is the weighting factor, which represents the estimated proportion of the stochastic risk resulting from tissue, T, to the stochastic risk when the whole body is uniformly irradiated for occupational exposures under certain conditions (ICRP 1977). Weighting factors for selected tissues are listed in Table D-4.

The ICRU (1980), ICRP (1984), and NCRP (1985) now recommend that the rad, roentgen, curie, and rem be replaced by the SI units: gray (Gy), Coulomb per kilogram (C/kg), Becquerel (Bq), and sievert (Sv), respectively. The relationship between the customary units and the international system of units (SI) for radiological quantities is shown in Table D-5.

Table D-4. Weighting Factors for Calculating Effective Dose Equivalent for Selected Tissues

	Weighting factor		
Tissue	ICRP60	NCRP115/ ICRP60	NRC
Bladder	0.040	0.05	–
Bone marrow	0.143	0.12	0.12
Bone surface	0.009	0.01	0.03
Breast	0.050	0.05	0.15
Colon	0.141	0.12	–
Liver	0.022	0.05	–
Lung	0.111	0.12	0.12
Esophagus	0.034	0.05	–
Ovary	0.020	0.05	–
Skin	0.006	0.01	–
Stomach	0.139	0.12	–
Thyroid	0.021	0.05	0.03
Gonads	0.183	0.20	0.25
subtotal	0.919	0.99	0.70
Remainder	0.081	0.05	0.30

ICRP60 = International Commission on Radiological Protection, 1990 Recommendations of the ICRP;
NCRP115 = National Council on Radiation Protection and Measurements. 1993. Risk Estimates for Radiation Protection, Report 115. Bethesda, Maryland; NRC = Nuclear Regulatory Commission.
NRC = Nuclear Regulatory Commission, Title 10, Code of Federal Regulations, Part 20

Table D-5. Comparison of Common and SI Units for Radiation Quantities

Quantity	Customary units	Definition	SI units	Definition
Activity (A)	curie (Ci)	3.7×10^{10} transformations s^{-1}	becquerel (Bq)	s^{-1}
Absorbed dose (D)	rad (rad)	10^{-2} Jkg^{-1}	gray (Gy) Jkg^{-1}	
Absorbed dose rate (Š)	rad per second (rad s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	gray per second (Gy s^{-1})	Jkg^{-1} s^{-1}
Dose equivalent (H)	rem (rem)	10^{-2} Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose equivalent rate ( )	rem per second (rem s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	sievert per second (Sv s^{-1})	Jkg^{-1} s^{-1}
Linear energy transfer (LET)	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}

Jkg^{-1} = Joules per kilogram; $Jkg^{-1}s^{-1}$ = Joules per kilogram per second; Jm^{-1} = Joules per meter; s^{-1} = per second

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