TOXICOLOGICAL PROFILE FOR CESIUM

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

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DISCLAIMER

UPDATE STATEMENT

A Toxicological Profile for Cesium, Draft for Public Comment was released in July 2001. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
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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.

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 was made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

Julie Louise Gerberding, M.D., M.P.H.
Administrator
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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).

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 1.6 How Can (Chemical X) Affect Children?
Section 1.7 How Can Families Reduce the Risk of Exposure to (Chemical X)?
Section 3.8 Children's Susceptibility
Section 6.6 Exposures of Children

Other Sections of Interest:

Section 3.9 Biomarkers of Exposure and Effect
Section 3.12 Methods for Reducing Toxic Effects

ATSDR Information Center

Phone: 1-888-42-ATSDR or (404) 498-0110 ***Fax:*** (770) 488-4178
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 Exposure*s—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

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@AOEC.ORG • Web Page: http://www.aoec.org/.

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 cesium. The panel consisted of the following members:

1. Ranjan Bose, M.B.B.S., M.D., New Substances Assessment and Controls Bureau, Health Canada, Ottawa, Ontario, Canada;

2. Darrell Fisher, Ph.D., Senior Scientist, Pacific Northwest National Laboratory, Richland, Washington;

3. Richard Leggett, Ph.D., Life Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee;

4. Bruce Muggenburg, D.V.M., Ph.D., Senior Scientist and Veterinary Physiologist, Toxicology Division, Lovelace Respiratory Research Institute, Albuquerque, New Mexico;

5. Herman Cember, C.H.P., Ph.D., P.E., Adjunct Professor, School of Health Sciences, Purdue University, Lafayette, Indiana; and

6. Marvin Goldman, Ph.D., Professor Emeritus of Radiation Biology, Department of Surgical and Radiological Sciences, University of California, Davis, California.

These experts collectively have knowledge of cesium's physical and chemical properties, toxicokinetics, 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.

CONTENTS

LIST OF FIGURES

LIST OF TABLES

1. PUBLIC HEALTH STATEMENT

This public health statement tells you about cesium 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. Stable (not radioactive) cesium (^{133}Cs) has been identified in at least 8 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2003). It was reported that ^{134}Cs (radioactive) has been found in at least 3 of the 1,636 current or former NPL sites and ^{137}Cs (radioactive) has been detected in at least 23 of the 1,636 current or former NPL sites. However, the number of sites evaluated for cesium is not known. As more sites are evaluated, the sites at which cesium 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 exposed to a substance when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance, or by skin contact. If the substance is radioactive, you may also be exposed to radiation if you are near it.

External exposure to radiation may occur from natural or man-made sources. Naturally occurring sources of radiation are cosmic radiation from space or radioactive materials in soil or building materials. Man-made sources of radioactive materials are found in consumer products, industrial equipment, atom bomb fallout, and to a smaller extent from hospital waste, medical devices, and nuclear reactors.

If you are exposed to cesium, 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 CESIUM?

Cesium is a naturally-occurring element found in rocks, soil, and dust at low concentrations. Granites contain an average cesium concentration of about 1 part of cesium in a million parts of granite (ppm) and sedimentary rocks contain about 4 ppm. Natural cesium is present in the environment in only one stable form, as the isotope ^{133}Cs. Pure cesium metal is silvery white in color and very soft, but pure cesium is not expected to be found in the environment. Pure cesium metal reacts violently with air and water, resulting in an explosion-like reaction. Cesium compounds do not react violently with air or water and are generally very soluble in water. The most important source of commercial cesium is a mineral known as pollucite, which usually contains about 5–32% cesium oxide (Cs_2O). No known taste or odor is associated with cesium compounds. Cesium is not mined or produced in the United States and very little is imported from other countries. There are relatively few commercial uses for cesium metal and its compounds. Sometimes cesium is used as a getter for residual gas impurities in vacuum tubes and as a coating in tungsten filaments or cathodes of the tubes. Crystalline cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionizing radiation into pulses of visible light for radiation detection and spectroscopy. Cesium is also used in highly accurate atomic clocks. For more information on the physical and chemical properties and on the production and use of cesium, see Chapters 4 and 5.

Radioactive forms of cesium are produced by the fission of uranium in fuel elements (fuel rods) during the normal operation of nuclear power plants, or when nuclear weapons are exploded. Radioactive forms of cesium are unstable and eventually change into other more stable elements through the process of radioactive decay. The two most important radioactive isotopes of cesium are ^{134}Cs and ^{137}Cs. Radioactive isotopes are constantly decaying or changing into different isotopes by giving off radiation. Each atom of ^{134}Cs changes into either xenon 134 (^{134}Xe) or barium 134 (^{134}Ba), neither of which is radioactive, while each atom of ^{137}Cs decays to barium 137 (^{137}Ba), which is also not radioactive. As ^{134}Cs and ^{137}Cs decay, beta particles and gamma radiation are given off. The half-life is the time it takes for half of that cesium isotope to give off its radiation and change into a different element. The half-life of ^{134}Cs is about 2 years and the half-life of ^{137}Cs is about 30 years.

Quantities of radioactive cesium, as well as other radioactive elements, are measured in units of mass (grams) or radioactivity (curies or becquerels). Both the curie (Ci) and the becquerel (Bq) describe the rate of decay and tell us how much a radioactive material decays every second. The becquerel is a new international unit known as the SI unit, and the curie is an older, traditional unit; both are currently used. A becquerel is the amount of radioactive material in which 1 atom transforms every second. One curie is the amount of radioactive material in which 37 billion atoms transform every second; this is approximately equivalent to the radioactivity of 1 gram of radium.

1.2 WHAT HAPPENS TO CESIUM WHEN IT ENTERS THE ENVIRONMENT?

Naturally-occurring cesium occurs in the environment mostly from the erosion and weathering of rocks and minerals. The mining and milling of certain ores can also release cesium to the air, water, and soil. Radioactive cesium is released to the environment during the normal operation of nuclear power plants, explosion of nuclear weapons, and accidents involving nuclear power plants or nuclear powered satellites or submarines.

Nonradioactive (stable) cesium can be neither created nor destroyed under typical environmental conditions, but can react with other compounds found in the environment and change into different cesium compounds. Radioactive decay decreases the concentration of ^{134}Cs and ^{137}Cs. Both stable and radioactive cesium are the same element and behave in a similar manner chemically and in the body. Cesium compounds can travel long distances in the air before being brought back to the earth by rainfall and gravitational settling. In water and moist soils, most cesium compounds are very soluble. Cesium binds strongly to most soils and does not travel far below the surface of the soil. Consequently, cesium is not readily available for uptake by vegetation through roots. However, radiocesium can enter plants upon falling onto the surface of leaves.

1.3 HOW MIGHT I BE EXPOSED TO CESIUM?

You can be exposed to stable or radioactive cesium by breathing air, drinking water, or eating food containing cesium. The level of cesium in air and water is generally very low. The concentration of natural cesium in air is generally less than 1 nanogram (1 nanogram equals 1/1,000,000,000 of a gram) per cubic meter of air (ng/m^3). The amount of cesium in drinking water is ordinarily about 1 microgram (1 microgram equals 1/1,000,000 of a gram) per liter of water (μg/L). On average, a person swallows about 10 μg of stable cesium per day in food and water, and breathes about 0.025 μg per day. Plants and animals contain cesium at concentrations of about 1–300 ng/g.

Radioactive cesium has been detected in surface water and in many types of food. This includes breast milk and pasteurized milk. The amount of radioactive cesium in food and milk is highly dependent upon several factors. The most important factor is whether or not there has been recent fallout from a nuclear explosion such as a weapons test or an accident that has occurred at a nuclear power plant. However, atmospheric testing of nuclear weapons was halted many years ago, and there have only been two major reactor accidents at nuclear plants where radiocesium was released in significant amounts. The two accidents occurred in Windscale, England in 1957 and Chernobyl, Russia in 1986. You should understand that cesium only contributed a small fraction of the total radioactivity released following these events. The radiological impacts in Europe from ^{137}Cs and ^{134}Cs released from the Chernobyl accident, however, were great. These included environmental dispersion of radiocesium and uptake in reindeer, caribou, and livestock. Furthermore, the consequences of external exposure to gamma radiation and beta particles are not unique to ^{137}Cs and ^{134}Cs, but are very similar for all gamma and beta emitting radionuclides. People who work in industries that process or use natural cesium or cesium compounds can be exposed to higher-than-normal levels of cesium. An estimated 16,461 workers (4,276 of these are female) are potentially exposed to natural cesium and cesium compounds in the United States. If you work in the nuclear power industry, you may also be exposed to higher-than-normal levels of radioactive cesium, but there are many precautionary measures taken at these facilities to minimize this exposure.

1.4 HOW CAN CESIUM ENTER AND LEAVE MY BODY?

Stable and radioactive cesium can enter your body from the food you eat or the water you drink, from the air you breathe, or from contact with your skin. When you eat, drink, breathe, or touch things containing cesium compounds that can easily be dissolved in water, cesium enters your blood and is carried to all parts of your body. Cesium is like potassium; it enters cells and helps to maintain a balance of electrical charges between the inside and the outside of cells so that cells can perform tasks that depend on those electrical charges. Cells like muscle cells and nerve cells require changing electrical charges in order to function properly and allow you to think and move.

Once cesium enters your body, your kidneys begin to remove it from the blood; some cesium is quickly released from your body in the urine. A small portion is also released in the feces. Some of the cesium that your body absorbs can remain in your body for weeks or months, but is slowly eliminated from your body through the urine and feces.

1.5 HOW CAN CESIUM 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.

You are not likely to experience any health effects that could be related to stable cesium itself. Animals given very large doses of cesium compounds have shown changes in behavior, such as increased activity or decreased activity, but it is highly unlikely that you would breathe, eat, or drink amounts of stable cesium large enough to cause similar effects. If you were to breathe, eat, drink, touch, or come close to large amounts of radioactive cesium, cells in your body could become damaged from the radiation that might penetrate your entire body, much like x-rays, even if you did not touch the radioactive cesium. You would probably experience similar effects if you were exposed to any other substance with similar radioactivity. You might also experience acute radiation syndrome, which includes such effects as nausea, vomiting, diarrhea, bleeding, coma, and even death. A number of people in Brazil, who handled radioactive cesium that was scavenged from a medical machine used for radiation therapy, became sick from exposure to the radiation; a few of them died. But people exposed to radioactive cesium that has been widely dispersed in air, water, soil, or foods following nuclear bombings or accidents have not been exposed to amounts large enough to cause the same effects.

1.6 HOW CAN CESIUM AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans.

Children can be affected by cesium in the same ways as adults. Stable cesium is not likely to affect the health of children, but large amounts of gamma radiation, from sources such as radioactive cesium, could damage cells and might also cause cancer. Short exposure to extremely large amounts of radiation might cause nausea, vomiting, diarrhea, bleeding, coma, and even death. In addition, if babies were to be exposed to enough radiation while in their mother's womb during the time when their nervous system is rapidly developing, they could experience changes in their brains that could result in changes in behavior or decreased mental abilities. However, it is unlikely that children or babies would be exposed to enough gamma radiation from a radioactive cesium source to do such damage to their bodies.

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

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

Since cesium is naturally found in the environment, we cannot avoid being exposed to it. However, the relatively low concentrations of stable cesium do not warrant any immediate steps to reduce exposure. You are unlikely to be exposed to high levels of radioactive cesium unless there is a fuel meltdown and accidental release at a nuclear power plant or a nuclear weapon has been detonated. In such cases, follow the advice of public health officials who will publish guidelines for reducing exposure to radioactive material when necessary.

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

Everyone has small amounts of cesium in their body. Laboratories use special techniques to measure the amount of cesium in body fluids such as blood and urine, as well as in feces or other human samples. This can give an indication of whether a person has been exposed to levels of cesium that are higher than those normally found in food, water, or air. Special radiation detectors can be used to detect if a person has absorbed radioactive cesium. It is difficult to determine if a person has been exposed only to external radiation from radioactive cesium. Health professionals examining people who have health problems similar to those resulting from radiation exposure would need to rely on additional information, such as the testing of blood samples for cell counts and chromosomal damage, in order to establish if such people had been affected by being near a source of radioactivity.

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 U.S. Nuclear Regulatory Commission (USNRC).

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; they are then 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 cesium include the following:

There are few guidelines for compounds of stable cesium. Based on eye irritation, the NIOSH has established a recommended exposure limit (REL) for cesium hydroxide of 2 mg/m^3 as a time-weighted average (TWA) for up to a 10-hour workday and a 40-hour workweek. The American Conference of Governmental Industrial Hygienists (ACGIH) has assigned cesium hydroxide a threshold limit value (TLV) of 2 mg/m^3 as a TWA for a normal 8-hour workday and a 40-hour workweek, based on respiratory and eye irritation.

The NRC established guidelines for radioactive cesium that include occupational inhalation exposure Derived Air Concentrations (DACs) of 0.00000004 μCi/mL (4×10^{-8} μCi/mL) for ^{134}Cs and 0.00000006 μCi/mL (6×10^{-8} μCi/mL) for ^{137}Cs. Annual Limits on Intake (ALIs) for on-the-job exposure are 100 μCi (1×10^{2} μCi) for ^{134}Cs and 200 μCi (2×10^{2} μCi) for ^{137}Cs.

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 contact ATSDR at the address and phone number below.

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.

Toxicological profiles are also available on-line at www.atsdr.cdc.gov and on CD-ROM. You may request a copy of the ATSDR ToxProfiles CD-ROM by calling the information and technical assistance toll-free number at 1-888-42ATSDR (1-888-422-8737), by email at atsdric@cdc.gov, or by writing to:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE
Mailstop E-32
Atlanta, GA 30333
Fax: 1-770-488-4178

For-profit organizations may request a copy of final profiles from the following:

National Technical Information Service (NTIS)
5285 Port Royal Road
Springfield, VA 22161
Phone: 1-800-553-6847 or 1-703-605-6000
Web site: http://www.ntis.gov/

2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO CESIUM IN THE UNITED STATES

Naturally-occurring cesium exists as the stable isotope (^{133}Cs) in the earth's crust at an average concentration of about 1 ppm in granites and 4 ppm in sedimentary rocks. The most important source of commercial cesium is the mineral pollucite, which usually contains about 5–32% Cs_2O by weight. The largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world's known supply. Cesium has very low mobility in soil surfaces. Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles. The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays, which can result in limited uptake of cesium by grass and plant material. Continental dust and soil erosion are the main emission sources of naturally-occurring cesium that is present in the environment. As a result of human activities, however, cesium is released into the environment globally in small amounts. Cesium has also been detected in the fly ash of hazardous waste incinerators and coal burning power plants. Cesium is deposited on plants and trees by wet and dry deposition and can be absorbed into the flora through its foliage. The deposited cesium can make its way to soil through litter decomposition. Due to its low relative abundance, limited use in industry, and relatively low level of toxicity observed in animal studies, exposure to stable cesium is not considered to be a significant public health concern.

Radioactive isotopes of cesium (^{134}Cs and ^{137}Cs) are formed during nuclear fission, in commercial applications such as the generation of electricity at nuclear power plants. However, high levels of ^{134}Cs and ^{137}Cs have been released to the environment, as a result of atmospheric nuclear weapons testing (which has been discontinued for many years) or underground weapons testing and the accident at the Chernobyl, Ukraine nuclear reactor site in 1986. Once released, these radioactive cesium isotopes persist in the environment, with the potential for adverse health effects. Following release to the atmosphere, radioactive cesium can travel thousands of miles before settling to earth, and is removed by wet and dry deposition. Radioactive cesium can also be released to soil or water in liquid effluents from spent fuel and fuel reprocessing plants.

The total amount of ^{137}Cs released from weapons testing through 1980 was estimated as 2.6x10^{7} Ci (9.6x10^{17} Bq), 76% of which was released in the northern hemisphere and 24% in the southern hemisphere.

The accident at the Chernobyl nuclear power plant resulted in the release of an estimated 5.4x10^{5} Ci (2.0x10^{16} Bq) of ^{134}Cs and 1.1x10^{6} Ci (4.0x10^{16} Bq) of ^{137}Cs into the atmosphere over Europe (see Agency for Toxic Substances and Disease Registry 1999 for details of other accidental releases). Routine activities at nuclear power plants and fuel-reprocessing stations also release ^{137}Cs and ^{134}Cs to the environment on a regular basis, but these are at such levels as to be considered insignificant.

Since the half-life for some radioactive isotopes of cesium is long (the half-life of ^{137}Cs is about 30 years and the half-life of ^{134}Cs is about 2 years), the general population is exposed to ^{137}Cs and ^{134}Cs for long periods of time after it is released from a nuclear accident or weapons test, with the greatest exposure occurring near the source. Although inhalation and dermal exposure is possible, oral ingestion of contaminated food items is the greatest source of internal exposure for both naturally-occurring and radioactive cesium. Workers employed in the mining and milling of pollucite ores and the production of cesium compounds are exposed to cesium through oral, dermal, and inhalation routes. Similar routes of exposure to ^{137}Cs and ^{134}Cs are experienced by workers employed in the nuclear industry. External exposure to beta and gamma radiation can also occur for workers employed in the nuclear industry as well as for the general population following an accidental release or weapons test.

As discussed in Appendix A, the average annual effective dose of ionizing radiation (including ^{134}Cs and ^{137}Cs) from anthropogenic sources to the U.S. population is very small in comparison to natural sources.

2.2 SUMMARY OF HEALTH EFFECTS

Information regarding health effects in humans that can be associated with exposure to higher-than-normal levels of stable cesium is restricted to an account of decreased appetite, nausea, and diarrhea in a man who repeatedly ingested experimental amounts of cesium chloride and reports of prolonged QT syndrome and associated cardiac arrhythmias in patients who ingested cesium chloride as a component of homeopathic remedies.

Exposure to radioisotopes of cesium is of much greater human health concern. Energy released by radioactive isotopes can result in significant damage to living cells. Both ^{134}Cs and ^{137}Cs emit beta particles and gamma rays, which may ionize molecules within cells penetrated by these emissions and result in tissue damage and disruption of cellular function. The most important exposure routes for radioisotopes of cesium are external exposure to the radiation released by the radioisotopes and ingestion of radioactive cesium-contaminated food sources. Inhalation and dermal exposure routes may also present a health hazard. The hazards of external exposure to ^{134}Cs and ^{137}Cs are similar to those of other gamma- and beta-emitting radionuclides.

Radiation absorbed doses are expressed in terms of the amount of energy absorbed per unit mass, in units called rad or gray (Gy) (see Appendix D and Agency for Toxic Substances and Disease Registry 1999 for a complete description of principles of ionizing radiation). Generally, acute radiation doses below 15 rad (0.15 Gy) do not result in observable adverse health effects. At doses in the range of 15–50 rad (0.15–0.5 Gy), subclinical responses such as chromosomal breaks and transient changes in formed elements of the blood may be seen in sensitive individuals. Symptoms of acute radiation syndrome are observed at radiation doses above 50 rad (0.5 Gy), characterized by transient hematopoietic manifestations, nausea and vomiting, and moderate leukopenia at doses near 100 rad (1 Gy), progressing through more serious hematopoietic symptoms, clinical signs, and gastrointestinal symptoms with increasing dose (100–800 rad or 1–8 Gy), and usually death in persons receiving total doses ≥1,000 rad (10 Gy). Other health effects from acute or continued exposure to ionizing radiation may include reproductive, developmental, and latent cancer effects.

Signs and symptoms of acute toxicity from external and internal exposure to high levels of radiation from ^{134}Cs or ^{137}Cs are typical of those observed in cases of high exposure to ionizing radiation in general. Depending on the radiation dose, symptoms may include those typical of acute radiation syndrome (vomiting, nausea, and diarrhea), skin and ocular lesions, neurological signs, chromosomal abnormalities, compromised immune function, and death.

Acute or repeated exposure of humans or animals to ionizing radiation (from radioisotopes of cesium or other radioactive elements) may result in reduced male fertility, abnormal neurological development following exposure during critical stages of fetal development, and genotoxic effects such as increased frequencies of chromosomal aberrations, T-lymphocyte point mutations, dominant lethal mutations, and reciprocal translocations.

Due to the ionizing properties of radionuclides such as ^{134}Cs and ^{137}Cs, increased cancer risk would be expected among exposed individuals. However, studies of increased cancer risk specifically associated with exposure of humans to radioactive cesium isotopes were not located. The only documented reports of health effects in humans exposed to cesium as the source of radiation are derived from accidental exposure to a ^{137}Cs source in 1987 in Goiânia, Brazil, and during 1996 and 1997 in Russia. Long-term cancer studies on exposed individuals have not been completed to date.

Animal studies indicate increased risk of cancer following external or internal exposure to relatively high doses of radiation from ^{137}Cs sources. Increased lifetime risk of mammary tumors was noted in female rats acutely exposed to whole-body radiation. Intravenous injection of ^{137}Cs (as cesium chloride) in dogs resulted in long-term increased risk of all cancers combined in males, and all cancers combined (excluding mammary cancer) in females.

Immunological and Lymphoreticular Effects. Humans who were accidentally exposed externally and internally to ^{137}Cs that resulted in estimated radiation absorbed doses of 100–700 rad (1–7 Gy), exhibited severe bone marrow depression. Similar effects were seen in dogs exposed to ^{137}Cs by intravenous injection, resulting in estimated bone marrow doses of 700–2,400 rad (7–24 Gy).

Reproductive Effects. Exposure to radioisotopes of cesium may result in reduced fertility in males, as evidenced by reduced concentrations of spermatozoa in men who had been exposed externally and internally to ^{137}CsCl approximately 1 month prior to testing. Reduced fertility, including sterility, was reported in male mice exposed to gamma radiation from ^{137}Cs either by total-body external radiation, which resulted in a total radiation dose of 300 rad (3 Gy) over a 19.5-day exposure period, or by single or repeated oral dosing, which resulted in estimated total testicular radiation doses of 300–385 rad (3–3.85 Gy), measured at 5 weeks post-treatment. No significant reduction in male fertility was seen from total testicular radiation doses in the range of 10–100 rad (0.1–1 Gy). Persistent germinal epithelium damage and azoospermia were reported in all long-term surviving dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 742 to 1,640 rad (7.42–16.40 Gy).

Developmental Effects. Developmental effects such as reduced post-natal body weight, impaired motor activity, morphological changes in the brain, reduced head size, and retarded odontogenesis and palatal closure have been reported in rats that had been exposed to radioactive cesium sources (^{137}Cs) *in utero* via whole-body external exposure of dams; effects were of largest magnitude when exposure occurred around gestational day 15. Reported developmental effects in similarly-exposed mice included significantly decreased brain weight and increased aggressive behavior. Atomic bomb survivors of

Hiroshima and Nagasaki, exposed to high levels of ionizing radiation *in utero* during weeks 8–15 or 16–25 post-ovulation, exhibited later signs of impaired cognitive function. Radiation-induced developmental effects would be expected in humans or animals exposed to similar levels of ionizing radiation from any ionizing radiation source, including a radiocesium source. Resulting adverse health effects would be due to the external, penetrating gamma radiation, not cesium *per se*.

Neurological Effects. Excess exposure to stable cesium appears to result in central nervous system effects. A man who voluntarily ingested cesium chloride daily for 36 days reported neurological signs that included feelings of euphoria, heightened sense perception, and tingling sensations within 15 minutes of dosing, in the absence of apparent adverse mental or motor skills. In animal studies, administration of cesium chloride has been reported to trigger stimulant and depressant central nervous system responses.

Since radioisotopes of cesium such as ^{134}Cs and ^{137}Cs emit beta particles and gamma rays capable of ionizing cells, acute radiation doses >3,000 rad (30 Gy) in humans would be expected to result in symptoms indicative of central nervous system syndrome that include immediate onset of violent nausea and vomiting, diarrhea, irrational behavior, circulatory system collapse, and neuromuscular incoordination, followed by convulsions, coma, and death within 48 hours (see Agency for Toxic Substances and Disease Registry 1999 for more detailed information on health effects from exposure to ionizing radiation).

Cancer. Studies that assess the risk of cesium-induced cancer are restricted to radioactive isotopes, not stable cesium. No human studies were located in which cancer incidence was specifically associated with exposure to radioisotopes of cesium. Due to the nature of ionizing radiation in general, carcinogenic effects similar to those observed in Japanese survivors of the 1945 atomic bombing incidents might be expected among individuals acutely exposed to high levels of radiation from a radioactive cesium source (see Agency for Toxic Substances and Disease Registry 1999 for a detailed discussion of the carcinogenic effects of ionizing radiation). However, it is unlikely that levels of ionizing radiation as high as those experienced by the survivors of the atomic bombing incidents would be experienced by individuals who might be exposed to a radiocesium source. Exceptions are reports of accidental human exposures to radioactive cesium sources in Goiânia, Brazil, in 1987 and Lilo, Georgia (Russia) in 1996 and 1997. However, the incidents are too recent for meaningful carcinogenicity data. The EPA Office of Radiation and Indoor Air (ORIA) has classified all radionuclides, including radioisotopes of cesium, as known human carcinogens.

Animal studies indicate increased risk of cancer following external or internal exposure to relatively high doses of radiation from ^{137}Cs sources. Increased lifetime risk of mammary tumors was noted in female rats acutely exposed to whole-body radiation. There were no significant differences between age groups irradiated at 8, 12, 16, 22, or 36 weeks of age, but irradiation at 64 weeks yielded fewer carcinomas than unirradiated controls. In lifetime studies of dogs administered single intravenous doses of ^{137}CsCl, which resulted in average initial body burdens ranging from 36.4 to 147 MBq/kg (1 to 4 mCi/kg), benign and malignant neoplasms were found in a variety of tissues and organs, with no apparent single target organ of toxicity.

2.3 MINIMAL RISK LEVELS

Inhalation MRLs

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for cesium due to the lack of suitable human or animal data regarding health effects following inhalation exposure to stable or radioactive cesium. Available information, considered relevant to inhalation exposure, is limited to two studies of dogs intravenously administered ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972). Adverse health effects included depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs. Striking similarities in the biokinetics, observed in laboratory animals exposed to ^{137}CsCl via either parenteral injection or inhalation or oral routes, indicate that adverse health effects might be common to all three routes of exposure (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). However, extrapolation of data across exposure routes was not considered to be a valid basis for the derivation of inhalation MRLs.

Oral MRLs

No acute-, intermediate-, or chronic-duration oral MRLs were derived for stable cesium due to the lack of suitable human or animal data regarding health effects following oral exposure to stable or radioactive cesium. Reports of health effects following oral exposure to stable cesium are limited. Daily ingestion of approximately 68 mg Cs/kg (as cesium chloride) for up to 36 days resulted in decreased appetite, nausea, and diarrhea, as well as neurological signs within 15 minutes following ingestion (Neulieb 1984). Prolonged QT syndrome and associated cardiac arrhythmias were reported in patients who ingested cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001). Animal studies regarding oral exposure to stable cesium are limited to LD_{50} studies that indicate relatively low toxicity for stable cesium compounds (Ghosh et al. 1990; Johnson et al. 1975; Khosid 1967). Information regarding human exposure to radioactive cesium is inadequate because no human data were available on health effects from oral exposure to radioactive cesium. Oral data regarding health effects in animals exposed to radioactive cesium are restricted to a single study in which only reproductive and genotoxic end points were reported (Ramaiya et al. 1994).

Due to striking similarities in the biokinetics, observed in laboratory animals exposed to ^{137}CsCl via either parenteral injection or inhalation or oral routes, it has been suggested that adverse health effects might be common to all three routes of exposure (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs were observed in dogs intravenously administered ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972). However, extrapolation of data across exposure routes was not considered to be a valid basis for the derivation of oral MRLs.

MRLs for External Exposure to Cesium Isotopes

Two MRLs have been derived for ionizing radiation (Agency for Toxic Substances and Disease Registry 1999) and are applicable to external exposure to radioisotopes of cesium:

- An MRL of 400 mrem (4.0 mSv) has been derived for acute-duration external exposure to ionizing radiation (14 days or less).

The acute MRL is based on results of a study by Schull et al. (1988) in which neurological effects of radiation, measured by intelligence test scores, were evaluated in children 10–11 years of age who had been exposed at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki. When IQ scores were regressed on radiation dose estimates, IQ diminished linearly with increasing dose, resulting in an estimated decrease in IQ score of approximately 25 points per 100 rad (100 rem in dose equivalent) or 0.25 points/rem (25 points/Sv). To derive the MRL of 400 mrem (4.0 mSv), the Agency for Toxic Substances and Disease Registry (1999) divided the dose associated with a predicted change of 0.25 IQ points (1 rem) by an uncertainty factor of 3 (for human variability/sensitive population). The Agency for Toxic Substances and Disease Registry (1999) noted that a change in IQ points of 0.25 is less than the reported difference of 0.3 IQ points between separated and unseparated identical twins (Burt 1966).

The NRC set a radiation exposure limit of 500 mrem (5 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks, ATSDR believes that the acute MRL of 400 mrem (4 mSv) is consistent with the NRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

- An MRL of 100 mrem/year (1.0 mSv/year) above background has been derived for chronic-duration external exposure to ionizing radiation (365 days or more).

The MRL is based on the BEIR V (1990) report that the average annual effective ionizing radiation dose to the U.S. population is 360 mrem/year (3.6 mSv/year) from all background sources of radiation (earth, cosmic rays, building materials, etc.), a dose not expected to produce adverse health effects. This dose is obtained mainly by naturally-occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. An uncertainty factor of 3 (for human variability) was applied to the NOAEL.

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 cesium, existing naturally as the stable (nonradioactive) isotope ^{133}Cs and in the form of radioactive isotopes produced during nuclear fission, the most abundant of which are ^{137}Cs and ^{134}Cs. This chapter 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. Section 3.2 contains a discussion of the chemical toxicity of stable cesium; radiation toxicity associated with exposure to radiocesium (primarily ^{137}Cs and ^{134}Cs) is discussed in Section 3.3. The chemical properties of stable and radioactive cesium isotopes are identical and are described in Chapter 4.

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

Since the average concentration of stable cesium in the earth's crust is low (on average about 1 ppm) and stable cesium is used only in small quantities in electronic and energy production industries, the risk of significant exposure to stable cesium via inhalation, oral, or dermal routes is expected to be small. Limited information is available on monitoring (or detection) of stable cesium in the environment and on health effects from exposure to stable cesium.

Decreased appetite, nausea, and diarrhea were reported in a man who ingested about 34 mg Cs/kg (as cesium chloride) after morning and evening meals for 36 days; this man also experienced apparent neurological changes within 15 minutes of dosing (Neulieb 1984). Prolonged QT syndrome and associated cardiac arrhythmias have been described in patients who have ingested cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001).

Animal studies indicate that cesium is of relatively low toxicity. Acute oral LD_{50} values for rats and mice range from 800 to 2,000 mg Cs/kg, cesium hydroxide being more toxic than cesium iodide or cesium chloride. Single oral doses of cesium chloride, administered to female mice at dose levels ranging from 125 to 500 mg/kg, have been shown to result in significant increases in chromosomal breaks in bone marrow cells (Ghosh et al. 1990, 1991).

No reports were located regarding adverse effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation or dermal exposure to stable cesium.

Radioactive isotopes of cesium are a greater health concern than stable cesium. The most important exposure routes are external exposure to the radiation emitted by the radioisotope and ingestion of radioactive cesium-contaminated food sources. Vascular plants do not accumulate large levels of cesium through root uptake because cesium is strongly adsorbed to soils. However, the deposition of radioactive debris on flora with large surface areas such as lichens or moss is significant. Animals that feed on this vegetation, such as reindeer and caribou, may ingest large quantities of radiocesium (and other radionuclides found in fallout). Human consumption of meat from such animals results in the internalization of these radionuclides (see Section 6.7 for more detailed information on the lichen-caribou-human food chain). Radioactive cesium particles may be found in the air following the release of nuclear fission products; however, no reports were located in which adverse health effects from inhalation of radioactive cesium were discussed. Dermal absorption has been observed in rats, as evidenced by traces of ^{137}Cs in the blood a few minutes following dermal application of ^{137}CsCl (Pendic and Milivojevic 1966), but no data were located on relative amounts and absorption rates.

Limited human data are available regarding health effects that can be exclusively associated with exposure to radioactive cesium sources such as ^{137}Cs and ^{134}Cs. These radionuclides are products of either neutron activation or nuclear fission and may, therefore, be released from sites where nuclear fission occurs, from radioactive material removed from such sites, or from leakage of radioactive cesium sources. Both ^{137}Cs and ^{134}Cs emit beta radiation (that travels short distances and can penetrate the skin and superficial body tissues) and gamma radiation (that penetrates the entire body). The radiation dose from these radionuclides can be classified as either external (if the source is outside the body) or internal (if the source is inside the body).

The external dose from cesium radionuclides arises primarily from the penetrating gamma rays that travel great distances in air. Beta radiation emitted outside the body is normally of little health concern unless the radioactive material contacts the skin. Skin contact can allow the beta radiation to pass through the epidermis to live dermal tissue where it becomes a major contributor to a radiocesium-generated radiation dose to the skin. At very high doses, the beta and gamma radiation can cause such adverse effects as erythema, ulceration, or even tissue necrosis.

Once radioactive cesium is internalized, it is absorbed, distributed, and excreted in the same manner as stable cesium. The internal radiation dose from cesium is a measure of the amount of energy that the beta and gamma emissions deposit in tissue. The short-range beta radiation produces a localized dose while the more penetrating gamma radiation contributes to a whole body dose. Molecular damage results from the direct ionization of atoms that are encountered by beta and gamma radiation and by interactions of resulting free radicals with nearby atoms. Tissue damage results when the molecular damage is extensive and not sufficiently repaired in a timely manner.

In radiation biology, the term *absorbed dose* is the amount of energy deposited by radiation per unit mass of tissue, expressed in units of rad or gray (Gy) (see Appendix D for a detailed description of principles of ionizing radiation. The term *dose equivalent* refers to the biologically significant dose, which is determined by multiplying the absorbed dose by radiation weighting factors for the type and energy of the radiations involved. Dose equivalent is expressed in units of rem or sievert (Sv). The radiation weighting factors are considered to be unity for the beta and gamma radiation emitted from ^{134}Cs and ^{137}Cs, so for these radionuclides, the absorbed dose (in rad or Gray) is equal to the dose equivalent (in rem or sievert). The dose equivalent from internalized cesium radionuclides is estimated using the quantity of material entering the body (via ingestion or inhalation), the biokinetic parameters for cesium (retention, distribution, and excretion), the energies and intensities of the beta and gamma radiation emitted, and parameters describing the profile of absorbed radiation energy within the body. If, for example, a person ingests a given activity of radiocesium (measured in curies [Ci] or becquerels [Bq]), the tissues of the body will absorb some of the energy of the emitted beta and gamma radiation in a pattern reflecting the kinetics of distribution and elimination of the ingested radiocesium, the rate at which the radioactive isotope decays to a stable form, and the age of the person at the time of ingestion, which affects both the biokinetics of the radiocesium as well as the potential length of time over which the tissues can be exposed to the radiation. The biodistribution of cesium will vary somewhat for uptake in muscle, fat, various organs, and the skeleton. Therefore, each tissue may receive a different dose equivalent. The total dose equivalent for the body will reflect the integration of the dose equivalents for the various tissues using a weighting scheme for the relative sensitivities of tissues and organs to short- and long-term effects.

The EPA has published a set of internal dose conversion factors for standard persons of various ages (newborn; 1, 5, 10, or 15 years of age; and adult) in its Federal Guidance Report No. 13 supplemental CD (EPA 2000). For example, the EPA has estimated that the dose equivalent following ingestion of 1 Bq of ^{137}Cs is 1.30×10^{-8} Sv (assuming an integration time of 50 years for an adult following the initial exposure). Age-specific dose coefficients for inhalation and ingestion of any of the radioactive isotopes of cesium by the general public can be found in ICRP publications 71 (ICRP 1995) and 72 (ICRP 1996), respectively. Dose coefficients for inhalation, ingestion, and submersion in a cloud of cesium radionuclides can be found in U.S. EPA Federal Guidance Report No. 11 (EPA, 1993a). Dose coefficients for external exposure to radioisotopes of cesium in air, surface water, or soil contaminated to various depths can be found in U.S. EPA Federal Guidance Report No. 12 (EPA, 1993b).

Adverse health effects resulting from external exposure to beta or gamma emissions from radioisotopes of cesium would be the same as those from other radioactive elements that release beta or gamma radiation, and would not be the result of exposure to cesium *per se* (refer to Section 2.2 for a discussion of dose-related symptoms of acute radiation exposure). Developmental and carcinogenic effects have been reported in Japanese survivors of acute high-dose external radiation from the atomic bombs detonated over Hiroshima and Nagasaki (see Agency for Toxic Substances and Disease Registry 1999 for a detailed description of the health effects related to ionizing radiation in general). Exposure to lower levels of radiation would be expected to result in decreased health risk. Although developmental and carcinogenic effects would be expected in individuals subjected to high radiation doses from any source of gamma radiation, non-accidental exposure to a level of significant health risk via a radiocesium source is not likely to occur.

Symptoms typical of cutaneous radiation syndrome (initial dermal erythema and subsequent ulceration) occurred among Russian military recruits who were accidentally exposed to a sealed source of ^{137}Cs (Gottlöber et al. 2000). Some of the exposed men also described symptoms of nausea, vomiting, and headache, which occurred at the onset of the dermal effects.

A number of individuals in Goiânia, Brazil, who experienced mixed external, dermal, and oral exposure to an opened ^{137}CsCl source, exhibited classic symptoms of acute radiation syndrome including vomiting, diarrhea, and nausea, as well as skin lesions from radiation burns, orofacial lesions, ocular injury, hematological effects (bone marrow aplasia, leukopenia, thrombocytopenia, lymphopenia, neutropenia), mild elevations of some liver enzymes, reduced sperm counts, and death (in four cases, attributed to infections resulting from reduced resistance) (Brandão-Mello et al. 1991; Gomes et al. 1990). External exposure was estimated based on frequencies of chromosomal aberrations in lymphocytes of exposed individuals at various times following exposure, while internal doses were estimated based on whole-body radiation counting and excretory levels of ^{137}Cs. The adverse effects were the result of beta and gamma radiation, not cesium *per se*.

Information regarding adverse effects in animals exposed to radioactive isotopes of cesium via natural routes (inhalation, oral, or dermal) is limited to observations of significantly reduced fertility and temporary sterility in male mice following single or repeated oral administration of radioactive cesium nitrate (Ramaiya et al. 1994). Postmating embryo mortality was associated with increased frequency of dominant lethal mutations. Studies of animals exposed to radiocesium via natural routes are not usually performed because levels great enough to cause significant adverse health effects would also pose a severe health risk to investigators. However, it has been shown that distribution patterns of ^{137}Cs are similar in animals exposed to relatively nontoxic levels of ^{137}CsCl by parenteral injection, inhalation exposure, or oral administration (Boecker et al. 1969a; Stara 1965). Therefore, the occurrence of depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues in dogs exposed to ^{137}CsCl via intravenous injection provide the most reasonable indication of health effects that would be expected in animals exposed to ^{137}CsCl by inhalation or oral exposure (Nikula et al. 1995, 1996).

Adverse neurological, developmental, reproductive, genotoxic, and cancer effects have been observed in animal studies employing external exposure to sealed radioactive cesium sources, and are the result of the external gamma radiation, not the cesium *per se*. Impaired motor activity, decreased thickness of cortical layers of the brain, and increased aggressive behavior were observed after the birth of rats that had been briefly exposed *in utero* to relatively high levels of external radiation from a ^{137}Cs source (Minamisawa et al. 1992; Norton and Kimler 1987, 1988). The most vulnerable developmental period was around gestational days 14–15. In another study, adverse developmental effects in fetal rats irradiated on gestational day 12 included reduced litter size, smaller head size, retarded odontogenesis, and cleft palate when examined on gestational day 18 (Saad et al. 1991, 1994). Significant increases in the formation rate of micronuclei were seen in blood cells of other fetal rats following irradiation of pregnant dams via a sealed ^{137}Cs source on gestational day 14 (Koshimoto et al. 1994). Significantly reduced fertility (including temporary sterility) was reported in male mice exposed to an external ^{137}Cs source for almost 20 days; an increased frequency of dominant lethal mutations was also indicated by increased postmating embryo mortality (Ramaiya et al. 1994). Increased lifetime risk of mammary tumors was noted in female rats that were exposed, between the ages of 8 and 36 weeks, to single whole-body doses of radiation from a ^{137}Cs source (Bartstra et al. 1998). Irradiation at 64 weeks, however, yielded fewer carcinomas than unirradiated controls.

3.2 DISCUSSION OF HEALTH EFFECTS OF STABLE CESIUM BY ROUTE OF EXPOSURE

Section 3.2 discusses the chemical toxicity of cesium. Radiation toxicity resulting from exposure to radiocesium is discussed in Section 3.3.

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, and dermal) 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.

Estimates of exposure levels posing minimal risk to humans (Minimal Risk Levels or MRLs) have been made for cesium. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure. 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 within a given route of exposure. MRLs are based on noncancerous health effects only and do not consider carcinogenic effects. MRLs can be derived for acute, intermediate, and chronic duration exposures for inhalation and oral routes. Appropriate methodology does not exist to develop MRLs for dermal exposure.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 1990b), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

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.

3.2.1 Inhalation Exposure

No reports were located regarding the following health effects in humans or animals following inhalation exposure to stable cesium:

3.2.1.1 Death

3.2.1.2 Systemic Effects

3.2.1.3 Immunological and Lymphoreticular Effects

3.2.1.4 Neurological Effects

3.2.1.5 Reproductive Effects

3.2.1.6 Developmental Effects

3.2.1.7 Cancer

3.2.2 Oral Exposure

Limited information is available regarding health effects following oral exposure of humans to stable cesium compounds. Symptoms of decreased appetite, nausea, diarrhea, and cardiac arrhythmia have been associated with consumption of cesium chloride (Bangh et al. 2001; Harik et al. 2002; Neulieb 1984; Saliba et al. 2001). Death was reported laboratory animals following oral administration of large doses of cesium compounds (Ghosh et al. 1990; Johnson et al. 1975; Khosid 1967).

3.2.2.1 Death

No reports were located regarding death in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No studies were located regarding death in animals following intermediate- or chronic-duration oral exposure to stable cesium. However, acute oral administration of cesium at high dose levels has resulted in observed mortality in rats and mice. In female mice administered cesium chloride, reported oral LD_{50} values range from 2,300 to 2,500 mg/kg (Ghosh et al. 1990; Khosid 1967). An acute oral LD_{50} value for cesium iodide is 2,386 mg/kg in rats (Johnson et al. 1975). Cesium hydroxide appears to be more highly toxic to rats than cesium chloride and cesium iodide, as evidenced by a lower LD_{50} value of 1,026 mg/kg (Johnson et al. 1975). Some of the toxic effects can be attributed to the strong alkaline nature of cesium hydroxide. Khosid (1967) estimated an acute oral LD_{50} value of 800 mg/kg for mice that were administered cesium hydroxide. No information was located regarding mortality in animals following oral administration of other compounds of stable cesium.

3.2.2.2 Systemic Effects

Decreased appetite, nausea, and diarrhea were reported in a man who ingested about 34 mg Cs/kg (as stable cesium chloride) after morning and evening meals for 36 days; this man also experienced apparent neurological changes within 15 minutes of dosing (Neulieb 1984). Prolonged QT syndrome and associated cardiac arrhythmias have been described in patients who have ingested cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001). No additional information was located regarding systemic effects in humans following oral exposure to stable cesium.

No animal studies were located in which the systemic effects that were observed following oral administration of compounds of stable cesium could be attributed to the presence of cesium. Gastrointestinal and respiratory effects noted in rats administered acute oral doses of cesium hydroxide may have been due to the alkaline properties of the compound rather than the biochemical behavior of cesium.

No data were located regarding, hematological, musculoskeletal, endocrine, dermal, ocular, or metabolic effects in humans or animals following oral exposure to stable cesium.

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

No reports were located regarding respiratory effects in animals following intermediate- or chronic-duration exposure to stable cesium. Congested, cyanotic lungs with petechial hemorrhages were observed in rats following oral treatment with single cesium iodide doses large enough to cause death. A bloody nasal exudate was seen in some relatively high-dose rats (Johnson et al. 1975).

Cardiovascular Effects. Individual case reports describe prolonged QT syndrome and associated cardiac arrhythmia in patients who consumed cesium chloride as a component of homeopathic remedies for cancer prevention (Saliba et al. 2001), tumor reduction (Harik et al. 2002), and cyst reduction (Bangh et al. 2001. No reports were located regarding cardiovascular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

Gastrointestinal Effects. No reports were located regarding gastrointestinal effects in humans following acute- or chronic-duration oral exposure to stable cesium, and only one report of intermediate-duration exposure was located. An investigator who voluntarily ingested about 34 mg Cs/kg (as cesium chloride, assuming 70 kg body weight) after each morning and evening meal (68 mg Cs/kg/day) for 36 days reported gradually decreased appetite, prenausea feelings, and diarrhea. The observations were self-described and effects were correlated to dietary habits during the course of the study (Neulieb 1984).

No reports were located regarding gastrointestinal effects in animals following intermediate- or chronic-duration oral exposure to stable cesium. In an acute lethality study of rats, administration of cesium hydroxide or cesium iodide at dose levels up to 910 or 1,217 mg Cs/kg, respectively, resulted in stomach and intestinal hemorrhage, bloody fluid exudate within the peritoneal cavity, and adhesions of abdominal organs. Rats receiving lethal doses of cesium iodide exhibited fluid-filled stomach (Johnson et al. 1975).

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

No reports were located regarding hepatic effects in animals following acute- or chronic-duration oral exposure to stable cesium. No significant effect on maternal liver weight was noted in rats consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

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

No reports were located regarding renal effects in animals following acute- or chronic-duration oral intake of stable cesium, and one report was located regarding intermediate-duration intake. In that study, no significant effect on maternal kidney weight was noted in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

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

No reports were located regarding body weight effects in animals following acute- or chronic-duration oral exposure to stable cesium. One intermediate-duration study found no significant effect on maternal body weight in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

3.2.2.3 Immunological and Lymphoreticular Effects

No reports were located regarding immunological or lymphoreticular effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding immunological or lymphoreticular effects in animals following acute- or chronic-duration oral exposure to stable cesium. In one study of intermediate-duration, no significant effect on maternal spleen weight was noted in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

3.2.2.4 Neurological Effects

No reports were located regarding neurological effects in humans following acute- or chronic-duration oral exposure to stable cesium. In one study of intermediate-duration exposure, an investigator, who voluntarily ingested about 34 mg Cs/kg/day (as stable cesium chloride) after morning and evening meals for 36 days, experienced general feelings of well-being, heightened sense perception, and tingling sensations in lips, cheeks, hands, and feet within 15 minutes of intake (Neulieb 1984). No self-reported adverse effects were noted in performance of mathematical tasks or in automobile driving skill.

No reports were located regarding neurological effects in animals following intermediate- or chronic-duration oral exposure to stable cesium. Rats administered unspecified acute gavage doses of cesium hydroxide exhibited initial signs of hyperexcitability followed by apathy and weakness during the course of 14 days of observation after dosing (Johnson et al. 1975).

3.2.2.5 Reproductive Effects

No reports were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

3.2.2.6 Developmental Effects

No reports were located regarding developmental effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding developmental effects in animals following acute- or chronic-duration oral exposure to stable cesium. In intermediate-duration studies, one investigator reported reduced body weight in offspring of mouse dams consuming approximately 115 mg Cs/kg/day during gestation and

40 mg Cs/kg/day during lactation (Messiha 1988b). Other observations included slight, but significant, changes in some organ weights and slight differences in activity of some hepatic enzymes among offspring of treated dams, relative to controls. Similar results were reported in the offspring of female mice consuming approximately 40 mg Cs/kg/day only during lactation (Messiha 1989b). However, gross and histopathologic examinations of the offspring, typical of well-designed developmental toxicity studies, were not performed in either of these studies, making them of little value for assessment of the developmental toxicity potential of cesium.

3.2.2.7 Cancer

No reports were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

3.2.3 Dermal Exposure

3.2.3.1 Death

No reports were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

3.2.3.2 Systemic Effects

No reports were located in which respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, body weight, or metabolic effects in humans or animals could be associated with dermal exposure to stable cesium.

Dermal Effects. No reports were located regarding dermal effects in humans following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

No reports were located regarding dermal effects in animals following intermediate- or chronic-duration dermal exposure to stable cesium. Cesium hydroxide was considered a nonirritant on intact skin and a mild irritant on abraded skin of rabbits 24 and 48 hours, respectively, following closed-patch application of a 5% solution; similar application of cesium iodide resulted in no observed irritation (Johnson et al. 1975).

Ocular Effects. No reports were located regarding eye irritation in humans resulting from ocular contact with stable cesium.

A 5% solution of cesium hydroxide was extremely irritating and caustic to the rabbit eye (Johnson et al. 1975). It is likely that this effect was the result of the caustic nature of the hydroxide rather than an effect due to cesium *per se*. Similar treatment with cesium iodide resulted in no evidence of ocular irritation.

3.2.3.3 Immunological and Lymphoreticular Effects

No reports were located regarding immunological or lymphoreticular effects in humans following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

No reports were located regarding immunological or lymphoreticular effects in animals following acute- or chronic-duration dermal exposure to stable cesium. There was no indication of a sensitization response in guinea pigs following repeated intracutaneous injections of 0.1% solutions of cesium hydroxide or cesium iodide (Johnson et al. 1975).

No reports were located regarding the following health effects in humans or animals following dermal exposure to stable cesium:

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 Other Routes of Exposure

3.2.4.1 Death

No data were located regarding death in humans or animals following exposure to stable cesium via routes other than inhalation, oral, or dermal exposure.

3.2.4.2 Systemic Effects

No data were located regarding respiratory, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, dermal, ocular, body weight, or metabolic effects in humans or animals following exposure to stable cesium via routes other than inhalation, oral, or dermal exposure.

Cardiovascular Effects. Cardiac arrhythmias were elicited in experimental animals following the injection of stable cesium chloride directly into the circulatory system (Brachmann et al. 1983; Levine et al. 1985; Murakawa et al. 1997; Patterson et al. 1990; Senges et al. 2000).

No reports were located in which the following health effects in humans or animals could be associated with exposure to stable cesium via routes other than inhalation, oral, or dermal exposure:

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

3.3 DISCUSSION OF HEALTH EFFECTS OF RADIOACTIVE CESIUM BY ROUTE OF EXPOSURE

Section 3.3 discusses radiation toxicity associated with exposure to radionuclides of cesium and is organized in the same manner as that of Section 3.2, first by route of exposure (inhalation, oral, and external) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, 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 NOAELs or LOAELs reflect the actual dose (levels of exposure) used in the studies. Refer to Section 3.2 for detailed discussion of the classification of endpoints as a NOAEL, less serious LOAEL, or serious LOAEL.

Refer to Appendix B for a User's Guide, which should aid in the interpretation of the tables and figures for Levels of Significant Exposure.

3.3.1 Inhalation Exposure

During and after nuclear accidents, such as the steam explosion that occurred at the Chernobyl nuclear power plant in 1986, significant amounts of ^{137}Cs (and to a lesser extent ^{134}Cs) may be released to the atmosphere in suspended particles and be widely dispersed through the air. Although radioactive cesium, suspended in the air following such accidents or re-suspended later from ground-deposited fallout (USNRC 1998), may be internalized via inhalation, there was no indication that inhalation was a significant route of exposure to radioactive cesium among individuals exposed externally by either being in the vicinity of a release or in areas receiving substantial ground-deposited fallout, or those exposed by ingestion of radioactive cesium-contaminated food following the Chernobyl accident (Balonov 1993).

No reports were located regarding health effects in humans or animals following inhalation exposure to radioactive cesium. Available human case reports and animal studies involving inhaled radioisotopes of cesium deal exclusively with biokinetics. Parenteral injection of ^{137}CsCl in laboratory animals has resulted in distribution patterns and tissue doses of ^{137}Cs that are similar to those resulting from inhalation or oral exposure (Boecker et al. 1969a; Stara 1965). For these reasons, it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as ^{137}CsCl, should be similar across the three routes of exposure (Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Therefore, depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs, effects that have been observed in dogs following intravenous administration of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972), would be expected to occur following inhalation exposure to air concentrations of ^{137}CsCl that would result in comparable ^{137}Cs blood concentrations (see Section 3.3.4 for additional information regarding exposure other than inhalation, oral, dermal, or external exposure).

3.3.1.1 Death

Although no studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium, dose-related decreased survival was observed in beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in animals exposed to air concentrations of $^{137}CsCl$ that would result in similar body burdens (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.2 Systemic Effects

No data were located regarding systemic effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, hematological effects similar to those observed in dogs that had received single intravenous injections of $^{137}CsCl$ (Nikula et al. 1995, 1996; Redman et al. 1972) would be expected in animals exposed to air concentrations of $^{137}CsCl$ that would result in body burdens similar to those attained via intravenous injection.

Hematological Effects. Depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of $^{137}CsCl$, which resulted in average initial body burdens ranging from 36.4 to 141.0 MBq/kg (1.0 to 3.8 mCi/kg) (Nikula et al. 1995; Redman et al. 1972). Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of $^{137}CsCl$ at levels resulting in average initial body burdens in the range of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1996). Similar effects would be expected in dogs exposed to air concentrations of $^{137}CsCl$ that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.3 Immunological and Lymphoreticular Effects

No data were located regarding immunological or lymphoreticular effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, severe bone marrow depression was observed in dogs exposed to $^{137}CsCl$ by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula et al. 1995). Similar effects would be expected in dogs exposed to air concentrations of $^{137}CsCl$ that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.4 Neurological Effects

No data were located regarding neurological effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium.

3.3.1.5 Reproductive Effects

No data were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, germinal epithelium damage and azoospermia were reported in dogs that had been administered ^{137}CsCl by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 7.42 to 16.40 Gy (742 to 1,640 rad) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.6 Developmental Effects

No data were located regarding developmental effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium.

3.3.1.7 Cancer

No data were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of ^{137}CsCl, which resulted in average initial body burdens ranging from 37 to 147 MBq/kg (1.0 to 4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.2 Oral Exposure

No reports were located regarding health effects in humans or animals that could be exclusively associated with oral exposure to radioisotopes of cesium. Available human case reports and animal studies involving ingested radioisotopes of cesium deal with biokinetics. Parenteral injection of ^{137}CsCl in laboratory animals has resulted in distribution patterns and tissue doses of ^{137}Cs that are similar to those resulting from inhalation or oral exposure (Boecker et al. 1969a; Stara 1965). For these reasons, it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as ^{137}CsCl, should be similar across the three routes of exposure (Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Therefore, depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs, effects that have been observed in dogs following intravenous administration of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972), would be expected to occur following oral administration of ^{137}CsCl in amounts that would result in comparable ^{137}Cs blood concentrations (see Section 3.3.4 for additional information regarding exposure other than inhalation, oral, dermal, or external exposure).

3.3.2.1 Death

No reports were located regarding death in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. In an event involving mixed external, dermal, and oral exposure of adults and children to an opened ^{137}CsCl source, significant short-term morbidity was followed in 50 patients and 4 deaths were reported within a few weeks among individuals with estimated radiation doses ranging from 4.5 to 6 Gy (450 to 600 rad) (Brandão-Mello et al. 1991).

Although no studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium, decreased survival was observed in young adult beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar results would be expected in animals following oral exposure to ^{137}CsCl levels that would result in similar body burdens (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.2.2 Systemic Effects

Acute radiation syndrome, characterized by nausea, vomiting, and diarrhea was reported in a number of individuals following mixed external, dermal, and oral exposure to an opened 50.9 TBq (1,375 Ci) ^{137}CsCl source in Goiânia, Brazil. Other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). Hematological effects similar to those observed in dogs that had received single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972) would be expected in animals following oral administration of ^{137}CsCl at levels that would result in body burdens similar to those attained via intravenous injection.

No data were located regarding, respiratory, cardiovascular, musculoskeletal, renal, endocrine, body weight, or metabolic effects in humans or animals following oral exposure to radioactive cesium.

Gastrointestinal Effects. Vomiting, diarrhea, and nausea were observed among eight patients treated for acute radiation exposure to an opened ^{137}CsCl source, both via external exposure and internal exposure (Brandão-Mello et al. 1991). These and other symptoms were classic symptoms of acute radiation syndrome. No reports were located regarding gastrointestinal effects in humans following intermediate- or chronic-duration oral exposure to radioactive cesium

No reports were located for animals regarding gastrointestinal effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Hematological Effects. No reports were located for humans regarding hematological effects following intermediate- or chronic-duration oral exposure to radioactive cesium. In the 1987 incident of overexposure to a scavenged medical radiation source containing 50.9 TBq (1,375 Ci) ^{137}CsCl, approximately 250 persons were contaminated externally, many of whom also were contaminated internally. Twenty individuals developed the acute radiation syndrome, 14 of whom developed bone marrow failure after having received whole-body radiation doses ranging from 1 to 7.0 Gy (100 to 700 rad). Four of these 14 heavily contaminated individuals died. These effects are typical signs and symptoms of the hemopoietic (blood forming) syndrome in which the blood forming cells in the bone marrow are killed. This results in sharp decreases of all the blood cells and consequent impairment of the immune system and anemia (Brandão-Mello et al. 1991; Gomes et al. 1990).

No reports were located for animals regarding hematological effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of ^{137}CsCl, which resulted in average initial body burdens ranging from 36.4 to 141.0 MBq/kg (1.0 to 3.8 mCi) (Nikula et al. 1995; Redman et al. 1972). Severely depressed blood cell counts were observed in 23 dogs that died from 20 to 52 days following single intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 122–164 MBq/kg (3.3–4.4 mCi/kg) (Nikula et al. 1996) and in 11 other dogs (Nikula et al. 1995) that died from 19 to 81 days following intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 72–141 MBq/kg (1.9–3.8 mCi/kg). Similar effects would be expected in dogs following oral administration of ^{137}CsCl at levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

Hepatic Effects. No reports were located that associate acute-duration oral exposure to radioactive cesium with hepatic effects. Mild elevations of aminotransferases (ALT/AST) were seen in a few patients hospitalized following radiation exposure to an opened ^{137}CsCl source (Brandão-Mello et al. 1991). Exposures were both external and internal. No reports were located for humans regarding hepatic effects following intermediate- or chronic-duration oral exposure to radioactive cesium.

No reports were located for animals regarding hepatic effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Dermal Effects. Reports of dermal effects are restricted to the accidental mixed external, dermal, and oral exposure of a number of individuals to an opened ^{137}CsCl source in which orofacial lesions, including oral bleeding and associated oral rash, mouth ulcers, acute oral candidiasis, and radiation dermatitis and depigmentation, were observed in 21 patients who had been acutely exposed at estimated radiation doses ranging from 1 to 7 Gy (100 to 700 rad) (Gomes et al. 1990). Some individuals exposed in the same incident exhibited typical radiation-induced skin lesions; the forearm was amputated in one individual with severe radiation injury (Brandão-Mello et al. 1991; Gomes et al. 1990).

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

Ocular Effects. No reports were located regarding ocular effects in humans following intermediate- or chronic-duration oral exposure to radioactive cesium. Among 20 patients hospitalized following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source, a few patients complained of lacrimation, hyperemia and edema of the conjunctiva, and ocular pain (Brandão-Mello et al. 1991). A few cases of protracted reduction in visual capacity were also reported, among which retinal injury was documented. In these cases, there was no change in lens transparency. These effects were due to the radiation, not to cesium *per se*.

No reports were located regarding ocular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.3.2.3 Immunological and Lymphoreticular Effects

No reports were located that associate immunological or lymphoreticular effects in humans with intermediate- or chronic-duration oral exposure to radioactive cesium. Severe bone marrow depression, characterized by a low white blood cell count and consequent immunodeficiency, developed in 14 patients hospitalized following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source resulting in estimated absorbed doses ranging from 1 to 7 Gy (100 to 700 rad) (Brandão-Mello et al. 1991).

No reports were located regarding immunological or lymphoreticular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, severe bone marrow depression was observed in dogs exposed to ^{137}CsCl by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula et al. 1995). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.2.4 Neurological Effects

No reports were located for humans or animals that associate neurological effects with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.3.2.5 Reproductive Effects

No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration oral exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following presumed acute radiation doses on the order of several hundred rad from an opened ^{137}CsCl source (Brandão-Mello et al. 1991). These individuals may have experienced mixed external, dermal, and oral exposure.

No reports were located regarding reproductive effects in animals following intermediate- or chronic-duration oral exposure to radioactive cesium. Significantly reduced fertility, expressed as the percentage of matings resulting in pregnancy (percent effective matings), was noted in male mice following a single oral administration of ^{137}Cs (as cesium nitrate) at activity levels that resulted in a total dose to the testis of approximately 3 Gy (300 rad) (Ramaiya et al. 1994). Complete, though temporary, sterility was evident at week 6 after dosing. By week 17, there were no significant differences in fertility between treatment and control groups. No significant reduction in male fertility was observed at activity levels resulting in cumulative doses to the testes ranging from 0.1 to 1 Gy (10 to 100 rad). Significantly reduced fertility was also evident in male mice administered daily oral doses of ^{137}Cs (as cesium nitrate) for 2 weeks that resulted in total testicular radiation doses of about 3.85 Gy (385 rad), measured at 5 weeks after treatment (Ramaiya et al. 1994).

Germinal epithelium damage and azoospermia were reported in dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 7.42 to 16.40 Gy (742 to 1,640 rad) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

The highest NOAEL values and all reliable LOAEL values for reproductive effects from oral exposure to radioactive cesium are presented in Table 3-1 and plotted in Figure 3-1.

Table 3-1 Levels of Significant Exposure to Cesium - Radiation Toxicity - Oral

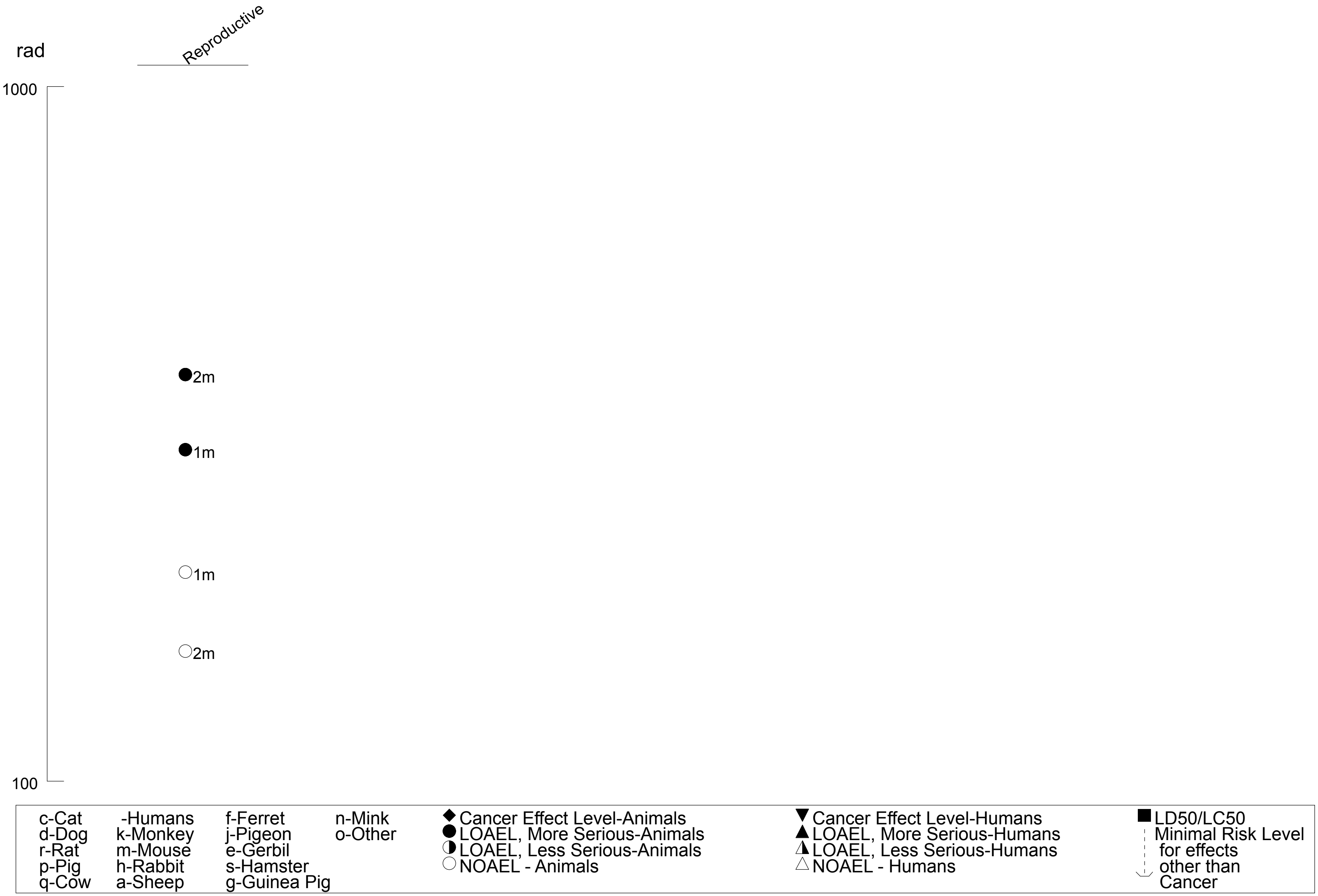
Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	ACUTE EXPOSURE						
	Reproductive						
1	Mouse (Hybrid)	Once (G)		200 M		300 M (temporary sterility)	Ramaiya et al. 1994 Cesium 137(as Cesium Nitrate)
2	Mouse (Hybrid)	2 wk 1x/d (G)		154 M		385 M (reduced fertility)	Ramaiya et al. 1994 Cesium 137(as Cesium Nitrate)

a The number corresponds to entries in Figure 3-1.

d=day(s); (G) = gavage; LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; wk = week(s)

Figure 3-1. Levels of Significant Exposure to Cesium - Radiation Toxicity - Oral

Acute (≤14 day)



3.3.2.6 Developmental Effects

No reports were located that associate developmental effects in humans or animals with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.3.2.7 Cancer

No reports were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of $^{137}CsCl$, which resulted in initial body burdens ranging from 37 to 147 MBq/kg (1.0 to 4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs following oral exposure to $^{137}CsCl$ levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.3 External Exposure

This section contains information regarding health effects related to external exposure to radioactive cesium sources. Radionuclides of cesium, such as ^{137}Cs and ^{134}Cs, emit both beta particles and gamma rays, which are a health hazard in living organisms because they are capable of ionizing atoms that they encounter. Beta particles can travel appreciable distances in air, but travel only a few millimeters in tissues. External exposure to beta particles may result in damage to skin and superficial body tissues, but is not a threat to internal organs unless the radiation source is internalized. Gamma radiation, on the other hand, can easily pass completely through the human body and cause ionization of atoms in its path. Several feet of concrete or a few inches of lead or other high-density shielding are required for protection from gamma rays. Because it is so highly penetrating, external gamma radiation emitted by radionuclides such as cesium is a radiation hazard to internal organs (Agency for Toxic Substances and Disease Registry 1999; EPA 1998).

The purpose of this section is to provide information regarding health effects associated with external exposure to a radioactive cesium source. External exposure to radioactive cesium is simply exposure to beta and gamma radiation; there is nothing unique to cesium *per se*. The same hazards exist from external exposure to any source of beta and gamma radiation. Refer to Agency for Toxic Substances and Disease Registry (1999) for a detailed description of health effects from external exposure to ionizing radiation in general.

3.3.3.1 Death

There are no reports of deaths in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Death was noted within a few weeks following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source that resulted in estimated radiation doses ranging from 4 to 6 Gy (400 to 600 rad) (Brandão-Mello et al. 1991). See Section 3.3.2.1 for more detailed information.

Significantly reduced survival was noted in rat fetuses following whole-body irradiation (via a ^{137}Cs source) of pregnant dams on gestational day 14 at acute radiation doses ≤4 Gy (400 rad); an LD_{50} value was about 5 Gy (500 rad) (Koshimoto et al. 1994). No reports were located regarding death in animals following intermediate- or chronic-duration external exposure to radioactive cesium.

3.3.3.2 Systemic Effects

Symptoms typical of cutaneous radiation syndrome (initial dermal erythema and subsequent ulceration) occurred among Russian military recruits who were accidentally exposed to a sealed source of ^{137}Cs during training in 1996 and 1997 (Gottlöber et al. (2000). Some of the exposed men also described symptoms of acute radiation syndrome (nausea, vomiting, and headache), which occurred at the onset of the dermal effects. Nausea, vomiting, and diarrhea, were reported in a number of individuals following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source in Goiânia, Brazil; other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). See Section 3.3.2.2 for a more detailed description of systemic effects following mixed exposure to radioactive cesium.

No reports were located regarding respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, ocular, body weight, metabolic, or other systemic effects in humans or animals that could be exclusively associated with external exposure to radioactive cesium.

Dermal Effects. Initial dermal erythema progressed to ulceration within a few weeks following initial accidental exposure of Russian military recruits to a sealed external source of ^{137}Cs gamma radiation (Gottlöber et al. 2000). However, there was no information regarding radiation doses to these individuals.

3.3.3.3 Immunological and Lymphoreticular Effects

No reports were located that associate immunological or lymphoreticular effects in humans with intermediate- or chronic-duration external exposure to radioactive cesium. Severe bone marrow depression, characterized by neutropenia and thrombocytopenia, developed in 14 patients hospitalized following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source resulting in estimated absorbed doses ranging from 1 to 7 Gy (100 to 700 rad) (Brandão-Mello et al. 1991). This effect was the result of the radiation, not the presence of cesium *per se*.

No reports were located regarding immunological or lymphoreticular effects in animals following acute-, intermediate-, or chronic-duration external exposure to radioactive cesium.

3.3.3.4 Neurological Effects

No reports were located that associate neurological effects in humans or animals with postnatal acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Neurological effects associated with *in utero* exposure are discussed in Section 3.3.3.6, Developmental Effects.

3.3.3.5 Reproductive Effects

No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration external exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following presumed acute exposure to an opened ^{137}CsCl source (Brandão-Mello et al. 1991). These individuals may have experienced mixed external, dermal, and oral exposure.

No reports were located regarding reproductive effects in animals following acute- or chronic-duration external exposure to radioactive cesium. Significantly reduced fertility, expressed as the percentage of matings resulting in pregnancy (% effective matings), was noted in male mice following external exposure to a ^{137}Cs source 23 hours a day for 19.5 days at a dose rate of 0.675 mGy/hour (6.75 mrad/hour), resulting in a total dose of 3Gy (300 rad) (Ramaiya et al. 1994). Complete sterility was evident in males by the third week following exposure termination. During weeks 1 and 2 after the cessation of treatment, significantly increased total and postimplantation embryo mortality was noted. These effects were the result of the radiation, not the presence of cesium *per se*.

All reliable LOAEL values for reproductive effects from external exposure to radioactive cesium are presented in Table 3-2 and plotted in Figure 3-2.

3.3.3.6 Developmental Effects

No reports were located that associate developmental effects in humans with acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Cells of the developing central nervous system are among the most sensitive to the effects of ionizing radiation in the developing fetus (Agency for Toxic Substances and Disease Registry 1999). Schull and Otake (1999) cite numerous reports in which impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed (during weeks 8–15 or 16–25 after ovulation) to ionizing radiation from the bombs. The small number (38) of mentally-impaired survivors makes it difficult to generalize a dose-response relationship. However, the data are compatible with either a threshold dose of 20–40 rad (0.2–0.4 Gy) or zero threshold linear response. Although such effects would be expected in individuals exposed to similar levels of external radiation from any source of gamma radiation, it is unlikely that such high levels would be achieved from any radiocesium source under situations of normal use.

No reports were located regarding developmental effects in animals following intermediate- or chronic-duration external exposure to radioactive cesium. Significantly reduced postnatal body weight, impaired motor activity, and decreased thickness within cortical layers of the brain were observed in young rats (7–21 days postpartum) exposed during gestation by whole-body radiation of pregnant dams via a sealed ^{137}Cs gamma radiation source at a rate of approximately 0.5 Gy/minute (50 rad/minute) for a total radiation dose of 0.75 or 1 Gy (75 or 100 rad) (Norton and Kimler 1987, 1988). The effects were of larger magnitude when the fetal rats were exposed on gestation day 15 rather than earlier (gestation days 11 or 13) or later (gestation day 17). Significantly smaller litter size, smaller head size, and retarded odontogenesis were observed in fetuses of pregnant mice exposed on gestation day 12 to whole-body irradiation from a sealed ^{137}Cs gamma radiation source for 4.9 minutes (resulting in a total radiation dose

Table 3-2 Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
ACUTE EXPOSURE							
Developmental							
1	Rat (Wistar)	Once Gd 13, 14, or 15			50 (increased micronuclei in hematocytes)	400 (31.5% fetal mortality)	Koshimoto et al. 1994 Cesium 137
2	Rat (Sprague-Dawley)	Once Gd 11 or 17				100 (altered motor function, decreased cortical thickness)	Norton and Kimler 1987 Cesium 137
3	Rat (Sprague-Dawley)	Once Gd 13, 15, or 17				75 (altered motor function, decreased cortical thickness, most prominent when exposed on Gd 15)	Norton and Kimler 1988 Cesium 137
4	Mouse (C57BL/6)	Once Gd 14			100 M (6% lower body weight, 9% lower brain weight, reduced brain size)		Minamisawa et al. 1990 Cesium 137
5	Mouse (C57BL/6)	Once Gd 14			100 M (16% lower body weight, increased aggressive behavior)		Minamisawa et al. 1992 Cesium 137
6	Mouse Swiss albino	Once Gd 12			400 (retarded odontogenesis)		Saad et al. 1991 Cesium 137

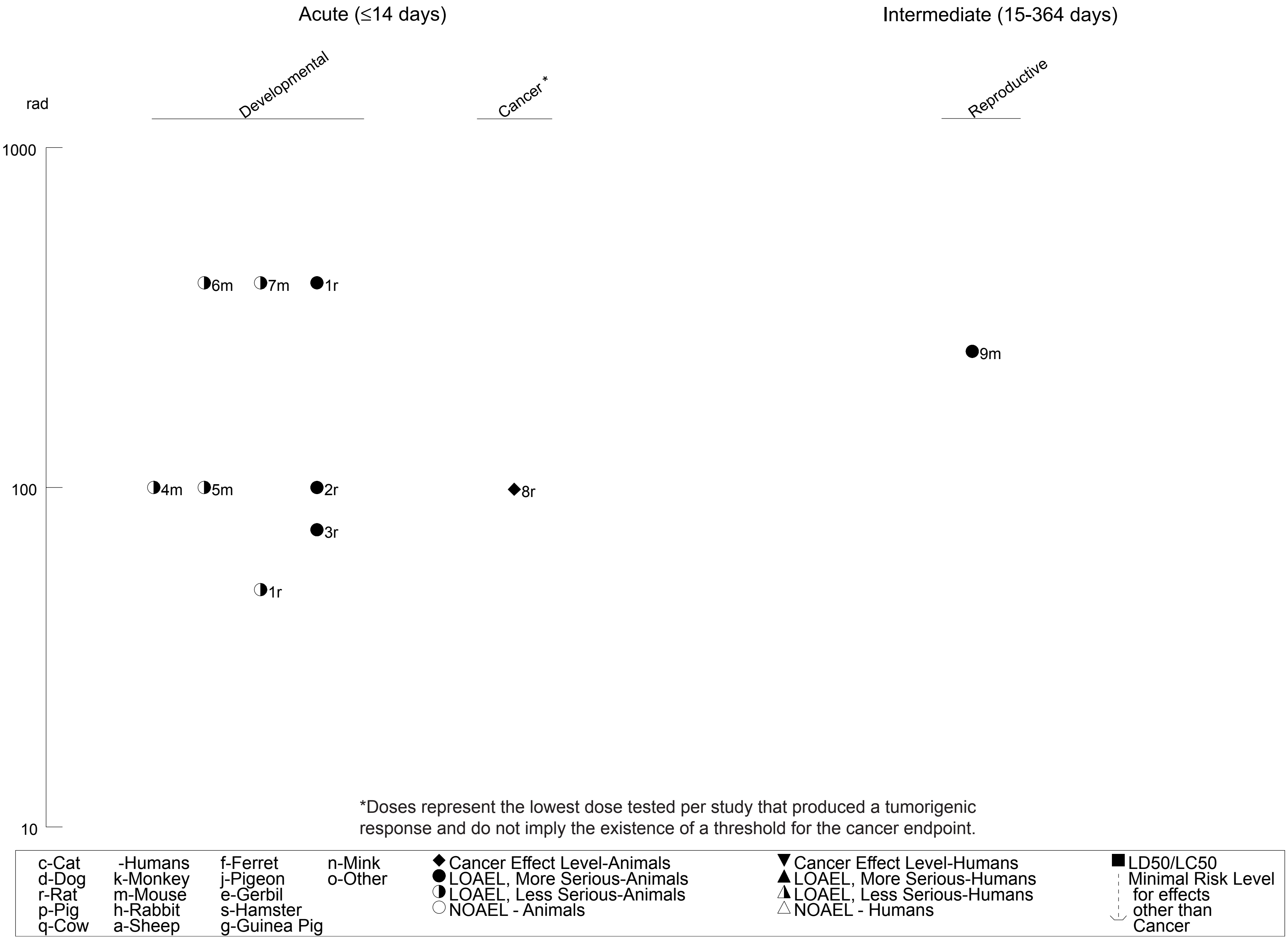
Table 3-2 Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
7	Mouse Swiss albino	Once Gd 12			400 (smaller litter size, smaller head size, delayed palatogenesis)		Saad et al. 1994 Cesium 137
	Cancer						
8	Rat WAG/Rij	Once at 8, 12, 16, 22, 36, or 64 wk of age				100 F (mammary carcinoma in 57/200 rats)	Bartstra et al. 1998 Cesium 137
	INTERMEDIATE EXPOSURE						
	Reproductive						
9	Mouse (Hybrid)	19.5 d 23 hr/d				300 M (reduced fertility)	Ramaiya et al. 1994 Cesium 137

a The number corresponds to entries in Figure 3-2.

d=day(s); F = Female; Gd = gestational day; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; wk = week(s).

Figure 3-2. Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation



*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer endpoint.

of 4 Gy or 400 rad) and examined on gestation day 18. Additionally, all exposed fetuses exhibited cleft palate on examination while normal closure was observed in all unexposed control fetuses (Saad et al. 1991, 1994). Significantly reduced survival was observed in rat fetuses following whole-body irradiation (via a sealed ^{137}Cs gamma radiation source) of pregnant dams on gestation day 14 at acute radiation doses of 4 Gy (400 rad) or greater. An LD_{50} value for fetuses was about 5 Gy (500 rad) (Koshimoto et al. 1994). Aggressive behavior was studied in male mice (100–135 days of age) exposed *in utero* on gestation day 14 through whole-body irradiation of pregnant dams via an external sealed ^{137}Cs gamma radiation source at total radiation doses of 1 or 2 Gy (100 or 200 rad) (Minamisawa et al. 1992). Incidences of aggressive behavior were significantly higher among irradiated groups, relative to untreated controls. The intensity of aggressive behavior was significantly higher only in the 2 Gy (200 rad) exposure group. Minamisawa et al. (1990) found dose-related significantly decreased brain weight in 6-month-old mice that had been irradiated on gestation day 14 at doses of 1–3 Gy (100–300 rad). In each of these studies (Koshimoto et al. 1994; Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988), the observed developmental effects were the result of radiation exposure, not the presence of cesium *per se*.

Kusama and Hasegawa (1993) designed a study to examine the relationship between fetal developmental stage at the time of external exposure to a sealed ^{137}Cs gamma radiation source and the occurrence of external malformations and growth retardation. Groups of pregnant ICR mice were irradiated once with 1.5 Gy (150 rad) at a dose rate of 0.2 Gy/minute (20 rad/minute) on 6-hour intervals during the period of organogenesis (gestation days 6.5–14). Fetuses were examined on gestation day 18. The authors reported peaks in the occurrence of exencephaly among fetuses irradiated during gestation days 6.5–8.75 and 10.25–10.75, and the highest peak occurred at gestation day 7.5. Peaks in the occurrence of cleft palate were seen in fetuses irradiated at gestation days 8.75 and 10.75. The most apparent reduction in body weight of irradiated fetuses, relative to controls, occurred in groups irradiated between gestation days 9.75 and 11. The observed developmental effects were the result of radiation exposure, not the presence of cesium *per se*.

All reliable LOAEL values for developmental effects from external exposure to radioactive cesium are presented in Table 3-2 and plotted in Figure 3-2.

3.3.3.7 Cancer

No reports were located regarding cancer in humans following acute-, intermediate-, or chronic-duration external exposure to radioactive cesium in particular. Due to the nature of ionizing radiation in general, carcinogenic effects similar to those observed in Japanese survivors of the 1945 atomic bombing incidents might be expected among individuals acutely exposed to high levels of radiation from a radioactive cesium source. In the only available reports of adverse health effects in humans following exposure to radioactive cesium (^{137}Cs, the accidental human exposures in Goiânia, Brazil in 1987 and Russia in 1996 and 1997), the incidents are too recent for meaningful data on the potential for carcinogenicity. No reports were located in which increased cancer risk could be associated with long-term exposure to low levels of ionizing radiation.

No reports were located regarding cancer in animals following intermediate- or chronic-duration external exposure to radioactive cesium. Increased lifetime risk of mammary tumors was observed in female WRG/Rij rats exposed to single whole-body doses of 1 or 2 Gy (100 or 200 rad) of ^{137}Cs gamma radiation at a dose rate of 75 rad/minute (0.75 Gy/minute) between the ages of 8 and 36 weeks (Bartstra et al. 1998). The excess normalized risk values for carcinoma were 0.9 and 2.2 for 1 and 2 Gy (100 and 200 rad) doses, respectively, with no significant differences between the age groups of 8, 12, 16, 22, or 36 weeks. Irradiation at 64 weeks, however, yielded fewer carcinomas than unirradiated controls. The excess normalized risk values were found to be -0.7 and -0.3 for 1 and 2 Gy (100 and 200 rad) doses, respectively. These effects were the result of the gamma radiation, not the presence of cesium *per se*.

3.3.4 Other Routes of Exposure

Although parenteral injection is not considered to be an exposure route of concern for the general population, it has been considered to be a good indicator of adverse health effects that would be expected in laboratory animals following the absorption of ^{137}CsCl into the blood if such animals were to be exposed via inhalation or oral routes (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). This argument is based on the results of biokinetic studies in dogs (Boecker et al. 1969a) in which intravenous injections of ^{137}CsCl resulted in temporal and tissue distribution patterns and tissue doses of ^{137}Cs that were similar to those resulting from inhalation exposure (Boecker et al. 1969a). Similar tissue distribution and retention kinetics were also shown in guinea pigs whether exposure to ^{137}CsCl had been via intraperitoneal, inhalation, or oral routes (Stara 1965).

In dogs, intravenous administration of soluble ^{137}CsCl has resulted in depression of a number of blood factors, severe bone marrow depression, germinal cell damage (males), and early death (Nikula et al. 1995, 1996; Redman et al. 1972). Long-term surviving dogs exhibited increased incidences of benign and malignant neoplasms in a variety of tissues and organs, with no apparent single target organ of toxicity.

3.3.4.1 Death

Dose-related decreased survival was observed in young adult beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 71.7–141 MBq/kg (1.9–3.8 mCi/kg) (Nikula et al. 1995). All six dogs in the highest exposure group died between 19 and 33 days following injection. The total whole body radiation dose to death in this group of dogs averaged 11.8 Gy (1,180 rad). Deaths were attributed to severe pancytopenia resulting from hematopoietic cell damage. In a study of 63 other beagle dogs, intravenous injection of ^{137}CsCl resulted in initial body burdens of approximately 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1996). These dogs, grouped according to age, were juveniles (142–151 days old), young adults (388–427 days old), or middle-aged adults (1,387–2,060 days old) at the time of injection. Early mortality, within 52 days following injection, was noted in 10/10 middle-aged dogs, 10/38 young adults, and 3/15 juveniles. Average initial ^{137}Cs body burdens among the early deaths were in the range of 133–147 MBq/kg (3.6–4.0 mCi/kg). The middle-aged dogs died significantly earlier ($p=0.002$) than the juvenile or young adult dogs, and middle-aged female dogs died significantly earlier ($p=0.002$) than middle-aged male dogs.

3.3.4.2 Systemic Effects

No data were located regarding respiratory, cardiovascular, gastrointestinal, musculoskeletal, hepatic, renal, endocrine, dermal, ocular, body weight, or metabolic effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

Hematological Effects. Hematological dyscrasia, characterized by severe thrombocytopenia and leukopenia, and death within 81 days were observed in 11 of 54 dogs that had been administered single intravenous injections of ^{137}CsCl (Nikula et al. 1995; Redman et al. 1972). The early deaths occurred in groups with average initial body burdens ranging from 71.7 to 141 MBq/kg (1.9 to 3.8 mCi) and cumulative doses to death in the range of 11.8–14.0 Gy (1,180–1,400 rad). Moderately to severely depressed blood cell counts were observed among 25 surviving dogs, some of which had been injected with lower levels of ^{137}CsCl that resulted in average initial body burdens of 36.4 or 51.7 MBq/kg (1.0 or

1.4 mCi/kg). Other treatment-related hematological effects included bone marrow aplasia, decreased platelet levels, and reduced packed-cell volume. In long-term surviving dogs, depressed blood values returned toward normal within during the first year after radiocesium injection. Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of ^{137}CsCl at levels resulting in average initial body burdens in the range of 64–162 MBq/kg (1.7–4.4 mCi/kg) (Nikula et al. 1996).

3.3.4.3 Immunological and Lymphoreticular Effects

Severe bone marrow depression was observed in dogs administered ^{137}CsCl by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula et al. 1995).

3.3.4.4 Neurological Effects

No data were located regarding neurological effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

3.3.4.5 Reproductive Effects

Persistent germinal epithelium damage and azoospermia were reported in all long-term surviving dogs that had been administered ^{137}CsCl by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 7.42 to 16.40 Gy (742 to 1,640 rad) (Nikula et al. 1995, 1996).

3.3.4.6 Developmental Effects

No data were located regarding developmental effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

3.3.4.7 Cancer

Benign and malignant neoplasms were found in a variety of tissues and organs of beagle dogs that had been administered single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996). Fifty-four young adult dogs at the Inhalation Toxicology Research Institute (ITRI) received amounts of ^{137}CsCl that resulted in average initial body burdens ranging from 37 to 141 MBq/kg (1.0 to 3.8 mCi/kg) (Nikula et al. 1995). In a study initiated at the Argonne National Laboratory (ANL), 63 beagle dogs, grouped according to age at study initiation (juveniles, 142–151 days old; young adults, 388–427 days old; and middle-aged adults, 1,387–2,060 days old), were administered single intravenous injections of ^{137}CsCl that resulted in initial body burdens of approximately 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1996). In both studies, dose-related increased incidences were observed for malignant neoplasms, malignant neoplasms excluding mammary neoplasms, all sarcomas considered as a group, all nonmammary carcinomas considered as a group, and malignant liver neoplasms. An increased risk for malignant thyroid neoplasms was seen in the ANL male dogs, but not in the ITRI males or females. In the ITRI (but not ANL) dogs, an increased relative risk for benign neoplasms excluding mammary neoplasms was observed. The occurrence of neoplasms in a diversity of tissues and organs results from the widespread distribution of cesium in the body.

3.4 GENOTOXICITY

Genotoxicity data for stable and radioactive cesium are summarized in Tables 3-3 and 3-4, respectively.

Evidence for genotoxic effects of stable cesium is limited to studies in which cesium chloride induced significantly increased incidences of chromosomal aberrations in human lymphocytes *in vitro* (Ghosh et al. 1993) (see Table 3-3) and mouse bone marrow *in vivo* (Ghosh et al. 1990, 1991; Santos-Mello et al. 2001) (see Table 3-4). Cesium sulfate was not mutagenic in *Escherichia coli (E. coli)* tester strains PQ37 and PQ35 either with or without metabolic activation in the SOS Chromotest (a bacterial colorimetric assay) at doses up to those resulting in significant toxicity (Olivier and Marzin 1987).

Increased frequency of point mutations in T-lymphocytes was observed in individuals in Goiânia, Brazil who had been exposed to an opened ^{137}CsCl source approximately 2.5 years prior to testing (Skandalis et al. 1997). The estimated dose from external radiation was 1.7 Gy (170 rad). The authors estimated internal doses based on whole-body counts and measured activity in urine and feces; however, realistic estimates were not reported. Among individuals exposed in the same incident, frequencies of

Table 3-3. Genotoxicity of Cesium *In Vitro*

		Result		
Species (test system)	End point	With activation	Without activation	Reference
Stable Cesium				
Mammalian cells:				
Human lymphocytes	Chromosomal aberrations	No data	+	Ghosh et al. 1993
Human lymphocytes	Micronuclei	No data	–	Santos-Mello et al. 1999
Prokaryotic organisms:				
Escherichia coli (PQ 37 and PQ 35)	Mutations	–	–	Olivier and Marzin 1987
Radioactive Cesium				
Mammalian cells:				
Human peripheral blood lymphocytes	Micronuclei	No data	+	Balasem and Ali 1991
Human peripheral blood lymphocytes	Chromosomal aberrations	No data	+	Doggett and McKenzie 1983
Human peripheral blood lymphocytes	Chromosomal aberrations	No data	+	Hintenlang 1993
Human peripheral blood lymphocytes	Chromosomal aberrations	No data	+	Iijima and Morimoto 1991
Human peripheral blood lymphocytes	Sister chromatid exchange	No data	–	Iijima and Morimoto 1991
Human spermatozoa	Chromosomal aberrations	No data	+	Mikamo et al. 1990, 1991
Human spermatozoa and zona-free hamster oocytes fertilization system	Micronuclei	No data	+	Kamiguchi et al. 1991
Mouse (BALB/c, SC3T3/W, Scid/St cells)	DNA double-strand breaks	No data	+	Biedermann et al. 1991
Chinese hamster ovary cells	Chromosomal aberrations, sister chromatid exchange	No data	+	Arslan et al. 1986

DNA = deoxyribonucleic acid; – = negative results; + = positive results

Table 3-4. Genotoxicity of Cesium *In Vivo*

Species (test system)	End point	Results	Reference
Stable Cesium			
Mammalian cells:			
Mouse bone marrow	Chromosomal aberrations	+	Ghosh et al. 1990, 1991
Mouse bone marrow	Micronuclei	+	Santos-Mello et al. 2001
Mouse bone marrow	Micronuclei	–	Santos-Mello et al. 1999
Radioactive Cesium			
Mammalian cells:			
Human peripheral blood lymphocytes	Chromosomal aberrations	+	Natarajan et al. 1998
Human peripheral blood lymphocytes	Chromosomal aberrations	+	Padovani et al. 1993
Monkey germ cells (male)	Reciprocal translocations	+	Tobari et al. 1988
Monkey germ cells (male)	Reciprocal translocations	+	Ramaiya et al. 1994
Mouse germ cells (male)	Reciprocal translocations	+	Ramaiya et al. 1994
Mouse germ cells (male)	Dominant lethal mutations	+	Ramaiya et al. 1994

+ = positive results; – = negative results chromosomal aberrations were used to estimate external radiation doses (Natarajan et al. 1998). No human reports were located in which genotoxic effects could be associated with specific radiation exposure levels, nor was there any information regarding potential for route-specific differences in observed genotoxic effects related to radioactive cesium exposure. Five years after the initial exposure to radioactive fallout from the Chernobyl accident of 1986, slightly greater frequencies of chromosomal aberrations were observed in peripheral blood lymphocytes of three groups of Byelorussian children (41 total) living in areas with ground contamination from ^{137}Cs fallout than in those of an Italian control group of 10 children (Padovani et al. 1993). Whole-body counts found an internally deposited ^{137}Cs activity range of 0.46–2.8 kBq (12-75 nCi) in children from Navrovl'a, an area (70 km from Chernobyl) exhibiting ^{137}Cs contamination of 550-1,500 GBq/km^2 (15–40 Ci/km^2). Internally deposited ^{137}Cs activity ranges of 0.044–0.4 kBq (1.2-10.8 nCi) and 7.7–32.3 kBq (208–872 nCi) were reported for children evacuated from the Chernobyl area soon after the accident to areas 200–300 km from Chernobyl with ^{137}Cs ground contamination of 40-400 GBq/km^2 (1–10 Ci/km^2), and children living in the Stolin area (250 km from Chernobyl) with ^{137}Cs ground contamination of 40–550 GBq/km^2 (1–5 Ci/km^2), respectively. Internalized activity was presumably from the consumption of ^{137}Cs-contaminated food. Although a small increase in the frequency of chromosomal aberrations in lymphocytes was observed, no pathology was apparent. These genotoxic effects were the result of the radiation, not the presence of cesium *per se*.

A dose-related increased frequency of micronuclei was observed in human peripheral blood lymphocytes exposed *in vitro* to gamma radiation from a sealed ^{137}Cs source at doses ranging from 0.05 to 6.00 Gy (5 to 600 rad) (Balasem and Ali 1991). This effect was the result of the radiation, not the presence of cesium *per se*.

In mice, genotoxic effects resulting from repeated oral exposure (daily administration for 2 weeks) to ^{137}Cs (as cesium nitrate) were compared with those elicited from external whole-body irradiation (23 hours/day for 19.5 days) using a ^{137}Cs source (Ramaiya et al. 1994). At comparable total radiation doses (approximately 3–4 Gy or 300–400 rad), both exposure scenarios resulted in similar increases in dominant lethal mutations among exposed male mice. Significant increases in the frequency of reciprocal translocations have been reported in spermatogonia of mice orally administered single doses of ^{137}Cs (as cesium chloride) that resulted in absorbed total body doses of approximately 3 Gy (300 rad) (Ramaiya et al. 1994).

Significant (dose-related) increases in the formation rate of micronuclei were seen in blood cells of fetal rats following irradiation of pregnant dams to total radiation doses of 0.5–4 Gy (50–400 rad), from a sealed ^{137}Cs gamma source, on gestation day 14 (Koshimoto et al. 1994). In crab-eating monkeys exposed to gamma radiation from an external ^{137}Cs source, increases in reciprocal translocations in spermatogonia were dose-related through the total absorbed dose range of 0.3–1.5 Gy (30–150 rad); it was also noted that the induction rate of translocations after acute high-dose-rate (0.25 Gy/minute or 25 rad/minute) exposure was about 10 times higher than that resulting from longer-term low-dose-rate (1.8×10^{-7} Gy/minute or 1.8×10^{-5} rad/minute) exposure (Tobari et al. 1988). These effects were the result of the radiation, not the presence of cesium *per se*.

Additional assays, performed *in vitro*, have also indicated that radioisotopes of cesium are genotoxic; specific end points include chromosomal aberrations and breaks, sister chromatid exchanges, and micronuclei (Arslan et al. 1986; Biedermann et al. 1991; Doggett and McKenzie 1983; Hintenlang 1993; Iijima and Morimoto 1991; Kamiguchi et al. 1991; Mikamo et al. 1990, 1991). Refer to Agency for Toxic Substances and Disease Registry (1999) for more information on the genotoxic effects of ionizing radiation.

3.5 TOXICOKINETICS

Numerous biokinetic studies have been performed in animals exposed internally to small amounts of the radioisotope ^{137}Cs. The biokinetic behavior of cesium has also been studied in humans either given tracer amounts of radiocesium or accidentally exposed to larger amounts.

Fractional absorption of inhaled or ingested cesium to the blood decreases with decreasing solubility of the carrier. Cesium taken into the body in soluble form is almost completely absorbed to blood. Cesium entering the respiratory or gastrointestinal tract as relatively insoluble particulates is mostly excreted in the feces. Cesium that comes into contact with the skin may be absorbed to some extent through the skin.

Following uptake by the blood, widespread distribution of cesium to all major soft tissues is observed in humans and animals. Cesium levels are slightly higher in skeletal muscle than other tissues. Distribution patterns in animals have been shown to be similar following exposure to soluble cesium compounds by inhalation, oral, and parenteral routes of exposure. Cesium crosses the placenta to the fetus. Cesium is also found in breast milk of a mother with an internal deposition.

Cesium is a close chemical analogue of potassium. Cesium has been shown to compete with potassium for transport through potassium channels and can also substitute for potassium in activation of the sodium pump and subsequent transport into the cell.

Excretion rates for ^{137}Cs have been studied in numerous populations exposed to nuclear fallout following incidents such as the Chernobyl accident. Biologically based pharmacokinetic models have been developed to describe relationships between intake and elimination. Experimental human studies have also been performed using tracer amounts of ^{134}Cs and ^{137}Cs. Urinary excretion is the primary route of elimination of cesium, and is independent of the route of exposure. Urinary to fecal ratios for cesium in humans have been found to range from 2.5:1 to 10:1. Radiocesium excretion rates were higher in males with muscular dystrophy than in normal, age-matched controls, and higher in pregnant women than in others who were not pregnant.

The elimination of cesium in humans appears to be age and sex related; long-term retention is principally a function of muscle mass. Elimination half-times are shorter in children than in adults, and shorter in women than in men.

3.5.1 Absorption

3.5.1.1 Inhalation Exposure

Inhalation exposure to relatively soluble cesium compounds will result in absorption of cesium in humans, although no reports were located that measured absorption of cesium following inhalation exposure. Evidence for absorption was presented by Miller (1964) via whole-body counts of ^{137}Cs (taken periodically for up to 285 days) in two men following occupational exposure to ^{137}Cs (as cesium sulfate) that was presumed to have been by inhalation. Distribution of ^{137}Cs was relatively uniform throughout the body, and steadily decreasing whole-body counts indicated that ^{137}Cs was eliminated from the body with a biological half-time of approximately 73–84 days. Additional indirect evidence of absorption was reported for an adult male who had been accidentally exposed to airborne ^{137}Cs twice in a 13-month period (Hölgye and Malý 2002). In this case, biological half-times for elimination of ^{137}Cs were 72 and 73 days.

Inhaled soluble cesium compounds are readily absorbed and distributed systemically in animals. Approximately 80% cesium absorption was observed in dogs acutely exposed to small amounts of aerosolized ^{137}Cs (as cesium chloride) (Boecker 1969a, 1969b). Deposition and distribution of cesium following inhalation exposure to radiolabeled cesium chloride was also observed in rats (Lie 1964; Stara and Thomas 1963) and guinea pigs (Stara 1965). Absorption was rapid following inhalation exposure.

3.5.1.2 Oral Exposure

It is generally accepted that cesium ingested as soluble cesium compounds is well absorbed by the gastrointestinal tract of humans and animals. Observations indicating that soluble cesium compounds were absorbed after ingestion by humans include: (1) low fecal excretory rates, (2) urinary excretory rates 4–10 times higher than those of fecal excretion, and (3) elimination half-times ranging from 45 to 147 days (Henrichs et al. 1989; Iinuma et al. 1965; Richmond et al. 1962; Rosoff et al. 1963). Henrichs et al. (1989) estimated an average cesium absorption of 78% in a group of 10 adult (5 male, 5 female) volunteers ingesting a meal of venison that was highly contaminated with ^{137}Cs and ^{134}Cs. Results from other controlled studies on human subjects indicate that absorption represented >90% of the cesium ingested in soluble form (Rosoff et al. 1963; Rundo 1964; Yamataga et al. 1966).

Absorption of ^{137}Cs from ingestion of radioactive fallout particles was found to be in the range of only 3%, indicating that such particles are relatively insoluble in biological fluids (LeRoy et al. 1966). Measurable amounts of ^{137}Cs were found in breast milk of women living in areas contaminated with radioactive fallout from the Chernobyl nuclear accident (Johansson et al. 1998). Based on whole body measurements of radioactivity in mothers and nursing infants and measured radioactivity in breast milk samples, it was estimated that 15% of the mothers' daily ^{137}Cs intake from contaminated food was transferred to the infant.

Animal studies support the findings in humans. Rapid absorption and widespread distribution of cesium was reported in guinea pigs administered single oral doses of soluble ^{137}Cs (as cesium chloride); fractional absorption data were not reported (Stara 1965). In rats orally administered single doses of highly insoluble irradiated fuel particles (mean diameter of 0.93 μm) containing ^{137}Cs and other radioactive elements, absorption of ^{137}Cs was found to be <10% (Talbot et al. 1993).

3.5.1.3 Dermal Exposure

Dermal absorption has been demonstrated in rats (Pendic and Milivojevic 1966). Traces of ^{137}Cs were observed in the blood of rats within a few minutes following the dermal application of ^{137}CsCl in aqueous solution. Approximately 3% of ^{137}CsCl applied to a surface area of several cm^2 was absorbed within 6 hours.

3.5.2 Distribution

3.5.2.1 Inhalation Exposure

Once absorbed, cesium is rapidly distributed throughout the body. Separate measurements of radioactivity from head, chest, upper abdomen, lower abdomen, thighs, legs, and feet indicated that ^{137}Cs was widely distributed throughout the bodies of two men who were occupationally exposed to ^{137}Cs (Miller 1964). Proportions of radioactivity in these body segments remained relatively constant from days 9 to 285 after exposure, indicating that ^{137}Cs was not likely to have been selectively accumulating in a particular region.

Animal studies also indicate a relatively uniform distribution of cesium following inhalation exposure to soluble cesium compounds (Boecker 1969a, 1969b; Stara 1965). Within 2 hours following exposure to aerosols of ^{137}Cs (as cesium chloride), up to 60% of the total body burden of ^{137}Cs was found in tissues other than respiratory or gastrointestinal tracts of dogs (Boecker 1969a). At 32 days post exposure, concentrations of ^{137}Cs in skeletal muscles, diaphragm, kidneys, and mandibular salivary gland were slightly higher than the whole-body average; concentrations in lung, skin, bone (femur, ribs), fat, and blood were somewhat lower (Boecker 1969b). Other tissues exhibited concentrations approximating the whole-body average. A relatively uniform distribution of ^{137}Cs in numerous body tissues, with the highest concentrations in skeletal muscle, was also observed in guinea pigs and rats exposed by inhalation (Lie 1964; Stara 1965; Stara and Thomas 1963).

3.5.2.2 Oral Exposure

Widespread distribution of cesium was observed in humans following oral exposure to soluble cesium compounds. In two human subjects orally administered ^{137}Cs (as cesium chloride), whole blood levels of ^{137}Cs within the first hour after administration amounted to approximately 2–3% of the amount administered, indicating that ^{137}Cs was rapidly absorbed and well distributed via the circulation (Rosoff et al. 1963).

Animal studies also showed relatively uniform distribution following oral exposure to soluble cesium compounds. Guinea pigs exhibited ^{137}Cs in numerous body tissues after receiving single oral doses of ^{137}Cs (as cesium chloride). The highest concentrations were found in skeletal muscle (Stara 1965). After the first day post administration, no significant differences in ^{137}Cs distribution patterns were observed between groups of guinea pigs exposed by inhalation, oral administration, or intraperitoneal injection of ^{137}Cs (as cesium chloride) (Stara 1965). Dogs and mice exhibited relatively uniform distribution of cesium throughout body tissues following chronic oral administration of ^{137}Cs (as cesium chloride) (Furchner et al. 1964). Cesium also crossed the placenta of animals and was found in breast milk. Newborn lambs exhibited lower tissue levels of ^{134}Cs than their mothers following oral administration of radiolabeled cesium chloride during pregnancy (Vandecasteele et al. 1989). The concentrations of ^{134}Cs in nursing lambs eventually exceeded the levels in their mothers.

3.5.2.3 Dermal Exposure

One report was located regarding distribution of cesium in animals following dermal exposure (Pendic and Milivojevic 1966). The investigators found widespread distribution of ^{137}Cs in rats within a few minutes following application of ^{137}CsCl solution to the skin. Although cesium was distributed throughout the body, it was deposited mainly in the kidneys, muscular tissues (particularly cardiac muscle), and liver.

3.5.2.4 Other Routes of Exposure

Comparative human and animal studies have shown that parenteral exposure to cesium compounds results in cesium distribution patterns similar to those observed following inhalation or oral exposure (Rosoff et al. 1963; Stara 1965). By 3 days following administration, ^{137}Cs was found to be distributed relatively uniformly among the body tissues of five cancer patients who died at various times following intravenous injection of a single tracer dose of ^{137}Cs (as cesium chloride). Time-related decreases were observed in ^{137}Cs retention by all tissues surveyed (Rosoff et al. 1963). Transfer of ^{137}Cs from pregnant dam to fetus has been shown in rats following intraperitoneal injection of radiolabeled cesium chloride (Mahlum and Sikov 1969).

3.5.3 Metabolism

Absorbed cesium behaves in a manner similar to that of potassium (Rundo 1964; Rundo et al. 1963). Both potassium and cesium are alkali metals that distribute throughout the body as cations, and are incorporated into intracellular fluids by active transport mechanisms. Cesium has been shown to compete with potassium for transport through potassium channels and can also substitute for potassium in activation of the sodium pump and subsequent transport into the cell (Cecchi et al. 1987; Edwards 1982; Hodgkin 1947; Latorre and Miller 1983; Sjodin and Beauge 1967). In both types of transport, movement of cesium is sluggish compared with that of potassium (Blatz and Magleby 1984; Coronado et al. 1980; Cukierman et al. 1985; Edwards 1982; Gay and Stanfield 1978; Gorman et al. 1982; Hille 1973; Reuter and Stevens 1980); that is, transport mechanisms generally favor potassium over cesium. Discrimination between potassium and cesium generally is greater for passive transport out of cells (selectivity ratios of Cs:K for a variety of tissues ranging from <0.02 to approximately 0.2) than for active transport into cells (selective Cs:K ratio approximating 0.25) (Leggett et al. 2003). This results in a greater residence time of cesium than potassium in muscle cells and hence in the whole body, since skeletal muscle contains most of the body's potassium or cesium at equilibrium. However, cesium appears to compete somewhat more favorably with potassium during transport out of red blood cells (Forth et al. 1963) or across or between epithelial cells (Cereijido et al. 1981; Greger 1981; Wright 1972).

3.5.4 Elimination and Excretion

3.5.4.1 Inhalation Exposure

Urinary excretion is the major route of elimination of cesium taken into the body in soluble form. In dogs exposed to ^{137}Cs (as cesium chloride) by inhalation, rates of excretion of ^{137}Cs in the urine and feces were highest in the first 3 days after exposure, accounting for approximately 12 and 3% of the initial body burden, respectively. Urinary and fecal excretion of ^{137}Cs continued at lower rates through 130 days of analysis. Rates of elimination were determined by measuring the cesium remaining in the organs of rats sacrificed at 9 time points during 120 days following treatment. The elimination rates for specific tissues (muscle, kidney, liver, and lung) were similar to the whole-body rates, indicating that cesium did not selectively accumulate in any particular tissues (Boecker 1969b). Within 2.5 days following inhalation exposure to ^{137}Cs (as cesium chloride), guinea pigs had eliminated approximately 50% of the initial ^{137}Cs body burden in the urine and feces (Stara 1965). The urinary to fecal ratio for excretion was approximately 3:1 throughout 60 days of post-exposure measurements, by which time, virtually all of the initial ^{137}Cs body burden had been eliminated. The urinary to fecal ratio for elimination of cesium in rats was about 3.5:1, with a biological half-time of elimination of approximately 4 days; 99% had been eliminated within 65 days following exposure (Stara and Thomas 1963).

No reports were located regarding routes of elimination and excretion of cesium in humans following inhalation exposure. However, kinetics of ^{137}Cs elimination in two adult males, accidentally exposed to ^{137}Cs (as cesium sulfate), were studied by whole-body measurements of gamma emission. The measured biological half-times were 73 and 84 days (Miller 1964). In the case of another adult male, accidentally exposed to airborne ^{137}Cs twice in a 13-month period, biological half-times for elimination were 72 and 73 days (Hölgye and Malý 2002).

In dogs exposed by inhalation, elimination rates of ^{137}Cs from specific tissues were similar to the rate of whole-body elimination, indicating that ^{137}Cs did not selectively accumulate in certain tissues, but was relatively uniformly eliminated from the body with a half-time of approximately 36–42 days (Boecker 1969b). Elimination rates of ^{137}Cs in guinea pigs and rats exposed by inhalation also did not vary significantly according to tissue type, although much shorter half-times of ^{137}Cs elimination (2.5 and 4 days) were observed for guinea pigs and rats, respectively (Stara 1965; Stara and Thomas 1963).

Relatively insoluble inhaled particles containing cesium were not absorbed in significant amounts and were more slowly eliminated from the lungs.

3.5.4.2 Oral Exposure

Urinary excretion is the primary route of elimination for cesium in humans. Among seven cancer or pulmonary patients who were administered single oral doses of ^{137}Cs (as cesium chloride), 7-day cumulative excretion of ^{137}Cs ranged from 7.0 to 17.3% of the administered activity. The urinary to fecal excretion ratio ranged from 2.5:1 to 10:1 (Rosoff et al. 1963). In a study of four Japanese volunteers orally administered single doses of ^{137}Cs (as cesium chloride), urinary to fecal excretion ratios ranging from 4.57:1 to 8.75:1 were calculated for excretory data collected after day 4 post administration. During the first 4 days after administration, excretory rates were consistently higher and the urinary:fecal excretory ratio was also somewhat higher (Iinuma et al. 1965). Based on the results of numerous reports of urinary and fecal excretion of Cs in human subjects, Leggett et al. (2003) reported an average urinary fraction (i.e., the ratio of cumulative urinary Cs to cumulative Cs in urine plus feces) of 0.86. Other sources of information on excretion rates of ^{137}Cs include numerous studies of populations exposed via fallout following atmospheric testing of nuclear weapons and from the Chernobyl accident, and mathematical models, such as those described in Section 3.5.5, have been developed to describe the relationships among intake, retention, and elimination of cesium.

Animal data support the findings in humans of urinary excretion as the major route of elimination of cesium following oral administration of soluble cesium compounds. Guinea pigs had eliminated approximately 50% of the initial ^{137}Cs body burden in the urine and feces within 2.5 days following administration (Stara 1965). The urinary to fecal ratio was within the range of 2–3:1 throughout 60 days of post exposure measurements, by which time, virtually all of the initial ^{137}Cs body burden had been eliminated.

Elimination half-times for cesium in the whole body, sometimes expressed in terms of whole-body radioactivity retention, have been reported by some investigators (Henrichs et al. 1989; Iinuma et al. 1967; Lloyd et al. 1973; Melo et al. 1997; Richmond et al. 1962; Rundo 1964). For example, among 10 volunteers who consumed ^{134}Cs- and ^{137}Cs-contaminated food, approximately 6% of the initial body burden was rapidly eliminated (average half-time of elimination of 0.3 days); the remaining 94% was eliminated more slowly (average half-time of elimination of 90 days) (Henrichs et al. 1989). In another oral study of four adult males, a mean elimination half-time was 135 days for ^{134}Cs and ^{137}Cs (Richmond et al. 1962).

Elimination rates for ^{137}Cs appear to be age- and sex-dependent, decreasing with age and lower in adult males than adult females. Results of studies of populations that consumed food containing ^{137}Cs from weapons testing fallout showed elimination half-times that varied from 15±5 days in infants to 100±50 days in adults (McCraw 1965). Similar studies after the Chernobyl accident found similar elimination half-times, which ranged from about 8 days for 1-year-old infants to about 110 days for adults (IAEA 1991). A 4-year study of 110 persons comprising a cross-section within an unspecified population indicated that children, 5–14 years of age, had the shortest elimination half-times (20 days, with no significant difference between males and females) (Boni 1969b). The elimination half-times in older groups were significantly longer (47 days for adolescent and adult females, 67 days in 15-year-old males, and 93 days in males 30–50 years of age). Melo et al. (1994) also reported age- and sex-related differences in elimination rates among individuals internally contaminated by ^{137}CsCl in the Goiânia, Brazil accident (see Agency for Toxic Substances and Disease Registry 1999 for a complete description of the incident). Elimination half-times for girls 1–4 years of age averaged 24 days. For 7–10-year-old girls and boys, the average elimination half-time was 37 days. Elimination half-times of 58 and 83 days were estimated for adolescent and adult males, respectively; compared with 46 and 66 days for adolescent and adult females, respectively. In the study of Melo et al. (1994), a high correlation was found between biological half-time for ^{137}Cs and weight for all age groups and sexes, except adult females.

Elimination rates of cesium may be altered by potassium intake. Following the intraperitoneal injection of ^{137}Cs in rats, a basal diet supplemented with 8–11% potassium resulted in cesium clearance of 60 days compared to about 120 days for rats receiving the unsupplemented basal diet that contained 1% potassium (Richmond and Furchner 1961). After 20 days on the diets, rats receiving supplemental potassium had body burdens of ^{137}Cs that were one-half those of the rats not receiving supplemental potassium. This finding shows that supplemental potassium reduces the uptake and increases the elimination of ingested ^{137}Cs.

Retention of cesium was lower in males suffering from muscular dystrophy than in age-matched controls. Older males with advanced signs of muscular dystrophy had lower retention than younger males exhibiting earlier stages of the disease (Lloyd et al. 1973).

Cesium crosses the placenta from mother to fetus. Measurable amounts of ^{137}Cs have been detected in human placenta and fetal tissue (Toader et al. 1996; Yoshioka et al. 1976). Cesium concentrations are higher in older fetuses than in younger ones (Toader et al. 1996). Pregnancy may increase the removal of cesium from the mother, as indicated by shorter elimination half-times during pregnancy relative to measurements taken before or after pregnancy or among nonpregnant controls (Bengtsson et al. 1964; Rundo and Turner 1966; Thornberg and Mattsson 2000; Zundel et al. 1969). Cesium has also been detected in human breast milk (Thornberg and Mattsson 2000).

3.5.4.3 Dermal Exposure

No reports were located regarding elimination and excretion of cesium in humans or animals following dermal exposure.

3.5.4.4 Other Routes of Exposure

Evidence for age-related differences in cesium elimination rates was observed in rats injected with ^{134}Cs or ^{137}Cs (as cesium chloride) at various ages. Retention of radiocesium from a single intraperitoneal injection of 0.5 μCi of carrier-free ^{134}CsCl increased with increasing age of rats at the time of dosing (Lengemann 1970). For example, the amount of radiocesium retained at 49 days after dosing was

>14 times greater in rats dosed at 4 months of age than in rats dosed at 1 month of age. Retention in rats injected at 21 months of age was approximately 1.9 times greater than that of the rats dosed at 4 months of age. Long-term retention also appears to be age related in dogs injected intravenously with $^{137}CsCl$; puppies 3–5 months of age exhibited elimination half-times that were shorter than those of adult dogs (Melo et al. 1997). Age-related increases in cesium retention rates were also observed in other young dogs from 61 to approximately 300 days old, after which cesium retention reached a plateau; the increases in cesium retention were similar to growth curves (Tyler et al. 1969). There is some indication, however, that retention of cesium is higher in neonatal rats than in weanling or adult rats (Lengemann 1969, 1970; Mahlum and Sikov 1969).

3.5.5 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 and Krishnan 1994; Andersen et al. 1987). 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 parameterization, (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

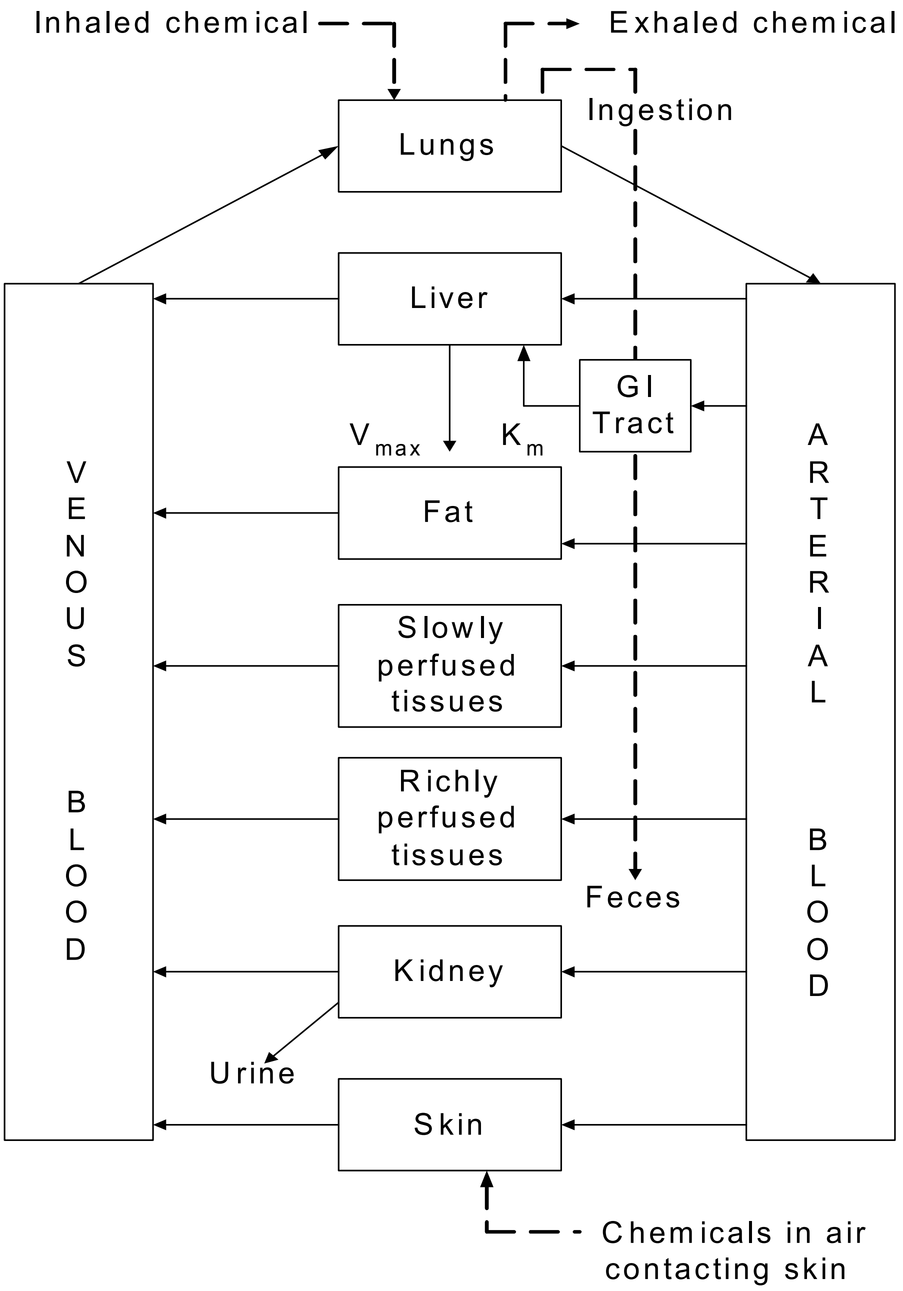
1994; Leung 1993). PBPK models for a particular substance require estimates of the chemical substance-specific physicochemical parameters, and species-specific physiological and biological parameters. The numerical estimates of these model parameters are incorporated within a set of differential and 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). Similar models have been developed for radionuclides. These PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals and radiation 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 3-3 shows a conceptualized representation of a PBPK model. Figures 3-4 through 3-7 show models for radionuclides in general or specifically for cesium.

The International Commission on Radiological Protection (ICRP 1994, 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 cesium compounds. The ICRP (1979, 1989, 1993) also developed a 2-compartment biokinetic model for human oral exposure that applies to cesium. EPA (1998) adopted the ICRP (1993, 1994, 1995) models for assessment of radiologic risks from cesium exposures. The National Council on Radiation Protection and Measurements (NCRP) 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 radiation doses for 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

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



Source: adapted from Krishnan et al. 1994

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.

to the report, NCRP provides the animal testing clearance data and equations fitting the data that supported the development of the human model for cesium.

Human Respiratory Tract Model for Radiological Protection (ICRP 1994)

Deposition. The ICRP (1994) developed a deposition model to describe the behavior of inhaled aerosols and vapors in the respiratory tract. This model was developed to estimate the fractions of radioactivity in breathing air that deposit in each anatomical region of the respiratory tract. ICRP (1994) provides inhalation dose coefficients, which can be used to estimate the committed equivalent and effective 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. The model may be adjusted for various segments of the population (e.g., sex, age, level of physical exertion). This model also allows the evaluation of the bounds of uncertainty in deposition estimates. Uncertainties arise from natural biological variability among individuals. The ICRP model is applicable to particulate aerosols containing cesium, but was developed for a wide variety of radionuclides and their chemical forms.

The ICRP deposition model may be used to estimate the amount of inhaled material that initially enters each compartment (see Figure 3-4). The model was developed with 5 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 the ET_1 region may be cleared either by dissolution and absorption into the blood or by nose-blowing. Particles deposited in each of the other regions may then be removed from each region and redistributed either upward into the respiratory tree by mucociliary clearance mechanisms, or to the lymphatic system and blood by different particle removal mechanisms.

For extrathoracic deposition of particles, the model is based on experimental data, where deposition is related to particle size and airflow parameters. The model scales deposition for women and children from adult male data. Similarly 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 3-5 provides reference respiratory values for the general Caucasian population and for several levels of physical activity.

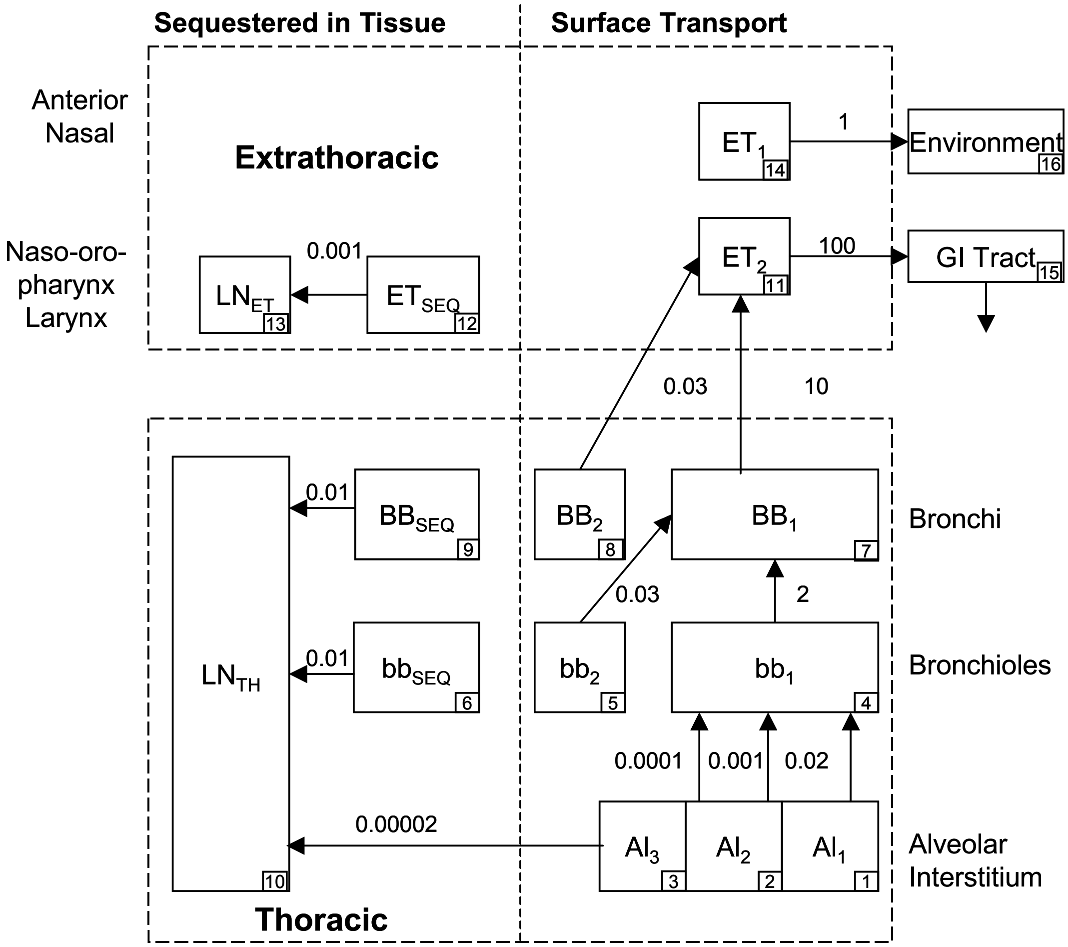
Table 3-5. Reference Respiratory Values for a General Caucasian Population at Different Levels of Activity[a]

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	Both:	0.3	0.31	17	0.33	0.38	19	0.58	1.12	32			
	Male:										0.841	2.22	44
	Female:										0.667	1.84	46
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 1994) for data from which these reference values were derived.
[b] B = ventilation rate; f_R = respiration frequency; V_T = tidal volume

H = hour; L = liter; m = meter; min = minute; N/A = not applicable

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



*Compartment numbers shown in lower right corners are used to define clearance pathways. The clearance rates, half-lives, and fractions by compartment, as well as the compartment abbreviations are presented in Table 3-6.

Source: ICRP 1994

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 compounds in the respiratory tract (see Figure 3-5). The ICRP (1994) 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 the inhaled gas is exhaled. Radiation doses from inhalation 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 that 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 that 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 Mechanical (Particle) Clearance. The clearance portion of the model identifies the principal clearance pathways within the respiratory tract. The model was developed to predict the retention and clearance of various radioactive materials. The compartmental model is linked to the deposition model (see Figure 3-4) and to reference values presented in Table 3-6. Table 3-6 provides clearance rates by biological processes only, not by radioactive decay, and deposition fractions for each compartment for insoluble particles. The table provides rates of insoluble particle transport for each of the compartments, expressed as a fraction per day and also as half-time. ICRP (1994) also developed modifying factors for some of the parameters, such as age, smoking, and disease status. Parameters of the clearance model are based on human data, 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 over a short time period (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. As particles dissolve, absorption rates also 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

Table 3-6. 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 3-6. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

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:

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

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

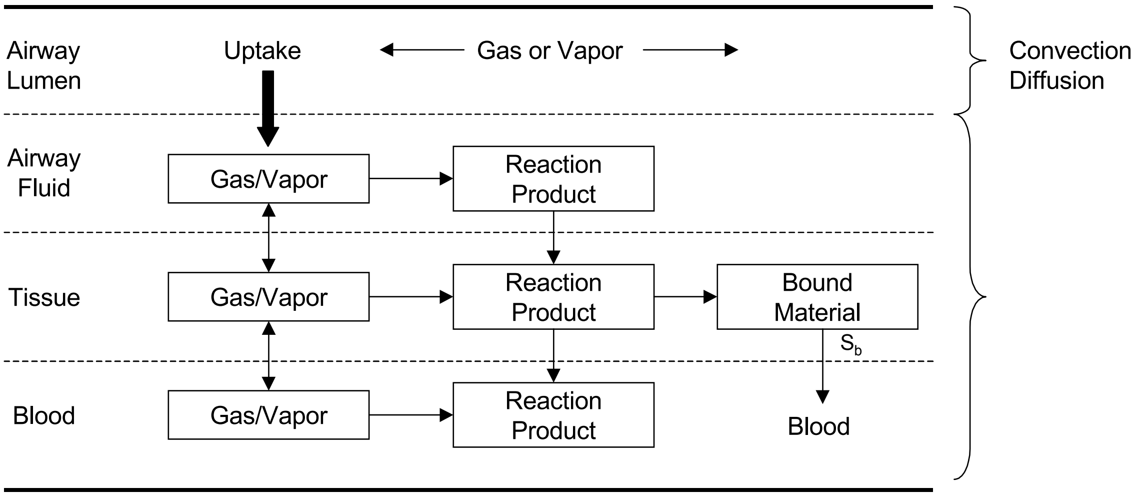
where:

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; GI tract = gastrointestinal tract; 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 3-5. Reaction of Gases or Vapors at Various Levels of the Gas-Blood Interface



Source: ICRP 1994

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. A majority of deposited particles are eventually swallowed. In the front part of the nasal passages (ET_1), nose blowing, sneezing, and wiping remove most of the deposited particles. Particles remain in the nasal passages for about a day. For particles with AMADs of 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 mucous fluids that cover the airways. In this region, particle clearance is completed within 15 minutes.

Ciliary action removes deposited particles from the bronchi and bronchioles. Though mucociliary action rapidly transports most particles deposited here toward the pharynx, a fraction of these particles is cleared more slowly. Evidence for this clearance 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 many as half of the bronchi- and bronchiole-deposited particles. In human bronchi and bronchiole regions, mucus movement is influenced by location. Movement is slower in areas closer to alveoli. 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 and both with 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 may be 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 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 representing different slow clearance rates, all of which are slow.

Particle clearance from the alveolar-interstitial region has been measured in human subjects. The ICRP model uses 2 half-times to represent clearance. About 30% of the particles have a 30-day half-time, and the remaining 70% are given a half-time of several hundred days. Over time, AI particle transport diminishes and some insoluble particles may remain 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. Absorption is essentially a 2-stage process, as shown in Figure 3-6. First, there is dissociation (dissolution) of particles. The dissolved molecules or ions then diffuse across capillary walls and are taken up by the blood. Immediately following dissolution, rapid absorption takes place. 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 ET2. Thus, for nose breathing, there is rapid absorption of approximately 25% of the deposit in ET and 50% for mouth breathing.

- 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 ET2. 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.

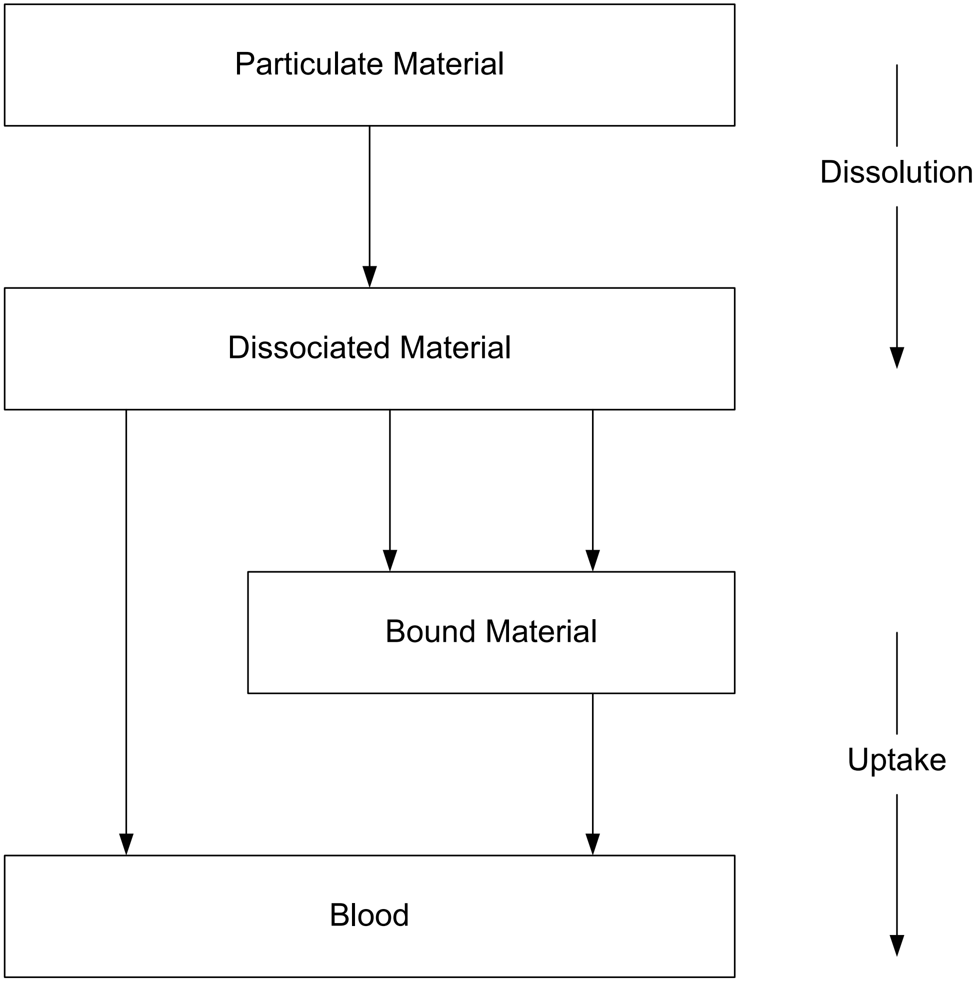
- For Type V, complete absorption (100%) is considered to occur instantaneously.

ICRP (1995) considers the experimental and human data to support the following classifications: cesium chloride and nitrate, Type F; cesium in irradiated fuel fragments, Type F or M; and cesium in fused aluminosilicate particles, M or S. ICRP (1995) recommends assigning all cesium aerosols to Type F in the absence of specific information supporting an alternative classification.

ICRP (1993) Cesium Biokinetic Model

Description of the Model. ICRP (1979, 1989, 1993) developed a 2-compartment model of the kinetics of ingested cesium in humans (Figure 3-6) that is applicable to infants, children, adolescents, and adults. The model is based on an age- and gender-specific model of Leggett (1986) in which compartment sizes and retention half-times were expressed as a function of the mass of potassium in the body. Ingested cesium is assumed to be completely absorbed into blood. Absorbed cesium is then assumed to distribute

Figure 3-6. The Human Respiratory Tract Model: Absorption into Blood



Source: ICRP 1994

uniformly in the body and to be eliminated from fast and slow elimination pools. The fraction of the total body cesium associated with the fast and slow pools, as well as the elimination half-times from each pool, are assumed to vary with age. The elimination half-times vary for ages 3 months, 1, 5, 10, 15 years, and adult (>15 years). The contribution of the fast pool decreases from 45% at age 5 years to 10% in adults. The elimination half-times of the fast pool decrease from 9.1 days at age 5 years to 2 days in adults, whereas the elimination half-times of the slow pool increase from 13–16 days in infants to 110 days in adults.

Validation of the Model. The extent to which the ICRP model has been validated is not described in ICRP (1993).

Risk Assessment. The ICRP biokinetic model has been used to establish radiation dose equivalents (Sv/Bq) of ingested ^{134}Cs, ^{136}Cs, and ^{137}Cs for ages 3 months to 70 years (ICRP 1993).

Target Tissues. The ICRP model is designed to calculate radiocesium intake limits based on radiation dose to all major organs.

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

Interroute Extrapolation. The ICRP model is designed to simulate oral exposure assuming 100% absorption of ingested cesium. The model is applicable to other routes of exposure if the extent of absorption for a particular exposure route is known.

Leggett et al. (2003) Cesium Biokinetics Model

Description of the Model. Leggett et al. (2003) developed a PBPK model of the kinetics of ingested or injected cesium in humans (Figure 3-7). The model includes compartments representing blood (plasma and red blood cells), brain, gastrointestinal tract, heart, kidney, liver, lung, pancreas, skin, and spleen. Transfers between plasma and tissues are simulated as first-order, flow-limited, rate constants (Table 3-7). Flow limitation is imposed by deriving the plasma-to-tissue rate constants in terms of tissue blood flows and empirically-derived arterial-venous extraction fractions (Table 3-8). Rate constants for transfers from tissues to plasma are derived from the product of the plasma-to-tissue rate constants and empirically-derived equilibrium ratios for cesium (fraction of body burden) in plasma and tissue (Table 3-9).

Table 3-7. Transfer Coefficients (d^{-1}) for a Reference Adult Male

Plasma to heart	14.128
Plasma to liver	19.515
Plasma to kidneys	67.108
Plasma to muscle	30.022
Plasma to gastrointestinal tract tissue	52.98
Plasma to stomach contents	4.516
Plasma to small intestine contents	1.0480
Plasma to large intestine contents	0.02
Plasma to spleen	5.298
Plasma to pancreas	1.766
Plasma to brain	0.424
Plasma to red marrow	5.298
Plasma to other skeleton	3.532
Plasma to skin	4.415
Plasma to lungs	4.415
Plasma to adipose tissue	8.83
Plasma to other 1[a]	8.826
Plasma to other 2[b]	0.00353
Plasma to red blood cells	1.8
Heart to plasma	8.073
Liver to plasma	2.204
Liver to small intestine	0.116
Kidneys to urinary bladder contents	1.678
Kidneys to plasma	31.876
Muscle to plasma	0.0751
Gastrointestinal tract tissue to plasma	8.191
Gastrointestinal tract tissue to liver	0.431
Gastrointestinal tract tissue to stomach contents	0.0333
Gastrointestinal tract tissue to small intestine contents	0.108
Gastrointestinal tract tissue to large intestine contents	0.0667
Spleen to plasma	5.033
Spleen to liver	0.265
Pancreas to plasma	1.678
Pancreas to liver	0.0883
Skin to plasma	0.867
Skin to excreta	0.0159
Brain to plasma	0.0848
Red marrow to plasma	0.706
Other skeleton to plasma	0.128
Lungs to plasma	1.472
Adipose tissue to plasma	1.766
Other 1[a] to plasma	0.692

Table 3-7. Transfer Coefficients (d^{-1}) for a Reference Adult Male

Other 2[b] to plasma	0.00141
Red blood cells to plasma	0.257
Urinary bladder contents to urine	12.0
Stomach to small intestine (contents)	40.0
Small intestine contents to plasma	28.215
Small intestine to large intestine (contents)	0.3
Small intestine contents to liver	1.485
Large intestine contents to feces	0.5

[a] Remaining tissues and fluids in the body with a relatively short turnover time.
[b] Small component of the remaining tissues and fluids in the body with a tenacious retention of Cs apparent in some long-term studies.

Source: Leggett et al. 2003

Table 3-8. Tissue-specific Extraction Fractions Assumed for Cesium

Tissues	Extraction fraction
Kidneys, gastrointestinal tract, heart	0.2
Liver, skin	0.05
Brain	0.002
All other tissues	0.1

Source: Leggett et al. 2003

Table 3-9. Tissue Masses, Cesium Equilibrium Distribution, and Tissue Blood Flow for a Reference Adult Male

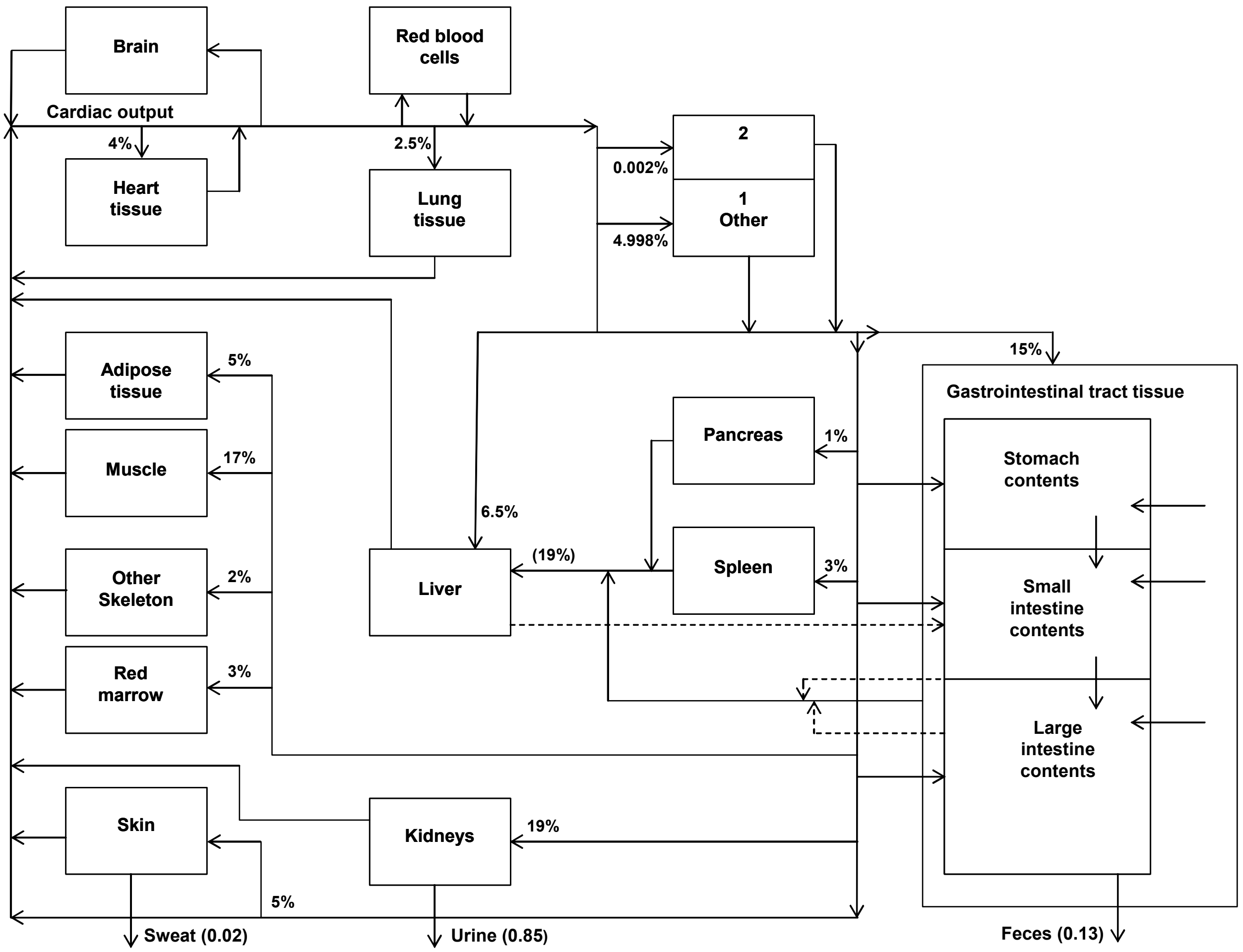
Compartment	Mass[a] (g)	Equilibrium cesium content[a] (fraction of total-body cesium)	Blood flow (FCO)
Adipose tissue[b]	12,000	0.01	0.05
Brain	1,450	0.01	0.12
Gastrointestinal contents	900	0.004	–
Gastrointestinal tract tissue	1,170	0.015	0.15
Heart	330	0.0035	0.04
Kidneys	310	0.004	0.19
Liver	1,800	0.02	0.065 (arterial) (0.19) (portal)
Lungs	500	0.006	0.025
Skeletal muscle	29,000	0.8	0.17
Plasma	3,100	0.002	–
Red blood cells	2,500	0.014	–
Skeleton	10,500	0.07	0.05
Red marrow		(0.015)	(0.03)
Bone and other tissue		(0.055)	(0.02)
Skin	3,300	0.01	0.05
Spleen	150	0.002	0.03
Pancreas	140	0.002	0.01
Other[c]	5,850	0.0305	0.05
Totals	73,000	1.00	1.00

[a] Without blood.
[b] Separable adipose tissue excluding yellow bone marrow.
[c] Remaining tissues and fluids in the body.

FCO = fraction of cardiac output

Source: Leggett et al. 2003

Figure 3-7. Directions of Flow of Cesium



Solid arrows represent plasma flow and broken arrows represent flow not involving plasma. Percentages indicate distribution of carbon monoxide. Numbers besides sweat, urine, and feces are fractions of cumulative excretion.

Source: Leggett et al. (2003).

Excretory pathways represented in the model include feces, sweat, and urine. Transfers between the central plasma flow and the gastrointestinal tract include secretion into the gastrointestinal tract and absorption to plasma.

Validation of the Model. The model has been evaluated for predicting observed blood and plasma cesium concentrations, whole-body retention of cesium, and urinary clearance of cesium in adults, following injections or ingestion of cesium (Leggett et al. 2003). Additional evaluations are reported, based on evaluations in rats and dogs. Model predictions corresponded reasonably well with observations.

Risk Assessment. The model is intended for predicting internal tissue doses to radiation following exposures to cesium for use in radiation risk assessment.

Target Tissues. The model is designed to calculate cesium concentrations in tissues, and includes major sites of accumulation (e.g., kidney, liver, and muscle).

Species Extrapolation. The model is designed for applications to human dosimetry and can be applied to other species (e.g., dog and rat) with modification of physiological parameters.

Interroute Extrapolation. The model is designed to simulate the intravenous or oral exposures to cesium and can be applied to other routes of exposure with the addition of simulations or estimates of the uptake of cesium into the central blood compartment.

3.6 MECHANISMS OF ACTION

3.6.1 Pharmacokinetic Mechanisms

Cesium is rapidly absorbed into blood following inhalation or oral exposure to soluble cesium compounds, as demonstrated by the rapid distribution of cesium activity after inhalation or ingestion. Approximately 80% absorption of ^{137}Cs was observed in dogs exposed to aerosols containing ^{137}Cs (as cesium chloride) (Boecker 1969a, 1969b). Oral ingestion of ^{134}Cs- and ^{137}Cs-contaminated food by volunteers resulted in approximately 78% absorption. Animal studies indicate that absorption rates from orally administered soluble cesium compounds are highest in the duodenum, followed in order by the jejunum, ileum, and colon. Very little absorption occurs in the stomach or caecum (Moore and Comar 1962, 1963). Absorption rates are higher in fasted rats than in fed rats, indicating that stomach contents may influence the rate of cesium absorption. Relatively insoluble forms of cesium compounds, which may sometimes be associated with irradiated fuel particles, are poorly absorbed by inhalation and oral exposure routes (Boecker et al. 1974, 1977; LeRoy et al. 1966; Talbot et al. 1993). Dermal retention, but not transdermal absorption, has been qualitatively demonstrated in humans (Rundo 1964). Dermal absorption was observed following application of ^{137}CsCl in aqueous solution to the skin of rats (Pendic and Milivojevic 1966). Traces of ^{137}Cs were observed in the blood of rats within a few minutes following application.

Once absorbed, cesium is rapidly distributed throughout the body, becoming incorporated into the intracellular fluid of numerous tissues. Animal studies indicate that distribution patterns are similar following absorption from inhalation or oral exposure and that concentrations of cesium within muscle tissue are somewhat higher than the whole-body average (Stara 1965). Comparative human and animal studies have shown that parenteral exposure to cesium compounds results in cesium distribution patterns similar to those following inhalation or oral exposure (Rosoff et al. 1963; Stara 1965).

Absorbed cesium behaves in a manner similar to that of potassium. Both potassium and cesium are alkali metals that are distributed throughout the body as cations, becoming incorporated into intracellular fluids. Cesium has been shown to compete with potassium for transport through potassium channels and can also substitute for potassium in activation of the sodium pump and subsequent transport into the cell (Cecchi et al. 1987; Edwards 1982; Hodgkin 1947; Latorre and Miller 1983; Sjodin and Beauge 1967). In both types of transport, movement of cesium is sluggish compared with that of potassium (Blatz and Magleby 1984; Coronado et al. 1980; Cukierman et al. 1985; Edwards 1982; Gay and Stanfield 1978; Gorman et al. 1982; Hille 1973; Reuter and Stevens 1980). Discrimination between potassium and cesium generally is greater for passive transport out of cells than for active transport into cells (Leggett et al. 2003). This results in a greater residence time of cesium than potassium in muscle cells and hence in the whole body, since skeletal muscle contains most of the body's potassium or cesium at equilibrium. Although the biokinetics of potassium and cesium may vary somewhat in such characteristics as relative affinity for various cell types and differing retention rates, the similarities allow elimination rates for potassium to be used as an index of elimination rates for cesium (USNRC 1983). Urinary excretion is the major route of elimination of cesium.

3.6.2 Mechanisms of Toxicity

Earth contains relatively small amounts of stable (nonradioactive) cesium. Cesium has few industrial applications. At environmental levels, stable cesium is not chemically toxic in animals. Cesium is not likely to be of toxic concern to humans exposed to cesium by inhalation, oral, or dermal contact. Although a number of investigators have reported cesium-induced alterations in behavior or cardiac activity in animals systems exposed to cesium chloride by parenteral injection, underlying mechanisms are not yet fully understood.

Cesium may have both depressant and antidepressant properties in rodents, as it was shown to decrease the conditioned avoidance response of pole-climbing (Bose and Pinsky 1983b) and to reduce vertical and horizontal motor activity (Bose and Pinsky 1981, 1984; Bose et al. 1981; Pinsky et al. 1980), while enhancing amphetamine-induced hyperactivity and reducing the locomotor depressive action of reserpine (Messiha 1978).

Increased vertical activity (rearing), but not horizontal activity, was observed in mice given repeated injections of cesium chloride (Johnson 1972). Rastogi et al. (1980) found no increase in behavioral activity in rats repeatedly injected with cesium chloride, but noted a number of biochemical changes in the brain that included a statistically significant rise in tyrosine hydroxylase activity that resulted in a slight but statistically significant increase in tyrosine levels, markedly enhanced levels of the neurotransmitters norepinephrine and dopamine, and increased levels of a norepinephrine metabolite (4-hydroxy-3-methoxyphenylglycol). Cesium appeared to block the uptake of norepinephrine by synaptosomes.

Cesium was shown to alter normal cardiac rhythm, triggering short-lived early after depolarizations (EADs) and polymorphic ventricular tachyarrhythmias (VTs) in canine myocardial muscle fibers and Purkinje cells (Brachmann et al. 1983; Levine et al. 1985; Murakawa et al. 1997; Patterson et al. 1990), effects that are similar to those observed in humans with congenital and acquired long QT syndrome (Bonatti et al. 1983). Prolonged QT syndrome and associated cardiac arrhythmia have been observed in patients who consumed cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001). Available animal data suggest that cesium-induced EADs and VTs were most likely the result of ionic imbalance due to reduced potassium permeability (Isenberg 1976) and imbalances of intra- and extracellular concentrations of calcium and sodium (Szabo et al. 1987).

Both ^{134}Cs and ^{137}Cs emit gamma radiation, and therefore, radioactive cesium is a health hazard. Highly penetrating gamma rays are the major cause of damage to tissues and internal organs following external overexposure to radioactive cesium. Once radioactive cesium is taken internally, cells of nearby tissues are at highest risk for damage due to the emission of beta particles. Radiation-induced damage in cells may be repaired quickly. Misrepaired damage may lead to permanent DNA changes and the potential for carcinogenesis. Very large acute radiation doses can damage or kill enough cells to cause the disruption of organ systems (acute radiation syndrome), harm to developing fetuses, and even death. Human and animal data indicate that radioactive cesium overexposure can result in adverse effects such as reduced fertility, abnormal neurological development, genotoxicity, and damage to blood-forming organs (Bartstra et al. 1998; Matsuda et al. 1985; Nikula et al. 1995, 1996; Padovani et al. 1993; Ramaiya et al. 1994; Skandalis et al. 1997; Tobari et al. 1988). For a more complete discussion of the mechanisms associated with the toxic effects of ionizing radiation, refer to Chapter 5 of the Toxicological Profile for Ionizing Radiation (Agency for Toxic Substances and Disease Registry 1999).

3.6.3 Animal-to-Human Extrapolations

No data were located to indicate significant interspecies differences in pharmacokinetics or health effects associated with exposure to stable or radioactive cesium.

3.7 TOXICITIES MEDIATED THROUGH THE NEUROENDOCRINE AXIS

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. Chemicals with this type of activity are most commonly referred to as *endocrine disruptors*. However, appropriate terminology to describe such effects remains controversial. The terminology *endocrine disruptors*, initially used by Colborn and Clement (1992), was also used in 1996 when Congress mandated the Environmental Protection Agency (EPA) to develop a screening program for "...certain substances [which] may have an effect produced by a naturally occurring estrogen, or other such endocrine effect[s]...". To meet this mandate, EPA convened a panel called the Endocrine Disruptors Screening and Testing Advisory Committee (EDSTAC), which in 1998 completed its deliberations and made recommendations to EPA concerning *endocrine disruptors.* In 1999, the National Academy of Sciences released a report that referred to these same types of chemicals as *hormonally active agents*. The terminology *endocrine modulators* has also been used to convey the fact that effects caused by such chemicals may not necessarily be adverse. Many scientists agree that chemicals with the ability to disrupt or modulate the endocrine system are a potential threat to the health of humans, aquatic animals, and wildlife. However, others think that endocrine-active chemicals do not pose a significant health risk, particularly in view 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 chemicals are derived from plants and are similar in structure and action to endogenous estrogen. Although the public health significance and descriptive terminology of substances capable of affecting the endocrine system remains controversial, scientists agree that these chemicals may affect the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body responsible for maintaining homeostasis, reproduction, development, and/or behavior (EPA 1997). Stated differently, such compounds may cause toxicities that are mediated through the neuroendocrine axis. As a result, these chemicals may play a role in altering, for example, metabolic, sexual, immune, and neurobehavioral function. Such chemicals are also thought to be involved in inducing breast, testicular, and prostate cancers, as well as endometriosis (Berger 1994; Giwercman et al. 1993; Hoel et al. 1992).

No studies were located regarding endocrine disruptive effects resulting from exposure to stable or radioactive cesium.

3.8 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).

Soluble cesium compounds are readily absorbed into body fluids and bloodstream and are widely distributed throughout the body (see Section 3.5 for detailed information). PBPK models are used to simulate potential age-related differences in deposition of inhaled cesium, as well as differences in elimination rates for absorbed cesium (see Section 3.5.5 for more information on PBPK models). Although inhalation exposure to environmental levels of stable or radioactive cesium is not considered to be a major health concern, age-related differences in physical properties of the respiratory system and ventilation patterns could result in differences in absorption rates of inhaled soluble or insoluble cesium compounds. Soluble cesium compounds are assumed to be completely absorbed in the gastrointestinal tract, with no adjustments for age. Available human and animal data do not indicate that age-related differences might exist for absorption and distribution of cesium following oral exposure. Since cesium is principally absorbed and distributed in ionic form (as Cs^+), any age-related differences in absorption following oral exposure would likely be the result of differences in diffusion rates and active transport mechanisms involved in the movement of cesium through extra- and intracellular fluids. Elimination rates for cesium appear to be age-related and may be most closely related to body mass. As described in detail in Section 3.5.4.2, young children exhibit whole-body biological elimination half-times that are much shorter than those of older children and adults (Boni 1969b; Melo et al. 1994). It is not known whether or not these age-related differences may be due to higher retention of cesium in adult tissues and lower rates of excretion.

Measurable amounts of ^{137}Cs have been found in the breast milk of women living in areas contaminated with radioactive fallout. Transfers to newborns and 1-year-old children were estimated to be approximately 40 and 50%, respectively (Johansson et al. 1998). Animal studies have shown that cesium crosses the placental barrier, but cesium is found in lower concentrations in the fetus than in maternal or placental tissues (Mahlum and Sikov 1969; Vandecasteele et al. 1989).

Although no information was located regarding age-related health effects in humans exposed to stable cesium, age-related differences in the pharmacokinetics of stable cesium could conceivably result in age-related differences in health effects. No studies were located regarding age-related differences in toxicity in animals exposed to stable cesium.

Most of the available information regarding age-related health effects from overexposure to cesium concern developmental effects related to *in utero* irradiation of human or animal fetuses from an external source of radiation. Impaired cognitive function was observed in atomic bomb survivors overexposed to ionizing radiation *in utero* during critical stages of neurological development (Schull and Otake 1999). Developmental toxicity studies employing external gamma radiation from a radioactive cesium source (or from any other gamma ray source) indicate that rats and mice are most sensitive to the effects of external radiation around gestation day 14. Effects observed following irradiation during this period include reduced survival, decreased brain size, smaller head size, and retarded odontogenesis (Koshimoto et al. 1994; Minamisawa et al. 1990; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994). When tested as adults, animals irradiated during this developmental period exhibit increased aggressive behavior

(Minamisawa et al. 1992; Norton and Kimler 1987, 1988). Although comparative studies of neurological effects in animals first irradiated as juveniles or adults were not located, it is apparent that there are critical stages of fetal developmental during which there is increased susceptibility to the effects of radiation. In these studies, although cesium was used as the gamma source, the effects were not unique to cesium. Similar results would be elicited by any gamma source.

3.9 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 cesium are discussed in Section 3.9.1.

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 cesium are discussed in Section 3.9.2.

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 3.11, Populations that are Unusually Susceptible.

3.9.1 Biomarkers Used to Identify or Quantify Exposure to Cesium

Stable or radioactive isotopes of cesium may be measured in samples of urine, blood, feces, or body tissues by a number of methods outlined in Section 7.1. Stable cesium is of little toxicological concern. However, overexposure to radioactive isotopes of cesium may pose a significant health risk. Internal exposure may be quantified by direct counting (*in vivo* measurements) of radioactive emission from the body using whole-body counters capable of distinguishing the gamma emissions that are unique to radioactive isotopes of cesium. Radioactivity can be accurately measured in blood, excrement, and tissue samples using scintillation counting. The biomarkers that may help quantify exposure to stable or radioactive cesium are similar in children and adults.

3.9.2 Biomarkers Used to Characterize Effects Caused by Cesium

There are no known biomarkers of effect for exposure to stable cesium. High-level external or internal exposure to radioactive cesium can result in bone marrow aplasia, reduced white blood cell counts, decreased hemoglobin and platelet levels, and increased frequencies of chromosomal aberrations in lymphocytes. Frequencies of chromosomal aberrations were used to estimate external radiation doses among individuals in Goiânia, Brazil who had been exposed to an opened ^{137}CsCl source (Natarajan et al. 1998). These results are not unique to radioactive cesium. Similar results would be expected following overexposure to any source of ionizing radiation.

3.10 INTERACTIONS WITH OTHER CHEMICALS

No data were located regarding interactions of cesium with other chemicals that might influence the toxicity of cesium.

3.11 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to cesium than will most persons exposed to the same level of cesium 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 cesium, or compromised function of organs affected by cesium. Populations who are at greater risk due to their unusually high exposure to cesium are discussed in Section 6.7, Populations with Potentially High Exposures.

Increased susceptibility to the toxic effects resulting from exposure to high levels of stable or radioactive cesium might be indicated among individuals with abnormally low potassium intake, those with compromised kidney function, and patients taking stimulant or depressant drugs for the treatment of mental disorders (see Sections 3.5 and 3.6 for detailed information on the toxicokinetics and mechanisms of action of cesium). Individuals with compromised immune function might be more susceptible to the adverse effects of radiation overexposure from a radioactive cesium source (or from any other gamma emitting radioactive source).

Evidence for potential age-related differences in susceptibility to stable or radioactive cesium toxicity is provided by studies of elimination rates for ^{137}Cs in humans (Boni 1969b; Melo et al. 1994; Toader et al. 1996). Elimination rates are higher in young children than adults, and higher in adult females than adult males, indicating that lower elimination rates could result in greater retention and, therefore, increased toxicity for a given intake. Animal studies support these findings (Lengemann 1970; Mahlum and Sikov 1969; Melo et al. 1996, 1997; Tyler et al. 1969). There is some indication, however, that retention of cesium is higher in neonatal rats than in weanling or young adult rats (Lengemann 1969, 1970; Mahlum and Sikov 1969) (see Sections 3.5.4.2 and 3.5.4.4 for more detailed information.

Pregnant women (Zundel et al. 1969) and individuals suffering from muscular dystrophy (Lloyd et al. 1973) exhibited decreased cesium retention, which may decrease susceptibility to stable or radioactive cesium-induced toxicity.

3.12 METHODS FOR REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to cesium. 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 cesium. 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 cesium:

Ellenhorn MJ, Schonwald S, Ordog G, et al., eds. 1997. Medical toxicology: Diagnosis and treatment of human poisoning. 2nd edition. Baltimore, MD: Williams & Wilkins, 1682-1723.

Haddad LM, Shannon MW, Winchester JF, eds. 1998. Clinical management of poisoning and drug overdose. 3rd edition. Philadelphia, PA: WB Saunders, 413-425.

NCRP Report No. 65. 1980. Management of persons accidentally contaminated with radionuclides. Bethesda MD: National Council on Radiation Protection and Measurements.

3.12.1 Reducing Peak Absorption Following Exposure

Because soluble cesium compounds are rapidly absorbed into blood following inhalation, oral, and dermal exposure, there are no prescribed methods for reducing peak absorption following exposure. Early counter measures that may aid in reducing peak absorption following oral exposure to stable or radioactive cesium include oral administration of insoluble Prussian blue (ferric hexacyanoferrate) that exchanges potassium for cesium, forming an insoluble complex that is eliminated through the feces (Oak Ridge Associated Universities 2003). Animal studies indicate that absorption of cesium may also be reduced by the administration of excess potassium (Richmond and Furchner 1961). Cathartics such as magnesium sulfate, as well as gastric lavage, will shorten the transit time of ingested cesium in the gastrointestinal tract (Ellenhorn et al. 1997; Gerber et al. 1992; Haddad et al. 1998). Countermeasures attempt to reduce the body burden of cesium following inadvertent exposure (see Section 3.12.2).

3.12.2 Reducing Body Burden

Oral administration of Prussian blue (potassium ferricyanoferrate) may enhance the fecal excretion of absorbed cesium (Ducousso et al. 1975; Ellenhorn et al. 1997; Gerber et al. 1992; Haddad et al. 1998;

Melo et al. 1996). Animal studies indicate that excretion of cesium may also be enhanced by the administration of excess potassium (Richmond and Furchner 1961). Higher plasma concentrations of potassium may increase the mobilization of cesium from tissues, and thus increase excretion.

3.12.3 Interfering with the Mechanism of Action for Toxic Effects

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

3.13 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 cesium 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 cesium.

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.

3.13.1 Existing Information on Health Effects of Cesium

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to stable and radioactive cesium are summarized in Figure 3-8 and 3-9, respectively. The purpose of these figures is to illustrate the existing information concerning the health effects of cesium. 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 (Agency for Toxic

Figure 3-8. Existing Information on Health Effects of Stable Cesium

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

Human

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

Animal

● Existing Studies

Figure 3-9. Existing Information on Health Effects of Radioactive Cesium

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

Human

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

Animal

● Existing Studies

Substances and Disease Registry 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.

As shown in Figure 3-8, limited information is available regarding health effects in humans following intermediate-duration oral exposure to stable cesium. No information is available regarding health effects in humans following inhalation or dermal exposure to stable cesium. Information is available on the health effects in animals exposed to stable cesium. However, the available information is mostly from acute oral LD_{50} studies, a single intermediate-duration oral study, and a study of male reproductive toxicity.

As shown in Figure 3-9, limited information is available regarding oral and dermal exposure to radioactive cesium. An accidental exposure of a number of individuals in Goiânia, Brazil resulted in adverse health effects that could be attributed to external and internal (oral and dermal) exposure to radiation from a radioactive cesium source. Information is also available for humans externally exposed to a radiocesium source that resulted in adverse dermal effects. Numerous reports are available regarding other cases of external and internal environmental exposure to radioactive isotopes of cesium in humans, especially from areas with significant amounts of radioactive fallout. However, at present, associations between exposure to environmental levels of radioactive cesium and adverse health effects have not been confirmed. Present environmental levels of radiocesium, therefore, might not represent overexposure to radiation.

Reduced sperm counts in mice were found following oral administration of radioactive cesium. Studies of dogs, intravenously administered $^{137}CsCl$, resulted in hematologic dyscrasia as early effects and tumors of various organs as late effects. Given by this route, the tissue distribution of ^{137}Cs is similar to that resulting from oral or inhalation exposure.

3.13.2 Identification of Data Needs

Acute-Duration Exposure. Results from human and animal studies indicate that stable cesium is of little acute oral toxicity concern. Toxicokinetic data regarding the widespread distribution of cesium absorbed following oral exposure indicate that dermal and inhalation exposure to stable cesium would not present a greater health concern than that posed by oral exposure. Acute-duration inhalation and oral MRLs were not derived for stable cesium due to a lack of human or animal data. To generate appropriate data for deriving acute-duration inhalation and oral MRLs for stable cesium, at least one comprehensive acute inhalation and one acute oral toxicity study would be needed of at least one animal species exposed to several dose levels.

Reports of adverse effects in humans that can be specifically attributed to acute exposure to radioactive cesium are restricted to the accounts of accidental external exposure (Gottlöber et al. 2000) and both external and internal (dermal and oral) exposure (Brandão-Mello et al. 1991) to an opened ^{137}CsCl source. Observed health effects were representative of those resulting from overexposure to other beta- and gamma-emitting sources of ionizing radiation. Animal data regarding acute oral exposure to radioactive cesium are limited to reports of dominant lethal mutations and reduced fertility in mice (Ramaiya et al. 1994). Acute-duration inhalation and oral MRLs were not derived for radioactive cesium due to a lack of human or animal data. To generate appropriate data for deriving acute-duration inhalation and oral MRLs for radioactive cesium, 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, great danger would be posed to investigators considering the exposure of laboratory animals to radioactive cesium at levels great enough to cause significant adverse health effects. Distribution patterns of ^{137}Cs are similar in animals exposed to relatively nontoxic levels of ^{137}CsCl by parenteral injection, inhalation exposure, or oral administration (Boecker et al. 1969a; Stara 1965). Therefore, the results of the intravenous injection studies in dogs (Nikula et al. 1995, 1996) provide the most reasonable indication of health effects that would be expected in animals exposed by inhalation or oral administration. An acute-duration MRL that was derived by Agency for Toxic Substances and Disease Registry (1999) for acute external exposure to ionizing radiation was considered to be appropriate as an acute-duration MRL for external exposure to ionizing radiation from a radioactive cesium source.

Intermediate-Duration Exposure. Limited information regarding intermediate-duration oral exposure in humans indicates that the nervous and cardiovascular systems are the most likely targets of toxicity for high-dose stable cesium (Bangh et al. 2001; Harik et al. 2002; Neulieb 1984; Saliba et al. 2001). Limited data indicate that intermediate-duration oral exposure of pregnant mice to stable cesium may adversely affect developing fetuses (Messiha 1988b, 1989b). Orally administered stable cesium has also been shown to be genotoxic to female mice (Ghosh et al. 1990, 1991). Intermediate-duration inhalation and oral MRLs were not derived for stable cesium due to the paucity of human or animal data. To generate appropriate data for deriving intermediate-duration inhalation and oral MRLs for stable cesium, 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.

No human or animal data are available in which intermediate-duration inhalation or oral exposure to radioactive cesium can be associated with adverse human health effects. When humans are exposed to high levels of radioactivity from a radiocesium source, as were the cases of acute exposure in Goiânia, Brazil (Brandão-Mello et al. 1991) and Russia (Gottlöber et al. 2000), such exposures should be of value in assessing potential health hazards. Animal studies could be designed to assess the health effects associated with intermediate-duration exposure to radioactive cesium. Such studies could be designed to also generate data regarding potential age-related differences in toxicity.

Chronic-Duration Exposure and Cancer. Since there are no studies pertaining to noncancer or cancer health effects in humans or animals following chronic-duration inhalation or oral exposure to stable cesium, no chronic-duration inhalation or oral MRLs were derived for stable cesium. Additional data from acute- and intermediate-duration animal studies might be helpful in determining the need for longer-term studies.

There are no data regarding noncancer or cancer health effects in humans following chronic-duration inhalation or dermal exposure to radioactive cesium. Low levels of radioactive cesium are found in the diets of individuals living in areas that have been contaminated with radioactive fallout; however, there is a lack of information regarding dose-response following chronic-duration oral exposure. No chronic-duration inhalation or oral MRLs were derived for radioactive cesium. A chronic-duration MRL that was derived by Agency for Toxic Substances and Disease Registry (1999) for chronic external exposure to ionizing radiation was appropriate as a chronic-duration MRL for external exposure to ionizing radiation from a radioactive cesium source. Long-term research into health effects associated with chronic exposure to radioactive cesium following incidents such as the Chernobyl nuclear accident may help to elucidate long-term noncancer and cancer health risks from chronic exposure to radionuclides of cesium.

Genotoxicity. No genotoxicity studies of *in vivo* exposure of humans to stable cesium compounds were located. Stable cesium (as cesium chloride) induced chromosomal aberrations in human lymphocytes *in vitro* (Ghosh et al. 1993) and chromosomal aberrations and micronuclei in mouse bone marrow *in vivo* (Ghosh et al. 1990, 1991; Santos-Mello et al. 2001). Cesium sulfate was not mutagenic in *E. coli* either with or without metabolic activation (Olivier and Marzin 1987). Studies in mammalian systems would be useful. Studies of workers exposed to known levels of stable cesium would be useful in establishing whether or not cesium is of genotoxicity concern in humans.

In vivo human data are limited to the findings of increased point mutations in T-lymphocytes and chromosomal aberrations among individuals who had been exposed to beta and gamma radiation from an opened ^{137}CsCl source (Natarajan et al. 1998; Skandalis et al. 1997) and apparently increased frequencies of chromosomal aberrations in lymphocytes of children living in areas contaminated by ^{137}Cs fallout following the Chernobyl nuclear accident (Padovani et al. 1993). *In vitro* exposure of human lymphocytes to a sealed ^{137}Cs gamma source resulted in an increased frequency of micronuclei (Balasem and Ali 1991). *In vivo* oral and external exposure of male mice to a ^{137}Cs gamma source resulted in increases in dominant lethal mutations (Ramaiya et al. 1994). Increases in the frequency of reciprocal translocations in spermatogonia were observed following oral exposure to ^{137}Cs in mice (Ramaiya et al. 1994) and external exposure to a ^{137}Cs source in crab-eating monkeys (Tobari et al. 1988). A number of *in vitro* genotoxicity assays have indicated chromosomal aberrations and breaks, sister chromatid exchanges, and micronuclei in animal cells (Arslan et al. 1986; Biedermann et al. 1991; Doggett and McKenzie 1983; Hintenlang 1993; Iijima and Morimoto 1991; Kamiguchi et al. 1991; Mikamo et al. 1990, 1991).

Human and animal studies show that external and internal exposure to radioactive cesium is a genotoxicity concern. External exposure to any gamma source would be expected to result in genotoxic effects similar to those observed following external exposure to radioisotopes of cesium (see Agency for Toxic Substances and Disease Registry 1999 for more information on ionizing radiation). Whenever possible, additional human studies should focus on exposure levels to establish dose-response relationships.

Reproductive Toxicity. There are no reports of reproductive effects in humans or animals exposed to stable cesium. Although stable cesium appears to be of relatively low toxicity concern, animal studies could be designed to assess the potential for adverse health effects (including reproductive effects) associated with repeated exposure. Human data could be collected from individuals occupationally exposed to significant levels of stable cesium.

Reports of human reproductive effects following exposure to radioactive cesium are limited to the findings of reduced sperm counts following external and internal exposure to an opened ^{137}CsCl source (Brandão-Mello et al. 1991). Mice, exposed to ^{137}Cs either orally or externally, exhibited reduced fertility (including complete sterility) (Ramaiya et al. 1994). Persistent germinal epithelium damage and azoospermia were observed in all long-term surviving dogs that had been administered intravenous injections of ^{137}Cs (Nikula et al. 1995, 1996). In cases of known human exposure to radioactive cesium, exposure-response relationships should be established when possible. Although reduced fertility has been shown in males, additional animal studies could be designed to assess the potential for reproductive toxicity in females.

Developmental Toxicity. There are no reports of developmental effects in humans exposed to stable cesium. One investigator reported reduced body weight and certain organ weights among offspring, as well as indications of altered activity of some hepatic enzymes among offspring of pregnant mouse dams repeatedly exposed (orally) to stable cesium (Messiha 1988b). The same investigator reported similar results in pups exposed (via their nursing mothers) only during lactation (Messiha 1989b). These studies did not include gross and histopathologic examination of the offspring. Well-designed animal studies could more completely assess the potential for developmental toxicity of stable cesium.

Although there are no reports of developmental effects in humans exposed specifically to radioisotopes of cesium, impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed to high levels of ionizing radiation during critical stages of neural development (Schull and Otake 1999; Schull et al. 1988). External exposure to sufficiently high doses of radiation from a radioactive cesium source would be expected to result in similar effects. *In utero* exposure of rat and mouse fetuses via whole body exposure of dams resulted in developmental effects such as reduced postnatal body weight, impaired motor activity, morphological changes in the brain, increased aggressive behavior, reduced brain and head size, and retarded odontogenesis and palatal closure (Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994). Continued monitoring of populations known to have been exposed to ionizing radiation (including radioactive cesium sources) should help to refine estimates of dose-response and the relationship to adverse health effects, including developmental toxicity.

Immunotoxicity. There are no reports regarding the immunotoxicity of stable cesium in humans or animals. Animal studies could be designed to assess these parameters, but such studies do not presently seem necessary.

Severe bone marrow depression was observed in individuals exposed externally and internally to a ^{137}Cs source. This effect is typical of individuals exposed to ionizing radiation (see Agency for Toxic

Substances and Disease Registry 1999 for additional information on the effects of ionizing radiation). A similar effect was observed in dogs given an intravenous injection of ^{137}CsCl (Nikula et al. 1995). Data should be collected from individuals known to have been exposed to ionizing radiation (including that from radioactive cesium sources). Additional animal studies could be designed to establish dose-response relationships.

Neurotoxicity. Data regarding neurological effects of stable cesium in humans are restricted to a single case of an investigator reporting feelings of euphoria, heightened sense perception, and tingling sensations within 15 minutes of ingesting oral doses of cesium chloride during a 36-day exposure period. No apparent adverse effects on mental or motor skills were observed (Neulieb 1984). Administration of cesium chloride to animals has triggered stimulant (Johnson 1972; Messiha 1978) and depressant (Bose and Pinsky 1981, 1983b, 1984; Bose et al. 1981; Pinsky et al. 1980) central nervous system responses. Additional animal studies could be designed to elucidate mechanisms responsible for the observed neurological effects.

Although there are no reports of neurotoxicity in humans exposed specifically to radioisotopes of cesium, impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed to high levels of external ionizing radiation during critical stages of neural development (Schull and Otake 1999; Schull et al. 1988). External exposure to sufficiently high doses of radiation from a sealed radioactive cesium gamma source would result in similar effects. *In utero* exposure of rat and mouse fetuses via whole-body exposure of dams resulted in impaired motor activity, morphological changes in the brain, increased aggressive behavior, and reduced brain and head size (Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994), effects that have been shown to be related to critical developmental stages. Neurotoxic effects, noted in humans suffering from acute radiation syndrome due to ionizing radiation exposure, are well-characterized (see Agency for Toxic Substances and Disease Registry 1999 for more detailed information on the effects of ionizing radiation). Such effects would be expected in humans exposed to sufficiently high doses of radiation from a radioactive cesium source. Additional well-designed animal studies might elucidate mechanisms of neurotoxicity, but do not presently appear to be needed.

Epidemiological and Human Dosimetry Studies. Stable cesium is ubiquitous in the earth's crust, but is found at such low environmental levels that the probability of human intake of toxic amounts of stable cesium is negligible. Although there is no apparent need for specifically designed epidemiological or human dosimetry studies regarding stable cesium, such data, collected from individuals occupationally exposed to significant amounts of cesium or persons living near toxic waste sites with significant levels of cesium, might be useful.

Due to accidental or intentional releases during nuclear fission, ^{134}Cs and ^{137}Cs can be found in air, soil, water, and food. There is concern for the health of humans living in close proximity to release or storage sites or in areas receiving significant amounts of radioactive fallout. Epidemiological studies of radiation doses typically involve estimates of exposure that are based on whole-body measurements of internally-deposited ^{134}Cs or ^{137}Cs or genotoxic effects such as chromosomal aberrations in peripheral blood lymphocytes. 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 radioisotopes of cesium; in particular, for cases of known internal exposure.

Biomarkers of Exposure and Effect.

Exposure. Both stable and radioactive isotopes of cesium may be detected in samples of urine, blood, feces, or body tissues. Due to the relatively long biological half-time of cesium (several months in humans), short-term exposures cannot be readily distinguished from longer-term ones. No new biomarkers of exposure are needed at this time.

Effect. No known biomarkers of effect exist for exposure to stable cesium. Although high radiation doses from internally deposited radioactive cesium can cause bone marrow aplasia, altered blood values, and increased chromosomal aberrations in lymphocytes (Brandão-Mello et al. 1991; Natarajan et al. 1998), these effects are not specific to radioactive cesium.

Absorption, Distribution, Metabolism, and Excretion. Human and animal data show that inhaled or ingested cesium (in soluble compounds) is rapidly absorbed into the blood (Boecker 1969a, 1969b; Henrichs et al. 1989; Lie 1964; Miller 1964; Stara 1965; Stara and Thomas 1963), whereas relatively insoluble forms of cesium are not readily absorbed into blood following inhalation or oral exposure (Boecker et al. 1974, 1977; Leroy et al. 1966; Talbot et al. 1993). Dermal absorption has been qualitatively (but not quantitatively) demonstrated in rats (Pendic and Milivojevic 1966). Additional studies could measure relative absorption rates for a variety of soluble and insoluble cesium compounds. Furthermore, studies could be designed to measure dermal absorption rates. Other studies could further assess the fate of relatively insoluble inhaled particles containing radioisotopes of cesium that may be retained in lung tissue for long periods of time.

Cesium absorbed via inhalation or ingestion has been shown to be rapidly distributed throughout the body in humans and animals (Boecker 1969a, 1969b; Furchner et al. 1964; Lie 1964; Miller 1964; Rosoff et al. 1963; Stara 1965; Stara and Thomas 1963). Once absorbed by pregnant women, cesium can pass the placental barrier and be absorbed by the conceptus. Absorbed cesium can also be found in the milk of lactating women (Thornberg and Mattsson 2000). Once cesium is absorbed into body fluids, distribution patterns in soft tissue are expected to be similar for any route of exposure since cesium is distributed throughout the body as the cation (Cs^+), much like potassium (K^+). Available studies appear to adequately describe the distribution of absorbed cesium. Additional studies could be designed to elucidate mechanisms whereby cesium ions may influence central nervous system activity.

Human and animal studies adequately describe elimination of absorbed cesium, primarily via the urine (Boecker 1969b; Hölgye and Malý 2002; Iinuma et al. 1965; Rosoff et al. 1963; Stara 1965; Stara and Thomas 1963). Age-related differences in elimination rates have been described in humans (Boni 1969b; Melo et al. 1994) and dogs (Melo et al. 1996). In cases of known human exposure to cesium, additional information may help to further assess age-related differences in the toxicokinetics of cesium.

Comparative Toxicokinetics. Available cesium toxicokinetic data in humans and various animal species indicate similar patterns of absorption, distribution, and elimination. The central nervous system appears to be a target for effects in humans (Neulieb 1984) and animals (Bose and Pinsky 1981, 1983b, 1984; Bose et al. 1981; Johnson 1972; Messiha 1978; Pinsky et al. 1980). Additional studies could be designed to elucidate and compare mechanisms responsible for central nervous system effects.

Methods for Reducing Toxic Effects. Oral administration of Prussian blue that exchanges potassium for cesium, cathartics that shorten the transit time of ingested cesium within the gastrointestinal tract, and gastric lavage may aid in reducing peak absorption of ingested cesium, but due to the rapid absorption of cesium from soluble cesium compounds, these measures would only be of potential benefit within a short time following initial exposure. Most countermeasures focus on reducing the body burden of absorbed cesium. The intestinal reabsorption of cesium that is excreted into the small intestine via the bile can be blocked by oral administration of Prussian blue, forming an insoluble cesium complex that is excreted in the feces. Animal studies also indicate that increased plasma concentrations of potassium may increase the mobilization of cesium from tissues, increasing its excretion (Richmond and Furchner 1961). There are no prescribed methods for interfering with mechanisms of action for toxic effects of cesium, since such mechanisms have not been elucidated.

Children's Susceptibility. Available information on age-related differences in health effects comes from *in utero* exposure to radiation from external ionizing radiation sources (including radioisotopes of cesium). Studies have shown neurological effects in humans and animals exposed during critical periods of central nervous system development (Koshimoto et al. 1994; Minamisawa et al. 1990; Norton and Kimler 1987, 1988; Schull and Otake 1999). Comparative studies of neurological effects in animals first irradiated as juveniles or adults are lacking. No information was located regarding age-related health effects in humans or animals exposed to stable cesium.

PBPK models account for potential age-related differences in deposition of inhaled cesium, as well as differences in elimination rates for absorbed cesium (see Section 3.5.5 for more information on PBPK models). Potential differences in absorption rates of inhaled cesium compounds could be due to age-related differences in physical properties of the respiratory system and/or ventilation patterns. PBPK models also could adjust for potential age-related differences in gastrointestinal absorption. If such differences exist, they would likely be the result of differences in diffusion rates and active transport mechanisms. Young children exhibit biological half-times for absorbed cesium that are shorter than those of older children and adults (Boni 1969b; Melo et al. 1994); these age-related differences are associated with body mass and could be the result of age-related differences in tissue retention and excretory rates.

Cesium is found in the breast milk of mothers with an internal cesium burden (Johansson et al. 1998; Thornberg and Mattsson 2000), and can be transferred to nursing infants (Johansson et al. 1998). Cesium has been shown to cross the placental barrier of animals, but concentrations of cesium in the fetal tissues are less than those in corresponding tissues of the mother (Mahlum and Sikov 1969; Vandecasteele et al. 1989). Data collected from areas containing elevated concentrations of radioactive cesium fallout might be of value in further assessing age-related transfer rates.

Biomarkers of exposure or effect are the same in adults and children (see Section 3.9 for detailed information on biomarkers of exposure and effect). There are no data on interactions of cesium with other chemicals in children. No pediatric-specific methods have been found to reduce peak absorption, or body burden, of cesium following exposure, although methods employed to reduce the body burden of cesium in adults are also effective in children.

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

3.13.3 Ongoing Studies

A single relevant ongoing study was identified in the Federal Research In Progress database (FEDRIP 2002). Dr. R. Albertini, from the University of Vermont, Burlington, Vermont, is quantitatively examining radiation-induced mutation at the hprt gene in human T-lymphocytes. Gamma radiation sources used in the study include ^{137}Cs.

4. CHEMICAL, PHYSICAL, and RADIOLOGICAL INFORMATION

4.1 CHEMICAL IDENTITY

Information regarding the chemical identity of cesium is located in Table 4-1.

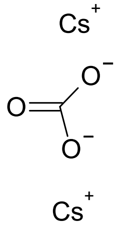
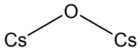
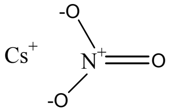
4.2 PHYSICAL, CHEMICAL, AND RADIOLOGICAL PROPERTIES

Information regarding the physical and chemical properties of cesium is located in Table 4-2.

Cesium is a silvery white, soft, ductile metal with only one oxidation state (+1). At slightly above room temperature, cesium exists in the liquid state. Compared to the other stable alkali metals, cesium has the lowest boiling point and melting point, highest vapor pressure, highest density, and lowest ionization potential. These properties make cesium far more reactive than the other members of the alkali metal group. When exposed to air, cesium metal ignites, producing a reddish violet flame, and forms a mixture of cesium oxides. Pure cesium reacts violently with water to form cesium hydroxide, the strongest base known, as well as hydrogen gas. The burning cesium can ignite the liberated hydrogen gas and produce an explosion. Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility.

There are several radioactive isotopes of cesium ranging from ^{114}Cs to ^{145}Cs (Helmers 1996). The radioactive isotopes have a wide range of half-lives ranging from about 0.57 seconds (^{114}Cs) to about 3×10^6 years (^{135}Cs) (Helmers 1996). The radioactive isotopes ^{137}Cs and ^{134}Cs are significant fission products because of their high fission yield, and their relatively long half-lives. The fission yield of ^{137}Cs in nuclear reactions is relatively high; about 6 atoms of ^{137}Cs are produced per 100 fission events (WHO 1983). ^{137}Cs has a radioactive half-life of about 30 years and decays by beta decay either to stable ^{137}Ba or a meta-stable form of barium (^{137m}Ba). The meta-stable isotope (^{137m}Ba) is rapidly converted to stable ^{137}Ba (half-life of about 2 minutes) accompanied by gamma ray emission whose energy is 0.662 MeV (ICRP 1983). Figure 4-1 illustrates this decay scheme. The first beta decay mode that forms ^{137m}Ba accounts for roughly 95% of the total intensity, while the second mode accounts for about 5% (WHO 1983). Radioactive ^{134}Cs primarily decays to stable ^{134}Ba by beta decay accompanied by gamma ray emissions or less frequently to stable ^{134}Xe by electron capture (EC) accompanied by a single gamma ray

Table 4-1. Chemical Identity of Cesium and Compounds

Characteristic	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide	Cesium nitrate
Synonym(s)	Caesium	Cesium monochloride	Dicesium salt	Cesium hydrate		
Registered trade name(s)	No data	No data	No data	No data	No data	No data
Chemical formula	Cs	CsCl	Cs_2CO_3	CsOH	Cs_2O	$CsNO_3$
Chemical structure	Cs	Cs—Cl		Cs—OH		
Identification numbers:						
CAS registry	7440-46-2	7647-17-8	534-17-8	21351-79-1	20281-00-9	7789-18-6
NIOSH RTECS	FK9225000	FK9625000	FK9400000	FK9800000	No data	
EPA hazardous waste	No data	No data	No data	No data	No data	No data
OHM/TADS	No data	No data	No data	No data	No data	No data
DOT/UN/NA/IMCO shipping	No data	No data	No data	No data	No data	No data
HSDB	No data	No data	No data	No data	No data	No data
NCI	No data	No data	No data	No data	No data	No data

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

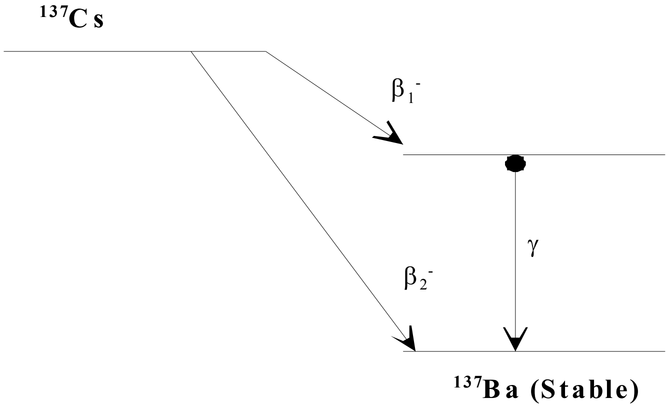
Table 4-2. Physical and Chemical Properties of Cesium and Compounds

Property	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide	Cesium nitrate
Molecular weight	132.906	168.36	325.82	149.91	281.81	194.91[b]
Color	Silvery-white	White	White	Colorless	Golden-yellow	White[b]
Physical state	Solid (liquid slightly above room temperature)	Solid	Solid	Solid	Solid	Solid[b]
Melting point	29 °C	646 °C	610 °C	272 °C	490 °C	414 °C[b]
Boiling point	685 °C	1290 °C	No data	No data	No data	No Data
Density, g/cm^3	1.93 (20 °C)[b]	3.988 (20 °C)[b]	4.24 (20 °C)[b]	3.68 (20 °C)[b]	4.65 (20 °C)[b]	3.66 (20 °C)[b]
Odor	No data	No data	No data	No data	No data	No data
Odor threshold:						
Water	No data	No data	No data	No data	No data	No data
Air	No data	No data	No data	No data	No data	No data
Solubility:						
Water	Reacts violently with water	1.87 kg/L (20 °C)	2.1 kg/L	4 kg/L (15 °C)	Very soluble in water[b]	Soluble[b]
Organic-solvent(s)		Soluble in ethanol[b]	Soluble in ethanol and ether[b]	Soluble in ethanol[b]		
Partition coefficients:						
Log K_{ow}	N/A	N/A	N/A	N/A	N/A	N/A
Log K_{oc}	N/A	N/A	N/A	N/A	N/A	N/A
Vapor pressure	0.0075 mmHg at 144.5 °C	No data	No data	No data	No data	No data
Henry's Law constant	N/A	N/A	N/A	N/A	N/A	N/A
Autoignition temperature	No data	No data	No data	No data	No data	
Flashpoint	No data	No data	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data	No data	No data
Conversion factors	No data	No data	No data	No data	No data	No data
Explosive limits	No data	No data	No data	No data	No data	No data

[a] Data from Burt 1993 unless otherwise specified.
[b] Lide 1998

N/A = not applicable

Figure 4-1. The Decay Scheme of ^{137}Cs



β = beta decay; γ= γ -ray emission

emission as depicted in Figure 4-2. The energy of the various gamma rays are in the range of 0.24–1.4 MeV. The half-life average energy of the beta transitions and intensity of the transitions for both ^{134}Cs and ^{137}Cs are summarized in Table 4-3.

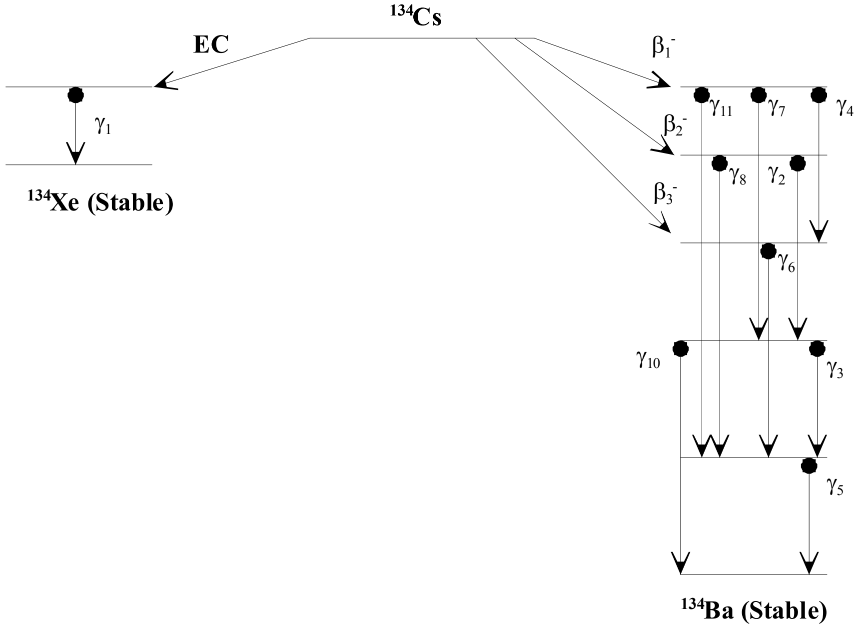
Table 4-3. Decay Properties of the Radioactive Isotopes of Cesium

Isotope	Half-life (years)	Decay mode	Intensity percent	Beta particle energy (MeV)
^{134}Cs	2.062	β_1^-	27	0.02309
		β_2^-	2.5	0.1234
		β_3^-	70	0.2101
^{137}Cs	30	β_1^-	94.6	0.1734
		β_2^-	5.4	0.4246

[a] ICRP 1983

[b] The ^{134}Xe daughter yield from the electron capture decay of ^{134}Cs is approximately $3x10^{-6}$.

Figure 4-2. The Decay Scheme of ^{134}Cs



EC = electron capture; β = beta decay; γ = gamma-ray emission

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

5.1 PRODUCTION

Cesium is the rarest of the naturally occurring alkali metals, ranking 40th in elemental abundance. Although it is widely distributed in the earth's crust, cesium is found at relatively low concentrations. Granites contain cesium at about 1 ppm of cesium and sedimentary rocks contain approximately 4 ppm of cesium. Cesium is found in muscovite, beryl, spodumene, potassium feldspars, leucite, petalite, and other related minerals. The most common commercial source of cesium is pollucite, which contains between 5 and 32% Cs_2O (Burt 1993). There are three basic methods of converting pollucite ore to cesium metal or related compounds: direct reduction with metals; decomposition with bases; and acid digestion. In each method, grinding of the mined ore to approximately 75 μm sized particles precedes chemical conversion.

Direct Reduction With Metals. Pollucite is directly reduced by heating the ore in the presence of calcium to 950 °C in vacuum, or in the presence of either sodium or potassium to 750 °C in an inert atmosphere (Burt 1993). Excessive amounts of reducing metal are required, and the resultant cesium metal is impure, requiring further distillation and purification.

Decomposition With Bases. Alkaline decomposition is carried out by roasting the pollucite ore with either a calcium carbonate-calcium chloride mix at 800–900 °C or with a sodium carbonate-sodium chloride mix at 600–800 °C followed by a water leach of the roasted mass. The resultant cesium chloride solution is separated from the gangue by filtration (Burt 1993).

Acid Digestion. Acid digestion of pollucite is the preferred commercial process for producing pure cesium. Hydrofluoric, hydrobromic, hydrochloric, and sulfuric acid may be employed in this method. Hydrofluoric and hydrobromic acid digestion yield the greatest cesium recovery, but the inherent difficulties of safely handling these acids limit their use. Digestion with hydrochloric acid takes place at elevated temperatures and produces a solution of mixed cesium chlorides, aluminum, and other alkali metals separated from the siliceous residue by filtration. The impure cesium chloride can be purified as cesium chloride double salts that are then recrystallized. The purified double salts are decomposed to cesium chloride by hydrolysis or precipitated with hydrogen sulfide (Burt 1993).

Sulfuric acid digestion is performed at 110 °C with a 35–40% solution of sulfuric acid, followed by a hot water wash and vacuum filtration. Cesium alum is crystallized from the leach filtrate by stage cooling to 50 °C and then to 20 °C and roasted in the presence of 4% carbon. The residue is leached to produce cesium sulfate solution, which can be converted to cesium chloride (Burt 1993).

Radioactive isotopes of cesium such as ^{134}Cs and ^{137}Cs are produced by nuclear fission in fuel rods in nuclear power plants and in fallout from nuclear weapons. Radiocesium can be recovered from fission products by digestion with nitric acid. After filtration to remove the waste, the radioactive cesium phosphotungstate is precipitated using phosphotungstic acid (Burt 1993). Other processes for the removal of ^{134}Cs and ^{137}Cs from radioactive waste involve solvent extraction using macrocyclic polyethers, or crown ethers and coprecipitation with sodium tetraphenylboron (Burt 1993).

5.2 IMPORT/EXPORT

The United States is 100% import-reliant for elemental cesium. No economic data, such as production volume, consumption, or import/export volumes of cesium, are available. Although there is no information regarding the countries shipping cesium or cesium compounds to the United States, it is believed that Canada is the major foreign source of cesium (USGS 1999). Other possible sources of cesium-bearing material include Germany and the United Kingdom.

5.3 USE

There are relatively few commercial uses for cesium metal and its compounds. Cesium is used as a getter (combines chemically with residual gas in partial vacuum to increase the vacuum) for residual gas impurities in vacuum tubes and as a coating to reduce the work function of the tungsten filaments or cathodes of the tubes. Crystalline cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionizing radiation into pulses of visible light (Burt 1993). Cesium is also used in magnetohydrodynamic power generators as a plasma seeding agent (Lewis 1997). Recently, cesium compounds have been employed as catalysts in organic synthesis, replacing sodium or potassium salts. One of the most interesting uses of cesium is in the production of highly accurate atomic clocks. When exposed to microwave radiation, the natural vibration of cesium atoms occurs at a frequency of 9,192,631,770 Hz, and 1 second in time is defined as the duration of 9,192,631,770 periods of radiation absorbed or emitted by the transition of ^{133}Cs atoms in two hyperfine levels of their ground state. Radioactive ^{137}Cs has been approved as the gamma ray source for the sterilization of wheat, flour, potatoes, surgical equipment and other medical supplies, and sewage sludge, and is also used as a calibration source in gamma ray spectroscopy (Lewis 1997). ^{137}Cs is also used in industrial radiography and for imaging of transport containers at border crossings. ^{131}Cs was recently approved by the U.S. FDA as an active source in radioactive seeds used to treat prostate cancer (FDA 2003).

5.4 DISPOSAL

Because of its high reactivity, special precautions are required for the handling and disposal of pure cesium metal. Cesium metal is usually stored and transported in stainless steel containers, which are contained in outer packing, to ensure that the metal remains in a dry, oxygen-free environment.

Most nonradioactive cesium minerals, compounds, and materials do not require special disposal and handling requirements. However, some chemical forms may be classified as hazardous materials if the compound is chemically reactive, flammable, or toxic. Care should be taken to read and understand all of the hazards, precautions, and safety procedures for each specific chemical form. In addition, all federal, state, and local laws and regulations should be investigated and subsequently followed with regard to disposal and handling of the specific chemical form of the cesium mineral, compound, or material.

Radioactive cesium requires special disposal and handling requirements. Radioactive waste containing ^{134}Cs and ^{137}Cs is usually grouped into four categories: low-level waste (LLW), high-level waste (HLW), mixed waste, and spent nuclear fuel.

Low-level waste is all radioactive waste that cannot be classified as either HLW, spent fuel, or mixed waste. Low-level does not necessarily mean low radioactivity or low environmental hazard. Low-level waste types that may be contaminated with ^{134}Cs and ^{137}Cs include both wet and dry wastes. Examples of the physical form of LLW are: spent ion exchange resins, filter sludges, filter cartridges, evaporator bottoms, compactible trash, noncompactible trash, irradiated components, ashes produced from the incineration of combustible material, contaminated detergents or solvents, organic liquids, and discarded contaminated equipment or tools. Of the LLW generated today, approximately 64% of the volume and 70% of the radioactivity is generated as a result of nuclear power plant activities or supporting fuel cycle operations. Other sources of LLW are commercial, academic, and government research laboratories and medical facilities. Radiocesium contamination accounts for only a small fraction of the activity of LLW. Nearly all of the ^{134}Cs and ^{137}Cs produced as a result of fission events remains trapped within the spent nuclear fuel rods (DOE 1996b). Low-level waste from Department of Energy (DOE) sources is currently disposed of at several DOE facilities across the United States. Only two sites accept non-DOE LLW: Barnwell, South Carolina and Richland, Washington (DOE 1996a). As required by the Federal Low Level Radioactive Waste (LLRW) Policy Act in 1980 and the 1985 amendments, states are required to build facilities to contain LLW generated from sources within its boundaries. The law encourages states to cooperate together in coordinating LLW disposal facilities. Many states have formed “compacts” to collaborate on construction of these LLW facilities. However, other than Barnwell, South Carolina and Richland, Washington, no other facility in the United States is accepting LLW from non-DOE sources (Eisenbud 1987). Over half of the LLW in the eastern United States is disposed of at the Barnwell site (Eisenbud 1987). The method of disposal for LLW has been to package the material in drums or boxes and bury the material in shallow pits and trenches. Approximately 3 million cubic meters of LLW generated has been disposed of in this way (DOE 1996a).

As defined by the Nuclear Waste Policy Act, HLW is “the highly radioactive material resulting from the reprocessing of spent nuclear fuel, including liquid waste produced directly in reprocessing and any solid material derived from such liquid waste that contains fission products in sufficient concentration...” (42 USC 100). Most HLW has been generated from the production of plutonium. A smaller fraction is related to the recovery of enriched uranium from naval reactor fuel. This waste typically contains highly concentrated solutions of relatively long-lived fission by-products such as ^{90}Sr and ^{137}Cs, hazardous chemicals, and toxic heavy metals. Liquid HLW is typically stored in large underground tanks of either stainless steel or carbon steel depending on whether they are acid or alkaline solutions. Approximately 100 million gallons of liquid HLW is stored in underground tanks in Washington, South Carolina, Idaho, and New York. These tanks contain a variety of radioactive liquids, solids, and sludges. Some of the liquid waste has been solidified into glass, ceramic slag, salt cake, and sludge. High-level waste in solid form is stored in underground bins (DOE 1996a).

Mixed waste contains both radioactive and chemically hazardous materials. All HLW is managed as mixed waste and some LLW is classified as mixed waste. Certain hazardous mixed wastes that contain radioactive isotopes have been incinerated in Oak Ridge, Tennessee (DOE 1996a). Spent nuclear fuel, such as fuel elements and irradiated targets used in nuclear reactors, are currently stored at the commercial nuclear power plants and DOE facilities where they were produced. Spent fuel is highly radioactive, due to the large concentration of fission products, and must be stored in special water-cooled pools that shield and cool the material. Nearly all of the DOE spent fuel, about 3,000 metric tons, is stored at four sites: Hanford, Savannah River, Idaho National Environmental and Engineering

Laboratory, and West Valley. Commercial reactors have generated >30,000 metric tons of spent fuel. The spent fuel from these facilities is located at >100 commercial nuclear reactor sites around the country.

The establishment of a HLW and spent fuel repository for both DOE and commercial waste is currently under evaluation at Yucca Flats, Nevada. This commercial waste storage facility will not be ready to accept spent fuel before 2010.

6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

Cesium has been identified in at least 8 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2003). It was reported that ^{134}Cs has been found in at least 3 of the 1,636 current or former NPL sites and ^{137}Cs has been detected in at least 23 of the 1,636 current or former NPL sites. However, the number of sites evaluated for cesium is not known. The frequency of these sites can be seen in Figures 6-1, 6-2, and 6-3. Of these sites, none are located in the Commonwealth of Puerto Rico.

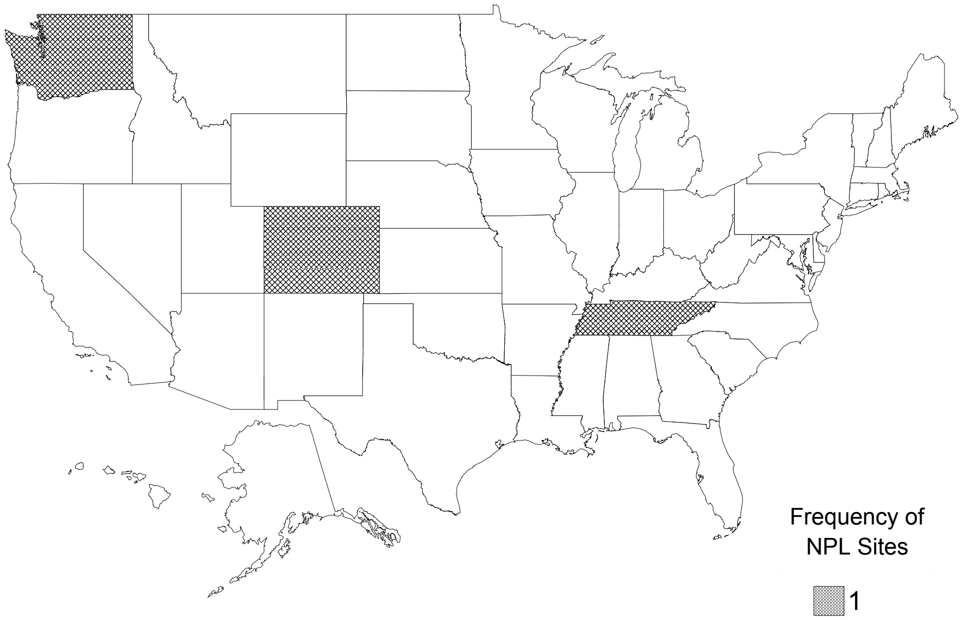
Naturally-occurring cesium and cesium minerals consist of only one stable isotope, ^{133}Cs. Cesium occurs in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993). Higher concentrations are found in lepidolite, carnallite, muscovite, beryl, spodumene, potassium feldspars, leucite, petalite, and related minerals. The most important source of commercial cesium is the mineral pollucite, which usually contains about 5–32% Cs_2O (Burt 1993). The largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world’s known supply. Smaller deposits are located in Zimbabwe, Namibia, Brazil, Scandinavia, Czechoslovakia, and the United States. Continental dust and soil erosion are the main emission sources of naturally occurring cesium present in the environment. Cesium is also released to the environment as a result of human activities. The mining of pollucite ores and the production and use of cesium compounds in the electronic and energy production industries contribute to its direct release to the environment. Cesium has also been detected in the fly ash of hazardous waste incinerators and coal burning power plants (Fernandez et al. 1992; Mumma et al. 1990). Since the production and use of cesium compounds are limited, and since the natural concentration of cesium in the earth's crust is low, stable ^{133}Cs is not often monitored or detected in the environment.

Of much greater concern is the release of radioactive forms of cesium to the environment, such as ^{137}Cs and ^{134}Cs. These and other radioactive isotopes were released to the environment as a result of atmospheric testing of nuclear weapons (carried out from 1945 to 1980) and accidents that occurred at nuclear power plants such as the incident at the Chernobyl nuclear power plant in 1986 and the accident at the Windscale nuclear weapons facility in the United Kingdom in 1957. Small amounts of ^{137}Cs and ^{134}Cs

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

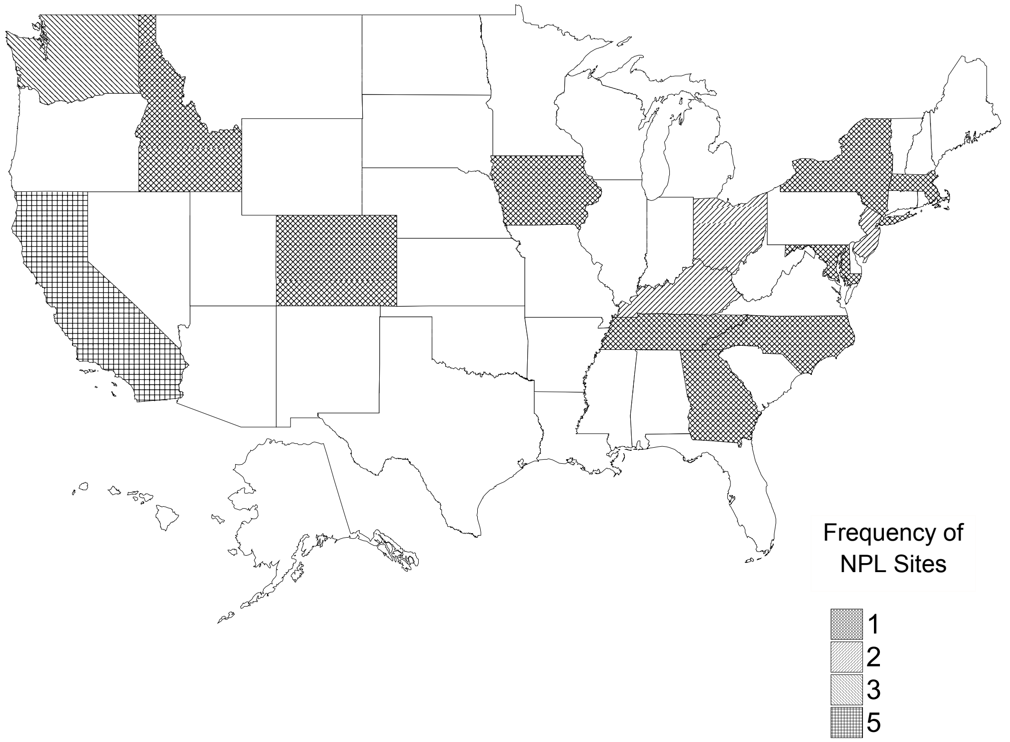
Derived from HazDat 2003

Figure 6-2. Frequency of NPL Sites with ^{134}Cs Contamination



Derived from HazDat 2003

Figure 6-3. Frequency of NPL Sites with ^{137}Cs Contamination



Derived from HazDat 2003

are also released in the airborne and liquid effluents during the normal operation of nuclear power plants. These levels are very low in comparison to the amounts released from weapons tests and accidents at nuclear power plants and are not expected to have a major impact upon human health. For the most part, testing of nuclear weapons has been discontinued by most nations for many years now. However, India and Pakistan have recently (May of 1998) conducted limited underground tests of nuclear weapons (UN 1998).

Radioactive materials are commonly referred to by activity rather than mass. The activity is the number of transformations (disintegrations) that the material undergoes in a given period of time. The most common units of activity are the curie (Ci) or the becquerel (Bq). One curie is equal to 3.7×10^{10} transformations per second (tps) and 1 becquerel is equal to 1 tps. For convenience, picocuries (pCi) are often reported for lower activities; $1 \text{pCi} = 1 \times 10^{-12} \text{Ci} = 0.037 \text{Bq}$.

Radioactive cesium is removed from the air by wet and dry deposition and can travel thousands of miles before settling to earth. Wet deposition is considered the most important pathway for the removal of radioactive cesium from the atmosphere. It is a complex process that depends upon meteorological conditions such as temperature, the microphysical structure of the clouds, and the rainfall rate, as well as the physical and chemical properties of the airborne cesium.

Cesium has very low mobility in soil. In general, it has been reported that cesium usually does not migrate below depths of about 40 cm, with the majority of cesium being retained in the upper 20 cm of the soil surface (Korobova et al. 1998; Takenaka et al. 1998). Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles (Paasikallio 1999). The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays. These factors can limit the uptake of cesium in grass and plant material. There are exceptional areas, however, where cesium fixation in soil is much less, resulting in greater transport in the soil and uptake in plants. Regions in Venezuela, Brazil, and Russia have been identified where the mobility of cesium is considerably greater than in other soils (LaBrecque and Rosales 1996; WHO 1983). Cesium is also deposited on plants and trees by wet and dry deposition and is absorbed into the flora through its foliage (Sawidis et al. 1990). The deposited cesium can make its way to soil through decomposition of the contaminated foliage.

Since the half-life for some radioactive isotopes of cesium is long (the half-life of ^{137}Cs is about 30 years and the half-life of ^{134}Cs is about 2 years), the general population is exposed to ^{137}Cs and ^{134}Cs for long periods of time after it is released from a nuclear accident or weapons test, with the greatest exposure occurring near the source. Although inhalation and dermal exposure is possible, oral ingestion of contaminated food items is the greatest source of internal exposure for both naturally occurring and radioactive cesium. Cesium is uniformly distributed throughout the whole body similar to potassium, and it does not accumulate in any one particular part of the body like iodine (thyroid) or strontium (bones). For this reason, radioactive cesium only poses significant health risks if a large amount has been ingested. Workers employed in the mining and milling of pollucite ores and the production of cesium compounds are exposed to cesium through oral, dermal, and inhalation routes. Similar routes of exposure to ^{137}Cs and ^{134}Cs occurs for workers employed in the nuclear industry. External exposure to gamma radiation can also occur for workers employed in the nuclear industry as well as for the general population. The health consequences of external exposure to gamma radiation are not unique to ^{137}Cs and ^{134}Cs, but are similar for all gamma emitting radionuclides.

6.2 RELEASES TO THE ENVIRONMENT

Throughout this chapter, the units used to express concentration or intakes of cesium are the same units reported by the authors. In most cases, values are expressed in mass units when referring to ^{133}Cs, while radioactive cesium isotopes are expressed in units of activity.

According to the Toxics Release Inventory (TRI), in 2000, there were no reportable releases of ^{133}Cs, or its compounds into the environment by commercial sources (TRI00 2002). The TRI data should be used with caution since only certain types of facilities are required to report. Therefore, it is not an exhaustive list. Facilities are required to report to TRI if they have 10 or more full-time employees, or if the facility is classified under Standard Industrial Classification (SIC) codes 20–39, if the facility manufactures or processes over 25,000 pounds of the chemical, or otherwise uses >10,000 pounds of the chemical in a calendar year.

In the United States, commercial nuclear power plant operators are required to report any detectable quantities of radioactive materials released to the environment (10 CFR 50.36a). Table 6-1 summarizes releases of ^{137}Cs and ^{134}Cs to the atmosphere and water for 1993 from pressurized water reactor (PWR) and boiling water reactor (BWR) nuclear power plants. Nearly all of the radioactive material reported as being released in effluents are from planned releases. Planned releases result from normal plant operation or from anticipated operational occurrences. The latter include unplanned releases of radioactive

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Boiling water reactors					
Browns Ferry[b]	Decatur, AL	0.033	0.18	3.5x10^{-4}	8.9x10^{-4}
Brunswick[b]	Wilmington, NC	2.9x10^{-4}	1.7x10^{-3}	No data	7.0x10^{-4}
Clinton	Clinton, IL	No data	No data	No data	No data
Cooper	Omaha, NE	9.3x10^{-3}	0.052	No data	No data
Dresden[b]	Joliet, IL	1.2x10^{-6}	0.025	No data	1.9x10^{-4}
Duane Arnold	Cedar Rapids, IA	No data	No data	No data	1.4x10^{-6}
Edwin I. Hatch	Baxley, GA	6.3x10^{-3}	0.044	No data	6.2x10^{-5}
Fermi	Laguna Beach, MI	No data	8.3x10^{-6}	No data	No data
Grand Gulf	Vicksburg, MS	3.5x10^{-4}	6.0x10^{-4}	2.1x10^{-6}	2.3x10^{-6}
Hope Creek	Wilmington, DE	No data	4.8x10^{-5}	No data	No data
Humbolt Bay[b]	Eureka, CA	No data	8.8x10^{-3}	No data	3.2x10^{-5}
James A. Fitzpatrick	Syracuse, NY	No data	4.1x10^{-5}	No data	6.6x10^{-6}
LaCrosse[b]	LaCrosse, WI	3.2x10^{-4}	0.010	No data	1.0x10^{-4}
LaSalle	Ottawa, IL	No data	No data	No data	No data
Limerick	Philadelphia, PA	2.0x10^{-3}	6.0x10^{-3}	No data	No data
Millstone	New London, CT	0.066	0.224	6.9x10^{-6}	8.9x10^{-5}
Monticello	St. Cloud, MN	No data	No data	No data	4.8x10^{-4}
Nine Mile Point	Oswego, NY	No data	No data	No data	1.9x10^{-5}
Oyster Creek	Toms River, NJ	No data	No data	No data	6.5x10^{-5}
Peach Bottom	Lancaster, PA	5.2x10^{-4}	1.4x10^{-3}	6.1x10^{-5}	3.0x10^{-4}
Perry	Painesville, OH	8.8x10^{-5}	1.6x10^{-4}	No data	No data
Pilgram	Boston, MA	No data	9.8x10^{-4}	2.4x10^{-5}	7.3x10^{-5}
Quad-Cites	Moline, IL	No data	3.6x10^{-3}	No data	1.8x10^{-4}
River Bend	Baton Rouge, LA	2.6x10^{-4}	2.2x10^{-3}	No data	No data
Shoreham	Brookhaven, NY	No data	No data	No data	No data
Susquehanna	Berwick, PA	No data	2.9x10^{-5}	No data	No data
Vermont Yankee	Brattleboro, VT	No data	No data	No data	9.9x10^{-5}
WNP-2	Richland, WA	6.3x10^{-3}	0.019	6.7x10^{-6}	7.2x10^{-6}
Total		**0.12**	**0.58**	**4.5x10^{-4}**	**3.3x10^{-3}**
Pressurized water reactors					
Arkansas One	Russellville, AR	0.054	0.11	No data	3.5x10^{-8}
Beaver Valley	Shippingport, PA	1.2x10^{-3}	2.3x10^{-3}	No data	No data
Big Rock Point	Charlevoix, MI	9.5x10^{-4}	0.012	1.1x10^{-5}	1.5x10^{-4}
Braidwood	Joliet, IL	6.5x10^{-4}	1.7x10^{-3}	No data	No data
Byron	Byron, IL	5.6x10^{-3}	1.5x10^{-3}	No data	No data
Callaway	Fulton, MO	5.3x10^{-4}	7.8x10^{-4}	No data	No data
Calvert Cliffs	Washington, DC	0.14	0.21	3.4x10^{-3}	4.2x10^{-3}

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Catawba	Rock Hill, SC	1.6×10^{-3}	3.9×10^{-3}	No data	No data
Comanche Peak	Glen Rose, TX	8.7×10^{-3}	8.7×10^{-3}	No data	No data
Crystal River	Tampa, FL	2.7×10^{-3}	9.3×10^{-3}	No data	3.1×10^{-6}
Davis-Besse	Toledo, OH	1.4×10^{-3}	6.2×10^{-3}	No data	No data
Diablo Canyon	San Luis Obispo, CA	0.036	0.064	3.3×10^{-5}	2.4×10^{-4}
Donald C. Cook	St. Joseph, MI	1.3×10^{-3}	6.3×10^{-3}	No data	7.3×10^{-7}
Fort Calhoun	Omaha, NE	1.3×10^{-3}	0.012	No data	1.7×10^{-6}
H.B. Robinson	Hartsville, SC	3.6×10^{-4}	4.1×10^{-4}	5.8×10^{-6}	1.8×10^{-5}
Haddam Neck	Middletown, CT	0.013	0.023	8.0×10^{-4}	8.8×10^{-3}
Harris	Raleigh, NC	2.9×10^{-4}	3.9×10^{-4}	No data	No data
Indian Point[b]	Peekskill, NY	3.0×10^{-3}	0.10	No data	7.0×10^{-4}
Joseph M. Farley	Dothan, AL	1.5×10^{-3}	3.6×10^{-3}	No data	No data
Kewaunee	Green Bay, WI	No data	2.6×10^{-6}	No data	2.0×10^{-6}
Maine Yankee	Wicassett, ME	1.4×10^{-3}	8.2×10^{-3}	No data	4.2×10^{-5}
McGuire	Charlotte, NC	1.6×10^{-3}	4.6×10^{-3}	No data	1.3×10^{-6}
North Anna	NW Richmond, VA	6.2×10^{-3}	9.2×10^{-3}	3.4×10^{-6}	8.2×10^{-5}
Oconee	Greenville, SC	2.7×10^{-3}	0.010	No data	3.9×10^{-4}
Palisades	South Haven, MI	2.4×10^{-4}	4.3×10^{-3}	No data	1.1×10^{-5}
Palo Verde	Phoenix, AZ	No data	No data	2.0×10^{-3}	1.7×10^{-3}
Point Beach	Manitowoc, WI	0.019	0.027	6.9×10^{-3}	6.9×10^{-3}
Prairie Island	Minneapolis, MN	2.8×10^{-3}	3.8×10^{-3}	1.5×10^{-5}	1.8×10^{-5}
R.E. Ginna	Rochester, NY	0.042	0.041	No data	5.2×10^{-6}
Rancho Seco[b]	Sacramento, CA	1.9×10^{-5}	3.6×10^{-4}	No data	No data
Salem	Wilmington, DE	0.81	1.0	No data	7.0×10^{-7}
San Onofre[b]	San Clemente, CA	0.49	0.57	2.5×10^{-5}	4.0×10^{-5}
Seabrook	Portsmouth, NH	No data	3.3×10^{-5}	No data	No data
Sequoyah	Daisy, TN	0.086	0.14	No data	No data
South Texas	Bay City, TX	3.4×10^{-3}	5.4×10^{-3}	No data	No data
St. Lucie	Ft. Pierce, FL	0.055	0.083	9.2×10^{-6}	2.1×10^{-5}
Summer	Columbia, SC	1.6×10^{-3}	3.3×10^{-3}	2.2×10^{-5}	2.9×10^{-5}
Surry	Newport News, VA	7.6×10^{-5}	0.011	No data	7.2×10^{-5}
Three Mile Island[b]	Harrisburg, PA	0.026	0.030	1.2×10^{-7}	4.7×10^{-6}
Trojan[b]	Portland, OR	1.0×10^{-3}	4.0×10^{-3}	No data	No data
Turkey Point	Florida City, FL	9.4×10^{-4}	5.5×10^{-3}	No data	9.4×10^{-7}
Vogtle	Augusta, GA	5.6×10^{-3}	7.3×10^{-3}	No data	No data
Waterford	New Orleans, LA	0.013	0.016	No data	No data
Wolf Creek	Burlington, KS	0.022	0.024	No data	No data

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Yankee Rowe[b]	Greenfield, MA	4.3×10^{-6}	6.0×10^{-5}	No data	1.0×10^{-7}
Zion	Waukegan, IL	0.014	0.029	2.4×10^{-4}	2.9×10^{-4}
Total		**1.88**	**2.85**	**0.013**	**0.023**

[a] Post office state abbreviations used.
[b] Facilities that are permanently or indefinitely shut down.

materials from miscellaneous actions such as equipment failure, operator error, or procedure error; these releases are not of such consequence as to be considered an accident.

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

Stable cesium is introduced into the atmosphere by resuspension of soil, accidental release from mining and milling pollucite, and emissions from hazardous waste incinerators or coal burning plants. These emissions are expected to be low since cesium occurs naturally in the earth's crust at low concentrations and only small amounts of pollucite are mined annually. Cesium was detected at concentrations of 10.8 and 6.11 mg/m^3 in the effluent of a coal-burning power plant in the western United States (Ondov et al. 1989) and has been identified in the fly ash from municipal incinerators (Mumma et al. 1990, 1991). Fly ash from five municipal waste incinerators in the United States contained cesium at concentrations of 2,100–12,000 ppm (EPA 1990a). Stable cesium has been identified in air at one of the eight NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003).

Radioactive isotopes of cesium such as ^{137}Cs and ^{134}Cs have been released to the atmosphere from atmospheric nuclear weapons testing, accidents at nuclear reactors, and nuclear-powered satellites burning up in the atmosphere upon re-entry. The total amount of ^{137}Cs released from weapons testing through 1980 was estimated as $2.6x10^7$ Ci ($9.6x10^{17}$ Bq), 76% of which was released in the northern hemisphere and 24% in the southern hemisphere (WHO 1983). On April 26, 1986, a steam buildup caused an explosion and fire at a nuclear power generating plant in Chernobyl, Russia, releasing an estimated $5.4x10^5$ Ci ($2.0x10^{16}$ Bq) of ^{134}Cs and $1.1x10^6$ Ci ($4.0x10^{16}$ Bq) of ^{137}Cs into the atmosphere over Europe (Watson 1987). Long-range atmospheric transport spread the radionuclides throughout the Northern Hemisphere. No airborne activity from Chernobyl has been reported south of the equator (Eisler 1995). By early May 1986, these radionuclides were readily detectable in environmental samples collected in North America (Huda et al. 1988). More recent estimates have put the total activity of ^{137}Cs released from the Chernobyl power plant as $2.3x10^6$ Ci ($8.5x10^{16}$ Bq) and $1.2x10^6$ Ci ($4.4x10^{16}$ Bq) for ^{134}Cs (Buzulukov and Dobrynin 1993). On January 24, 1978, the Soviet nuclear-powered satellite Cosmos 954 re-entered earth's atmosphere over the Canadian Arctic, releasing an estimated 86 Ci of ^{137}Cs (Barrie et al. 1992). In October 1957, an accident at the Windscale nuclear weapons plant at Sellafield in the United Kingdom resulted in the release of 595 Ci of ^{137}Cs (Agency for Toxic Substances and Disease

Registry 1999). Routine activities at nuclear power plants and fuel-reprocessing stations also release ^{137}Cs and ^{134}Cs to the environment on a regular basis. Radiocesium released in airborne effluents from the normal operation of nuclear power plants is considered low in comparison to releases from past atmospheric weapons tests and the major releases following accidents at nuclear power plants. In 1998, it was reported that 1.3×10^{-4} Ci of ^{134}Cs and 5.1×10^{-3} Ci of ^{137}Cs were released to the atmosphere from the Savannah River plutonium processing site in South Carolina (DOE 1998b). In 1993, the Nuclear Regulatory Commission (NRC) estimated that 0.013 Ci of ^{134}Cs and 0.023 Ci of ^{137}Cs were released in airborne effluents from 30 PWR nuclear power plants operating in the United States (USNRC 1993). It was also estimated that 4.6×10^{-4} Ci of ^{134}Cs and 3.3×10^{-3} Ci of ^{137}Cs were released in airborne effluents from 28 BWR nuclear power plants (USNRC 1993). The total airborne and liquid releases of ^{134}Cs and ^{137}Cs from the individual nuclear power plants are summarized in Table 6-1.

Radioactive ^{134}Cs was not identified in air at the 3 NPL hazardous waste sites where it was detected in some environmental media, but ^{137}Cs was identified in air at 5 of the 23 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003).

6.2.2 Water

Cesium can be released to water surfaces during the mining, milling, and production process of pollucite ore. The natural erosion and weathering of rocks will also lead to cesium's introduction into ground and surface water. Stable cesium was identified in groundwater and surface water at four and one of the eight NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2003).

The dumping of high and low level radioactive wastes into the Arctic waters by the former Soviet Union has also led to the release of ^{137}Cs and ^{134}Cs as well as other radioactive nuclides into these waters. In the past, the majority of radioactive cesium released to water surfaces in North America arose from deposition following atmospheric nuclear weapons testing conducted by the United States, primarily during the 1960s (Robbins et al. 1990). Radioactive cesium can be introduced to water from nuclear power plants (accidents and normal operation) and at facilities that produce weapons grade plutonium and uranium. During the period of 1961–1973, it was estimated that about 514 Ci of ^{137}Cs was emitted to the Savannah River watershed due to the activities at the Savannah River plutonium processing plant (Olsen et al. 1989). It was further noted that about 18% of this total (92 Ci) drained directly into the Savannah River (Olsen et al. 1989). In 1998, it was reported that 1.0×10^{-4} Ci of ^{134}Cs and 0.19 Ci of ^{137}Cs were released in liquid effluents from the Savannah River plutonium processing site in South Carolina (DOE 1998b). In 1993, the NRC estimated that 1.88 Ci of ^{134}Cs and 2.85 Ci of ^{137}Cs were released in liquid effluents from 30 PWR nuclear power plants operating in the United States (NRC 1993a). It was also estimated that 0.12 Ci of ^{134}Cs and 0.58 Ci of ^{137}Cs were released in liquid effluents from 28 BWR nuclear power plants (NRC 1993a). The EPA reported that the total on-site liquid discharge of ^{137}Cs from containment ponds at the Nevada Test Site was 0.0017 Ci in 1997 (EPA 1999c). It was estimated that 1,622 Ci of ^{137}Cs and 811 Ci of ^{134}Cs were released to the cooling pond surrounding the Chernobyl nuclear power plant following the accident in 1986 (UNSCEAR 1996).

Radioactive ^{134}Cs was not identified in water at the three NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003). However, ^{137}Cs was identified in groundwater and surface water at 5 and 3 of the 23 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2003).

6.2.3 Soil

Anthropogenic sources of stable ^{133}Cs releases to soils include the mining, milling, and processing of pollucite ore. It is also found in the ash of coal burning power plants and municipal waste incinerators. Stable cesium was detected at concentrations of 0.44–2.01 ppm in the bottom ash of municipal solid waste incinerators operating in the United States (Mumma et al. 1990) and at concentrations of 3–23 ppm from a municipal waste incinerator operating in Barcelona, Spain (Fernandez et al. 1992). Stable cesium was identified in soil at one of the eight NPL hazardous waste sites and in sediment at one of the eight NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003).

Radioactive cesium has been released to soil surfaces by underground nuclear weapons testing, fallout from the accident at the Chernobyl nuclear power plant, and fallout from atmospheric weapons testing. About 1,400 underground tests have been performed worldwide, with a total explosive yield of about 90 megatons (Agency for Toxic Substances and Disease Registry 1999). Small amounts of ^{137}Cs and ^{134}Cs are also released to soil from the normal operation of nuclear power plants and the storage of spent fuel rods. Not including the 30-km exclusionary zone, an area of approximately 2.4×10^4 km^2 near the Chernobyl nuclear power plant was contaminated with ^{137}Cs at a deposition density $>5.4 \times 10^{-5}$ Ci/m^2 following the accident in 1986 (UNSCEAR 1996). Within the exclusionary zone the contamination density may have been 2 orders of magnitude greater in limited areas (UNSCEAR 1996). The mean deposition density of ^{137}Cs and ^{134}Cs in four different soils in Devoke, United Kingdom for May 1986 were reported as 3.7×10^{-7}–5.4×10^{-7} Ci/m^2 and 1.0×10^{-7}–1.8×10^{-7} Ci/m^2, respectively (Hilton et al. 1993). The concentrations of ^{137}Cs in eight sediment cores of the Danube River, Austria were about 540 pCi/kg in April 1985, but increased to approximately 27,000–81,000 pCi/kg in October 1986, following the accident at the Chernobyl nuclear power plant (Rank et al. 1990). The deposition of ^{137}Cs attributed to the accident at the Chernobyl nuclear power plant in sediment at five different sites in Lake Constance, Germany ranged from 2.7×10^{-7} to 2.1×10^{-6} Ci/m^2, while the concentration attributed to fallout from nuclear weapons testing since 1963 ranged from 1.4×10^{-7} to 5.4×10^{-7} Ci/m^2 (Richter et al. 1993). It was estimated that 2,973 Ci of ^{137}Cs and 1,622 Ci of ^{134}Cs were released to the sediments in the cooling pond surrounding the Chernobyl nuclear power plant following the accident in 1986 (UNSCEAR 1996). The deposition density of ^{137}Cs in 123 soil cores collected at the Idaho National Engineering and Environmental Laboratory (INEEL), a site for stored transuranic waste in the United States, ranged from 1.6×10^{-8} to 3.4×10^{-7} Ci/m^2 (DOE 1998a). The deposition density of ^{137}Cs in soils from Idaho, Montana, and Wyoming ranged from 3.0×10^{-9} to 1.1×10^{-7} Ci/m^2, and it was assumed that its origin was fallout from the Nevada Test Site (DOE 1998a). The mean deposition density of ^{137}Cs in the top layer (0–8 cm) of soils near the Chernobyl nuclear power plant in 1988 was 8.6×10^{-5} Ci/m^2 and the mean deposition density of ^{134}Cs was 1.9×10^{-5} Ci/m^2 (Mikhaylovskaya et al. 1993).

It was reported that ^{134}Cs was identified in soil at one site, but was not detected in sediment at the three NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003). It was also reported that ^{137}Cs was identified in soil and sediment at 14 and 5 of the 23 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2003).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

The transport and partitioning of particulate matter in the atmosphere is largely dependent upon the physical properties of the matter such as size and density as well as the meteorological conditions such as temperature, the microphysical structure of the clouds, and rainfall rate. The particle size of ^{137}Cs released to the atmosphere following the Chernobyl nuclear accident was in the range of 0.1–10 μm, (Hirose et al. 1993). Particles <5 μm in diameter usually have low deposition velocities and are transported long distances before being removed from the atmosphere. Atmospheric cesium is deposited on land and water via wet and dry deposition and the deposited cesium may be re-suspended to the atmosphere by disturbances that occur on the ground such as vehicular traffic and construction activity.

The wet deposition velocity of ^{137}Cs at Tsukauba, Japan from May 5 to May 30, 1986 ranged from 0.0026 to 0.110 m/second, and the largest value recorded was during a period of heavy rainfall (Hirose et al. 1993). The mean deposition velocity (wet and dry) of ^{137}Cs measured in Prague, Czechoslovakia was reported as 0.08 m/second from 1989–1992 and the mean flux rate was 1,108 pCi/m^2-year (Rybacek et al. 1994).

Since cesium does not volatilize from water, transport of cesium from water to the atmosphere is not considered likely, except by windblown sea sprays. Most of the cesium released to water will adsorb to suspended solids in the water column and ultimately be deposited in the sediment core. Cesium can also bioconcentrate and has been shown to bioaccumulate in both terrestrial and aquatic food chains. Mean bioconcentration factors (BCF) for ^{137}Cs of 146, 124, and 63 were reported for fish, brown macroalgae, and molluscs, respectively (Fisher et al. 1999). Mean BCF values of 92, 58, 39, and 150 were reported for ^{137}Cs in cod, haddock, plaice, and whiting, respectively (Steele 1990). In a study of aquatic organisms inhabiting the Ottawa River, a 4-fold increase of ^{137}Cs levels was observed with each trophic level (Rowan et al. 1998). The levels of ^{137}Cs in lake trout from Great Slave Lake, Canada were consistently higher than levels found in food sources and a biomagnification factor of 1.9 was calculated for lake trout, relative to their food sources. The biomagnification factor was 3.5 for large mature trout populating the lake (Rowan et al. 1998). It was shown that the bioconcentration and bioaccumulation of ^{137}Cs by aquatic organisms is significantly reduced in waters with a large humic content and high levels of potassium cations (Penttila et al. 1993). Because of the high potassium concentration in oceans, the transfer of ^{137}Cs and ^{134}Cs to fish is much greater in freshwater and the activity of freshwater fish may be 100 times that of ocean fish, given the same cesium concentration in the water (WHO 1983).

In soil surfaces, cesium has low mobility in comparison to other metals and usually does not migrate below a depth of 40 cm. The major portion of cesium is retained in the upper 20 cm of the soil surface (Korobova et al. 1998; Ruse and Peart 2000; Takenaka et al. 1998). Vertical migration patterns of ^{137}Cs in four agricultural soils from southern Chile indicated that approximately 90% of the applied cesium was retained in the top 40 cm of soil, and that in one soil, essentially 100% was bound in the upper 10 cm (Schuller et al. 1997). Migration rates of radiocesium were derived from the depth distribution profiles and were in the range of 0.11 to 0.29 cm/year (Schuller et al. 1997). The vertical migration patterns of ^{90}Sr and ^{137}Cs produced from the atomic bomb exploded in Nagasaki, Japan were studied over a 40-year period (Mahara 1993). Over this period, 95% of the cesium remained in the top 10 cm of the soil surface and no cesium was detected below a depth of 40 cm. In contrast, only 70% of ^{90}Sr was located within a depth of 10 cm and a small percentage was detectable below a depth of 200 cm. The *in situ* vertical migration rate of ^{90}Sr was calculated as 0.42 cm/year and the migration rate of ^{137}Cs was 0.10 cm/year (Mahara 1993). The distribution of ^{137}Cs deposited in eastern France due to the Chernobyl accident was studied recently in four soil types with differing land use. The soils were all high in clay content, organic matter content never exceeded 10% in any of the soils, and none of the soils had been ploughed or tilled since the Chernobyl accident in 1986. In all cases, the majority of ^{137}Cs was detected in the upper 10 cm of soil, with only 1–2% migrating to depths of 40 cm or more (Renaud et al. 2003). Soil adsorption coefficients (K_d) of five radionuclides (^{54}Mn, ^{60}Co, ^{65}Zn, ^{85}Sr, and ^{137}Cs) were measured for 36 agricultural soils collected in Japan. It was determined that ^{137}Cs had the largest median K_d of all five radionuclides, and that a positive correlation was observed between the adsorption coefficient and exchangeable potassium content in the soil (Yasuda et al. 1995). No correlations were observed for other soil properties such as pH, water content, cation exchange capacity, and exchangeable calcium. Other studies have reported that clay and zeolite minerals strongly bind cesium cations and can therefore reduce the bioavailability of cesium and the uptake in plants by irreversibly binding cesium in interlayer positions of the clay particles (Paasikallio 1999). Experiments conducted by growing plants in a peat soil showed that the introduction of zeolites into the soil-plant system decreased the uptake of ^{134}Cs in plants by a factor of 8 (Shenber and Johanson 1992). The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays and zeolites (Hakem et al. 1997). Soils rich in organic matter adsorb cesium, but the cesium adsorbed in the organic fraction is readily exchangeable and highly available for plant uptake (Sanchez et al. 1999). Regions in Venezuela, Brazil, and Russia have been identified where a great deal of rain is encountered, the soil is peaty or podzolic (a type of forest soil characterized by high leachability), and the mobility of cesium is considerably greater than in other soils (LaBrecque and Rosales 1996; WHO 1983).

The plant/soil concentration ratio (activity/kg of plant/activity/kg of soil) of ^{137}Cs for field crops in southern Finland ranged from 0.01 to 0.26. In northern Finland, this ratio ranged from 0.01 to 2.29, with the lowest values occurring in clay and silt soils and the highest values observed in fine sands (Paasikallio et al. 1994). The plant/soil concentration ratios for a series of vegetables and grains decreased in the following order: lettuce, cabbage>carrot, potato>cereals, onion; for fruits, the order was: black-currant>strawberry>apple (Paasikallio et al. 1994). The mean plant/soil concentration ratios of ^{137}Cs for trees at the Hanford Waste Site in the United States were 0.03 (roots), 0.06 (cores), and 0.02 (leaf/twig) (Landeen and Mitchell 1986).

6.3.2 Transformation and Degradation

6.3.2.1 Air

When pure cesium metal is exposed to air, an explosion-like oxidation occurs, forming a mixture of cesium oxides (Cs_2O, Cs_2O_2, and Cs_2O_3). Cesium compounds released to the atmosphere will eventually settle to earth by wet and dry deposition. Radioactive forms of cesium such as ^{137}Cs and ^{134}Cs are continuously transformed to stable isotopes of barium or xenon by the natural process of radioactive decay. The pathways and mechanisms of these reactions have been described in Chapter 4.

6.3.2.2 Water

When pure cesium metal is released to water, a vigorous reaction occurs yielding cesium hydroxide (CsOH), the strongest base known, and hydrogen gas, which may ignite spontaneously. In general, cesium compounds are very water soluble, in which cesium is present in the ionic form as the Cs^+ cation. Under normal environmental conditions, Cs^+ cations are neither degraded nor transformed, but may adsorb to suspended solids and sediment in the water column, forming insoluble complexes.

6.3.2.3 Sediment and Soil

Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility. Cesium cations have low hydration energy and can react with clay minerals, zeolites, or soils with a high percentage of exchangeable potassium, forming insoluble, immobile complexes.

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to cesium depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Radioactive cesium is measured in units of activity, not mass. A great deal of monitoring data for radioactive cesium in environmental samples prior to, and shortly following, the accident at the Chernobyl nuclear power plant on April 26, 1986 has been included in this chapter.

6.4.1 Air

Data reporting the background levels of ^{133}Cs in the atmosphere are limited. Since the amount of cesium mined and milled is small, it is expected that background concentrations in the atmosphere will be low. The concentration of ^{133}Cs measured in the South Pole during 1974–1975 was reported in the range of 0.072–0.14 pg/m^3, with a mean of 0.10 pg/m^3 (Maenhaut et al. 1979). The maximum airborne concentrations of ^{133}Cs at the Tera Nova Bay Station in the Arctic were reported as 20–90 pg/m^3 during the 1989–1990 Italian expedition and 10–60 pg/m^3 for the 1990–1991 expedition (Chiavarini et al. 1994). The average concentration of ^{133}Cs in precipitation collected in Lennox, Massachusetts during the summer of 1984 was reported as 0.0075 μg/L, with a range from 0.0050 to 0.024 μg/L (Dasch and Wolff 1989).

Radioactive ^{134}Cs and ^{137}Cs have been detected at various concentrations (activities) in the atmosphere following the accident at the Chernobyl nuclear power plant on April 26, 1986. The greatest concentrations were observed in locations throughout Russia and Europe, but detectable levels were measured globally, including in North America. The air concentrations of ^{134}Cs above the destroyed reactor were 1,756.8 pCi/m^3 on May 8 and 54.0 pCi/m^3 on May 18 (Buzulukov and Dobrynin 1993). The concentrations of ^{137}Cs in the air above the destroyed reactor were 8,918.9 pCi/m^3 on May 8 and 135.1 pCi/m^3 on May 18 (Buzulukov and Dobrynin 1993). Atmospheric concentrations of ^{134}Cs in Belgrade, Yugoslavia were 5.4x10^{-4} (May 1, 1986), 160.5 (May 2, 1986), 145.0 (May 3, 1986), and 25.4 (May 4, 1986) pCi/m^3, while atmospheric concentrations of ^{137}Cs were 0.001 (May 1, 1986), 324 (May 2, 1986), 276 (May 3, 1986), and 49 (May 4, 1986) pCi/m^3 (Ajdacic and Martic 1990). Cesium concentrations decreased significantly over time as advection, wet deposition, and dry deposition lowered the atmospheric concentrations. The average monthly atmospheric concentrations of ^{137}Cs in Belgrade that were measured during 1991–1996 ranged from 1.3x10^{-4} to 2.0x10^{-3} pCi/m^3, with a pronounced maximum during the winter months (Todorovic et al. 1999). Monitoring data from Prague, Czechoslovakia indicated that there was a gradual decrease in the atmospheric concentration of ^{137}Cs from May 1986 to February 1992. Maximum concentrations were observed immediately following the accident at the Chernobyl nuclear power plant (approximately 0.05 pCi/m^3) and gradually decreased over a 6-year period (Rybacek et al. 1994). The atmospheric concentration of ^{134}Cs decreased more rapidly, presumably due to the shorter half-life of ^{134}Cs. The concentration of ^{137}Cs in the atmosphere of Thessaloniki, Greece ranged from 8.1x10^{-4} to 0.044 pCi/m^3 from July 1987 to December 1988, and the concentration of ^{137}Cs in rainfall ranged from 0.27 to 34 pCi/L from November 1986 to February 1989 (Papastefanou et al. 1989). During a heavy rainfall event occurring on May 6, 1986, ^{137}Cs was detected at a concentration of 46,000 pCi/L (Papastefanou et al. 1989). The concentration of ^{137}Cs in Tsukuba, Japan during May 1986 ranged from about 0.054 to 1.6 pCi/m^3 (Hirose et al. 1993). The average atmospheric concentrations of ^{134}Cs and ^{137}Cs in eastern Canada were reported as 0.024 and 0.046 pCi/m^3, respectively, during May 10–24, 1986 (Huda et al. 1988). The maximum atmospheric concentration of ^{137}Cs measured in New York City in May 1986 was reported as 0.26 pCi/m^3 (Feely et al. 1988). The average concentrations of ^{137}Cs and ^{134}Cs in Barrow, Alaska were reported as 0.027 and 0.019 pCi/m^3, respectively, for the month of May 1986 (DOE 1986).

6.4.2 Water

The background concentration of ^{133}Cs in fresh water lakes and rivers is ordinarily in the range of 0.01 to 1.2 µg/L, and the concentration in seawater is about 0.5 µg/L (WHO 1983). Stable cesium was detected in streams that feed into the Tamagawa River, Japan at concentrations of 9x10^{-4} to 0.093 µg/L (Tanizaki et al. 1992). Studies from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) have shown some areas contain high levels of ^{133}Cs in natural waters and sediment (Fernandez-Turiel et al. 1995). The cesium concentration in fresh water systems of this region ranged from 0.58 to 3.69 µg/L (Fernandez-Turiel et al. 1995). The concentrations of 19 trace metals were studied in drinking water and groundwater supplies in southern Nigeria. Stable cesium was detected in groundwater and drinking water at mean concentrations of 0.61 µg/L (range 0.09–3.72 µg/L) and 0.35 µg/L (range 0.05–4.32 µg/L), respectively (Asubiojo et al. 1997). The mean concentration of cesium in drinking water was lower than the mean concentration of any of the other trace elements analyzed.

High and low level radioactive wastes were dumped by the former Soviet Union into remote Arctic waters, leading to the release of radioactive cesium into the Kara and Barents Seas. The level of ^{137}Cs in surface water of the Barents Sea and Kara Sea was 0.14 and 0.16 pCi/L, respectively, and ^{137}Cs was also detected in deep water of the Barents Sea at a concentration of 0.15 pCi/L (Fisher et al. 1999). The concentration of ^{137}Cs in the Black Sea ranged from 2.7 to 8.1 pCi/L for the period of 1991–1996, with the exception of the spring of 1992, when concentrations as high as 43 pCi/L were observed (Strezov et al. 1999). Preliminary measurements taken in the vicinity of the nuclear submarine, the Kursk, which accidentally sank in the Barents Sea in August 2000, indicate that that no leakage of radionuclides from the reactor has occurred and only background levels of radioactive cesium (and other radionuclides) have been observed in water and sediment near the submarine (Amundsen et al. 2002). From 1988 to 1991, the mean concentrations of ^{137}Cs and ^{134}Cs along the Spanish coast of the Mediterranean Sea were 0.13 and 0.0072 pCi/L, respectively (Molero et al. 1999). Due to its shorter half-life, ^{134}Cs was detected in all

14 samples collected in 1988 and 1989, but only in 3 samples collected in 1990 and 1991, suggesting that the ^{134}Cs levels observed in surface Mediterranean waters during this period were due mainly to Chernobyl-related deposition. The ^{137}Cs concentration incorporated into the Mediterranean Sea near the Spanish coast from the post-Chernobyl fallout was about 0.032 pCi/L, which was approximately a 33% increase over previous levels (Molero et al. 1999). Maximum ^{137}Cs and ^{134}Cs levels in the immediate vicinity of nuclear power plants located on the southern Catalan shore of the Mediterranean were 0.57 and 0.059 pCi/L (Molero et al. 1999). Concentrations of ^{137}Cs in lakes and streams in Devoke, United Kingdom decreased exponentially from a maximum concentration of about 8.1 pCi/L on May 6, 1986 to about 0.027 pCi/L 1,200 days later (Hilton et al. 1993). The mean concentration of ^{137}Cs in six lakes located in central Finland ranged from 111 pCi/L in 1987 to 8.1 pCi/L in 1989 (Penttila et al. 1993).

The concentration of ^{137}Cs and ^{134}Cs in groundwater at 18 U.S. Department of Energy (DOE) facilities was reported in the range of 2.7×10^{-3} to 1.83×10^{3} pCi/L (DOE 1992). The concentration of ^{137}Cs measured in groundwater wells at Carlsbad, New Mexico (the site of Project GNOME) ranged from 99 to 6.8×10^{5} pCi/L in 1997 (EPA 1999c). The concentration of ^{137}Cs in groundwater at the Chernobyl nuclear power plant was in the range of 40.5–1,100 pCi/L in 1988 and 29.7–129.7 pCi/L in 1989 (Prister et al. 1990). The mean concentration of ^{137}Cs in drinking water in Poland in 1974 was reported as 0.2 pCi/L (Glowiak et al. 1977b). These concentrations in water may be compared to the federal radiation safety standards. For continuous ingestion over a lifetime, the maximum concentrations of ^{134}Cs and ^{137}Cs in drinking water are limited to 900 and 1,000 pCi/L respectively; these limits assume no other intake of radioactivity. If other radioisotopes are ingested, then the intake limits for each must be adjusted proportionately (USNRC 1999a).

6.4.3 Sediment and Soil

Stable cesium occurs naturally in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993). Others have estimated cesium concentration of granites as high as 5 ppm (WHO 1983). Stable cesium was detected in dust samples from roadside and pedestrian traffic in Nagpur, India at concentrations of 1.53–3.63 μg/g, with the largest value obtained in the vicinity of a metals factory (Chutke et al. 1995). ^{133}Cs was detected at concentrations ranging from 0.9 to 2.2 μg/g in alluvial sediments in the Sava River, Croatia (Vertacnik et al. 1997). The concentration of ^{133}Cs in river sediment from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) was 2.28–6.20 μg/g (Fernandez-Turiel et al. 1995).

The concentration of ^{137}Cs in soils of Thessaloniki, Greece ranged from 1,440 to 35,324 pCi/kg (average 8,154 pCi/kg) and the concentration of ^{134}Cs ranged from about 270 to 5,676 pCi/kg during the period of August 1986 to February 1989, with most of the fallout attributed to the accident at the Chernobyl nuclear power plant (Papastefanou et al. 1989). The concentration of ^{137}Cs in 10 uncultivated fields from southern England ranged from 0 to 946 pCi/kg, with the highest levels contained in the top 10 cm of the soil surface (Owens et al. 1996). The concentration of ^{137}Cs in five cultivated fields ranged from 0 to 540 pCi/kg, and the concentrations were well distributed from the surface to the plough layer (Owens et al. 1996). The concentration of ^{137}Cs in three soils in Hong Kong receiving a large amount of rainfall ranged from 32 to 201 pCi/kg (Ruse and Peart 2000). The concentration of ^{137}Cs in sediment from the Ribble Estuary, England near the British Nuclear Fuels Laboratory ranged from 270 to 1,351 pCi/kg (Brown et al. 1999). The average concentration of ^{137}Cs in sediment from Lake Ontario was reported as 8,108 pCi/kg, and suspended solids from the Niagara River contained 324 pCi/kg, measured during the 1983–1986 sampling period (Platford and Joshi 1989). The average concentrations of ^{137}Cs in uncultivated soils in northern Poland were reported as 616–4,170 pCi/kg from 1988 to 1991 (Pietrzak-Flis et al. 1994). The mean concentration of ^{137}Cs in surface soil samples from the Los Alamos nuclear laboratory test site during the period of 1974–1996 was reported as 611 pCi/kg (Fresquez et al. 1998). Concentrations of ^{137}Cs around the perimeter of the site and background concentrations off the site were reported as 589 and 419 pCi/kg , respectively. The concentration of ^{137}Cs and ^{134}Cs in soils and sediments at 18 U.S. DOE facilities was reported to range from 20 to 4.69×10^7 pCi/kg (DOE 1992). The concentration of ^{137}Cs in sediment from the Savannah River ranged from 5 to 100 pCi/kg in 1986, while the concentration in suspended solids and particulate matter ranged from 240 to 4.324×10^6 pCi/kg (Olsen et al. 1989). The mean concentration of ^{137}Cs in soil at the Hanford Site in the United States was 4,540 pCi/kg (Landeen and Mitchell 1986). The mean concentration of ^{137}Cs in soils taken from two high-elevation sites in northern Colorado ranged from 4,054 to 7,027 pCi/kg (Ulsh et al. 2000).

The concentration of ^{137}Cs in sediment near the nuclear submarine, the Kursk, which accidentally sank in the Barents Sea in August 2000, ranged from about 18.9 to 40.5 pCi/kg (Amundesen et al. 2002). These levels are considered typical background levels in the Barents Sea and are attributed to releases from the Sellafield reprocessing facility in the United Kingdom, Chernobyl related fallout, and deposition from past nuclear weapons tests.

6.4.4 Other Environmental Media

Information on the concentrations of ^{137}Cs, ^{134}Cs, and ^{133}Cs in various animals is shown in Table 6-2. Concentrations are dependent on the location, date, and level of exposure. For example, the concentrations of ^{137}Cs in bullhead catfish inhabiting an abandoned nuclear reactor reservoir at the Savannah River site in South Carolina were as high as 1.54x10^{5} pCi/kg (McCreedy et al. 1997), but concentrations in various freshwater species of fish in the Ottawa River were in the range of 54 to 351 pCi/kg (Rowan et al. 1998). After the accident at the Chernobyl nuclear power plant, the average concentrations of ^{137}Cs in perch, pike, and roach obtained from 52 freshwater lakes in Finland were 55,811, 66,297, and 15,270 pCi/kg, respectively, in 1988. By contrast, the mean concentrations in 1992 had fallen to 14,324, 18,567, and 5,892 pCi/kg, respectively (Sarkka et al. 1996). The concentration of total ^{137}Cs and ^{134}Cs measured in body tissue from sheep in Ireland from 1989 to 1991 ranged from about 2,700 to 10,811 pCi/kg, with highest levels observed during November of 1989 (McGee et al. 1993).

The concentrations of ^{133}Cs in plants from Britain were studied over a 1-year period. Concentrations of 50–300 ng/g were measured, with the highest levels observed during the summer and fall months. The median concentration of ^{133}Cs in poplar leaves collected in Bulgaria was reported as 75 ng/g, while concentrations in land plants ranged from 30 to 440 ng/g (Djingova et al. 1995). In contrast to the radioactive isotopes of cesium, concentrations of stable ^{133}Cs depend primarily on the root uptake from soil and not on atmospheric deposition. Lichens and mosses have been shown to trap and retain ^{137}Cs and ^{134}Cs more than vascular plants due to their relatively large surface area. Lichens and mosses from northern Greece contained ^{137}Cs levels of 6.6x10^{4}–5.1x10^{5} pCi/kg during the period of 1989–1991 (Papastefanou et al. 1992) and moss samples from Finland collected in 1988–1989 contained 4.3x10^{4}–9.7x10^{5} pCi/kg (Penttila et al. 1993). The mean concentration of ^{137}Cs in three species of lichens collected in August 1986 from Megalopolis, Greece were 2.6x10^{4}–3.3x10^{4} pCi/kg, while the mean concentrations for the same three species of lichens collected in October 1996 had fallen to 3,324–7,892 pCi/kg (Riga-Karandinos and Karandinos 1998). Mushrooms, lichens, and mosses obtained near Manitoba, Canada in August 1986 contained ^{137}Cs at mean concentrations of 6.4x10^{5}, 8.6x10^{4}, and 8.4x10^{4} pCi/kg, respectively (Mihok et al. 1989). Since caribou and reindeer consume large amounts of this vegetation during the winter months, elevated levels of ^{137}Cs and ^{134}Cs have been detected in these animals.

Stable cesium has been detected infrequently in food products at low concentrations. The average concentration of stable ^{133}Cs in 110 onion samples collected in Denmark was 0.21 μg/kg, with a range of

Table 6-2. Concentration of ^{133}Cs, ^{134}Cs, and ^{137}Cs in Animals

Species	Location and date	^{133}Cs (µg/g)	^{134}Cs (Bq/kg)	^{137}Cs (Bq/kg)	Source
Royal albatross (N=3)	Indian Ocean (1994)	0.009–0.025 (0.014 mean); liver	No data	No data	Kim et al. 1998
Black-footed albatross (N=18)	North Pacific (1985)	0.007–0.049 (0.022 mean); liver	No data	No data	Kim et al. 1998
Black-browed albatross (N=9)	Indian Ocean (1994)	0.013–0.042 (0.022 mean); liver	No data	No data	Kim et al. 1998
White-capped albatross (N=3)	Indian Ocean (1994)	0.011–0.039 (0.029 mean); liver	No data	No data	Kim et al. 1998
Yellow-nosed albatross (N=4)	Indian Ocean (1994)	0.0029–0.079 (0.022 mean); liver	No data	No data	Kim et al. 1998
Grey-headed albatross (N=10)	Indian Ocean (1994)	0.011–0.031 (0.016 mean); liver	No data	No data	Kim et al. 1998
Northern giant petrel (N=6)	Indian Ocean (1994)	0.005–0.034 (0.015 mean); liver	No data	No data	Kim et al. 1998
Northern fulmar (N=17)	North Pacific (1985)	0.008–0.036 (0.016 mean); liver	No data	No data	Kim et al. 1998
Bluefin tuna (N=14)	Newfoundland (1990)	0.08–0.24 (0.13 mean); muscle	No data	No data	Hellou et al. 1992a
Cod (N=12)	Newfoundland (1990)	0.16–0.24 (0.19 mean); muscle	No data	No data	Hellou et al. 1992b
Cod (N=10)	Newfoundland (1990)	0.14–0.36 (0.23 mean); muscle	No data	No data	Hellou et al. 1992b
Pilot whale (N=9)	North Atlantic (1987–1996)	0–0.010 (0.006 mean); liver	No data	No data	Becker et al. 1997
White-sided dolphin (N=4)	North Atlantic (1987–1996)	0.027–0.042 (0.032 mean); liver	No data	No data	Becker et al. 1997
Beluga whale (N=15)	Arctic (1987–1996)	0.021–0.046 (0.031 mean); liver	No data	No data	Becker et al. 1997
Ringed seal (N=13)	Arctic (1987–1996)	0.0045–0.048 (0.020 mean); liver	No data	No data	Becker et al. 1997
Woodcock (N=24)	Ireland (1986)	No data	3.9–206.4; muscle	6.2–565.5; muscle	Pearce 1995
Duck (N=5)	Ireland (1986)	No data	2.2–14.3; muscle	6.4–18.0; muscle	Pearce 1995
Snipe (N=5)	Ireland (1986–1987)	No data	1.0–5.4; muscle	3.6–16.9; muscle	Pearce 1995
Reindeer (N=8)	Northern Sweden (1986–1987)	No data	No data	900 (mean); muscle	Ahman and Ahman 1994
Deer (N=11)	Los Alamos (1991–1998)	No data	No data	2,516 (mean); muscle	Fresquez et al. 1999a

Table 6-2. Concentration of ^{133}Cs, ^{134}Cs, and ^{137}Cs in Animals

Species	Location and date	^{133}Cs (μg/g)	^{134}Cs (Bq/kg)	^{137}Cs (Bq/kg)	Source
Deer (N=11)	Los Alamos (1991–1998)	No data	No data	888 (mean); bone	Fresquez et al. 1999a
Caribou (N=18)	Saskatchewan (1995)	No data	No data	58 (mean); bone	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	228 (mean); liver	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	367 (mean); muscle	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	553 (mean); kidney	Thomas and Gates 1999
Caribou (N=36)	Alaska (1987)	No data	No data	26–232; neck	Allaye-Chan et al. 1990
Caribou (N=36)	Alaska (1987)	No data	No data	28.4–201.1; shoulder	Allaye-Chan et al. 1990
Caribou (N=36)	Alaska (1987)	No data	No data	30.2–166.5; back	Allaye-Chan et al. 1990

not detected to 0.98 μg/kg (Bibak et al. 1998). By comparison, other elements such as calcium and potassium had mean concentrations of 2x10^5 and 1.6x10^6 μg/kg, respectively. The concentration range of ^{133}Cs in wheat flour samples collected in Pakistan was 6.7–11.2 ppb, but ^{133}Cs was not detected from wheat flour samples from America (Ahman et al. 1994).

Levels of ^{137}Cs were below detection limits for all foods analyzed for in the U.S. Food and Drug Administration (FDA) Total Diet Study in 1991–1996 with the exception of honey (Capar and Cunningham 2000). The concentration of ^{137}Cs in honey from the 1995 Market Basket Survey was 6.7 Bq/kg (181.1 pCi/kg), which is almost 200 times lower than the regulatory level of ^{137}Cs in foods. The average concentrations of total ^{137}Cs and ^{134}Cs in milk powder, infant milk powder, infant cereal, meat, lentil, wheat, and macaroni samples from Saudi Arabia were 514, 351, 486, 162, 270, 676, and 351 pCi/kg, respectively (Abdul-Majid et al. 1992). For the month of June 1986, the average concentrations of total ^{137}Cs and ^{134}Cs in milk, green vegetables, fruit, lamb, and beef were reported as 3,243, 2,703, 2,703, 8,108, and 1,622 pCi/kg in high deposition areas of the United Kingdom (Cumbria, north Wales, Scotland, northern Ireland, and the Isle of Man) (Mondon and Walters 1990). The concentration of total ^{137}Cs and ^{134}Cs was <676 pCi/kg in each of these food sources in areas of low deposition during this time frame. The maximum concentration of ^{137}Cs in pasteurized milk from 65 cities in the United States was 14 pCi/L in May 1989 (EPA 1989). The concentration of ^{137}Cs in fresh milk from Chester, New York and pasteurized milk samples from New York City in May 1986 ranged from about 5.4 to 18.9 pCi/L (Feely et al. 1988). Using radiological surveys from 1978 and 1985–1986, the concentration of ^{137}Cs in 44 adult food groups from the Rongelap Island and Rongelap Atoll was in the range of 0.52–13,000 pCi/kg (Robinson and Phillips 1989).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

General population exposure to cesium occurs by ingestion of food and drinking water, by inhalation of ambient air, and dermal contact with cesium in soil. Since stable ^{133}Cs is found in low concentrations in the environment, the exposure to the general population is also low. The National Health And Nutritional Examination Survey (NHANES), conducted by the Centers for Disease Control and Prevention (CDC), reported that the geometric mean concentration of ^{133}Cs in the urine of 2,464 U.S. residents in various population groups was as follows: children aged 6–11 years, 4.87 μg/L; adolescents aged 12–19 years, 4.54 μg/L; adults aged 20 or older, 4.26 μg/L (CDC 2003). Based upon gender, males had a higher geometric mean concentration (4.83 μg/L), as compared to females (3.94 μg/L). Occupational exposure to ^{133}Cs occurs primarily through inhalation and dermal contact at workplaces where pollucite is mined or cesium compounds are manufactured or used. The National Occupational Exposure Survey (NOES) conducted by the National Institute for Occupational Safety and Health (NIOSH) from 1981 to 1983 has estimated that 16,461 workers (4,276 of these were female) were potentially exposed to cesium and cesium compounds in the United States (NOES 1989). The NOES database does not contain information on the frequency, level, or duration of the exposure of workers to any of the chemicals listed therein. They are surveys that only provide estimates of workers potentially exposed to the chemicals. The median concentration of ^{133}Cs in the lungs of metal workers in northern Sweden was 0.016 μg/g and was lower than the median concentration of 0.021 μg/g for a control group that was not occupationally exposed (Hewitt 1988). The range of ^{133}Cs concentrations in lung tissue of coal miners from the United Kingdom was 0.07–0.91 μg/g (Hewitt 1988).

Exposure to radioactive cesium is of greater concern from a health perspective than exposure to stable cesium. The primary source of radioactive cesium in the environment is due to fallout from past atmospheric nuclear weapons tests and the Chernobyl accident. Additional contributions from the normal operation of nuclear power plants and other nuclear facilities are small by comparison. Current exposure of the general U.S. population to ^{134}Cs and ^{137}Cs is expected to be low since atmospheric testing of nuclear weapons was discontinued in 1963 and Chernobyl-related fallout was low in the United States. As discussed in Appendix A, the average annual effective dose of ionizing radiation (including ^{134}Cs and ^{137}Cs) to the U.S. population from anthropogenic sources is negligible in comparison to natural sources, especially radon and its decay products.

The average daily intake (AVDI) of ^{137}Cs and ^{134}Cs was estimated for adult males residing in the Ukraine in 1994, based upon total diet samples. The mean intake of ^{137}Cs was estimated as 109 pCi/day and the mean intake of ^{134}Cs was estimated as 8.1 pCi/day (Shiraishi et al. 1997). Based on dietary patterns and the concentration of radiocesium in food sources, the total dietary intakes of ^{134}Cs and ^{137}Cs for typical adults residing in Croatia for the month of May 1986 were estimated as 2.8×10^4 and 5.9×10^4 pCi, respectively (Lokobauer et al. 1988). The mean monthly levels of ^{137}Cs in human muscle tissue in Graz, Austria were reported as 1,519 (July 1986 to June 1987), 1,049 (July 1987 to June 1988), 340.5 (July 1988 to June 1989), and 202.7 pCi/kg (July 1989 to June 1990), with a maximum value of 9,584 pCi/kg observed in an individual during September 1986 (Rabitsch et al. 1991). The monthly averages for ^{134}Cs were about half of those reported for ^{137}Cs. By comparison, the maximum concentration of ^{137}Cs in muscle tissue from Harwell, England in 1959 was reported as 224 pCi/kg, the mean concentration in muscle tissue from Massachusetts during January 1961 to June 1962 was 100 pCi/kg, and the mean concentration in human muscle tissue obtained from Japan during April to December 1963 was reported as 119 pCi/kg (Rabitsch et al. 1991). The mean concentrations of ^{137}Cs in the urinary excretion of Italians in northern Italy were 7.3 and 6.2 pCi/day in 1995 and 1996, respectively (Ropolo and Cesana 1997). These values were about two orders of magnitude less than values reported for measurements taken in 1987. The mean concentration of ^{137}Cs in brain, heart, liver, gonads, muscle, bone, and teeth were 0.440, 1.860, 0.490, 2.440, 0.017, 0.106, and 0.23 pCi/g, respectively, for adult cadavers over 34 years of age in Poland during 1975 (Glowiak et al. 1977a). The mean body burdens of ^{137}Cs for adults in Helsinki, Viitasaari, and Ammans, Finland from 1987 to 1994 are given in Table 6-3 (Rahola and Suomela 1998).

Persons employed at nuclear power facilities and waste disposal sites are potentially exposed to higher levels of ^{137}Cs and ^{134}Cs than the is general population. The NOES conducted by NIOSH from 1981 to 1983 estimated that 13,148 workers (1,294 of these were female) were potentially exposed to ^{134}Cs and ^{137}Cs in the United States (NOES 1989).

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 Section 3.7, 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 1993a).

As for adults in the general population, exposures of children to cesium occur from normal ingestion of food and drinking water, inhaling air, and dermal contact with cesium in soil. No information on cesium levels in amniotic fluid, meconium, cord blood, or neonatal blood was available.

Radioactive cesium was detected in several pasteurized milk and breast milk samples worldwide, and since children tend to consume large amounts of milk, this represents an important source of childhood

Table 6-3. The Mean Body Burdens of ^{137}Cs for Adults in Finland from 1987 to 1994[a]

Location	Date	Concentration (pCi/kg)
Helsinki, Finland	1987	649
	1988	568
	1989	405
	1990	297
	1991	246
	1992	214
	1993	195
	1994	181
Viitasaari, Finland	1987	3,514
	1988	1,946
	1989	1,595
	1990	1,081
	1991	838
	1992	676
	1993	649
	1994	514
Ammans, Finland	1987	2,892
	1988	3,108
	1989	2,243
	1990	2,486
	1991	1,568
	1992	1,405
	1993	865
	1994	811

[a] Rahola and Suomela 1998

exposure. The maximum concentration of ^{137}Cs in pasteurized milk from 65 cities in the United States was 14 pCi/L in May 1989 (EPA 1989). Concentrations of ^{137}Cs in human milk samples from several U.S. cities from 1956 to 1961 were <20 pCi/L (Eaman 1986). The overall ^{137}Cs concentrations in milk taken 7 days postpartum from 37 mothers in two Italian hospitals were 5.8–115 pCi/L (Eaman 1986). In a study of females from northern Sweden, ^{137}Cs was detected in breast milk from 8 out of 12 mothers at concentrations of 7.3–178.4 pCi/kg (Johansson et al. 1998). The infants of these mothers who were breast-fed had whole-body levels of ^{137}Cs in the range of 45.9–675.7 pCi/kg (Johansson et al. 1998). Based on dietary patterns and the concentration of radiocesium in food sources, the total dietary intakes of ^{134}Cs and ^{137}Cs for children (10 years old) residing in Croatia for May–June 1986 were estimated as 43,000 and 190,000 pCi, respectively (Lokobauer et al. 1988). For infants (1 year of age), it was estimated that the total intakes of ^{134}Cs and ^{137}Cs were 46,000 and 170,000 pCi, respectively (Lokobauer et al. 1988). The total intakes of ^{134}Cs and ^{137}Cs for adults during this same time period were estimated as 40,000 and 84,000 pCi, respectively (Lokobauer et al. 1988). The higher intakes of ^{134}Cs and ^{137}Cs for infants and children were linked to a greater consumption of contaminated milk.

The tendency of young children to ingest soil, either intentionally through pica or unintentionally through hand-to-mouth activity, is well documented. These behavioral traits can result in ingestion of cesium present in soil and dust. Soil ingestion may be a potentially important exposure pathway in areas that have historically had a great deal of ^{134}Cs and ^{137}Cs deposited onto soil surfaces from the accident at the Chernobyl nuclear power plant or fallout from weapons testing. Playing in contaminated soil could also lead to dermal and external exposure. Ingested cesium is adsorbed strongly to soils and may not be in bioavailable form. In a study in which 102 healthy volunteers were fed ^{134}Cs-contaminated soil pellets, only about 1% of the original amount was absorbed and on average, about 60% of the intake was excreted within 48 hours (LeRoy et al. 1966).

It is unlikely that children whose parents are employed at nuclear power generating plants and facilities that store or handle radioactive waste are exposed to ^{134}Cs and ^{137}Cs from parents' clothing or items removed from the workplace because workers pass through exit monitors at nuclear power plants, and the extensive use of outerware that remains in the plant prevents these types of incidents. It is also unlikely that children are exposed to ^{133}Cs from parents' clothing or items that have been removed from the workplace if the parents are employed in the mining, milling, or processing of pollucite ore. Other home exposures are unlikely since household items or products used in crafts, hobbies, or cottage industries do not contain significant amounts of cesium.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Few populations are likely to be exposed to high levels of ^{133}Cs. Persons residing in the vicinity of pollucite mines and workers employed in the mining, milling, and production of cesium may be exposed to higher levels than the general population.

Human exposure to ^{134}Cs and ^{137}Cs can be external, due to exposure from a radioactive cloud and contaminated environmental media after deposition, or internal, via inhalation and ingestion of contaminated food or drinking water. In both cases, populations residing near the source are exposed to potentially higher doses than populations far removed from the source. Humans residing near areas where nuclear weapon testing was previously conducted may have been exposed to higher doses of radiation from ^{134}Cs and ^{137}Cs, both internally and externally, than the general population. Populations residing in southern Utah and Nevada were exposed to radioactive cesium (and many other radionuclides) due to testing conducted at the Nevada Test Site (NTS). A total of 100 surface or near-surface tests with a total explosive yield of about 1 megaton were performed at the Nevada test site between 1951 and 1962. The dust from these tests also drifted over the continental United States, producing varying degrees of exposure for remote populations depending upon the meteorological conditions. For example, the greatest non-local fallout from one of the tests was received in New York State in 1953, some 2,000 miles away from the source, due to wet deposition (Agency for Toxic Substances Disease Registry 1999). About 500 underground tests were also performed at the NTS, but underground testing rarely leads to significant off-site contamination (Agency for Toxic Substances Disease Registry 1999). The EPA Office of Radiation and Indoor Air conducts off-site environmental monitoring around former U.S. nuclear test areas. The 1997 report concluded that the current exposure to populations around the NTS from radionuclides, including ^{137}Cs and ^{134}Cs, was negligible in comparison to background levels (EPA 1999c).

Populations residing in the vicinity of nuclear power plants may also be exposed to higher levels of ^{134}Cs and ^{137}Cs than the general population due to airborne and liquid effluents from these plants. Persons employed in these facilities are also likely to be exposed to greater levels than the general population, particularly those employees who must handle radioactive waste material. However, despite the potential for exposure, increased body burdens of radioactive cesium have not been observed among the population of workers in nuclear facilities or among members of the off-site general public.

Human populations that received a large amount of fallout from the Chernobyl nuclear accident were potentially exposed to high levels of radiocesium. These populations were primarily located in the Ukraine and northern Europe that received a great deal of rainfall in the weeks following the Chernobyl accident. Not including the 30-km exclusionary zone, an area of approximately 2.4×10^4 km^2 near the plant was contaminated with ^{137}Cs at a deposition density >5.4×10^{-5} Ci/m^2 (UNSCEAR 1996). Within the exclusionary zone, the contamination density may have been a factor of about 100 greater in limited areas (UNSCEAR 1996). The Bryansk-Belarus region about 200 km northeast of the reactor had deposition densities as high as 1.3×10^{-4} Ci/m^2, and the Kaluga-Tula-Orel location approximately 500 km northeast of the reactor had deposition densities of about 1.6×10^{-5} Ci/m^2 (Agency for Toxic Substances Disease Registry 1999). While cesium is not considerably taken up by the roots of vascular plants, the deposition of radioactive debris on flora with large surface areas such as lichens or moss is significant (see Section 6.4.4). As a result, animals that feed on this vegetation such as caribou or reindeer may ingest large quantities of radiocesium. Nordic or Eskimo populations that use these animals as an important source for food are exposed to larger quantities of ^{137}Cs and ^{134}Cs than the general population (Allaye-Chan et al. 1990; WHO 1983). The average concentration of ^{137}Cs in the skeletal muscle of a herd of caribou from Alaska was in the range of 76–133 Bq/kg in 1987 (Allaye-Chan et al. 1990). According to the National Council on Radiation Protection and Measurements, the maximum nonoccupational radiocesium intake is 300,666 Bq/year (8.1×10^6 pCi/year) for adults (NCRP 1977). At the maximum average skeletal muscle concentration (133 Bq/kg), an annual consumption of over 2,260 kg of caribou meat would be required to reach this limit (assuming no other intake sources). Using the mean ^{137}Cs level of 900 Bq/kg in the muscle of reindeer obtained from northern Sweden in 1986–1987 (Ahman and Ahman 1994), over 330 kg of contaminated meat would have to be consumed to reach the maximum intake level. Populations residing in the Marshall Islands were exposed to higher levels of ^{137}Cs than the general population due to nuclear weapons testing conducted by the United States from 1946 to 1958. Using radiological surveys from 1978 and 1985–1986, the AVDI of ^{137}Cs due to food ingestion was estimated for seven age groups residing in the Rongelap Atoll (Robinson and Phillips 1989). The intakes in pCi/day were as follows: 0–3 months of age, 424; 4–8 months of age, 556; 9 months to 1.4 years of age 773; 1.5–3 years of age, 517; 4–11 years of age, 594; 12–17 years of age, 761; over 18 years of age, 1,085 (Robinson and Phillips 1989). This corresponds to an annual intake of 3.96×10^5 pCi/year for adults over 18 years of age.

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 cesium 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 cesium.

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 and Chemical Properties. As shown in Table 4-2, the relevant physical and chemical properties of cesium and its compounds are known (Burt 1993; Lide 1998). In general, cesium compounds are very water soluble and primarily exist in ionic form in aqueous environments. Cesium adsorbs strongly to soils and is not very mobile (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). The radioactive decay modes of the two most important cesium isotopes, ^{134}Cs and ^{137}Cs, are also well known (ICRP 1983), and no additional physical or chemical data are required to predict the environmental fate of cesium.

Production, Import/Export, Use, Release, and Disposal. Knowledge of a chemical's production volume is important because it may indicate possible environmental contamination and human exposure. If a chemical's production volume is high, there is an increased probability of general population exposure via consumer products and environmental sources such as air, drinking water, and food. Data concerning production volumes, import, and use of commercially significant cesium compounds are not available. No information was found for cesium in the TRI. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. This database will be updated yearly and should provide a list of industrial production facilities and emissions. The United States is 100% import reliant for stable ^{133}Cs. No data are available on production volume, consumption, or import/export volumes of cesium. Although there is no information regarding the countries shipping cesium or cesium compounds to the United States, it is believed that Canada is the major source of cesium (USGS 1999). Data regarding the U.S. production (if any) of cesium and its compounds as well as import/export statistics would be useful. The amount of ^{134}Cs and ^{137}Cs released in airborne and liquid effluents from nuclear power plants in the United States is known (USNRC 1993).

Environmental Fate. Information is available to permit the assessment of the environmental fate and transport of cesium in air (Hirose et al. 1993; Rybacek et al. 1994), water (Burt 1993; WHO 1983), and sediment and soil (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). Cesium compounds are water soluble, and only one oxidation state (+1) is observed under environmental conditions (Burt 1993). Cesium released to the atmosphere may be carried long distances before being deposited onto soil and water surfaces by wet and dry deposition (Hirose et al. 1993). Most of the cesium released to water will adsorb to suspended solids in the water column and ultimately be deposited in the sediment core. In soil surfaces, cesium is strongly adsorbed in the upper layers and generally has very low mobility (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). This is particularly true for soils with high potassium content or soils rich in clay. The radioactive decay modes of the two most important cesium isotopes, ^{134}Cs and ^{137}Cs, have been described in Chapter 4 (ICRP 1983).

Bioavailability from Environmental Media. The bioavailability of cesium in environmental media is well understood and no data needs are identified at this time. For the most part, cesium is adsorbed strongly in the surface of most soils and is not readily bioavailable (Paasikallio 1999). In a study in which 102 healthy volunteers were fed ^{134}Cs-contaminated soil pellets, only about 1% of the original intake was absorbed, and on average, about 60% of the original amount was excreted within 48 hours (LeRoy et al. 1966). In peaty or podzolic soils with low clay content, cesium is reversibly adsorbed to the organic fraction and is expected to be in bioavailable form (Sanchez et al. 1999). Cesium uptake in vascular plants has been demonstrated (Djingova et al. 1995; Willey and Martin 1995). Increasing the clay or potassium content results in lower uptake by plants (Shenber and Johanson 1992). It has also been shown that fish residing in waters with high concentrations of humic material and potassium, such as oceans, have lower whole-body cesium concentrations than fish in freshwater where the concentration of dissolved potassium is lower for the same cesium concentration in the water (WHO 1983).

Food Chain Bioaccumulation. Cesium bioaccumulates in both aquatic and terrestrial food chains (Rowan et al. 1998; WHO 1983). Mean BCFs for ^{137}Cs of 146, 124, and 63 were reported for fish, brown macroalgae, and molluscs, respectively (Fisher et al. 1999). Cesium accumulates in aquatic organisms from both food sources and from cesium dissolved in the water column or adsorbed to suspended solids and sediments. The lichen-caribou-human food chain is an important route of human exposure and has been well studied (Allaye-Chan et al. 1990; WHO 1983). No additional data are required to assess the potential for human exposure to cesium through food chain bioaccumulation.

Exposure Levels in Environmental Media. Stable cesium and radioactive cesium have been detected in air (Ajdacic and Martic 1990; Chiavarini et al. 1994; Dasch and Wolff 1989; Rybacek et al. 1994; Todorovic et al. 1999), water (Asubiojo et al. 1997; DOE 1992; Fisher et al. 1999; Prister et al. 1990; Strezov et al. 1999; WHO 1983), soil/sediment (Burt 1993; DOE 1992, 1998a; Ruse and Peart 2000; WHO 1983), and food (Ahman et al. 1994; Bibak et al. 1998; Mondon and Walters 1990). Due to the large surface area of lichens and moss, they can collect a great deal of atmospheric nuclear fallout and often have high concentrations of ^{134}Cs and ^{137}Cs (Papastefanou et al. 1992; Penttila et al. 1993). Grazing animals such as reindeer and caribou that feed on large quantities of lichens or moss may potentially ingest large amounts of radioactive cesium, and this cesium may be transferred to humans who consume these animals as a meat source (Allaye-Chan et al. 1990; WHO 1983). Continued monitoring data on ^{134}Cs and ^{137}Cs in environmental media are needed to increase knowledge of human exposure to ^{134}Cs and ^{137}Cs.

Exposure Levels in Humans. Monitoring data are available for the levels of ^{134}Cs and ^{137}Cs in human populations (Glowiak et al. 1977b; Lokobauer et al. 1988; Rabitsch et al. 1991; Rahola and Suomela 1998; Ropolo and Cesana 1997). Most of the current data are for areas of eastern Europe and Russia. Limited data are available regarding the levels of ^{133}Cs in humans (Hewitt 1988). Stable cesium was detected in the urine of U.S. residents at geometric mean concentrations of about 4–5 μg/L depending upon age and gender (CDC 2003). More information regarding the background concentration of ^{133}Cs in human populations would be useful, but given the ubiquitous distribution of cesium at low levels in the environment, background levels are unlikely to approach levels that would reflect cesium toxicity. Since the levels of radioactivity change over time, continued monitoring data on the levels of ^{134}Cs and ^{137}Cs in humans are needed to evaluate potential risks to human health at current exposure levels.

Exposures of Children. Children, like adults, are not expected to be over exposed to ^{133}Cs. Existing data show that children are exposed to ^{134}Cs and ^{137}Cs after major releases. These exposures are primarily related to dietary intake and the intake of contaminated milk (Lokobauer et al. 1988). Dietary consumption patterns suggest that the weight-adjusted intake of radioactive cesium for children may be greater than for adults following a nuclear release (Lokobauer et al. 1988). In a study of females from northern Sweden, ^{137}Cs was detected in breast milk from 8 out of 12 mothers at concentrations of 7.3–178.4 pCi/kg (Johansson et al. 1998). The infants of these mothers who were being breast-fed had whole-body levels of ^{137}Cs in the range of 45.9–675.7 pCi/kg (Johansson et al. 1998). Additional data regarding the transfer of ^{134}Cs and ^{137}Cs from breast milk and contaminated pasteurized milk samples to children, as well as body burden studies on children, would be useful in assessing the potential risk that these radionuclides would pose following a major release.

Child health data needs relating to susceptibility are discussed in Section 3.13.2, Identification of Data Needs: Children's Susceptibility.

Exposure Registries. Cesium is currently one of the chemicals for which a subregistry has been established in the National Exposure Registry. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to cesium exposure.

6.8.2 Ongoing Studies

The database of federal research programs in progress (FEDRIP 2002) indicates several current projects that may fill some existing data gaps and add to the current database of knowledge. Studies are in progress to identify methods for more effective removal of radioactive cesium from contaminated soils and wastes. Researchers at the Universitaet des Saarlandes, Saarbruecken (Germany) are seeking cost-effective methods to remove gamma-emitting radionuclides, such as ^{137}Cs and ^{134}Cs, from radioactive waste commonly produced at nuclear power plants. Researchers at the Oak Ridge National Laboratory, Environmental Sciences Division are studying the environmental fate and behavior of selected radionuclides in terrestrial and aquatic environments. Dr. Michael Blaylock of Phytotech Incorporated is investigating the use of metal-accumulating plant species to extract and remove the radionuclides cesium and strontium from contaminated soils ("phytoextraction"). Rufus L. Chaney from the Beltsville Agricultural Research Center in Beltsville, Maryland is researching the feasibility of developing plants, soil, and plant management practices that can cost-effectively phytoextract radionuclides and heavy metals from contaminated soils. Dr. Heit from the U.S. DOE is conducting measurements regarding the fallout of nuclear debris from both atmospheric weapons tests and accidental atmospheric releases to determine the mechanisms of transport and deposition and to verify and correct fallout models. Work is being performed by Dr. Kinkead at the Los Alamos National Laboratory regarding the separation of cesium and strontium from high-level radioactive waste. Dr. Leon Kochian from the Agricultural Research Center in Ithaca, New York is investigating the bioaccumulation of ^{137}Cs in plants grown in contaminated soils.

7. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, measuring, and/or monitoring cesium, its metabolites, and other biomarkers of exposure and effect to cesium. 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 cesium and its radioisotopes into the human body occurs by ingestion, inhalation, or penetration through skin (IAEA 1988; NCRP 1977, 1985). The amounts of cesium in the body can be assessed by bioassay *(in vivo* and *in vitro)* measurements. *In vivo* measurements are made with whole-body counters. *In vivo* measurement techniques are commonly used to measure body burdens of cesium radioisotopes, but can not be used to measure the body content of the stable isotope of cesium. *In vitro* measurements provide an indirect estimate of internally deposited cesium (both the stable and radioactive isotopes), by techniques that measure cesium in body fluids, feces, or other human samples (Gautier 1983). Examples of these analytical techniques are given in NCRP Report No. 87 (1987) and are also listed in Table 7-1 for stable cesium and Table 7-2 for radioactive cesium.

7.1.1 *In Vivo* Cesium Measurements

In vivo measurement techniques are the most direct and widely used approach for assessing the burden of cesium radioisotopes in the body. The *in vivo* measurement of these radioisotopes within the body is performed with various radiation detectors and associated electronic devices that are collectively known as whole-body counters. These radiation detectors usually employ sodium iodide (NaI), hyperpure germanium, or organic liquid scintillation detectors to measure the 605 (98%) and 796 keV (85%) gamma

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

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Sample dried, ashed, and irradiated	INAA	1×10^{-5} µg/g	No data	Oughton and Day 1993
Soft tissue	Sample ashed and then concentrated by precipitation with AMP, extracted with sodium phenylboron	Flame photometry	0.005 µg/g	96–99%	Feldman and Rains 1964
Feces	Sample dried, ashed, and irradiated	INAA	1×10^{-5} µg/g	No data	Oughton and Day 1993

AMP = ammonium molybdophosphate; INAA = instrument neutron activated analysis

Table 7-2. Analytical Methods for Determining Radiocesium in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Sample is acidified and concentrated on a KCFC column	γ-spectrometry with NaI detector	6 pCi/L	98%	Boni 1966
Urine	Sample transferred to Marinelli beaker and counted	γ-spectrometry with NaI detector	100 pCi/L	98%	Gautier 1983
Urine	Sample transferred to Marinelli beaker and counted	γ-spectrometry with NaI(Tl) detector	2 pCi/L	No data	Cahill and Wheeler 1968
Soft tissue	Sample wet-ashed	γ-spectrometry	No data	No data	Baratta et al. 1969
Soft tissue	Sample directly counted in detector	γ-spectrometry	5 pCi/g	No data	Rabon and Johnson 1973
Soft tissue	Sample digested in acid, oxidized with $HClO_4$, concentrated by precipitated with AMP, purified by resin column, precipitated with hexachloroplatinic acid	β-counter	0.1 pCi/g	40–85%	Nevissi 1992
Feces	Sample directly counted in detector	γ-spectrometry with NaI detector	0.14 nCi/L	No data	Lipsztein et al. 1991
Human milk	Sample directly counted in detector	γ-spectrometry with NaI detector	0.001 pCi/L	No data	Risica et al. 1994

KCFC = potassium cobalt ferrocyanide

rays from the decay of ^{134}Cs, and 662 keV (89.9%) gamma rays that are emitted from the decay of ^{137}Cs (Palmer et al. 1976). ^{134}Cs and ^{137}Cs distribute uniformly in muscle and soft tissue of the body. The photons emitted by ^{134}Cs and ^{137}Cs are easily detected and quantitated using whole-body counting techniques (NCRP 1987; Palmer et al. 1976; Sun et al. 1997). Many configurations of the whole-body counter and scanning methods have been utilized, ranging from unshielded single-crystal field detectors to shielded, multi-detector scanning detectors (IAEA 1962, 1970, 1972, 1976, 1985; NCRP 1987; Palmer et al. 1976). 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. Additionally, care must be exercised to ensure that external contamination with radioactive cesium or other gamma-emitting radioisotopes are not present on the clothing or skin of the individual to be scanned. *In vitro* measurements of cesium (see Section 7.1.2) are used in conjunction with whole-body counting when monitoring individuals working with cesium, especially in conjunction with the assessment of individuals who have experienced accidental exposures to cesium (Bhat et al. 1973).

Whole-body counters are calibrated using tissue-equivalent phantoms. These phantoms are constructed to mimic the shape and density of the anatomical structure using tissue-equivalent materials such as water-filled canisters or masonite (Barnaby and Smith 1971; Bhat et al. 1973; Sun et al. 1997). For example, the bottle mannequin absorber (BOMAB) consists of a series of water-filled polyethylene canisters constructed into seated or reclined human forms (Sun et al. 1997). Cesium standards are measured either as point sources along the phantom or dissolved within the water-filled canisters. Comparisons of the actual counts obtained from the phantom to the known activity of the cesium standards are used to determine the efficiency of the counting technique and, thus, to provide the basis for calibrating the counting system.

Assessment of short- and long-term retention of cesium radioisotopes takes into account the turnover rate for cesium within the human body. Although the physical half-life of ^{137}Cs is 30 years, the biological and effective half-life of cesium inside the body is approximately 110 days (NCRP 1987; Rundo and Newton 1964). This relatively high turnover rate for cesium in the body is due to the high solubility of cesium in body fluids that allows for the rapid uptake (e.g., absorption of ingested cesium through the gut) and elimination of cesium into and from the body (e.g., excreted through urine) (NCRP 1987). For acute and chronic exposures to cesium, the estimates of cesium retention are determined from direct, whole-body measurements. Models for cesium in the human body have been developed for estimating the short- and long-term retention of cesium based on whole-body measurement techniques (ICRP 1979, 1989, 1993; NCRP 1987; Sun et al. 1997).

7.1.2 *In Vitro* Cesium Measurements

In vitro analyses of cesium are routinely performed in support of an *in vivo* monitoring program or in situations where direct *in vivo* measurements can not be obtained. Urinalysis is the preferred sample for *in vitro* analyses of cesium, although other sample types, such as feces, tissue, bone, or blood, can also be analyzed. Urinalysis is an optimum method for assessing the clearance of soluble cesium. Fecal analysis is used to assess the clearance of ingested, insoluble cesium (Baratta et al. 1969; Gautier 1983; Ide and McInroy 1975; NCRP 1987).

The *in vitro* analysis of the stable isotope of cesium, ^{133}Cs, in human samples (e.g., urine, tissue, feces) is performed by a number of methods that have the selectivity and sensitivity to measure cesium in biological matrices. These methods include spectrophotometry, instrumental neutron activation analysis (INAA), and inductively coupled plasma mass spectrometry (ICP-MS) (Dreizen et al. 1970; Iyengar and Woittiez 1988; Paschal et al. 1996). Of these methods, the INAA and ICP-MS methods offer the greatest detection sensitivity and are the preferred method of analysis for cesium in human samples (Iyengar and Woittiez 1988).

For the *in vitro* analysis of the cesium radioisotopes ^{134}Cs and ^{137}Cs in human samples, a number of analytical methods can be used to measure the cesium radioisotopes directly in the samples without requiring an extensive sample preparation procedure. In the radiochemical analysis of cesium in urine, a 24-hour urine collection (approximately 2 L) is obtained, followed by the transfer of a 1 L aliquot to a Marinelli beaker for counting in a gamma-ray spectrometer (Gautier 1983). This simple procedure offers high recoveries of cesium (98%) and the minimum detection sensitivity (100 pCi/L) that is required to evaluate individuals for exposures to radioactive cesium (Gautier 1983). Similar methods are also used for the analysis of cesium radioisotopes in tissues, feces, and blood (Table 7-1). Mass spectrometry techniques have also been employed to measure cesium radioisotopes in human samples.

Accuracy of *in vivo* and *in vitro* measurements of cesium is determined through the use of standard, certified solutions or radioactive sources with known concentrations or activities of cesium. National Institute of Standards and Technology (NIST) traceable standards for ^{133}Cs can be obtained through a number of commercial sources. The primary source of certified cesium radioisotope standards is the NIST. Gamma-ray point sources for ^{137}Cs (standard reference material [SRM] 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [8 μCi]) and standard solutions of ^{137}Cs (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST.

7.2 ENVIRONMENTAL SAMPLES

Two common approaches are available for measuring cesium radioisotopes in the environment. Cesium radioisotopes 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 quantitation of cesium. However, quantitation of the stable cesium isotope in environmental samples is generally conducted in the laboratory.

7.2.1 Field Measurements of Cesium

In situ measurement techniques are useful for the rapid characterization of radionuclide contamination in the environment, such as soils, sediments, and vegetation, and for monitoring personnel for exposure to radionuclides. The measurement of gamma ray-emitting radionuclides such as ^{134}Cs and ^{137}Cs in the environment is conducted with portable survey instruments such as Gieger-Mueller detectors, sodium iodide scintillation detectors, and gamma-ray spectrometers. The use of gamma-spectrometers in field survey equipment is preferred for measuring cesium in the field because of its energy selectivity and detection sensitivity. The relatively high energy and penetrance of the gamma ray that is emitted during the decay of ^{134}Cs and ^{137}Cs provide an advantage for assessing the level of cesium. These gamma-ray spectrometers are equipped with a high purity germanium detector that is able to separate the 602, 662, and 796 keV gamma rays emitted from ^{134}Cs and ^{137}Cs from the gamma rays emitted from other radionuclides; for example, ^{40}K (USNRC 1997). Minimum detectable activities (MDAs) of 0.005 Bq/g for ^{137}Cs are routinely achieved using p-type germanium gamma spectrometers with 10-minute counting times (USNRC 1997). Computational methods have been derived to aid in determining the concentrations and distributions of ^{134}Cs and ^{137}Cs in different soil types and depths (Fülöp and Ragan 1997; Hillmann et al. 1996; USNRC 1997). The concentrations and distributions of ^{134}Cs and ^{137}Cs that have been derived from the computational analysis of the survey data are often verified by laboratory-based analyses of soil samples procured from the survey area.

7.2.2 Laboratory Analysis of Environmental Samples

Analytical methods for measuring cesium and cesium radioisotopes in environmental samples (e.g. air, water, soil, and biota) are summarized in Tables 7-3 (^{133}Cs) and 7-4 (^{134}Cs, ^{137}Cs). The methods that are commonly used in the analysis of ^{133}Cs are based on instrumental analytical techniques such as spectrophotometry, instrumental neutron activation analysis, and mass spectrometry. The analysis of ^{134}Cs and ^{137}Cs can be determined either as total mass or total activity, depending on the analytical technique that is used. Typically, radiochemical methods of analysis employing gamma spectrometry techniques are used to quantitate ^{134}Cs and ^{137}Cs in environmental samples. However, spectrophotometric and mass spectrometry techniques have been used to determine the total mass of ^{134}Cs and ^{137}Cs in samples. Using the specific activity of ^{137}Cs (89 μCi/μg), it can be deduced that a sample with activity of 1 μCi of ^{137}Cs contains roughly 0.011 μg of ^{137}Cs.

The analysis of cesium in air is based on the measurement of cesium in aerosols or particles that become trapped on cellulose or glass fiber filters after a measured amount of air is pulled through the filters. For the analysis for ^{133}Cs, the filter is solvent extracted and the extracted metals are analyzed by INAA (Gone et al. 2000). Analysis of ^{134}Cs and ^{137}Cs can be performed directly from the filter, or by following some sample preparation (e.g., ashing or solvent extraction), using gamma spectrometry (Kanapilly et al. 1983; Kolb 1971; Krieger et al. 1976).

For the analysis of cesium in water, a broad array of sample preparation and detection methodologies are available (see Tables 7-3 and 7-4). Different standardized methods that can directly measure cesium or its radioactive isotopes within a water sample using INAA or radiochemical techniques with minimal sample preparation and good detection sensitivities (10–20 pCi/L), precision (4–9%), and bias (-5–1%) (ASTM 1999; EPA 1980). Other methods are available that preconcentrate cesium from natural or potable waters when interfering impurities are present or the activity of the cesium radioisotopes are too low (<30 pCi/L) for quantitation by gamma spectrometry (APHA 1998; Frigieri et al. 1980). This preconcentration of cesium can be achieved either through precipitation with molybdate compounds, for example, or through chromatographic techniques using columns packed with resins that specifically bind cesium (EPA 1980; Frigieri et al. 1980; Gaur 1996; Petrow and Levine 1967).

The quantity of cesium and its radioisotopes in soil, sediments, vegetation, and biota is determined using detection methods similar to those described above (Tables 7-3 and 7-4). Analysis of the stable cesium isotope by INAA and ICP-MS requires either digesting the sample in acid or ashing the sample before

Table 7-3. Analytical Methods for Determining Cesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Water	Sample is purified by passage through Dowex 50-X8 resin, concentrated with NCFC column	Electrothermal-AA	1 μg/L	99% at 10–100 μg/L	Frigieri et al. 1980
River water	Sample concentrated by precipitation with AMP, purified by extraction with sodium tetraphenylboron	Flame photometry	0.010 μg/L	94.5%	Feldman and Rains 1964
River water	Sample is purified by passage through Dowex 50-X8 resin, concentrated with NCFC column	Electrothermal-AA	1 μg/L	99% at 10–100 μg/L	Frigieri et al. 1980
Lake water	Filtered samples were irradiated	INAA	0.010 μg/L	90%	Hakonson and Whicker 1975
Sea water	Sample was precipitated with sodium tetraphenyl-borate, and the precipitate was neutron irradiated	INAA	0.008 μg/L	92%	Taskaev 1987
Sea water	Sample precipitated with AMP, purified by extraction with sodium tetraphenyl-boron	Flame photometry	0.010 μg/L	94.5%	Feldman and Rains 1964
Mineral and thermal waters	Direct aspiration of sample into graphite furnace	Graphite furnace-AA	0.00185 μg/L	92.3–100.9%	Bermejo-Barrera et al. 1989
Groundwater	Sample purified by ultracentrifugation	ICP-MS	0.010 μg/L	No data	Probst et al. 1995
Groundwater	Sample purified by ultracentrifugation	ICP-AES	0.010 μg/L	No data	Probst et al. 1995
Soil	Sample was pre-ashed, digested with acid	Electrothermal-AA	0.09 mg/g	80–85%	Anderson et al. 1996
Soil	Sample was dried, ground, and irradiated	INAA	0.003 ng/g	No data	Oughton and Day 1993
Soil	Sample digested with acid	ICP-MS	0.011 μg/g	95%	Robb et al. 1995
Sediment	Sample was dried and irradiated	INAA	0.010 μg/g	90%	Hakonson and Whicker 1975
Silicate rock	Sample digested in HF/H_2SO_4	Graphite furnace-AA	0.05 μg/L	76%	Grobenski et al. 1983

Table 7-3. Analytical Methods for Determining Cesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Vegetation	Sample ashed and irradiated	INAA	$1x10^{-5}$ μg/g	No data	Oughton and Day 1993
Vegetation	Sample prepared by microwave digestion	ICP-MS	$2x10^{-5}$ μg/g	95–105%	Dombovári et al. 2000

AA = atomic absorption; AMP = ammonium molybdophosphate; ICP-AES = inductively coupled plasma-atomic emission spectrometry; ICP-MS = inductively coupled plasma-mass spectrometry; INAA = instrumental neutron activation analysis; NCFC = ammonium hexacyanocobalt ferrate

Table 7-4. Analytical Methods for Determining Radiocesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (occupational)	Sample filter was solvent extracted	Scintillation counter with NaI detector	No data	95%	Kanapilly et al. 1983
Air (ambient)	Sample filter digested in acid, cesium precipitated with chloroplatinate	γ-spectrometry with Ge/Li detector	0.01 fCi/m^3	80%	Krieger et al. 1976
Drinking water	Direct count of sample	γ-spectrometry with Ge/Li detector	10 pCi/L	No data	EPA 1980
Drinking water	Direct count of sample	γ-spectrometry with Ge detector	<2 pCi/L	92–100%	APHA 1998
Fresh water	Sample concentrated with Dowex 1x8/KCFC mixed ion exchange column	γ-spectrometry with NaI detector	3 pCi/L	99%	Boni 1966
River water	Sample precipitated with AMP, concentrated with Dowex-50 cation exchange column	Scintillation counter with NaI detector	<7 fCi/L	99%	Kahn et al. 1957
River water	Sample concentrated on Dowex 50W-X8 column	γ-spectrometry with Ge(Li) detector	2 pCi/L (50 L sample)	97%	Luetzelschwab 1976
Lake water	Sample concentrated on ACFC column	γ-spectrometry with NaI(Tl) detector	No data	97%	Eyman and Kevern 1975
Water and waste water	Direct count of sample	γ-spectrometry with Ge/Li detector	<2 pCi/L	92–100% at 2–94 pCi/L	ASTM 1999
Sea water	Sample purified by passage through chelating resin, concentrated with KCFC ion exchange column	γ-spectrometry with NaI detector	0.07 pCi/L	98%	Boni 1966
Soil	Sample dried and crushed	γ-spectrometry with Ge(Li) detector	0.05 pCi/g	No data	Arnalds et al. 1989
Soil	Sample mixed with 5% Ag and compressed into disc	GDMS	0.2 pg/g	No data	Betti et al. 1996
Sediment	Sample extracted with acid, concentrated by precipitation with AMP, solvent extracted with sodium tetraphenylboron	γ-spectrometry with Ge(Li) detector	No data	96%	Eyman and Kevern 1975

Table 7-4. Analytical Methods for Determining Radiocesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Biota	Sample ashed, oxidized with $HClO_4$, concentrated by precipitation with AMP, puriifed by resin column, precipitated with hexa-chloroplatinic acid	β-counter	0.1 pCi/g	No data	Nevissi 1992

ACFC = ammonium hexacyanocobalt ferrate; AMP=ammonium molybdophosphate; ICP-MS = inductively coupled plasma-mass spectrometry; KCFC = potassium cobalt ferrocyanide; GDMS = glow discharge mass spectrometry analysis. In some cases where interfering compounds or materials may be present, additional sample concentration or purification may be required. For the radioisotopes of cesium, direct detection of ^{134}Cs and ^{137}Cs within the neat sample can be performed using gamma spectrometry detection methods.

The detection limits, accuracy, and precision of any analytical methodology are important parameters in determining the appropriateness of a method to quantitate a specific analyte at the desired level of sensitivity within a particular matrix. The MDA refers to the intrinsic detection capability of a measurement procedure (sampling through data reduction and reporting) (USNRC 1984). Several factors influence the MDA, including background count 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 MDAs may vary between laboratories using the same or similar measurement procedures.

The accuracy of a measurement technique in determining the quantity of a particular analyte in environmental samples is dependent on the reliability of the calibrating technique. Thus, the availability of standard, certified radiation sources with known concentrations of cesium and its radioisotopes is required in order to insure the reliability of the calibration methods and accuracy of cesium measurements in environmental samples. NIST traceable standards for ^{133}Cs can be obtained through a number of commercial sources. The primary source of certified cesium radioisotope standards is the NIST. Gamma-ray point sources for ^{137}Cs (SRM 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [8 μCi]) and standard solutions of ^{137}Cs (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST. SRMs are also available containing the stable (SRM 1944 [sediment], SRM 2710 and 2711 [soil]) and radioactive isotopes of cesium (SRM 4350 [sediment] and SRM 4357 [sediment]).

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 cesium 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 cesium.

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 cesium and its radioisotopes in human tissues and body fluids.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods with the required sensitivity and accuracy are available for measuring cesium, both total and isotopic, in environmental matrices (Tables 7-2 and 7-4). Knowledge of the levels of cesium in various environmental media, along with appropriate modeling (see Chapters 4 and 6), can be used to evaluate potential human exposures through inhalation and ingestion pathways.

Whether in the environment or in the human body, cesium radioisotopes will undergo radioactive decay to nonradioactive isotopes (see Chapter 4). Current analytical methods, such as mass spectrometry, have the necessary resolution and sensitivity to detect and quantitate these decay products.

7.3.2 Ongoing Studies

Current research trends in the quantitation of cesium and its radioisotopes are focused on improving the selectivity and detection sensitivity of cesium in biological and environmental samples. Mass spectrometry approaches, such as double focusing sector field inductively coupled mass spectrometry or time-of-flight selected ion monitoring systems, are being developed further to provide the required selectivity and sensitivity to rapidly measure cesium in the presence of other trace metals in complex environmental samples. Cesium-selective electrodes are being developed into a highly-selective, rapid detection technique for measuring cesium in environmental samples and waste streams. Current efforts are focused on the development of electrode membranes that contain cesium binding agents, such as crown ether derivatives. New cesium-selective resins, for example lanthanum-based resins or montmorillonite clays, are being developed and tested for selectivity and recovery of cesium.

The database of federal research programs in progress (FEDRIP 2002) indicates several current projects that may fill some existing data gaps and add to the current database of knowledge. W.H. Aberth from Antek Incorporated, located in Palo Alto, California is attempting to increase the sensitivity to which cesium can be analyzed in human tissue through the use of a cluster ion gun in liquid dynamic secondary ion mass spectrometry (SIMS).

8. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding stable cesium in air, water, and other media are summarized in Table 8-1. The regulations regarding radioactive cesium are summarized in Table 8-2.

No MRLs were derived for inhalation or oral exposure to stable or radioactive cesium. Two MRLs, derived by the Agency for Toxic Substances and Disease Registry (1999) for external exposure to ionizing radiation, are applicable to external exposure to radioisotopes of cesium. An MRL of 400 mrem (4.0 mSv) was derived for acute-duration external exposure (14 days or less), based on cognitive learning deficit in children who had been exposed to ionizing radiation at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki (Schull et al. 1988). An MRL of 100 mrem/year (1.0 mSv/year) above background was derived for chronic-duration external exposure (365 days or more), based on the BEIR V (1990) report that the average annual effective ionizing radiation dose to the U.S. population is 360 mrem/year (3.6 mSv/year), a dose not expected to produce adverse health effects.

The EPA has not derived reference concentrations (RfCs) or reference doses (RfDs) for stable or radioactive cesium (IRIS 2002). The IRIS database does not provide cancer assessments for radioisotopes of cesium. This function is the responsibility of the EPA Office of Radiation and Indoor Air (ORIA). All radionuclides, including radioisotopes of cesium, are classified as known human (Group A) carcinogens. This classification is based on results of epidemiological studies of Japanese atomic bomb survivors, underground uranium miners, radium dial painters, and patients subjected to a variety of radiation treatments, as well as results of laboratory animal research and mammalian tissue culture studies. ORIA has published cancer slope factors (mortality and morbidity cancer risk estimates) for all known radionuclides, by various exposure routes (inhalation, drinking water ingestion, food ingestion, soil ingestion, immersion in a cloud, and external exposure from contaminated soil) for five age groups and 14 radiogenic cancer sites (EPA 2000). Slope factors for ^{137}Cs and ^{134}Cs are listed in Table 8-2.

Table 8-1. Regulations and Guidelines Applicable to Cesium

Agency	Description	Information	Reference
<u>INTERNATIONAL</u>			
Guidelines:			
IARC		No data	IARC 2000
<u>NATIONAL</u>			
Regulations and Guidelines:			
a. Air			
ACGIH	TLV (8-hour TWA for a 40-hour workweek)		ACGIH 2000, 2001
	Cesium hydroxide (based on upper respiratory tract, skin, and eye irritation)	2 mg/m^3	
EPA		No data	
NIOSH	REL (10-hour TWA for a 40-hour workweek		NIOSH 1992, 2000
	Cesium hydroxide (based on skin, eye, and respiratory irritation)	2 mg/m^3	
OSHA		No data	
b. Water		No data	
c. Food		No data	
d. Other		No data	
<u>STATE</u>		No data	

ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; IARC = International Agency for Research on Cancer; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; REL = recommended exposure limit; TLV = threshold limit value; TWA = time weighted average

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium

Agency	Description	Information	Reference
INTERNATIONAL			
Guidelines:			
IARC		No data	IARC 2000
NATIONAL			
Regulations and Guidelines:			
a. Air			
ACGIH		No data	ACGIH 2000
EPA	Detection limits for man-made beta particle and photon emitters	10 pCi/L	EPA 1999a 40 CFR 141.25
NIOSH		No data	NIOSH 2000
NRC	Effluent concentrations–air		NRC 1999a 10 CFR 20 App B
	^{134}Cs	2x10^{-10} μCi/mL	
	^{137}Cs	2x10^{-10} μCi/mL	
b. Water			NRC 1999a 10 CFR 20 App B
NRC	Effluent concentrations–water		
	^{134}Cs	9x10^{-7} μCi/ml	
	^{137}Cs	1x10^{-6} μCi/ml	
c. Food			FDA 1998
FDA	Derived intervention level[a] (DIL; Bq/kg food) in accidentally-contaminated human food		
	^{134}Cs	930	
	^{137}Cs	1360	
d. Other			
EPA	Concentration levels for environmental compliance		EPA 1999a 40 CFR 61 App E
	^{134}Cs	0.7x10^{-14} Ci/m^3	
	^{137}Cs	1.9x10^{-14} Ci/m^3	
	Carcinogenicity–slope factors[b]		EPA 2002
	Lifetime risk per pCi–ingestion		
	Water		
	^{134}Cs	4.22x10^{-11}	
	^{137}Cs	3.04x10^{-11}	
	Food		
	^{134}Cs	5.14x10^{-11}	
	^{137}Cs	3.74x10^{-11}	
	Soil		
	^{134}Cs	5.81x10^{-11}	
	^{137}Cs	4.33x10^{-11}	
	Lifetime risk per pCi–inhalation		
	^{134}Cs	1.65x10^{-11}	
	^{137}Cs	1.19x10^{-11}	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium

Agency	Description	Information		Reference
NATIONAL (*cont.*)				
	External exposure–risk/year per pCi/g soil			EPA 2002
	^{134}Cs	$7.10\text{x}10^{-6}$		
	^{137}Cs	$5.32\text{x}10^{-10}$		
	^{137}Cs (plus disintegration products)	$2.55\text{x}10^{-6}$		
NRC	Occupational inhalation exposure			NRC 1999a
	ALIs			10 CFR 20 App B
	^{134}Cs	100 µCi		
	^{137}Cs	200 µCi		
	DACs			
	^{134}Cs	$4\text{x}10^{-8}$ µCi/mL		
	^{137}Cs	$6\text{x}10^{-8}$ µCi/mL		
	Quantities of licensed material requiring labeling			NRC 1999b 10 CFR App C
	^{134}Cs	10 µCi		
	^{137}Cs	10 µCi		
STATE				
a. Air				
Michigan	Gross beta particle activity			MI Dept Environ Quality 2000
	^{134}Cs	15 pCi/L		
b. Water		No data		
c. Food		No data		
d. Other				
Louisiana	Quantity required for consideration of need for emergency plan for responding to a release	Release fraction	Quantity	LA Dept Environ Quality 2000
	^{134}Cs	0.01	2,000 Ci	
	^{137}Cs	0.01	3,000 Ci	

[a] The FDA-recommended Derived Intervention Level (DIL) for radionuclides of cesium, is defined as the DIL for the most sensitive age group (adults) that was calculated from the most limiting Protective Action Goal (PAG; 5 mSv committed effective whole body dose equivalent).

[b] EPA classifies all radionuclides as Group A (known human) carcinogens. Radionuclide risk coefficients, or 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). Ingestion values are tabulated separately for ingestion of tap water, dietary intakes (food), and incidental soil ingestion. 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 limitations on intake; CFR = Code of Federal Regulations; DAC = derived air concentrations; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; IARC = International Agency for Research on Cancer; NIOSH = National Institute for Occupational Safety and Health; NRC = Nuclear Regulatory Commission; OSHA = Occupational Safety and Health Administration

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10. GLOSSARY

Some terms in this glossary are generic and may not be used in this profile.

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 $I=I_oe^{-\mu x}$, 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.

10. GLOSSARY

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 180° to each other.

Annual Limit on Intake (ALI)—The derived limit for the amount of radioactive material taken into the body of an adult worker by inhalation or ingestion in a year. It is the smaller value of intake of a given radionuclide in a year by the reference man that would result in a committed effective dose equivalent of 5 rem or a committed dose equivalent of 50 rem to any organ or tissue.

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 Mass Number—See Mass Number.

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 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).

- **Terabecquerel (TBq)—**One trillion becquerel.
- **Gigabecquerel (GBq)—**One billion becquerel.
- **Megabecquerel (MBq)—**One million becquerel.
- **Kilobecquerel (kBq))—**One thousand becquerel.
- **Millibecquerel (mBq)—**One-thousandth of a becquerel.
- **Microbecquerel (µBq)—**One-millionth of a becquerel.

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^-).

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.

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.

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—A long-term, continuous exposure to a chemical or radioactive material. For example, 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 that originate outside the earth's atmosphere and interact with the atmosphere to produce a shower of secondary cosmic rays.

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)—An instrument for detecting beta particle radiation. Beta particles are detected by ionization of the counter gas which results in an electrical impulse at an anode wire.

Counter, Geiger-Mueller (GM counter)— Highly sensitive, gas-filled radiation-measuring device that detects (counts) individual photons or particulate radiation.

Counter, Scintillation—The combination of a crystal or 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 $3.7x10^{10}$ nuclear transformations per second. The activity of 1 gram of radium is approximately 1 Ci.

Attocurie (aCi)—One-thousandth of a femtocurie ($3.7x10^{-8}$ disintegrations per second).
Femtocurie (fCi)—One-billionth of a microcurie ($3.7x10^{-5}$ disintegrations per second).
Megacurie (MCi)—One million curies ($3.7x10^{16}$ disintegrations per sec).
Microcurie (μCi)—One-millionth of a curie ($3.7x10^{4}$ disintegrations per sec).
Millicurie (mCi)—One-thousandth of a curie ($3.7x10^{7}$ disintegrations per sec).
Nanocurie (nCi)—One-billionth of a curie ($3.7x10^{1}$ disintegrations per sec).
Picocurie (pCi)—One-millionth of a microcurie ($3.7x10^{-2}$ disintegrations per second.

Daughter Products—See Progeny and Decay Product

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.

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

Delta Ray—An electron removed from an atom of a medium that is irradiated, or through which radiation passes, during the process of ionization (also called secondary electron). Delta rays cause a track of ionizations along their path.

Derived Air Concentration (DAC)—The concentration of radioactive material in air that, if breathed by the reference man for a working year of 2000 hours under conditions of light work (at a rate of 1.2 liters of air per hour), would result in an intake of one ALI (see Annual Limit on Intake).

Deterministic Effect—A health effect, the severity of which varies with the dose and for which a threshold is believed to exist (also called a non-stochastic effect).

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 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. The NRC defines it as the product of the absorbed dose, the quality factor, and all other modifying factors at the location of interest. ICRP has changed its definition to be the product of the absorbed dose and the radiation weighting factor. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

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, 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).

Committed Dose Equivalent ($H_{T,50}$)—The dose equivalent to organs or tissues of reference (T) that will be received from an intake of radioactive material by an individual during the 50 years following the intake.

Committed Effective Dose Equivalent ($H_{E,50}$)—The sum of the products of the weighting factors applicable to each of the body organs or tissues that are irradiated and the committed dose equivalent to those organs or tissues.

Effective Dose—A dose value that attempts to normalize the detriment to the body (for cancer mortality and morbidity, hereditary effects, and years of life lost) from a non-uniform exposure to that of a uniform whole body exposure. Effective dose is calculated as the sum of products of the equivalent dose and the tissue weighting factor (w_T) for each tissue exposed. ($E = \sum D_{T,R}\ w_R\ w_T$).

Effective Dose Equivalent (H_E)—This dose type is limited to internal exposures and is the sum of the products of the dose equivalent to the organ or tissue (H_T) and the weighting factors (w_T) applicable to each of the body organs or tissues that are irradiated. ($H_E = \sum w_T\ H_T$).

Equivalent Dose—A dose quantity that places the biological effect of all radiation types on a common scale for calculating tissue damage. Alpha particles, for example, are considered to cause 20 times more damage than gamma rays. Equivalent dose is calculated as the sum of products of the average absorbed dose (in gray) in an organ or tissue ($D_{T,R}$) from each type of radiation and the radiation weighting factor (w_R) for that radiation ($\sum D_{T,R}\ w_R$).

External Dose—That portion of the dose equivalent received from radiation sources outside the body.

Internal Dose—That portion of the dose equivalent received from radioactive material taken into the body.

Limit—A permissible upper bound on the radiation dose.

Maximum Permissible Dose (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 Rate—The amount of radiation dose delivered per unit time. Generically, the rate at which radiation dose is delivered to any material or tissue.

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 radiation 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). $1\ eV = 1.6 \times 10^{-12}$ 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. Gravitationally, "potential energy" is the energy inherent in a mass because of its spatial relation to other masses. Chemically or radiologically, “potential energy” is the energy released when a chemical reaction or radiological transformation goes to completion. "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.

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.

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)—Subjection to ionizing radiation or to a radioactive material. For example, exposure in air is 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 with emission of several neutrons, 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, Effective—See Half-Time, Effective.

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-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}.

$$\text{Effective half-time} = \frac{\text{Biological half-time} \times \text{Radioactive half-life}}{\text{Biological half-time} + \text{Radioactive half-life}}$$

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.

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.

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).

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$_{(Lo)}$ (LD_{Lo})—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.

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. This is averaged over a number of particles.

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.

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.

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 the medium (typically water) by using the energy released from the nuclear fission of uranium or plutonium isotopes 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—Any radionuclide nuclide which, upon disintegration, yields a new nuclide (termed the progeny or daughter), 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.

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.

Pharmacokinetics—The dynamic behavior of a material in the body, used to predict 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.

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.

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.

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: $E = h\nu$.

Population dose—See Collective dose.

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 daughter 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.

Type of radiation	Quality Factor
X, gamma, or beta	1
Alpha particles	20
Neutrons of unknown energy	10
High energy protons	10

Rad—The traditional 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).

Radiation Weighting Factor (also called Quality Factor)—In radiation protection, a factor (1 for x-rays, gamma rays, beta particles; 20 for alpha particles) weighting the absorbed dose of radiation of a specific type and energy for its effect on tissue.

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.

Radionuclide—Any radioactive isotope of any element. Approximately 5,000 natural and artificial radioisotopes have been identified.

Radiosensitivity—Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are used comparatively, rather than absolutely.

Reference Dose (RfD)—An estimate 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 kVp x rays) required to produce an identical biological effect in a particular experimental organism or tissue (see Quality Factor).

Rem—The traditional 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).

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 minutes continually. No more than four excursions are allowed per day, and there must be at least 60 minutes 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). The sievert is also the SI unit for effective dose equivalent, which is the sum of the products of the dose equivalent to each organ or tissue and its corresponding tissue weighting factor.

Specific-Activity—Radioactivity per unit mass of a radionuclide, expressed, for example, as Ci/gram or Bq/kilogram.

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).

Standardized Mortality Ratio (SMR)—A ratio of the observed number of deaths and the expected number of deaths in a specific standard population.

Stochastic Effect—A health effect that occurs randomly and for which the probability of the effect occurring, rather than its severity, is assumed to be a linear function of dose without a threshold (also called a nondeterministic effect).

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 deposits 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 similar concepts 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.

Tissue Weighting Factor (W_t)—Organ- or tissue-specific factor by which the equivalent dose is multiplied to give the portion of the effective dose for that organ or tissue. Recommended values of tissue weighting factors are:

Tissue/Organ	Tissue Weighting Factor
Gonads	0.70
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Esophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder (adrenals, brain, upper large intestine, small intestine, pancreas, spleen, thymus, and uterus)	0.05

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 absorption, distribution and elimination of toxic compounds in the living organism.

Toxicosis —A diseased condition resulting from poisoning.

Transformation, Nuclear—The process of radioactive decay 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 internal conversion electron emission.

Tritium—The hydrogen isotope with one proton and two neutrons in the nucleus (Symbol: ^3H). 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, Prefixes—Many units of measure are expressed as submultiples or multiples of the primary unit (e.g., 10^{-3} curie is 1 mCi and 10^{3} becquerel is 1 kBq).

Factor	Prefix	Symbol	Factor	Prefix	Symbol
10^{-18}	atto	A	10^{3}	kilo	k
10^{-15}	femto	F	10^{6}	mega	M
10^{-12}	pico	p	10^{9}	giga	G
10^{-9}	nano	N	10^{12}	tera	T
10^{-6}	micro	M	10^{15}	peta	P
10^{-3}	milli	M	10^{18}	exa	E
10^{-2}	centi	C			

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 (or No-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 LEVELS 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 agency wide 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-32, Atlanta, Georgia 30333.

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name:	Radioactive Cesium
CAS Number:	Multiple
Date:	March 11, 2004
Profile Status:	Draft 3, Post-Public
Route:	[ ] Inhalation [ ] Oral [X] External
Duration:	[X] Acute [ ] Intermediate [ ] Chronic
Species:	Human

Minimal Risk Level: 4 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv (400 mrem)

Reference:

Schull WJ, Otake M, Yoshimaru H. 1988. Effect on intelligence test score of prenatal exposure to ionizing radiation in Hiroshima and Nagasaki: A comparison of the T65DR and DS86 dosimetry systems. Radiation Effects Research Foundation. RERF TR 3-88. Research project 24-62.

Burt C. 1966. The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. Brit J Psychol 57(1 & 2):137-153.

Experimental design:

Schull et al. (1988) study: Schull et al. (1988) evaluated the quantitative effect of exposure to ionizing radiation on the developing fetal and embryonic human brain. The end point measured was changes in intelligence test scores. The effects on individuals exposed *in utero* to the atomic bombing of Hiroshima and Nagasaki were based on the original PE86 samples (n=1,759; data on available intelligence testing) and a clinical sample (n=1,598). The original PE86 sample included virtually all prenatally exposed individuals who received tissue-absorbed doses of 0.50 Gy or more. There were many more individuals in the dose range of 0–0.49 Gy in the PE86 sample than in the clinical sample. The clinical sample does not include children prenatally exposed at distances between 2,000 and 2,999 m in Hiroshima and Nagasaki. Children exposed at greater distances or not present in the city were selected as controls. In 1955–1956, Tanaka-B (emphasis on word-sense, arithmetic abilities, and the like, which were associated with the more subtle processing of visual clues than their simple recognition and depended more on connectedness) and the Koga (emphasis on perception of spatial relationships) intelligence tests were conducted in Nagasaki and the Koga test in Hiroshima.

Burt (1966) study: This study determined differences in intelligence in monozygotic twins reared together (n=95) and apart (n=53). All tests conducted in school consisted of (1) a group test of intelligence containing both nonverbal and verbal items, (2) an individual test (the London Revision of the Terman-Binet Scale) used primarily for standardization and for doubtful cases, and (3) a set of performance tests, based on the Pitner-Paterson tests and standardization. The methods and standard remained much the same throughout the study. Some of the reasons for separation of the twins were given as follows: death of the mother (n=9), unable to bring them up properly, mother's poor health (n=12), unmarried (n=6), and economic difficulties. The children were brought up by parents or foster parents (occupation ranged from unskilled to professional). IQ scores in the study group ranged from 66 to 137. The standard deviation of the group of separated monozygotic twins was reported at 15.3 as compared to 15.0 of ordinary siblings. Twins brought up in different environments were compared with those brought up in similar circumstances.

Effects noted in study and corresponding doses:

Schull et al. (1986) study: No evidence of radiation-related effect on intelligence was observed among individuals exposed within 0–7 weeks after fertilization or in the 26th or subsequent weeks. The highest risk of radiation damage to the embryonic and fetal brain occurs 8–15 weeks after fertilization under both dosimetric systems. The regression of intelligence score on estimated DS86 uterine absorbed dose is linear with dose, and the diminution in intelligence score is 21–29 points per Gy for the 8–15-week group and 10–26 points per Gy for the 16–25-week group. The results for 8–15 weeks applies regardless whether or not the mentally retarded individuals were included. The cumulative distribution of test scores suggested a progressive shift downwards in individual scores with increasing exposure. The mean IQ scores decrease significantly and systematically with uterine or fetal tissue dose within the 8–15- and 16–25-week groups.

In summary, analysis of intelligence test scores at 10–11 years of age of individuals exposed prenatally showed that:

There is no evidence of a radiation-related effect on intelligence scores among those individuals exposed within 0–7 weeks of fertilization or in the 26th week of gestation and beyond.

The cumulative distribution of test scores suggests a progressive shift downwards in intelligence scores with increasing exposure to ionizing radiation (dose-response relationship).

The most sensitive group was the 8–15 weeks exposure group. The regression in intelligence scores was found to be linear, with 1 Gy dose resulting in a 21–29 point decline in intelligence scores.

There was no indication of groups of individuals with differing sensitivities to radiation.

Burt (1966) study: The average intelligence of the twins measured on a conventional IQ scale (SD=15) was 97.8 for the separated monozygotes, 98.1 for monozygotes brought up together, 99.3 for the dizygotes as compared with 100.2 for the siblings, and 100.0 for the population as a whole. The difference of 0.3 IQ point between the separated and unseparated identical twins is considered a NOAEL for this study.

Dose and end point used for MRL derivation:

[X] NOAEL [ ] LOAEL 0.3 IQ point reduction in twins, between those raised together and those raised apart.

Uncertainty Factors used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability/sensitive population)

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose:
Not applicable

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information which lend support to this MRL: Husen (1959) reported a study involving 269 pairs of Swedish monozygotic (identical) twins where the intrapair IQ difference was 4 IQ points for a combination of twins raised together and apart. This is somewhat lower than the value of 7 IQ points for identical twins raised apart, and just larger than the range of IQ scores for Washington, DC children repetitively tested (Jacobi and Glauberman 1995).

Supporting evidence for the acute MRL is provided by Jacobi and Glauberman (1995). Children in the 1st, 3rd, and 5th grades born in Washington, DC were tested, and average IQ levels of 94.2, 97.6, and 94.6 were reported. The range of 3.4 IQ points is considered to be a LOAEL for this study, which, if used for MRL derivation, would yield an MRL of 0.004 Sv (3.4 IQ points x 1 Sv/25 IQ points ÷ 30 [10 for use of a LOAEL and 3 for a sensitive population]).

Additional supporting evidence for the acute MRL is provided by Berger et al. 1997, in a case study of accidental radiation injury to the hand. A Mexican engineer suffered an accidental injury to the hand while repairing an x-ray spectrometer. The day after the accident, his symptoms included a tingling sensation and itching in the index and middle fingers. On days 4 and 7, a "pinching" sensation, swelling, and slight erythema were observed. By day 7, the tip of his index fingers was erythematous and a large blister developed with swelling on other fingers. On day 10, examination by a physician showed that the lesions had worsened and the fingers and palms were discolored. On day 10, he was admitted to the hospital where hyperbaric oxygen therapy was administered without success. One month after the accident, the patient entered the hospital again with pain, discoloration, and desquamation of his hand. Clinical examination showed decreased circulation in the entire hand, most notably in the index and middle finger. Total white blood count decreased to 3,000/µL (normal range 4,300–10,800/µL). Cytogenic studies of peripheral blood lymphocytes revealed four dicentrics, two rings, and eight chromosomal fragments in the 300 metaphases studied. The estimated whole body dose was reported to be 0.382 Gy (38.2 rad). This dose is a potential LOAEL for acute ionizing radiation and would yield an MRL of 0.004 Sv (0.38 Sv ÷100 [10 for use of LOAEL and 10 for sensitive human population]).

The NRC set a radiation exposure limit of 0.5 rem (50 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks ATSDR believes that the conservative acute MRL of 4 mSv is consistent with the NRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

Calculations:

Given: 0.3 IQ point is a NOAEL. A 1 Sv dose results in a 25 IQ point reduction (range=21–29 points; mean=25) and provides a conversion factor from IQ prediction to radiation dose. Assume that the radiation dose and the subsequent reduction in IQ is a linear relationship.

MRL = NOAEL x CF ÷ UF
MRL = 0.3 x 1/25 ÷ 3
MRL = 0.004 Sv =4 mSv (400 mrem)

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CAS Number:	Multiple
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Profile Status:	Draft 3, Post-Public
Route:	[ ] Inhalation [ ] Oral [X] External
Duration:	[ ] Acute [ ] Intermediate [X] Chronic
Species:	Human

<u>Minimal Risk Level</u>: 1 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv/year (100 mrem/year)

<u>Reference</u>: BEIR V. 1990. Health effects of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. National Academy Press. Washington, DC.

<u>Experimental design</u>: Not applicable

<u>Effects noted in study and corresponding doses</u>: No individual studies were identified that could be used to base a chronic-duration external exposure MRL that did not result in a cancer-producing end point. However, two sources of information were identified that did provide doses of ionizing radiation that have not been reported to be associated with detrimental effects (NOAELs). These sources provide estimates of background levels of primarily natural sources of ionizing radiation that have not been implicated in producing cancerous or noncancerous toxicological endpoints. BEIR V states that the average annual effective dose to the U.S. population is 3.6 mSv/year. A total annual effective dose equivalent of 3.6 mSv (360 mrem)/year to members of the U.S. population is obtained mainly by naturally occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. The largest contribution (82%) is from natural sources, two-thirds of which is from naturally occurring radon and its decay products. Specific sources of this radiation are demonstrated in Table A-1.

The annual dose of 3.6 mSv per year has not been associated with adverse health effects or increases in the incidences of any type of cancers in humans or other animals.

Dose and end point used for MRL derivation: 3.6 mSv/year

[X] NOAEL [ ] LOAEL 3.6 mSv/year

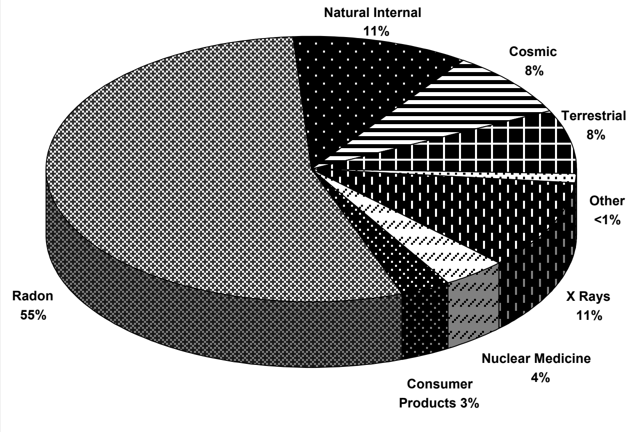
<u>Uncertainty Factors used in MRL derivation</u>:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability)

<u>Was a conversion used from ppm in food or water to a mg/body weight dose</u>? No.

Table A-1. Average Annual Effective Dose Equivalent from Ionizing Radiation to a Member of the U.S. Population[a]

	Effective dose equivalent	
Source	mSv	Percent of total dose
Natural		
Radon[b]	2.0	55
Cosmic	0.27	8.0
Terrestrial	0.28	8.0
Internal	0.39	11
Total natural	3.0	82
Artificial		
Medical		
X-ray	0.39	11
Nuclear	0.14	4.0
Consumer products	0.10	3.0
Other		
Occupational	<0.01	<0.3
Nuclear fuel cycle	<0.01	<0.03
Fallout	<0.01	<0.03
Miscellaneous[c]	<0.01	<0.03
Total artificial	0.63	18
Total natural and artificial	3.6	100



[a] Adapted from BEIR V, Table 1-3 , page 18.
[b] Dose equivalent to bronchi from radon daughter products
[c] DOE facilities, smelter, transportation, etc.

<u>If an inhalation study in animals, list the conversion factors used in determining human equivalent dose</u>: Not applicable.

<u>Was a conversion used from intermittent to continuous exposure</u>? No.

<u>Other additional studies or pertinent information which lend support to this MRL</u>: ICRP has developed recommended dose limits for occupational and public exposure to ionizing radiation sources. The ICRP recommends limiting public exposure to 1 mSv/year (100 mrem/year), but does note that values at high altitudes above sea level and in some geological areas can sometimes be twice that value (≥2 mSv). In Annex C of ICRP 60, the commission provides data that suggests increasing the dose from 1 mSv to 5 mSv results in a very small, but detectable, increase in age-specific human mortality rate. ICRP states that the value of 1 mSv/year was chosen over the 5 mSv value because 5 mSv/year (500 mrem/year) causes this increase in age specific mortality rate, and 1 mSv/year (100 mrem/year) is typical of the annual effective dose from background, less radon (ICRP 1991). The 1 mSv estimate may underestimate the annual exposure to external sources of ionizing radiation to the U.S. population, as it does not include radiation from radon. Conversely, the 5 mSv estimate may be high, in that increases in mortality rate been reported. The most useful estimate appears to be the BEIR V estimate of 3.6 mSv, in that it accounts for an annual exposure to radon, is specific to the U.S. population, has not been associated with increases mortality, and it falls short of the 5 mSv value associated with small increases in human mortality.

Calculations:

MRL = $\text{NOAEL}_{(\text{ADJ})} \div \text{UF}$
MRL = 3.6 mSv/year ÷ 3
MRL = 1.20 mSv/year
MRL = 1.0 mSv/year =100 mrem/year above background

<u>Agency Contact (Chemical Manager):</u> Malcolm Williams, Ph.D.

APPENDIX B. USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in nontechnical 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 a health effects summary 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 that they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, and 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.

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, ATSDR has derived 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.

MRLs 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 3.10, Interactions with Other Substances, and Section 3.11, 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 that the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgment, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgment 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 no-observed-adverse-effect level (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 levels of significant exposure (LSE) Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables and figures 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, 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 NOAELs, 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 Sample LSE Table 3-1 (page B-6)

(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. Typically when sufficient data exist, 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.

(2) Exposure Period. Three exposure periods—acute (<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 two "18r" data points in sample 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 3.5, 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 “Chemical X” via inhalation for 6 hours per day, 5 days per week, for 13 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, one systemic effect (respiratory) was investigated.

(8) NOAEL. A 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 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 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 that the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Sample Figure 3-1 (page B-7)

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 acute and intermediate 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, the open circle designated 18r identifies a NOAEL critical end point in the rat upon which an intermediate inhalation exposure MRL is based. 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 one of three studies for which CELs were derived. The diamond symbol refers to a CEL 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

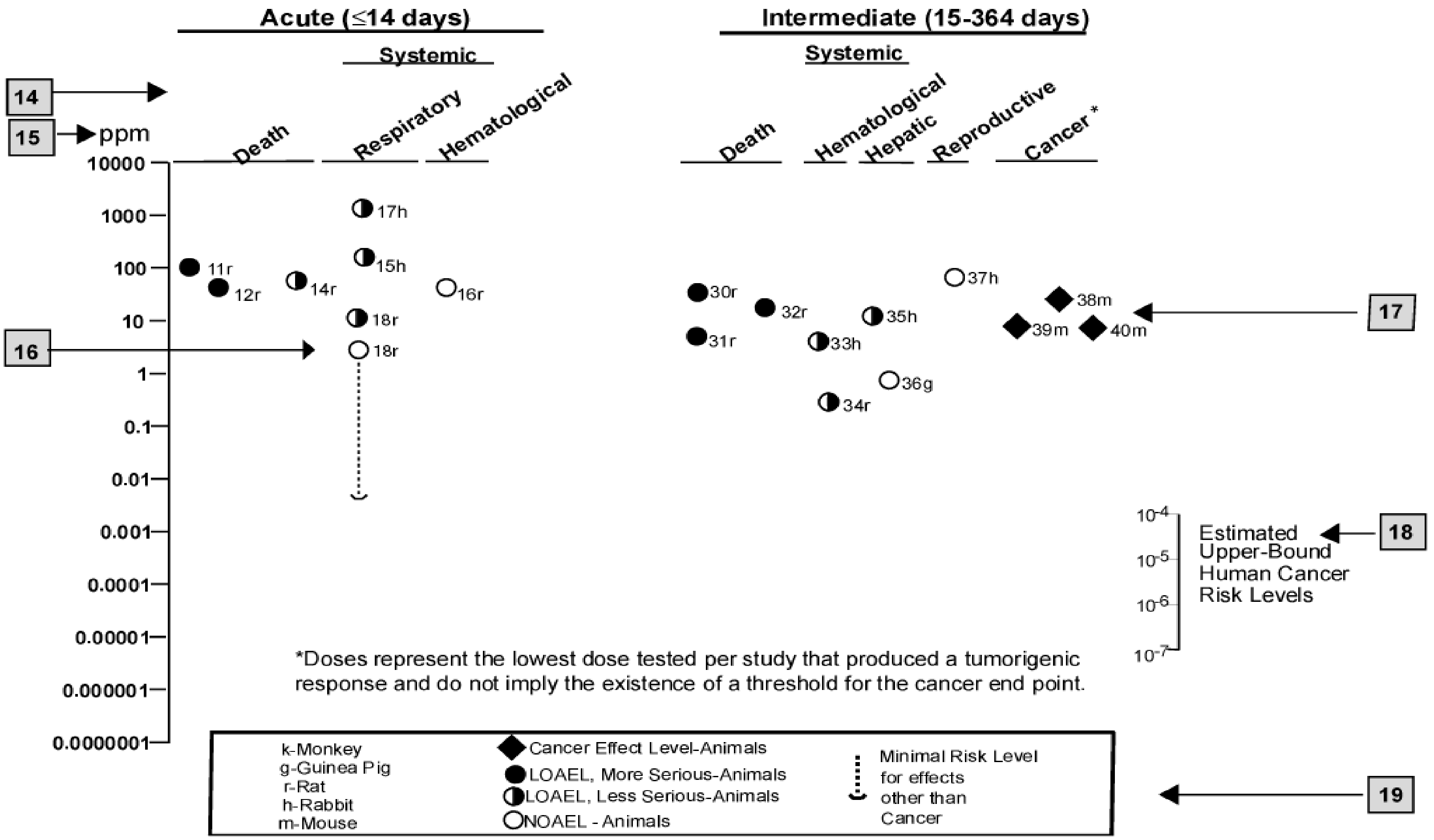
1 → TABLE 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation

						LOAEL (effect)		
	Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	Less serious (ppm)	Serious (ppm)	Reference
2 →	INTERMEDIATE EXPOSURE							
		5	6	7	8	9		10
3 →	Systemic	↓	↓	↓	↓	↓		↓
4 →	18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
	CHRONIC EXPOSURE							
	Cancer						11 ↓	
	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

12 → a The number corresponds to entries in Figure 3-1.
b Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5x10-3 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).

SAMPLE

Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation



*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer end point.

APPENDIX C. ACRONYMS, ABBREVIATIONS, AND SYMBOLS

Some terms are generic and may not be used in this profile.

ACGIH	American Conference of Governmental Industrial Hygienists
ACOEM	American College of Occupational and Environmental Medicine
ADI	acceptable daily intake
ADME	absorption, distribution, metabolism, and excretion
AED	atomic emission detection
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
ALI	annual limit on intake
ALT	alanine aminotransferase
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
AOEC	Association of Occupational and Environmental Clinics
AP	alkaline phosphatase
APHA	American Public Health Association
AST	aspartate aminotransferase
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
BMD	benchmark dose
BMR	benchmark response
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
CI	confidence interval
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
DAC	derived air concentration
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/NA/IMCO	Department of Transportation/United Nations/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
FR	Federal Register
FSH	follicle stimulating hormone
g	gram
GC	gas chromatography
gd	gestational day
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IARC	International Agency for Research on Cancer
IDLH	immediately dangerous to life and health
ILO	International Labor Organization
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_{50}	lethal concentration, 50% kill
LC_{Lo}	lethal concentration, low
LD_{50}	lethal dose, 50% kill
LD_{Lo}	lethal dose, low
LDH	lactic dehydrogenase
LH	luteinizing hormone
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
LT_{50}	lethal time, 50% kill
m	meter
MA	*trans,trans*-muconic acid

MAL	maximum allowable level
mCi	millicurie
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
MFO	mixed function oxidase
mg	milligram
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
mmol	millimole
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
NCEH	National Center for Environmental Health
NCI	National Cancer Institute
ND	not detected
NFPA	National Fire Protection Association
ng	nanogram
NHANES	National Health and Nutrition Examination Survey
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NLM	National Library of Medicine
nm	nanometer
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
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
OPPT	Office of Pollution Prevention and Toxics, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OR	odds ratio
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
pg	picogram
PHS	Public Health Service
PID	photo ionization detector
pmol	picomole
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	pretreatment standards for new sources
RBC	red blood cell
REL	recommended exposure level/limit
RfC	reference concentration
RfD	reference dose
RNA	ribonucleic acid
RQ	reportable quantity
RTECS	Registry of Toxic Effects of Chemical Substances
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
SGOT	serum glutamic oxaloacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SIC	standard industrial classification
SIM	selected ion monitoring
SMCL	secondary maximum contaminant level
SMR	standardized 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 carbon
TPQ	threshold planning quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TWA	time-weighted average
UF	uncertainty factor
U.S.	United States
USDA	United States Department of Agriculture
USGS	United States Geological Survey
USNRC	United States Nuclear Regulatory Commission
VOC	volatile organic compound
WBC	white blood cell

WHO	World Health Organization

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	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 materials (NORMs) exist 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 the activity of a radionuclide per mass of that radionuclide. If properly qualified, it can refer to activity per unit mass of related materials, such as the element itself or a chemical compound labeled with the radionuclide. 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 (ɣ) (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 defined as the activity of a radionuclide per mass of that radionuclide. 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:

$$T_{eff} = (T_{biol} \text{ x } T_{rad}) / (T_{biol} + T_{rad}).$$

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	474 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, as well as the electron density of the material with which it interacts, 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. The alpha particles emitted by a given radionuclide have the same energy and intensity combination. 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 (β) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (β-) or a positively charged electron, termed a positron (β+). Although the precise definition of "beta emission" refers to both β- and β+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the β+ particle.

D.2.4.2.1 Beta Negative Emission. Beta particle (β-) 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 of betas is much less in tissue than in air. 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 (β+) 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 β emitters of equal energy.

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

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[1] Neutrinos also accompany negative beta particles and positron emissions

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 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 (and neutron doses, where applicable) 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 radionuclide depends on the energy absorbed per unit mass by the irradiated tissue. For a radionuclide distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the radionuclide. 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 radionuclide 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 essential 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, 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 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. Radiation-induced leukemia has the shortest latent period at about 2 years, while other radiation induced cancers, such as osteosarcoma, have latent periods greater than 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 the 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 (or Equivalent Dose)

Dose equivalent (as measured in rem or sievert) is a special radiation protection quantity that is used for administrative and radiation safety purposes to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. ICRP (1990) changed this term to equivalent dose, but it has not yet been adopted by the USNRC or DOE.

The USNRC defines 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 or radiation weighting 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 and radiation weighting 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)	Radiation weighting factor (w_r)*
X, gamma, or beta radiation	1	1
Alpha particles, multiple-charged particles, fission fragments and heavy particles of unknown charge	20	0.05
Neutrons (other than thermal >> 100 keV to 2 MeV), protons, alpha particles, charged particles of unknown energy	10	20
Neutrons of unknown energy	10	
High-energy protons	10	0.1
Thermal neutrons		5

*Absorbed dose in rad equal to 1 rem or the absorbed dose in gray equal to 1 sievert.

Source: USNRC. 2004. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C. NCRP 1993

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 kVp x-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor (or radiation weighting factor) used in calculations of dose equivalents for radiation safety purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). Any RBE value applies only to a specific biological end point, in a specific exposure, under specific conditions to a specific species. There are no generally applicable values of RBE since RBEs are specific to a given exposure scenario.

D.5.3 Effective Dose Equivalent (or Effective Dose)

The absorbed dose is usually defined as the mean energy imparted per unit mass to 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. ICRP (1990) changed this term to effective dose, but it has not yet been adopted by the USNRC or DOE.

The effective dose equivalent, H_E, is

$$H_E = \text{(the sum of) } W_t\, H_t$$

where H_t is the dose equivalent (or equivalent dose) in the tissue t, W_t is the tissue weighting factor in that tissue, 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). Tissue weighting factors for selected tissues are listed in Table D-4.

D.5.4 SI Units

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. Tissue Weighting Factors for Calculating Effective Dose Equivalent and Effective Dose for Selected Tissues

	Tissue weighting factor	
Tissue	NCRP115/ ICRP60	USNRC/ICRP26
Bladder	0.05	–
Bone marrow	0.12	0.12
Bone surface	0.01	0.03
Breast	0.05	0.15
Colon	0.12	–
Esophagus	0.05	–
Gonads	0.20	0.25
Liver	0.05	–
Lung	0.12	0.12
Skin	0.01	–
Stomach	0.12	–
Thyroid	0.05	0.03
Remainder	0.05	0.30
Total	1.00	1.00

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
USNRC = 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	10^{-2} Jkg^{-1}	gray (Gy)	Jkg^{-1}
Absorbed dose rate (Ď)	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	10^{-2} Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose equivalent rate (Ĥ)	rem per second (rem s^{-1})	10^{-2} Jkg^{-1}s^{-1}	sievert per second (Sv s^{-1})	Jkg^{-1} s^{-1}
Effective dose	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Equivalent dose (H)	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-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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APPENDIX E. INDEX