

TOXICOLOGICAL PROFILE FOR
RADON

Agency for Toxic Substances and Disease Registry
U.S. Public Health Service

In collaboration with:

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FOREWORD

The Superfund Amendments and Reauthorization Act (SARA) of 1986 (Public Law 99-499) extended and amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). This public law directed the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare toxicological profiles for hazardous substances which are most commonly found at facilities on the CERCLA National Priorities List and which pose the most significant potential threat to human health, as determined by ATSDR and the Environmental Protection Agency (EPA). The lists of the 250 most significant hazardous substances were published in the Federal Register on April 17, 1987, on October 20, 1988, on October 26, 1989, and on October 17, 1990.

Section 104(i)(3) of CERCLA, as amended, directs the Administrator of ATSDR to prepare a toxicological profile for each substance on the list. Each profile must include the following content:

- (A) An examination, summary, and interpretation of available toxicological information and epidemiological evaluations on the hazardous substance in order 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 which present a significant risk to human health of acute, subacute, and chronic health effects, and
- (C) Where appropriate, an identification of toxicological testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

This toxicological profile is prepared in accordance with guidelines developed by ATSDR and EPA. The original guidelines were published in the Federal Register on April 17, 1987. Each profile will be revised and republished as necessary, but no less often than every three years, as required by CERCLA, as amended.

The ATSDR toxicological profile is intended to characterize succinctly the toxicological and adverse health effects information for the hazardous substance being described. Each profile identifies and reviews the key literature (that has been peer-reviewed) that describes a hazardous substance's toxicological properties. Other pertinent literature is also presented but 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.

Foreword

Each toxicological profile begins with a public health statement, which describes in nontechnical language a substance's relevant toxicological properties. Following the public health statement is information concerning significant health effects associated with exposure to the substance. The adequacy of information to determine a substance's health effects is described. Data needs that are of significance to protection of public health will be identified by ATSDR, the National Toxicology Program (NTP) of the Public Health Service, and EPA. The focus of the profiles is on health and toxicological information; therefore, we have included this information in the beginning of the document.

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 our assessment of all relevant toxicological testing and information that has been peer reviewed. It has been reviewed by scientists from ATSDR, the Centers for Disease Control, the NTP, and other federal agencies. It has also been reviewed by a panel of nongovernment peer reviewers and is being made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.



William L. Roper, M.D., M.P.H.
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Disease Registry

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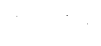
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1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about radon and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,177 sites on its National Priorities List (NPL). Radon has been found above background levels at five of these sites. However, we do not know how many of the 1,177 NPL sites have been evaluated for radon. As EPA evaluates more sites, the number of sites at which radon is found may change. The information is important for you because plutonium may cause harmful health effects and because these sites are potential or actual sources of human exposure to radon.

When a radioactive chemical 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 as a radioactive chemical. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous substance such as radon, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS RADON?

Radon is a naturally occurring colorless, odorless, tasteless radioactive gas that is formed from the normal radioactive decay of uranium. Uranium is present in small amounts in most rocks and soil. It slowly breaks down to other products such as radium, which breaks down to radon. Some of the radon moves to the soil surface and enters the air, while some remains below the soil surface and enters the groundwater (water that flows and collects underground). Uranium has been around since the earth was formed and has a very long half-life (4.5 billion years), which is the amount of time required for one-half of uranium to break down. Uranium, radium, and thus radon, will continue to exist indefinitely at about the same levels as they do now.

Radon also undergoes radioactive decay and has a radioactive half-life of about 4 days. This means that one-half of a given amount of radon will be changed or decayed to other products every 4 days. When radon decays, it divides into two parts. One part is called radiation, and the second part is called a daughter. The daughter, like radon, is not stable; and it also divides into radiation and another daughter. Unlike radon, the daughters are metal and easily attach to dust and other particles in the air. The dividing

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of daughters continues until a stable, nonradioactive daughter is formed. During the decay process, alpha, beta, and gamma radiations are released. Alpha particles can travel only a short distance and cannot go through your skin. Beta particles can penetrate your skin, but they cannot go all the way through your body. Gamma radiation, however, can go all the way through your body. Thus there are several types of decay products that result from radon decay. You will find more information about the physical and chemical properties of radon in Chapter 3, about its uses in Chapter 4, and about your potential for exposure in Chapter 5.

1.2 HOW MIGHT I BE EXPOSED TO RADON?

Since radon is a gas and radon daughters are often attached to dust, you are exposed to them primarily by breathing them in. They are present in nearly all air. However, background levels of radon in outdoor air are generally quite low, about 0.003 to 2.6 picocuries of radon per liter of air. A picocurie is a very small amount of radioactivity equal to one quintillionth ($1/10^{18}$) of an ounce of radon. In indoor locations, such as homes, schools, or office buildings, levels of radon and daughters are generally higher than outdoor levels. Indoor radon levels are generally about 1.5 picocuries radon per liter of air. Cracks in the foundation or basement of your home may allow increased amounts of radon to move into your home. You may also be exposed to radon and daughters by drinking water obtained from wells that contain radon. Average levels of radon in groundwater are about 350 picocuries of radon per liter of water. However, most radon in water is rapidly released into the air and can be breathed in. In some areas of the country the amount of uranium and radium in some rock types, such as phosphate or granite, is high. In these areas radon levels in outdoor air or in groundwater will generally be higher. You will find more information on exposure to radon in Chapter 5.

1.3 HOW CAN RADON ENTER AND LEAVE MY BODY?

Radon and its radioactive daughters can enter your body when you breathe them in or swallow them. By far, the greater amounts are breathed in. Most of the radon is breathed out again. However, some radon and most of the daughters remain in your lungs and undergo radioactive decay. The radiation released during this process passes into lung tissue and is the cause of lung damage. Some of the radon that you swallow with drinking water passes through the walls of your stomach and intestine. After radon enters your blood stream most (greater than 90%) of the radon goes to the lungs where you breathe most of it out. This occurs very shortly after it is taken in. Any remaining radon undergoes decay. Radon that does not go to the lungs goes to other organs and fat where it may remain and undergo decay. There is very limited information on whether radon gas can penetrate the skin, but some radon may be able to pass through the skin when you bathe in water containing radon. You will find more information on behavior of radon in the body in Chapter 2.

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1.4 HOW CAN RADON AFFECT MY HEALTH?

Long-term exposure to radon and radon daughters in air increases your chances of getting lung cancer. When exposures are high, noncancer diseases of the lungs may occur, such as thickening of certain lung tissues. While noncancer health effects may occur within days or weeks after exposure to radon, it will be several years before cancer effects become apparent. This is known from studies of workers exposed to radon in mines, primarily uranium miners, and from tests on laboratory animals. Although radon is radioactive, it gives off little gamma radiation. Therefore, harmful health effects from external exposure (when the chemical does not come into direct contact with your body) are not likely to occur. In addition, it is not known if radon causes health effects other than to the lung. Also, the effects of drinking water or eating food containing radon are not known. You will find more information on the health effects of radon in Chapter 2.

1.5 WHAT LEVELS OF EXPOSURE HAVE RESULTED IN HARMFUL HEALTH EFFECTS?

In studies of uranium miners, workers exposed to radon levels of 50 to 150 picocuries of radon per liter of air for about 10 years have shown an increased frequency of lung cancer. Although there is some uncertainty as to how much exposure to radon increases your chances of getting lung cancer, the greater your exposure to radon, the greater your chance of developing lung cancer. Even small exposures may increase your risk of developing lung cancer, especially if you smoke cigarettes. Tables 1-1 and 1-2 were derived from animal and human data for short-term or long-term exposure, as described in Chapter 2 and in Table 2-1. This information provides a basis for comparison to radon levels that you might encounter in the air. As seen in Tables 1-3 and 1-4, there is no information on the effects of radon if you drink water or eat food containing radon.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO RADON?

Radon in human tissues is not detectable by routine medical testing. However, several of its decay products can be detected in urine and in lung and bone tissue. These tests, however, are not generally available to the public and are of limited value since they cannot be used to accurately determine how much radon you were exposed to, nor can they be used to predict whether you will develop harmful health effects. You will find more information on methods used to investigate levels of radon in Chapters 2 and 6.

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

EPA recommends that all homes should be monitored for radon. If testing shows levels less than 4 picocuries radon per liter of air, then no action is necessary. For levels above this, follow-up measurements should be taken. If

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TABLE 1-1. Human Health Effects from Breathing Radon*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of humans to air containing specific levels of radon are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (pCi/L)</u>	<u>Length of Exposure</u>	<u>Description of Effects**</u>
100	Occupational (10 years)	Severe lung damage.

*See Section 1.2 for a discussion of exposures encountered in daily life.

**These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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TABLE 1-2. Animal Health Effects from Breathing Radon

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air (pCi/L)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
2.2x10 ⁸	1 day	Death in mice
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (pCi/L)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
2.6x10 ⁵	Life	Damage to lung tissue in hamsters.
5.5x10 ⁵	50 days	Lung damage in dogs.
4.8x10 ⁶	Life	Abnormal growth of cells in lung in rats.

*These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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TABLE 1-3. Human Health Effects from Eating or Drinking Radon*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of humans to food containing specific levels of radon are not known.
<u>Levels in Water</u>		The health effects resulting from short-term exposure of humans to water containing specific levels of radon are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of humans to food containing specific levels of radon are not known.
<u>Levels in Water</u>		The health effects resulting from long-term exposure of humans to water containing specific levels of radon are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

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TABLE 1-4. Animal Health Effects from Eating or Drinking Radon

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of animals to food containing specific levels of radon are not known.
<u>Levels in Water</u>		The health effects resulting from short-term exposure of animals to water containing specific levels of radon are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of animals to food containing specific levels of radon are not known.
<u>Levels in Water</u>		The health effects of long-term exposure of animals to water containing specific levels of radon are not known.

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follow-up levels are 20 picocuries radon per liter of air or higher, the home owner should consider some type of procedure to decrease indoor radon levels. The Mine Safety and Health Administration (MSHA) uses a standard of 4 Working Level Months (WLM) per year for people who work in mines. (Working Level Months combine the amount with length of exposure.) You will find more information on guidelines and standards in Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your State Health or Environmental Department or:

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

This agency can also give you information on the location of the nearest occupational and environmental health clinics. Such clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.

2. HEALTH EFFECTS

2.1 INTRODUCTION

This chapter contains descriptions and evaluations of studies and interpretation of data on the health effects associated with exposure to radon. Its purpose is to present levels of significant exposure for radon based on toxicological studies, epidemiological investigations, and environmental exposure data. This information is presented to provide public health officials, physicians, toxicologists, and other interested individuals and groups with (1) an overall perspective of the toxicology of radon and (2) a depiction of significant exposure levels associated with various adverse health effects.

Radon is a relatively inert noble gas that does not readily interact chemically with other elements. However, radon is a radioactive element and evaluation of the adverse health effects due to exposure to radon requires a slightly different approach than other chemicals. Radioactive elements are those that undergo spontaneous transformation (decay) in which energy is released (emitted) either in the form of particles, such as alpha and beta particles, or photons, such as gamma or X-ray. This disintegration or decay results in the formation of new elements, some of which may themselves be radioactive, in which case they will also decay. The process continues until a stable (nonradioactive) state is reached (See Appendix B for more information).

The decay rate or activity of radioactive elements has traditionally been specified in curies (Ci). The activity defines the number of radioactive transformations (disintegrations) of a radionuclide over unit time. The curie is approximately 37 billion disintegrations (decay events) per second (3.7×10^{10} transformations per second). In discussing radon, a smaller unit, the picocurie (pCi) is used, where 1 pCi is equal to 1×10^{-12} Ci. In international usage, the S.I. unit (the International System of Units) for activity is the Becquerel (Bq), which is equal to one disintegration per second or about 27 pCi. (Information for conversion between units is given in Chapter 9.) In the text of this profile, units expressed in pCi are followed by units in Bq contained in parentheses. The activity concentration of radon or another radionuclide in air is expressed in Ci/liter (L) of air (Bq/m^3). The activity concentration is a description of the exposure rather than the dose. In radiation biology the term dose refers specifically to the amount of radiant energy absorbed in a particular tissue or organ and is expressed in rad (or grays).

When radon decays, it and its daughters (decay products) emit alpha and beta particles as well as gamma radiation. However, the health hazard from radon does not come primarily from radon itself, but rather from the radioactive products formed in the decay of radon. These products, called "radon daughters" or "radon progeny," are also radioactive (See Chapter 3 for more information on the chemical and physical properties of radon). Unlike

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radon, the radon daughters are heavy metals and readily attach themselves to whatever they contact. The main health problems arise when radon daughters or dust particles carrying radon daughters are inhaled. Radon daughter particles attach to lung tissue and decay, resulting in the deposition of radiation (in the form of alpha particles) in the lung tissue.

Because it was not feasible to routinely measure the individual radon daughters, a unit termed the "Working Level" (WL) was introduced by the U.S. Public Health Service. The WL unit is a measure of the amount of alpha radiation emitted from the short-lived radon daughters (polonium-218, polonium-214, and lead-214) and represents any combination of short-lived radon progeny in one liter of air that will release 1.3×10^5 million electron volts (MeV) of alpha energy during decay. One WL is equivalent to 2.08×10^5 joules per cubic meter of air (J/m^3).

To convert between units of radon-222 radioactivity (Ci or Bq) and the potential alpha energy concentration (WL or J/m^3), the equilibrium between radon gas and radon daughters must be known (See Chapter 9 for conversion formula). When radon is in equilibrium with its progeny, that is, when each of the short-lived radon daughters is present at the same activity concentration in air as radon-222, then 1 WL equals 100 pCi radon-222/L of air. However, when removal processes other than radioactive decay are operative, such as with ventilation, the concentration of short-lived daughters will be less than the equilibrium amount. In such cases an equilibrium factor (F) is applied. For example, if the equilibrium factor is 0.5, then 200 pCi radon-222/L of air is equivalent to 1.0 WL; if the equilibrium factor is 0.3, then 1 WL corresponds to 333 pCi radon-222/L of air.

An additional unit of measurement used to describe human exposure to radon and radon progeny is the Working Level Month (WLM), which expresses both the intensity and duration of exposure. One WLM is defined as the exposure of a person to radon progeny at a concentration of 1.0 WL for a period of 1 working month (WM). A working month is assumed to be 170 hours. The S.I. unit for WLM is joule-hour per cubic meter of air ($\text{J}\cdot\text{h}/\text{m}^3$); 1 WLM is equal to $3.6 \times 10^3 \text{ J}\cdot\text{h}/\text{m}^3$.

The WL and the WLM have been used to describe human exposure in occupational settings for uranium and other hard rock miners. Since the WLM represents both the intensity and duration of exposure, it alone does not provide enough information to determine the actual activity concentrations of radon in the air. For example, exposure to radon and radon daughters at 1 WL (100 pCi radon-222/L of air) for 100 working months (WM) results in a cumulative dose of 100 WLMs; exposure to 100 WL (10,000 pCi radon-222/L of air) for 1 WM also results in a cumulative dose of 100 WLMs

For both human and animal studies, exposures expressed in WLs were converted to pCi radon-222/L of air. The unit of activity, the curie (or Becquerel), is the appropriate unit to describe radon levels in the

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environment, Unless otherwise stated by the authors of the studies reviewed, the equilibrium factors assumed for the conversion of WLs to pCi were 1.0 for animal studies and 0.5 for occupational epidemiological studies. For several of the epidemiological studies, exposure categories were expressed in WLMs without specific information concerning duration of radon exposure; therefore, for these studies dose conversions were not made. In this text and in the Supplemental Document, whenever possible radon levels are expressed in activity concentrations of pCi/L of air, pCi/kg of body weight, or pCi/L of water (along with the corresponding units in Becquerels).

Radon-222 is a direct decay product of radium-226, which is part of the decay series that begins with uranium-238 (see Chapter 3, Figure 3-1). Thorium-230 and thorium-234 are also part of this decay series. Uranium, thorium, and radium are the subject of other ATSDR Toxicological Profiles. Other isotopes of radon, such as radon-219 and radon-220, are formed in other radioactive decay series. However, radon-219 usually is not considered in the evaluation of radon-induced health effects because it is not abundant in the environment (Radon-219 is part of the decay chain of uranium-235, a relatively rare isotope) and has an extremely short half-life (4 seconds). Radon-220 is also usually not considered when evaluating radon-related health effects. While the average rate of production of radon-220 is about the same as radon-222, the amount of radon-220 entering the environment is much less than that of radon-222 because of the short half-life of radon-220 (56 seconds). All discussions of radon in the text refer to radon-222.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals address the needs of persons living or working near hazardous waste sites, the data in this section are organized first by route of exposure -- inhalation, oral, and dermal -- and then by health effect -- death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods -- acute, intermediate, and chronic.

Levels of significant exposure for each exposure route and duration (for which data exist) 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 levels of exposure used in the studies. LOAELs have been classified into "less serious" or "serious" effects. These distinctions are intended to help the users of the document identify the levels of exposure at which adverse health effects start to appear, determine whether or not the intensity of the effects varies 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 on the tables and figures may differ depending on the user's perspective. For example, physicians concerned with the interpretation of clinical findings in exposed persons or with the identification of persons with the potential to develop such disease

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may be interested in levels of exposure associated with "serious" effects. Public health officials and project managers concerned with response actions at Superfund sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAEL) or exposure levels below which no adverse effects (NOAEL) have been observed.

For certain chemicals, levels of exposure associated with carcinogenic effects may be indicated in the figures. These levels reflect the actual doses associated with the tumor incidences reported in the studies cited.

2.2.1 Inhalation Exposure

Levels of significant exposure for the inhalation route for acute, intermediate, and chronic exposure duration (for which data exist) are presented in Table 2-1 and illustrated in Figure 2-1.

2.2.1.1 Death

No deaths in humans have been reported as the result of acute radon exposure. However, several epidemiological studies of individuals exposed over long periods have reported significant increases in early mortality due to cancer and nonneoplastic (noncancer) diseases (see Section 2.2.1.8 for a discussion on cancer). Descriptions of cancer mortality were presented by exposure categories, i.e., WLM categories; however, deaths due to noncancer respiratory effects were generally reported for the total cohort. Increased mortality as a result of nonneoplastic respiratory diseases, such as emphysema and pulmonary fibrosis, has been reported in United States uranium miners exposed to radon and radon daughters at concentrations in the range of 100 to 10,000 pCi radon-222/L of air (3,700 to 370,000 Bq/m³) (Lundin et al. 1971; Waxweiler et al. 1981). The concentration of radon and radon daughters in mine air was reported to result in cumulative exposures of from 50 WLM to levels equal to or greater than 3,720 WLM. The incidence of mortality from respiratory diseases other than cancer and tuberculosis has been reported for uranium miners and related to cumulative exposure expressed in WLMs (Archer et al. 1976). As exposure increased, the number of cases per 1,000 individuals exposed also increased. However, there are a number of confounding factors to consider in all of these studies, including exposure to other agents, ethnicity, smoking history, and work experience. The cases of nonneoplastic respiratory diseases reported in these miners cannot be attributed solely to radon or radon daughters but may be due to exposure to silica, to other mine pollutants, to smoking, or to other causes.

In a more recent study (Roscoe et al. 1989) mortality from nonmalignant respiratory disease was reported for a cohort of white nonsmoking uranium miners. Deaths from these diseases were twelve times higher in uranium miners than in nonsmoking United States veterans. Causes of death in the cohort included silicosis, chronic obstructive pulmonary disease, fibrosis, and emphysema. However, the exposure history of the individuals having these

TABLE 2-1. Levels of Significant Exposure to Radon - Inhalation

Figure Key	Species	Exposure Frequency/ Duration	Effect	NOAEL (pCi/L)	LOAEL (Effect)		Reference
					Less Serious (pCi/L)	Serious (pCi/L)	
ACUTE EXPOSURE							
Death							
1	Mouse	1d 5-40hr				2.2x10 ⁸ (30 day LD50) ^a	Morken 1955
Systemic							
2	Mouse	1d 5-40hr	Hemato			2.2x10 ⁸ (anemia)	Morken 1955
INTERMEDIATE EXPOSURE							
Death							
3	Rat	4-6mo 2d/wk 1hr/d		3.0x10 ³			Chameaud et al. 1984
4	Rat	lifespan 2d/wk 90hr/wk				4.8x10 ⁶ (dec lifespan)	Palmer et al. 1973
5	Mouse	lifespan 150hr/wk				4.2x10 ⁵ (dec lifespan)	Morken and Scott 1966
6	Hamster	lifespan 2d/wk 90hr/wk				4.8x10 ⁶ (dec lifespan)	Palmer et al. 1973
Systemic							
7	Rat	lifespan 2d/wk 90hr/wk	Resp Other		4.8x10 ⁶ (dec bw)	4.8x10 ⁶ (metaplasia) ^b	Palmer et al. 1973
8	Mouse	lifespan 150hr/wk	Resp Hemato Other		4.2x10 ⁵ (dec lymphocytes) 4.2x10 ⁵ (dec bw)	4.2x10 ⁵ (metaplasia)	Morken and Scott 1966

TABLE 2-1 (Continued)

Figure Key	Species	Exposure Frequency/ Duration	Effect	NOAEL (pCi/L)	LOAEL (Effect)		Reference
					Less Serious (pCi/L)	Serious (pCi/L)	
9	Mouse	lifespan 2d/wk 90hr/wk	Resp Other		4.8x10 ⁶ (dec bw)	4.8x10 ⁶ (fibrosis)	Palmer et al. 1973
10	Hamster	lifespan 2d/wk 90hr/wk	Resp Other		4.8x10 ⁶ (dec bw)	4.8x10 ⁶ (metaplasia)	Palmer et al. 1973
11	Dog	1-50d 5d/wk 20hr/d	Resp			5.5x10 ⁵ (fibrosis) ^c	Morken 1973
Cancer							
12	Rat	2.5-8wk 4d/wk 3-6hr/d				3.0x10 ⁵ (CEL-lung)	Chameaud et al. 1982
13	Rat	25-115 d 4-5hr/d				7.5x10 ⁵ (CEL-lung)	Chameaud et al. 1974
14	Rat	4-6mo 2d/wk 1hr/d				3.0x10 ³ (CEL-lung)	Chameaud et al. 1984
CHRONIC EXPOSURE							
Death							
15	Hamster	lifespan 5d/wk 6hr/d		3.1x10 ⁵			Pacific Northwest Laboratory 1978
Systemic							
16	Human	>1mo-18yr (occup)	Resp			>1.0x10 ² (tuberculosis)	Waxweiler et al. 1981
17	Hamster	lifespan 5d/wk 6hr/d	Resp Hemato Other	3.1x10 ⁵	2.6x10 ⁵ (dec bw)	2.6x10 ⁵ (hyperplasia) ^d	Pacific Northwest Laboratory 1978

TABLE 2-1 (Continued)

Figure Key	Species	Exposure Frequency/ Duration	Effect	NOAEL (pCi/L)	LOAEL (Effect)		Reference
					Less Serious (pCi/L)	Serious (pCi/L)	
Cancer							
18	Human	0.5-23yr (occup)				3.4x10 ² (CEL-lung)	Gottlieb and Husen 1982
19	Human	(occup)				2.0x10 ² (CEL-lung)	Morrison et al. 1981
20	Human	0-14yr (occup)				1.0x10 ² (CEL-lung) ^e	Solli et al. 1985
21	Human	>29yr (occup)				6.0x10 ¹ (CEL-lung)	Edling and Axelson 1983
22	Human	>1yr->20yr (occup)				5.0x10 ¹ (CEL-lung)	Damber and Larsson 1985
23	Human	48wk/yr 48hr/wk (occup)				5.0x10 ¹ (CEL-lung)	Howe et al. 1987
24	Human	>10yr (occup)				>3.0x10 ¹ (CEL-lung)	Snihs 1974
25	Human	>2-30yr (res)				1.5x10 ⁰ (CEL-lung)	Svensson et al. 1987
26	Human	(occup)				2.4x10 ² (CEL-lung)	Fox et al. 1981
27	Human	>1mo-18yr (occup)				1.0x10 ² (CEL-lung)	Waxweiler et al. 1981
28	Human	>1mo-30yr (occup)				4.0x10 ² (CEL-lung)	Roscoe et al. 1989

^a2.2x10⁸ presented in Table 1-2.

^b4.8x10⁶ presented in Table 1-2.

^c5.5x10⁵ presented in Table 1-2.

^d2.6x10⁵ presented in Table 1-2.

^e100 presented in Table 1-1.

NOAEL=no-observed-adverse-effect level; LOAEL=lowest-observed-adverse-effect level; pCi/L=picocurie per liter; d=day; hr=hour; wk=week; mo=month; CEL=Cancer Effect Level; yr=year; hemato=hematological; resp=respiratory; occup=occupational; dec=decreased; bw=body weight; res=residential

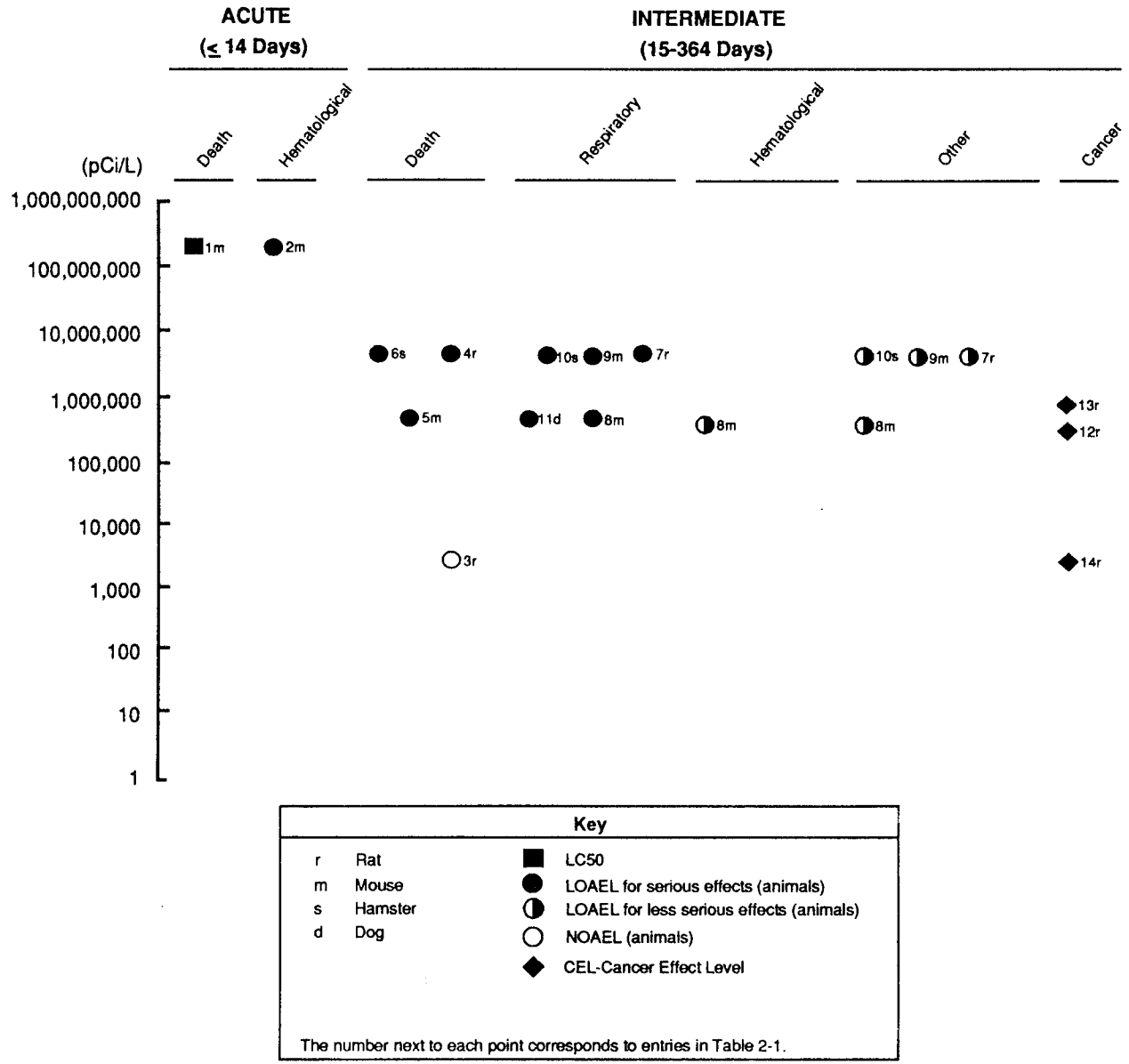


FIGURE 2-1. Levels of Significant Exposure to Radon - Inhalation

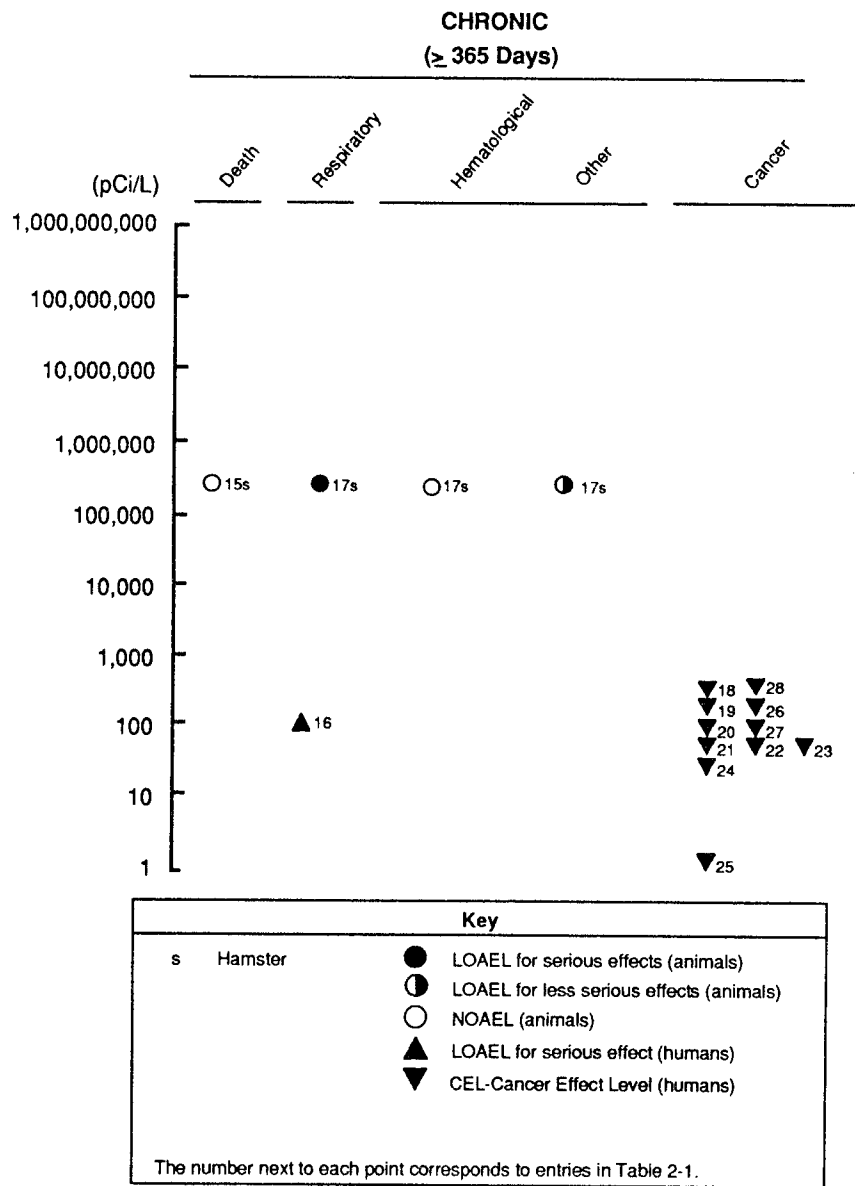


FIGURE 2-1 (Continued)

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diseases was not reported. Excepting cigarette smoking, this study has all of the confounding factors mentioned previously.

Mortality due to nonneoplastic respiratory diseases was not significantly elevated in other uranium mining cohorts including miners in Czechoslovakia (Sevc et al. 1988) or Ontario, Canada (Muller et al. 1985). Although environmental radon levels were not reported in either the Czechoslovakian or Canadian studies, cumulative occupational exposure to radon and radon daughters were estimated at levels up to about 600 WLMs. A statistically significant excess of mortality due to chronic nephritis and renal sclerosis was also reported in the United States uranium miner cohort, although it is unclear whether this was related to exposure to radon, uranium ore, or other mining conditions or to nonmining factors (Waxweiler et al. 1981).

The acute lethal effects of radon and radon daughters have been studied in mice. A 30-day LD₅₀ was estimated based on single exposures via inhalation to radon and radon daughters at a concentration of 2.2×10^8 pCi/L (8.1×10^9 Bq/m³) for 5 to 40 hours (Morken 1955). After 40 hours of exposure, 100% of the exposed mice died within 2 weeks (cause of death was not reported), while no deaths occurred within 60 days following an exposure of 26 hours or less.

Significant decreases in the lifespan of laboratory animals exposed to high doses of radon and radon daughters were reported by several investigators (Cross 1987; Morken 1973; Morken and Scott 1966; Palmer et al. 1973). Respiratory system insult contributed to the death of treated animals in these studies, although the actual cause of death was not reported. The lifespan of male and female mice (median lifespan of controls was 79 and 98 weeks) was reduced by 55% and 42%, respectively, as a result of continuous exposure (150 hours/week) to 4.2×10^5 pCi radon-222/L of air (1.6×10^7 Bq/m³) for up to 47 weeks (Morken and Scott 1966). Emaciation, reddening of the ears, and abnormal grooming was observed preceding death. Pseudoparalysis was observed in mice which died a few days after exposure (Morken and Scott 1966). A similar decrease in lifespan was observed in rats and hamsters following exposure to 4.8×10^5 pCi radon-222/L of air (1.8×10^8 Bq/m³) for 90 hours/week (Palmer et al. 1973). All animals in the Palmer et al. (1973) study died by the fourth month of treatment, while all treated animals in the Morken and Scott (1966) study died by the eleventh month. At lower concentrations (3,000 pCi radon-222/L of air [1.1×10^5 Bq/m³] for 2 hours/week, 6 months) the lifespan of rats was not decreased (Chameaud et al. 1984).

2.2.1.2 Systemic Effects

No studies were located regarding cardiovascular, gastrointestinal, musculoskeletal, hepatic, dermal, or ocular effects in humans or animals after inhalation exposure to radon and radon daughters.

Respiratory Effects. Adverse respiratory effects have been observed in humans under occupational conditions and in laboratory animals exposed to

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radon and radon daughters. Epidemiology studies of miner cohorts report an increased frequency of chronic, nonneoplastic lung diseases, such as emphysema and pulmonary fibrosis, among uranium miners in the United States (Lundin et al. 1971; Roscoe et al. 1989; Waxweiler et al. 1981) and among Cornish tin miners (Fox et al. 1981), and chronic interstitial pneumonia among Canadian uranium miners (Muller et al. 1985). Chronic lung disease was reported to increase with increasing cumulative exposure to radiation and with cigarette smoking (Archer 1980). In addition, nonsmoking uranium miners were also reported to have increased deaths from nonmalignant respiratory disease compared to a nonsmoking United States veteran cohort (Roscoe et al. 1989).

Alterations in respiratory function in United States uranium miners have been reported (Archer et al. 1964; Samet et al. 1984a; Trapp et al. 1970). Analyses among United States uranium miners indicated a loss of pulmonary function with increasing cumulative exposure (Archer et al. 1964) and with the duration of underground mining (Samet et al. 1984a). Evaluations of these respiratory end points did not permit assessment of the effects of each of the other possible mine pollutants, such as ore dust, silica, or diesel-engine exhaust. The individual contributions of these factors to the observed adverse respiratory effects are not defined.

No studies were located regarding the respiratory effects of radon and radon daughters in laboratory animals following acute exposure. Respiratory toxicity occurred in mice, hamsters, dogs, and rats following exposure to radon and radon daughters for intermediate exposure durations. Chronic inflammation (radiation-induced pneumonitis), pneumonia, and/or fibrosis of varying degrees in the alveolar region occurred in most animals exposed to radon and radon daughters (4.2×10^5 to 4.8×10^6 pCi radon-222/L of air [1.6×10^7 to 1.8×10^8 Bq/m³]) for 4 to 150 hours/week for 10 to approximately 45 weeks (Chaumeaud et al. 1974; Morken 1973; Morken and Scott 1966; Palmer et al. 1973). In these studies, the relationship of dose, temporal dosing pattern, and length of exposure to onset of effects is unclear since the time of onset of effects was rarely reported or effects were reported only when animals died or were sacrificed.

In Palmer et al. (1973), rats, mice, and hamsters, were exposed to radon [4.8×10^8 pCi radon/L of air (1.8×10^5 Bq/m³)] via inhalation for approximately 90 hours per week, in two continuous 45-hour periods. These animals were allowed to die, or were sacrificed when moribund, after which they were histopathologically examined. At four months of exposure, only one of the rodents remained alive. The radiation effects observed in these animals, which included interstitial pneumonitis or septal fibrosis, were found at post-mortem examination. Therefore the onset of respiratory effects could not be determined.

In a study conducted by Morken and Scott (1966), mice were to be exposed to 4.2×10^5 pCi radon/L of air (1.6×10^7 Bq/m³) 150 hours/week for life. However, by week 15 of the experiment the median lifetime of the animals had been decreased by 50%. However, the cause of this decreased lifespan -was not

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reported. The authors then sacrificed the remaining animals (15 treated mice and 3 control mice) for purposes of histopathological examination. Tracheal effects, including thickening of the mucous membrane, inflammation of the mucous glands, and destruction of cells lining the trachea, were observed. However, the onset of these effects could not be determined. In Morken (1973) 9 mice and dogs were exposed to radon for intermediate periods of time and then sacrificed at designated times post-exposure. In mice exposed to 5.5×10^5 pCi radon/L of air (2.0×10^7 Bq/m³) for 10, 15, 20, or 25 weeks, lesions of the trachea and bronchi were observed immediately post-exposure, but by eight weeks post-exposure tissues appeared normal. However, with increasing time post-exposure, the epithelial lining of the terminal bronchiole became flattened or disappeared. At long intervals after exposure to radon for 25 weeks, non-specific pulmonary effects, including small foci of interstitial fibrosis, were observed in mice. In dogs exposed to radon for one to 50 days [5.5×10^5 pCi of radon/L of air (2.0×10^7 Bq/m³)], no significant effects were observed in treated dogs immediately post-exposure compared to untreated controls. At one and two years post-exposure, there was a "probable increasing relation" to dose of small foci of chronic inflammation. At three years post-exposure, this relation had disappeared in dogs exposed to low doses of radon up to 800 WLM, but was still considered "probable" for the larger doses. However, a definite time of onset of respiratory effects in either mice or dogs could not be determined from the results of this study.

Respiratory effects similar to those observed following intermediate exposure have also been observed in laboratory animals following chronic exposure to radon and radon daughters. Respiratory lesions, mainly squamous metaplasia, were observed in the bronchioalveolar region of hamsters 8 months following initiation of lifetime exposure to 2.6×10^5 pCi radon-222/L of air (9.6×10^6 Bq/m³) for 30 hours/week (Pacific Northwest Laboratory 1978).

Pulmonary fibrosis in rats, hamsters, and dogs and emphysema in hamsters and dogs occurred following exposure to radon and radon daughters and uranium ore dust (Cross et al. 1984, 1985, 1986; Pacific Northwest Laboratory 1978). In hamsters emphysema was produced as a result of exposure to uranium ore alone, diesel exhaust alone, and radon and radon daughters alone; however, emphysema was not observed in hamsters at cumulative doses of radon of less than 7,000 WLM (Pacific Northwest Laboratory 1978). Fibrosis occurred in hamsters following exposure to radon and radon daughters at a cumulative dose of 8,000 WLM in combination with uranium ore and diesel exhaust, but not with radon and radon daughters alone at cumulative exposure at approximately 7,000 WLM. However, the incidence of bronchial hyperplasia was significantly greater in hamsters receiving radon and radon daughters alone. In dogs the combination of uranium ore dust and radon and radon daughters produced more severe emphysema and fibrosis than uranium ore dust alone; however, radon and radon daughters alone were not tested in dogs (Pacific Northwest Laboratory 1978). Fibrosis, but not emphysema, was observed in rats exposed to radon and radon daughters and uranium ore dust (Cross et al. 1984, 1985). These studies are discussed further in Section 2.6.

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Renal Effects. A statistically significant increase in mortality due to kidney disease, characterized by chronic nephritis and renal sclerosis, was reported among United States uranium miners (Waxweiler et al. 1981) and in Canadian miners at the Eldorado mines (Muller et al. 1985). Kidney toxicity has been induced experimentally in animals exposed to uranium (ATSDR 1990a). Kidney disease was not reported among other mining cohorts and no studies were located regarding renal effects in laboratory animals following inhalation exposure to radon. It is not clear whether the kidney effects observed by Waxweiler were due to radon, uranium ore, or other mining and nonmining factors.

Hematological Effects. No studies were located regarding hematological effects in humans after inhalation exposure to radon.

Hematological effects have been observed in mice following acute and chronic exposure to radon and radon daughters. The extent and severity of the hematological effects in mice were exposure related. Effects following acute exposure, either a single or multiple exposures, were transient. Recovery to control values occurred within a shorter time post-exposure after a single acute exposure than with multiple exposures. Chronic exposure of mice to radon-222 resulted in dose-related alterations to the hematological system.

Following a single exposure to mice of 1.76×10^8 pCi radon-222/L of air (6.5×10^9 Bq/m³), transient decreases in erythrocytes, reticulocytes, platelets, and white blood cells were observed immediately post-exposure (Morken 1961). Platelets and white blood cells returned to control levels by 50 days post-exposure, and reticulocytes increased 50% to 100% over controls within 2 to 3 weeks, but returned to normal about one year after exposure. Erythrocyte counts remained depressed for one-year post-exposure (Morken 1961). In mice exposed 2 or 4 times at concentrations of 2.11×10^8 pCi radon-222/L of air (7.8×10^9 Bq/m³), erythrocyte counts remained depressed compared to controls, while platelets and neutrophils rapidly decreased and then recovered within 2 weeks (Morken 1964). After multiple exposures, lymphocyte counts remained lower for longer periods of time compared to single exposures, indicating that recovery was affected by larger or repeated doses (Morken 1964). These effects are based on results observed in small numbers of animals.

In mice, lifetime exposure to 4.2×10^5 pCi radon-222/L of air (1.6×10^7 Bq/m³), 150 hours/week resulted in mild, progressive anemia in male mice and a decrease in lymphocyte count in male and female mice, which was linearly related to cumulative dose as expressed in working level months (WLMs) (Morken and Scott 1966). However, no hematological effects were observed in hamsters exposed to 3.1×10^5 pCi radon-222/L of air (1.1×10^7 Bq/m³) (Pacific Northwest Laboratory 1978).

Other Systemic Effects. Exposure to radon and radon daughters at concentrations ranging from 2.6×10^5 to 4.8×10^6 pCi radon-222/L of air (9.6×10^6

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to 1.8×10^8 Bq/m³), 30 to 150 hours/week, resulted in a significant decrease in body weight in hamsters (Pacific Northwest Laboratory 1978), mice (Morken and Scott 1966; Palmer et al. 1973), and rats (Palmer et al. 1973). There was no explanation given for these weight losses and food consumption was not reported in any of the studies.

2.2.1.3 Immunological Effects

No studies were located regarding immunological effects in humans and animals after inhalation exposure to radon.

2.2.1.4 Neurological Effects

No studies were located regarding neurological effects in humans after inhalation exposure to radon. Two guinea pigs exposed to approximately 4.7×10^{10} to 5.8×10^{10} pCi (1.7×10^9 to 2.15×10^9 Bq) radon for 1 to 2% hours became drowsy, their respiration increased, and after several hours, they died (Proescher 1913). Autopsy showed that both animals died from respiratory paralysis caused by central nervous system failure. The study has many limitations, such as the use of only two animals and the possibility that oxygen deprivation contributed to the respiratory failure. A causal relationship between central nervous system failure and radon exposure was not established.

2.2.1.5 Developmental Effects

No studies were located regarding developmental effects in humans and animals after inhalation exposure to radon.

2.2.1.6 Reproductive Effects

No maternal or fetal reproductive effects in humans have been attributed to exposure to radon and radon daughters. However, a decrease in the secondary sex ratio (males:females) of the children of male underground miners may be related to exposure to radon and radon daughters (Dean 1981; Muller et al. 1967; Wiese and Skipper 1986). The secondary sex ratio of the first born children of uranium miners was decreased with cumulative exposure to radon and radon daughters in miners whose median age at the time of conception was less than 25 years of age but was increased with cumulative exposure to radon and radon daughters in miners whose median age at the time of conception was greater than 25 years of age (Waxweiler and Roscoe 1981). This age effect was also observed when the miners were analyzed according to race.

No studies were located regarding reproductive effects in animals following inhalation exposure to radon and radon daughters.

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2.2.1.7 Genotoxic Effects

Some epidemiologic studies have indicated that radon and radon daughters may produce genotoxic effects in persons exposed in occupational and environmental settings. Brandom et al. (1978) reported a higher incidence of chromosomal aberrations among uranium miners exposed to radon and radon daughters at cumulative exposures ranging from <100 to >3,000 WLM, as compared to their matched controls. A clear exposure-related increase was observed for the groups exposed to 770 to 2,890 WLM with a sharp decrease at the highest dose group (>3,000 WLM). The cause of the reversal in exposure-response at the highest dose is unclear. Increases in chromosomal aberrations were also reported among spa-house personnel and in area residents in Badgastein, Austria, who were chronically exposed to radon and radon decay products present in the environment (Pohl-Rfiling and Fischer 1979, 1982; Pohl-Rtiling et al. 1976). A study by Tuschl et al. (1980) indicated a stimulating effect of repeated low-dose irradiation on DNA-repair in lymphocytes of persons occupationally exposed to radon (3,000 pCi/L of air [1.1×10^5 Bq/m³]). The study further indicated higher DNA-repair rates in juvenile cells than in fully differentiated cells.

Evidence of chromosomal aberrations was equivocal in an animal study. Rabbits exposed to high natural background levels of radon-222 (12 WLM) for over 28 months displayed an increased frequency of chromosomal aberrations (Leonard et al. 1981). However, when a similar study was conducted under controlled conditions (10.66 WLM), chromosomal aberrations were not found. According to the authors, the increased chromosomal aberrations in somatic cells of rabbits exposed to natural radiation were mainly due to the gamma radiation from sources other than radon.

Exposure of Sprague-Dawley male rats to radon at cumulative doses as low as 100 WLM resulted in an increase in sister chromatid exchanges (SCEs) in bone marrow by 600 days post-exposure (Poncy et al. 1980). At 750 days postexposure, the number of SCEs reached 3.21 per cell. The SCEs in the 500 and 3,000 WLM groups reached constant values of 3.61 and 4.13 SCEs per cell. In the high-dose group (6,000 WLM), SCEs continued to increase from 100 to 200 days after exposure, reaching a mean value of 3.5 SCE per cell. In controls SCEs were constant with age (2.4 per cell).

2.2.1.8 Cancer

Significant excesses in deaths from lung cancer have been identified in epidemiology studies of uranium miners and other hard rock miners. Statistically significant excesses in lung cancer deaths have been reported in uranium miners in the United States (Archer et al. 1973, 1976, 1979; Gottlieb and Husen 1982; Hornung and Meinhardt 1987; Lundin et al. 1971; Roscoe et al. 1989; Samet et al. 1984b, 1989; Wagoner et al. 1964; Waxweiler et al. 1981), Czechoslovakia (Sevc et al. 1988), and Canada (Howe et al. 1986, 1987; Muller et al. 1985).

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The results of these studies are consistent and demonstrate that the frequency of respiratory cancer mortality increased with increasing exposure to radiation (cumulative WLMs). Statistically significant excesses in lung cancer deaths were present after cumulative exposures of less than 50 WLMs in the Czechoslovakian cohort (Sevc et al. 1988) and at cumulative exposures greater than 100 WLMs in the cohorts from the United States and Ontario, Canada (Muller et al. 1985; Samet et al. 1989; Waxweiler et al. 1981). These studies indicate that lung cancer mortality was influenced by the total cumulative radiation exposure, by the age at first exposure, and by the timecourse of the exposure accumulation. Most deaths from respiratory cancers occurred 10 or more years after the individual began uranium mining (Lundin et al. 1971). Among uranium miners, epidermoid, small cell undifferentiated, and adenocarcinoma were present with increased frequency, while large-cell undifferentiated and other morphological types of lung cancer were seen less frequently (Archer et al. 1974).

The evidence for radon daughter-induced lung cancer is further supported by epidemiological studies conducted among nonuranium hard rock miners. The lung cancer mortality rate was also statistically higher in iron ore miners in Sweden (Damber and Larsson 1982; Edling and Axelson 1983; Jorgensen 1984; Radford and Renard 1984; Snihs 1974); metal miners in the United States (Wagoner et al. 1963); zinc-lead miners in Sweden (Axelson and Sundell 1978); tin miners in England (Fox et al. 1981); phosphate miners in Florida (Checkoway et al. 1985; Stayner et al. 1985); in a niobium mine (Solli et al. 1985); and Newfoundland fluorspar miners (Morrison et al. 1985). In some of these mines, the main source of radon and radon daughters was from radon dissolved in groundwater. Based on measurements of radon concentrations in mine air, significant excesses in lung cancer mortality were reported at concentrations of 30 pCi radon-222/L of mine air (111 Bq/m³) and greater (Snihs et al. 1974). Since exposure was for at least 10 years, the cumulative exposure to workers was approximately 36 WLMs or greater. This excess cancer mortality occurred at cumulative exposures as low as 5 WLMs (Howe et al. 1987) but generally at cumulative doses greater than 100 WLMs.

In a subcohort of 516 white nonsmoking uranium miners (drawn from a larger cohort of United States uranium miners), mean exposure was reportedly 720 WLM. For this cohort the mortality risk for lung cancer was found to be 12-fold greater than that of nonsmoking, nonmining United States veterans. No lung cancer deaths were found in nonsmoking miners who had exposure less than 465 WLMs (Roscoe et al. 1989). Unlike the nonmining cohort, the miners in the subcohort may have been exposed to other mining pollutants, e.g., diesel exhaust and silica dusts. The contribution of these factors was not considered in the analysis.

Several case-control studies have examined the association between lung cancer and housing construction materials, or between lung cancer and residential radon exposure. The majority of these have been conducted in Sweden (Axelson and Edling 1980, Axelson et al. 1979, 1981; Edling 1984; Svensson 1987, 1989). The Axelson studies examined the association between

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housing type and lung cancer risk. Residences of cases (having died from lung cancer) and controls (having died from noncancer causes) were classified into three categories: having lived in wooden house without basements; brick, concrete, or granite houses with basements; and a mixed category (all other types of houses). No radon measurements were taken in these homes. However, previous studies in Sweden had shown that, in general, the wooden structures had lower radon levels than brick or concrete structures. Axelson reported a statistically significant trend for increased lung cancer deaths associated with residence in mixed category houses or in stone houses with basements (Axelson and Edling 1980, Axelson et al. 1979). These studies were adjusted for age and sex, but not for smoking history. An additional study based on the same approach (lung cancer associated with type of residence) did measure radon levels in residences of interest (Edling et al. 1984). Wooden houses without basements had mean levels of 1.1 pCi/L (42 Bq/m³), wooden houses with a basement on radiation producing ground or plaster houses had mean levels of 4.6 pCi/L (170 Bq/m³), while all other types of houses had mean levels of 1.5 pCi/L (57 Bq/m³). Again, the association of incidence of lung cancer, adjusted for age and sex, and additionally for smoking, with type of residence and with radon levels, showed a significantly increasing trend (Axelson et al. 1981, Edling et al. 1984). All of the above studies have one or more methodological limitations, such as small cohort size and limited or no measurement of radon levels in homes.

Another study of a Swedish cohort has also reported significant correlation between incidence of lung cancer, type of residence, and radon exposure, although only 10% of residences were monitored for radon. In addition, it correlates levels of exposure with particular types of lung cancer. Association of exposure with lung cancer, adjusted for smoking, age, and degree of urbanization, was strongest for small cell carcinoma of the lung (Svensson et al. 1989). This particular type of lung tumor has also been reported in cohorts of United States uranium miners.

A study of lung cancer in adult white residents in Maryland reported an association of lung cancer with age, sex, and smoking. Lung cancer rates were highest in houses which had concrete walls and in houses without basements but with concrete slabs, but this association was very slight (Simpson and Comstock 1983).

Identification of specific cancer effect levels, i.e., the environmental concentration of radon and radon daughters, was not feasible for all of the epidemiological studies because of the quality of the exposure information provided. Environmental levels of radon and radon daughters, expressed in pCi radon-222/L of air, present in mines were measured at various times; however, actual measurements of radon and radon daughter levels were not available for every mine and for every year of exposure. Rather, actual measurements along with estimates of radon daughter levels based on extrapolations from actual measurements were then combined with individual work histories to derive estimates of cumulative radon daughter exposure for each individual, reported in WLMs. Workers were then classified into cumulative WLM exposure

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categories. For example, in the United States uranium mining cohort radon and radon daughter levels in mines were measured from 1950 to 1968 and ranged from >100 to >10,000 pCi radon-222/L of air ($>3.7 \times 10^3$ to $>3.7 \times 10^5$ Bq/m³) (>0.5 to >50 WLMs) across various mines and years. Miners were employed in the mines for 4 to 28 years with an average length of employment of 15 years (Saccomanno et al. 1988). The resulting exposure categories ranged from >120 WLM to $\geq 3,720$ WLMs. Only a few of the epidemiological studies provided enough exposure information to express exposures in pCi radon-222/L of air. However, the quality of the exposure measurements does not alter the conclusion that, based on the epidemiology studies, exposures to radon and radon daughters at cumulative doses greater than 100 WLMs resulted in excesses in lung cancer mortality, with the exception of the nonsmoking cohort reported by Roscoe et al. (1989), which reported excesses in lung cancer at higher doses.

No studies were located regarding cancer in laboratory animals following acute exposure to radon and radon daughters. Lung tumors have been observed in rats following intermediate exposure at concentrations as low as 3,000 pCi radon-222/L of air (1.1×10^5 Bq/m³) 2 hours/week for 4 months (Chameaud et al. 1984) and up to 3×10^6 pCi radon-222/L of air (1.1×10^8 Bq/m³) 12 hours/week for 2 weeks (Chameaud et al. 1974, 1982a, 1982b). The mean time to death with tumor in the Chameaud et al. (1984) study was approximately 112 weeks, which is close to the normal lifespan for a rat (104 weeks). In Chameaud et al. (1980)s lung cancers were not observed in rats until the 24th month of the study. These studies would indicate that the latency period for radon-induced lung tumors is long. No treatment-related cancers were observed in dogs, mice, or rats following exposure to radon and radon progeny alone [5.5×10^5 to 1×10^6 pCi radon-222/L of air (2.0×10^7 to 3.7×10^7 Bq/m³)], 25 to 150 hours/week (Morken 1973). In this study, dogs were exposed for 1 to 50 days, mice (three separate experiments) for 8 weeks to life, and rats for 24 weeks. However, the dog study was terminated at 3 years; the rat study only reported results through the twelfth month of the study; and two of the mouse studies had lifespan shortening. Some of the changes observed may have been preneoplastic. However, based on the results from the Chameaud et al. (1980, 1984) studies, lifespan shortening or the early termination of experiments may have precluded the development of tumors. In the remaining mouse study reported by Morken (1973), mice were sacrificed beginning at 60 weeks of age, following exposure to radon for 10, 15, 20, or 25 weeks, at 10 week intervals until all of the mice were killed (110 weeks). No treatment-related cancers were reported. However, reviewers of this study (Cross 1987) report that laboratory room air containing dusts and oil and water droplets may be a confounding factor in this study. The influence of these confounding factors is uncertain, but may have led to a more rapid solubilization of radon progeny causing a decrease in observed lung effects.

In other studies in which a significant increase in the incidence of lung cancer was not reported, the respiratory lesions that were observed following exposure to radon and radon daughters alone were considered by the authors to be "precancerous" (Morken and Scott 1966; Pacific Northwest Laboratory 1978; Palmer et al. 1973). In the Morken and Scott (1966) study, destructive

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hyperplastic and metaplastic lesions were observed in the trachea and bronchioles of mice following exposure to 4.2×10^5 pCi radon-222/L of air (1.6×10^7 Bq/m³) 150 hours/week for life, but no carcinomas were observed. However, there was a significant shortening of lifespan in the study, with many of the animals dead at 35 weeks of age. Because of this lifespan shortening, the animals may not have lived long enough to develop tumors. In a study reported by Palmer et al. (1973), no treatment-related cancers were observed in mice, rats, or hamsters exposed to 4.8×10^5 pCi radon-222/L of air (1.8×10^8 Bq/m³), but precancerous respiratory effects were observed in mice and rats, such as hyperplasia. The lack of cancer may be attributed to the fact that all of the animals but one were dead by the fourth month of the study. However, the cause of death was not reported. In a separate study, "precancerous" respiratory effects (fibrosis) were observed in dogs exposed to 1.1×10^5 pCi radon-222/L of air (4.1×10^6 Bq/m³) (Pacific Northwest Laboratory 1978). The lack of cancer observed in dogs may be due to lifespan shortening (4 years in treated versus 7 years in the normal dog), although the lifespan of untreated controls in this study was comparable.

Following chronic exposure to radon and radon daughters alone, no statistically significant increase in the incidence of any type of tumor was observed in hamsters exposed to 3.1×10^5 pCi radon-222/L of air (1.1×10^7 Wm3> , 30 hours/week for life, although pulmonary fibrosis and bronchial hyperplasia were observed (Pacific Northwest Laboratory 1978). Hamsters may be resistant to alpha radiation-induced lung cancer since no lung tumors were produced in hamsters exposed to another alpha-emitter, plutonium (ATSDR 1990b).

Lung cancer was reported in laboratory animals by Chameaud et al. (1974), Cross et al. (1982a, 1982b, 1984), and Stuart et al. (1970) following chronic administration of radon and radon daughters in conjunction with air pollutants, such as cigarette smoke, uranium ore dusts, or diesel exhaust (see Section 2.6).

2.2.2 Oral Exposure

No studies were located regarding the following health effects in humans or animals after oral exposure to radon and radon daughters.

2.2.2.1 Death

2.2.2.2 Systemic Effects

2.2.2.3 Immunological Effects

2.2.2.4 Neurological Effects

2.2.2.5 Developmental Effects

2.2.2.6 Reproductive Effects

2.2.2.7 Genotoxic Effects

An increase in chromosomal aberrations in lymphocytes was observed in 18 Finnish people of different ages chronically exposed to radon in household

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water at concentrations of 2.9×10^4 to 1.2×10^6 pCi radon-222/L of water (1.1×10^3 to 4.4×10^4 Bq/L) compared with people who did not have a history of exposure to high radon levels (Stenstrand et al. 1979). This study also indicated that the frequencies of chromosomal aberrations and multiple chromosomal breaks were more common in older people than in younger people exposed to radon. Although the radon was in household water, it is probable that much of this radon volatilized and was available to be inhaled. Therefore, this route of exposure includes both oral and inhalation routes.

2.2.2.8 Cancer

Limited information was located regarding cancer in humans after exposure to radon and radon daughters in water. Radon levels were measured in 2,000 public and private wells in 14 counties in Maine (Hess et al. 1983). The county averages were compared to cancer rate by county to determine any degree of correlation. Significant correlation was reported for all lung cancer and all cancers combined, when both sexes were combined, and for lung tumors in females. The authors note that correlation does not demonstrate causation and that confounding factors (e.g., smoking) exist. In addition, exposure from radon in these water supplies could have been by the inhalation route as well as the oral route.

No studies were located regarding cancer in animals after oral exposure to radon and radon daughters.

2.2.3 Dermal Exposure

No studies were located regarding the following health effects in humans or animals after dermal exposure to radon and radon daughters.

2.2.3.1 Death

2.2.3.2 Systemic Effects

2.2.3.3 Immunological Effects

2.2.3.4 Neurological Effects

2.2.3.5 Developmental Effects

2.2.3.6 Reproductive Effects

2.2.3.7 Genotoxic Effects

2.2.3.8 Cancer

A statistically significant increase in the incidence of basal cell skin cancers (103.8 observed vs. 13.0 expected) was observed in uranium miners exposed occupationally for 10 years or more to approximately 3.08 pCi/L of air (1.74×10^2 Bq/m³) resulting in 6.22 pCi (0.23 Bq) radon-222/cm² skin surface area (Sevcova et al. 1978). The authors believe that the causal agent may be exposure to radon and radon daughters. However, they acknowledge that exposure to other agents in the uranium mining environment, as well as minor traumas of the skin, may also play a role in the incidence of skin cancer. Increased incidences of skin cancer have not been reported in other uranium

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miner cohorts or for workers in other types of mining, such as metal or coal mines.

No studies were located regarding cancer in animals after dermal exposure to radon and radon daughters.

2.2.4 Other Routes of Exposure

2.2.4.1 Death

A single intravenous injection of 1.6×10^{10} pCi (6.0×10^8 Bq) radon-222/kg body weight in equilibrium with its decay products resulted in a 56% decrease in the average lifespan of mice (Hollcroft et al. 1955). This decrease was believed to be due to radiation-induced renal failure as indicated by inflammatory lesions and atrophy of the renal cortex as seen in most of the radon treated animals. The study by Hollcroft et al. (1955) has methodological deficiencies including an erratic schedule for sacrifice of animals and the failure to examine animals that died from acute radiation injury. Many other causes of such renal effects are known and the relevance of these effects is questionable following near lethal doses of radon.

2.2.4.2 Systemic Effects

No studies were located regarding respiratory, cardiovascular, gastrointestinal, musculoskeletal, hepatic, or dermal/ocular effects in humans or animals after exposure to radon and radon daughters by other routes of exposure.

Hematological Effects. No studies were located regarding hematological effects in humans after exposure to radon and radon daughters by other routes.

A single intravenous injection of 1.6×10^8 pCi (6.0×10^8 Bq) radon-222/kg body weight in equilibrium with its decay products in mice resulted in a decrease in red blood cell count within 2 weeks, which remained depressed until death of the mice at about 150 to 180 days (Hollcroft et al. 1955). The anemia observed was associated with the observed renal failure in these animals. White blood cell counts were decreased immediately post-exposure, but soon returned to normal levels. (See Section 2.2.4.1 for limitations of Hollcroft et al. 1955.)

Renal Effects. No studies were located regarding renal effects in humans after exposure to radon and radon daughters by other routes.

A decrease in kidney weight, extreme shrinkage of the cortex, and infiltration of fat into the lining of the renal tubules and eventual renal failure occurred in mice given a single intravenous injection of 1.6×10^{10} pCi (6.0×10^8 Bq) radon-222/kg body weight in equilibrium with its decay products

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(Hollcroft et al. 1955). Renal failure may have caused the observed anemia (see Hematological Effects), weight loss (see Other Effects), and decrease in lifespan observed in these mice. (See Section 2.2.4.1 for limitations of Hollcroft et al. 1955.)

Other Effects. A single intravenous injection of radon at a concentration of 1.6×10^{10} pCi (6.0×10^8 Bq) radon-222/kg body weight in equilibrium with its decay products resulted in a decrease in body weight in mice, possibly due to renal failure (Hollcroft et al. 1955). (See Section 2.2.4.1 for limitations of Hollcroft et al. 1955.)

No studies were located regarding the following effects in humans or animals after exposure to radon and radon daughters by other routes.

- 2.2.4.3 Immunological Effects
- 2.2.4.4 Neurological Effects
- 2.2.4.5 Developmental Effects
- 2.2.4.6 Reproductive Effects
- 2.2.4.7 Genotoxic Effects
- 2.2.4.8 Cancer

2.3 TOXICOKINETICS

In radiation biology the term dose has a specific meaning. Dose refers to the amount of radiation absorbed by the organ or tissue of interest and is expressed in rad (grays). Estimation of this radiation dose to lung tissue or specific cells in the lung from a given exposure to radon and radon daughters is accomplished by modeling the sequence of events involved in the inhalation, deposition, clearance, and decay of radon daughters within the lung. While based on the current understanding of lung morphometry and experimental data on radon and radon daughter toxicokinetics, different models make different assumptions about these processes, thereby resulting in different estimates of dose and risk. These models are described in numerous reports including Bair (1985), BEIR IV (1988), EPA (1988a), ICRP (1978), James (1987), NEA (1983), and NCRP (1984).

In this section the toxicokinetics of radon is described based on the available experimental data rather than descriptions derived from models. The toxicokinetics of radon, as it relates to the development of adverse health effects in exposed populations, is further complicated by the transformation of radon to radon daughters. These progeny may be present with radon in the environment and inhaled or ingested along with radon and/or they may be formed in situ from the transformation of the radon absorbed in the body.

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2.3.1 Absorption

2.3.1.1 Inhalation Exposure

The primary route of exposure to radon and its progeny is inhalation. The degree of deposition and the subsequent absorption of inhaled radon and progeny is determined by physiological parameters, such as respiration rate and tidal volume; and physical properties, such as the particle size distribution of the carrier aerosols and of the unattached fraction, the equilibrium state, and solubility coefficients (Crawford-Brown 1987; Holleman et al. 1969; Jacobi 1964).

Since radon is an inert gas, its movement across membranes is driven by solubility coefficients (Crawford-Brown 1987) and it may be readily absorbed by crossing the alveolar membrane. Most inhaled radon will be exhaled before it can decay and deposit a significant radiation dose to the lung tissue, due to the relatively long half-life of radon gas (McPherson 1980).

The radioactive decay of radon results in the formation of long- and short-lived daughter products which may attach to the surface of aerosol particles and, when inhaled, deposit on the mucus lining of the respiratory tract through impaction, sedimentation, or diffusion (Altshuler et al. 1964). It is assumed that the short-lived daughters, polonium-218, lead-214, and bismuth-214, remain in the mucus layer (James 1987); however, absorption of deposited radon daughters from the lung into the blood stream also may occur (Jacobi 1964; Morken and Scott 1966). The deposited radon daughters appear to act as soluble substances and are released from the dust particles after they undergo solvation (ICRP 1966). The long-lived radon daughter products (lead-210, bismuth-210, and polonium-210) contribute little to the radiation dose to lung tissue because they have a greater likelihood of being physically removed by ciliary action or absorbed by macrophages before they can decay and deliver a significant radiation dose (McPherson 1980). The absorption characteristics and rates of mucus clearance in various parts of the respiratory tract are uncertain (James 1987).

The total respiratory deposition of radon daughters in human subjects has been determined experimentally by George and Breslin (1967, 1969), Holleman et al. (1969), and Shapiro (1956) to range from 18% to 51% of the inhaled amount and to be dependent on tidal volume, particle size, and breathing rate. In general, deposition increases with increasing tidal volume, with smaller particle size, and with changes in normal breathing rates. Respiratory deposition has also been measured in casts of the human larynx and trachea by Chamberlain and Dyson (1956) who determined an average deposition of about 22% of the inhaled, uncombined radon activity at a breathing rate of 20 L/minute. The important sites for deposition of aerosols were determined by the use of casts of the human tracheobronchial tree to be at or near the first bifurcations of the bronchi (Cohen 1987; Martin and Jacobi 1972). According to Cohen (1987), the nonuniform deposition for bifurcations as compared with airway lengths suggests that the dose from radon daughter deposition will be

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about 20% greater than estimated for uniform deposition. Estimation of dose to the respiratory tract has been extensively studied using models (BEIR IV, 1988; EPA 1989a; Harley and Pasternak 1982). Both the studies of human lung casts and the information derived from models indicate that most deposition occurs in the tracheobronchial region of the lung; other regions (nasopharyngeal and pulmonary) receive much smaller doses (BEIR IV 1988).

From a study in rats, Cohn et al. (1953) were able to conclude that the radiation exposure per unit area is greater for the bronchi than for any other lung tissue and, that the radiation dose to the respiratory tract from the progeny was 125 times greater than from radon alone.

2.3.1.2 Oral Exposure

Exposure to radon by the oral route occurs from dissolution of radon in drinking water and, of the total radon dissolved in water, 30% to 70% may be lost by aeration and would be available for inhalation (Dundulis et al. 1984; Holoway and Turner 1981). Another study reported a loss of 15% to 25% radon to the air from drinking (Suomela and Kahlos 1972). Based on the time-course of radon elimination in expired air, it appears that the majority of radon absorption following ingestion in water occurs in the stomach and small intestine, and only 1% to 3% of the ingested radon remains to enter the large intestine to be available for absorption (Dundulis et al. 1984). Studies with other inert gases indicated that the small intestine plays a major role in gastrointestinal uptake of these inert gases (Tobias et al. 1949).

The rate of absorption of radon from the gastrointestinal tract depends on the stomach contents and the vehicle in which it is dissolved. Experimental data from humans who ingested radon dissolved in water indicate that radon is rapidly absorbed from the stomach and small intestines, and that greater than 90% of the absorbed dose is eliminated by exhalation in less than 1 hour (Hursh et al. 1965). Absorption of radon also may occur in the large intestine. This is based on experimental data where exhalation of radon continues at lower concentrations for a longer time after administration when radon dissolved in drinking water is ingested on a full stomach as compared to ingestion of radon on an empty stomach (Meyer 1937). The absorption of radon following ingestion of a meal high in fat is delayed (Vaternahm 1922). Radon is present in exhaled air at higher concentrations and at later times after ingestion of oil or fat emulsions containing radon than with water containing radon (Vaternahm 1922).

Ingested radon progeny may not contribute significantly to the radiation dose to the stomach as they may not penetrate the mucus lining to a great extent (Von Döbeln and Lindell 1964). Production of daughter products in situ, following absorption of radon in the gastrointestinal tract, will primarily result in a radiation dose to the gastrointestinal wall (Von Döbeln and Lindell 1964). The ingestion of radon may also result in exposure to lung tissue due to absorption from the gastrointestinal tract with transport by way

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of the systemic circulation to the lung with subsequent decay to daughter products occurring in the lung (Crawford-Brown 1987).

2.3.1.3 Dermal Exposure

Data regarding the absorption of radon following dermal exposure are very limited. Dermal absorption of radon has been measured in subjects after bathing in a radon-water spa (Furuno 1979; Pohl 1965) or after application of a radon-containing ointment to the intact skin (Lange and Evans 1947). After bathing for 5 to 15 minutes, radon-222 concentrations in expired air reached approximately 0.9% of that in the water and ranged from 17.9 to 49.1 pCi radon-222/L of air (662 to 1817 Bq/m³) compared to pre-bath levels of less than 1 pCi radon-222/L of air (37 Bq/m³). Radon concentrations in the water were reported by the authors as 5,800 pCi (215 Bq) radon-222/kg. However, the relative contributions of the dermal and inhalation routes cannot be determined (Furuno 1979). Radon concentrations in blood reached 0.85% to 1% of the radon concentration in the bath water, which was 1.8×10^5 pCi (4.9×10^6 Bq) radon-222/L of water, after 30 to 40 minutes of bathing while breathing compressed air (Pohl 1965). Approximately 4.5% of the radon applied in ointment to intact skin was measured in expired air within 24 hours following application (Lange and Evans 1947).

2.3.1.4 Other Routes of Exposure

No studies were located regarding absorption of radon or its progeny in humans and laboratory animals after exposure by other routes.

2.3.2 Distribution

2.3.2.1 Inhalation Exposure

The distribution of radon once it is absorbed or deposited in the lung is a function of its physical properties. Radon progeny, especially the longlived daughters, that have been deposited in the lungs are partially removed by the mucociliary blanket, which then carries the particles to the trachea and the gastrointestinal tract. Following chronic exposure in humans, lead-210, a stable daughter product, has been found in bone (Black et al. 1968; Blanchard et al. 1969; Cohen et al. 1973; Fry et al. 1983) and in teeth (Clemente et al. 1982, 1984). After prolonged exposure, radon concentrations in body organs can reach 30% to 40% of inhaled concentrations (Pohl 1964).

Fat appears to be the main storage compartment in rats following inhalation exposure. In rats following an acute exposure to radon, concentrations of radon and radon daughters were much higher in the omental fat than in any of the other tissues examined, followed by the venous blood, brain, liver, kidney, heart, muscle tissues, and testes (Nussbaum and Hursh 1957). Radon reached equilibrium in the fat in about 6 hours compared to 1 hour in all other tissues. This may be due to the nonuniformity of blood perfusion within this tissue.

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2.3.2.2 Oral Exposure

After radon enters the gastrointestinal tract, it is absorbed into the blood stream and then distributes to different organs and tissues (Crawford-Brown 1987). This transfer from the gastrointestinal tract to the blood was dependent upon the emptying patterns of the stomach into the upper intestine, stomach content, fat content of meals, and time of meal in relation to radon ingestion (Hursh et al. 1965; Suomela and Kahlos 1972; Vaternahm 1922; Von Dobein and Lindell 1964). No age-dependent differences in radon distribution from the gastrointestinal tract should be evident due to rapid equilibration in the body (Crawford-Brown 1983). However, changes in the mass of fatty tissue would be expected to affect distribution processes since radon is more soluble in fat than in other tissues (Crawford-Brown 1987).

According to Hursh et al. (1965), in humans greater than 90% of ingested radon is distributed to the lung where it is rapidly exhaled. Of the remaining administered dose of radon, 5% is distributed to the liver, 1.6% to the kidneys, and 2% to lung tissue (Holoway and Turner 1981). Acute exposure of human subjects to 1.3×10^5 to 2.83×10^5 pCi (4.9×10^3 to 1.05×10^2 Bq) radon-222/L of water resulted in a whole body accumulation of 1.9×10^3 to 1.22×10^4 pCi (70 to 450 Bq) bismuth-214, a radon decay product. The biological half-life of radon in these individuals ranged from 30 to 50 minutes (Suomela and Kahlos 1972).

From a chronic study in laboratory animals where 3.6 pCi (0.13 Bq) of radon was administered daily for 1 year, a body accumulation of 5 pCi (0.19 Bq) lead-210/g of tissue, 0.08 pCi (3.0×10^{-3} Bq) polonium-210/g of tissue, and 0.003 pCi (1.1×10^{-4} Bq) bismuth-210/g of tissue was reported (Fernau and Smereker 1933). Radon is very soluble in fat with its distribution coefficient in fat greater than in any other organ or tissue (Nussbaum and Hursh 1957). This storage of radon in body fat is a constant source of lead-210, polonium-210, and other progeny (Djuric et al. 1964). The presence of lead-210 and polonium-210 are not unique to radon exposure and are also found in cigarette smoke and food (NCRP 1984b).

In addition to the available data on distribution in humans and laboratory animals, many different models exist which estimate distribution in humans (EPA 1988a; ICRP 1978).

2.3.2.3 Dermal Exposure

No studies were located regarding distribution in humans or laboratory animals after dermal exposure to radon or its progeny.

2.3.2.4 Other Routes of Exposure

No studies were located regarding distribution of radon or its progeny in humans or laboratory animals after exposure by other routes.

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2.3.3 Metabolism

Radon is an inert noble gas that does not readily interact chemically with cellular macromolecules. Radon does not undergo metabolism in biological systems.

2.3.4 Excretion

2.3.4.1 Inhalation Exposure

Most of the inhaled radon will be eliminated by exhalation before it can decay and deposit a significant radiation dose to the lung tissue. The longlived radon progeny are, to some extent, physically removed before they can decay and deposit a radiation dose (McPherson 1980). The biological half-time for radon daughters in the pulmonary region has been reported to range from 6 to 60 hours and in the tracheobronchial region to range from 10 minutes to 4.8 hours (Altshuler et al. 1964; Jacobi 1964, 1972). The biological half-time in fat tissue has two components, a fast component of 21 minutes and a slow component of 130 minutes (Nussbaum and Hursh 1957).

Long-lived radon progeny (lead-210) have been reported to be excreted in the urine of uranium miners at 1 to 18 years following exposure. This excretion of lead-210 results from a slow release of the daughters from bone. Concentration of lead-210 in bone has been shown to correlate with cumulative exposure to radon and radon daughters in WLM (Black et al. 1968).

Experiments in rats and mice indicated that polonium-214 may be retained in the lung following inhalation exposure. The retention efficiency of polonium-214, a stable daughter product, in the lung was 2% and 2.2% of the administered activity in rats and mice, respectively, immediately following acute inhalation exposure (Doke et al. 1973).

2.3.4.2 Oral Exposure

Following ingestion of radon dissolved in water, greater than 90% of the absorbed radon was eliminated by exhalation within 100 minutes. By 600 minutes, only 1% of the absorbed amount remained in the body (Hursh et al. 1965). The biological half-time for removal of radon from the body ranges from 30 to 70 minutes depending on whether the stomach is empty or full and on fat content in the diet (Hursh et al. 1965; Suomela and Kahlos 1972; Vaternahm 1922). The presence of food in the stomach may result in a marked delay in the removal of radon from the body due to an increased emptying time of the stomach during which time a portion of the radon may decay (Hursh et al. 1965). The biological half-life in the blood of humans has been reported to be 18 minutes for 95% of the administered dose and 180 minutes for the remaining 5% (Hursh et al. 1965). The longer half-life for the remaining 5% may be due to storage and subsequent removal from tissues. The effective half-life for removal of radon was reported as 30 minutes (Andersson and Nilsson 1964).

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The transfer rate of radon from the gastrointestinal tract and subsequent elimination from the respiratory tract was found to be dependent on the pattern of emptying of the stomach into the small intestines (i.e., with or without a meal), and the accompanying vehicle (i.e., water or fat). After ingestion of radon dissolved in drinking water on an empty stomach, radon in exhaled air rapidly increased reaching a maximum concentration in exhaled air 5 to 10 minutes after ingestion (Meyer 1937). With ingestion of radon in drinking water with or after a meal, radon elimination in expired air is delayed and varies in concentration with time, reflecting absorption from the small intestines as it receives a portion of the stomach contents (i.e., with stomach emptying patterns) (Meyer 1937). After ingestion of radon dissolved in olive oil on an empty stomach, elimination in expired air reached a maximum concentration 50 minutes post-ingestion, then declined; however, when administered in olive oil after a meal, radon in expired air remained constant from 10 minutes to 5 hours after ingestion (Vaternahn 1922). These data suggest that radon is eliminated in expired air more rapidly from a water vehicle than a fat or oil vehicle and this elimination occurs over a longer period of time when ingested with a meal than on an empty stomach.

When radon dissolved in water was ingested on a full stomach, the exhalation of radon reached a maximum at 5 to 15 minutes then declined. This was then followed by a second peak about 20 minutes after ingestion. When ingestion of radon occurred "some time" after a meal, the second radon peak in exhaled air was delayed and was followed by further peaks (Meyer 1937). These subsequent peaks were explained by the absorption of radon from the intestine after it has received portions of the stomach content (Meyer 1937).

2.3.4.3 Dermal Exposure

Information on the excretion of radon and its progeny following dermal exposure is very limited. Within 24 hours, 4.5% of the radon, which was applied as a salve to intact human skin, was eliminated by exhalation, while 10% was exhaled after application of the radon to an open wound (Lange and Evans 1947). Bathers breathing compressed air while immersed in radon-containing water had exhaled approximately one-third of radon measured in blood immediately after bathing (Pohl 1965). By 6 to 8 minutes after bathing, these persons were exhaling one-half of the amounts exhaled immediately after bathing. The author stated that the remaining radon which distributed to fatty tissue was excreted more slowly.

2.3.4.4 Other Routes of Exposure

Experiments in animals have reported the retention of radon after exposure by the intraperitoneal and intravenous routes. After intravenous administration, 1.6% to 5.0% of the administered activity was retained in the animals after 120 minutes (Hollcroft and Lorenz 1949). Retention was greatest after intraperitoneal administration at 120 minutes, but by 240 minutes it was nearly the same for both routes of administration. These authors also reported that the amount of radon retained in tissues was greater in obese

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mice than in normal mice, especially after intraperitoneal administration (Hollcroft and Lorenz 1949). Radon retention has also been studied in dogs after intravenous administration of radium-226. The amount of radon in bone was found to increase with increasing time after injection (Mays et al. 1975).

2.4 RELEVANCE TO PUBLIC HEALTH

Growing concern in the late 1940s that the inhalation of radon and radon daughters was contributing to the adverse health effects observed in underground miners stimulated the conduct of epidemiological investigations and initiated animal studies with radon and radon daughters. Earlier inhalation studies had been conducted with radon only, but evidence from radon dosimetry studies indicated the involvement of radon daughters rather than just radon (Bale 1951). Epidemiological studies further suggested that the major health effects observed in miners might be attributed to radon daughters. Both human and animal studies indicate that the lung and respiratory system are the primary targets of radon daughter-induced toxicity. The evidence indicates that inhalation of radon decay products results in radiation damage to tissues in which these products are deposited. Nonneoplastic respiratory disease and lung cancer have been reported in humans and animals exposed to radon and radon daughters by inhalation.

Death. No deaths in humans following acute exposure to radon have been reported. Following long-term exposure, significant increases in early mortality due to nonneoplastic respiratory diseases have been reported among uranium miners. Because mortality due to nonneoplastic diseases is not generally reported by exposure categories (i.e., WLM categories), it is not clear what exposure concentration or duration of exposure in these mining cohorts is associated with this increased mortality. In addition, these nonneoplastic respiratory deaths cannot be attributed solely to radon but may result from exposure to other mine air pollutants. Reduction in lifespan due to respiratory disease as a result of exposure to high levels of radon or radon daughters has been reported in various animal studies. Based on the evidence in animals, it is apparent that death due to respiratory disease may result after exposure to radon at very high levels. However, it is unclear to what extent low-level environmental exposure to radon and radon daughters may increase the risk of death due to nonneoplastic respiratory disease.

Respiratory Effects. Respiratory disease characterized as emphysema, fibrosis, or pneumonia has been reported in both humans and animals with inhalation exposure to high levels of radon and radon daughters. In addition to deaths due to nonneoplastic respiratory disease, some studies have reported reductions in respiratory function. In all of the occupational cohorts, miners were concomitantly exposed to other mine pollutants, such as ore dust, other minerals, or diesel-engine exhaust. The contribution of these pollutants, as well as cigarette smoking, to the induction of nonneoplastic respiratory disease is unclear. As reported in Section 2.6, Interactions With Other Chemicals, the combination of radon and radon daughters along with ore

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dust or other pollutants enhanced the incidence and severity of adverse respiratory effects in laboratory animals as compared to either toxicant alone. Induction of this type of respiratory disease may occur primarily at doses that exceed those commonly found in the environmental setting; however, the radiation dose that would result in either pulmonary dysfunction or pulmonary disease is not known. The adverse respiratory effects observed appear to be consistent with alpha radiation damage that may occur at high doses in slower regenerating tissues such as the lung (see Appendix B). That being the case, production of respiratory tissue damage in the lungs may not be immediately apparent, especially at low environmental exposures.

Hematological Effects. No information on the hematological effects of radon in humans was located in the available literature. Alterations in hematological parameters following exposure to radon have been reported in animals. The extent and severity of the hematological effects were related to the level of exposure and the exposure duration, and red blood cells appear to be more sensitive to the effects of radon than white blood cells. Following acute exposure by the inhalation or intravenous routes, decreases in the number of red blood cells and white blood cells occurred immediately postexposure. Red blood cells remained depressed for the remaining life of the treated animals, while white blood cells returned to normal levels postexposure. Following repeated exposures, white blood cell counts remained depressed for longer periods of time and, with chronic exposure, depression in white blood cell counts was linearly related to the cumulative exposure. The animal studies indicate chronic exposure of humans to radon may result in similar alterations in the hematopoietic system.

Renal Effects. Evidence of kidney disease has been reported in United States uranium miners (Waxweiler et al. 1981). In that survey, chronic and unspecified nephritis was elevated after a 10-year latency. The nephrotoxicity of soluble uranium in animals has been documented (ATSDR 1990a). Due to their relatively short half-lives, the alpha-emitting radon daughters present in the lung undergo radioactive decay before they move to other organs, in contrast to other alpha-emitting radionuclides, such as uranium or plutonium (ATSDR 1990a, 1990b), which may translocate from the lung to irradiate other tissues. Nevertheless, direct evidence of renal dysfunction or impairment resulting from inhalation or oral exposure to radon and radon daughters alone is lacking.

Neurological Effects. No information on neurological effects in humans exposed to radon was located in the available literature. Only one animal study attributed the toxic effects observed to the action of radon on the central nervous system. This study reported respiratory paralysis due to central nervous system depression; however, the study has numerous flaws (see Section 2.2.1.4) that limit its usefulness and render the reported results questionable.

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Reproductive Effects. Recent epidemiological studies have raised speculation that inhalation exposure to radon and radon daughters during uranium mining may be associated with effects on reproductive outcome. A decrease in the secondary sex ratio (the ratio of male to female children) of children of underground miners following employment in uranium mines was reported (Dean 1981; Muller et al. 1967). Waxweiler and Roscoe (1981), however, found the secondary sex ratio to increase in older men but to decrease in younger men. If the father is exposed to radiation, an increase in the number of male children might be expected due to the relative resistance of the Y chromosome as compared to the X chromosome (Waxweiler and Roscoe 1981). In a preliminary study, Wiese and Skipper (1986) reported a decrease in the secondary sex ratio, although not statistically significant, in children born to underground uranium and potash workers. No other information exists on reproductive effects in other epidemiological investigations or animal studies. Therefore, these observations of alterations in secondary sex ratios are suggestive of possible effects but are not conclusive evidence that radon can produce reproductive toxicity in persons environmentally exposed to radon.

Genotoxic Effects. Increases in chromosomal aberrations have been reported among uranium miners and among personnel employed at a radon spa in Austria following inhalation exposure. Increases in chromosomal aberrations were also reported in a small group of people living in an area with high radon concentrations in their water supply. As stated in Section 2.3 on toxicokinetics, radon rapidly escapes from water; therefore, the probable major route of exposure in this cohort also was inhalation. In addition, increased DNA-repair rates in lymphocytes were observed in another occupational cohort. The increased DNA repair rates may reflect increases in DNA damage. DNA repair enzymes may be induced in response to DNA damage. The implications of this information for environmental exposures are unclear. In the case of the miner occupational cohorts, cumulative exposures were greater than 100 WLM and ranged up to 6,000 WLM. Also, the animal data are inconclusive and do not clearly establish a link between genotoxicity and radon exposure. A summary of the genotoxicity studies is given in Table 2-2.

Cancer. Numerous epidemiological studies have demonstrated a causal association between lung cancer mortality and exposure to radon in combination with radon daughters. The majority of these epidemiological data have been collected from occupational cohorts exposed to radon and radon daughters during mining operations. Despite the variety of conditions reported for the mines (including dust concentrations, type of ore mined, and ventilation rates) and differences in the cohorts (including levels of exposure, length of follow-up, smoking habits, and ages of exposure), exposure to radon in mining operations is clearly directly associated with lung cancer mortality.

Some of these studies indicate that lung cancer mortality was influenced by the total cumulative radiation exposure, by the age at first exposure, and by the time-course of the exposure accumulation. The length of the induction

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TABLE 2-2. Genotoxicity of Radon-222 In Vivo

End Point	Species (Test System)	Result	Reference
MAMMALIAN SYSTEMS			
Chromosomal aberrations	Human (peripheral lymphocytes)	+	Pohl-Rüling et al. 1976, 1987; Pohl-Rüling and Fischer 1979, 1982, 1983; Brandom et al. 1972, 1978
	Human (whole body lymphocytes)	+	Stenstrand et al. 1979
	Rabbit (somatic cells)	-	Leonard et al. 1981
DNA repair	Human (lymphocytes)	+	Tuschl et al. 1980
Sister chromatid exchanges	Rat (bone marrow cells)	+	Poncy et al. 1980
INVERTEBRATE SYSTEMS			
Dominant lethal	<u>Drosophila melanogaster</u>	(+)	Sperlich et al. 1967

+ = positive result

- = negative result

(+) = positive or marginal result

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latency, that is, the time from the start of mining to the development of cancer, is strongly dependent on the age at which a man starts mining (Archer 1981). The data indicate that the older a man is when he starts mining, the shorter his induction-latency period will be. The Czechoslovakian data indicate that the frequency of attributable lung cancer mortality rises steeply with increasing age at the start of mining, corresponding to decreasing induction-latency periods (Sevc et al. 1988). There is evidence that the induction-latency period is also dependent on the exposure rate and total radiation exposure, that is, the lower the exposure rate, the longer a group must be followed to evaluate the lung cancer risk (Archer et al. 1979). According to Sevc et al. (1988) lung cancer mortality for the same cumulative WLMS was greater in the subcohort with higher exposures early in their work history, compared to those with nearly equal yearly exposure or the subcohort with lower initial exposure which increased to higher levels in later years. Additional support for the role of radon as a causative agent in lung cancer is provided by the results of the studies of nonuranium hard rock miners, which also showed an increased mortality rate from lung cancer.

Some of these studies also indicated that underground miners who were cigarette smokers had a higher incidence of radiation-induced lung cancer mortality than did miners who were nonsmokers, and that the induction-latency period was substantially shorter for smokers than for nonsmokers (Archer 1981). A study of a nonsmoking cohort of uranium miners clearly indicated an increased mortality risk for lung cancer for the cohort (Roscoe et al. 1989). In addition, increases in lung cancer among American Indian uranium miners, who had a low frequency of lung cancer in the nonexposed general population compared to rates in the white United States population and a low frequency of cigarette smoking, support the conclusion that radiation is the primary cause of lung cancer among uranium miners (Gottlieb and Husen 1982; Samet et al. 1984b; Sevc et al. 1988). A comprehensive evaluation of risk estimates from various mining cohorts can be found in BEIR IV (1988).

Several studies of residential exposure to radon and radon daughters also indicate an increased risk of lung cancer (Axelson and Edling 1980; Axelson et al. 1971, 1981; Edling et al. 1984; Svennson et al. 1987, 1989). These studies are primarily case-control studies that involve a small number of subjects and have exposure estimates that are limited or based on surrogates. A more recent study has reported on a much larger cohort and has provided some exposure information (Svennson et al. 1989). These studies support the evidence obtained from the occupational cohorts. Radon concentrations in environmental settings are not expected to be at levels as high as those encountered in mining operations nor would they be expected to be combined with dusty conditions or diesel exhaust exposure, two features of the exposure of several of the examined cohorts. However, the BEIR IV (1988) Committee indicated that the risk from occupational or residential exposure to radon is the same per unit dose.

Studies in animals confirm and support the conclusions drawn from the epidemiological data. When all animal data are combined and reviewed, four

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variables surface which appear to influence the efficiency of radon daughters to produce lung cancer in laboratory animals (Cross et al. 1984). These variables include: cumulative exposure to radon and radon daughters, exposure rate to radon and radon daughters, unattached fraction of radon daughters, and disequilibrium of radon daughters. Another factor which may influence the tumorigenic potential of radon and radon daughters is exposure in conjunction with other pollutants, such as uranium ore dust or cigarette smoke (see Section 2.6 for a discussion of interactions of radon with other chemicals). The ability of radon daughters, alone or in conjunction with uranium ore dusts, to produce lung tumors in laboratory animals appears to increase with an increase in exposure until lifespan shortening reverses the trend, with a decrease from high exposure rate to low exposure rate, and with increasing unattached fraction and disequilibrium.

In general, the pattern of results from the epidemiological studies and animal experiments clearly indicates a risk due to radon and radon daughter exposure. Although individual studies have particular shortcomings that may make that conclusion less supportable for the individual study, the pattern over all the studies is convincing. Positive associations between exposure to radon daughters and lung cancer have been found in occupational settings for various types of mining operations, in various ethnic groups around the world, and under various concomitant exposure conditions. In some of these occupational settings, concomitant exposure to other pollutants, such as ore dust, diesel engine exhaust, or other minerals, such as silica, may have occurred. The possible impact of these other pollutants on radon daughter-induced lung cancer is unclear (see Section 2.6).

2.5 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).

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 or cell 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 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 (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 biologic 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

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mineral nutrients such as copper, zinc and selenium). Biomarkers of exposure to radon are discussed in Section 2.5.1.

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 epithelium cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are often not substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by radon are discussed in Section 2.5.2.

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. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, biologically effective dose, or target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 2.7, "POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE."

2.5.1 Biomarkers Used to Identify or Quantify Exposure to Radon

Biomarkers of exposure to radon and its progeny include the presence of radon progeny in several human tissues and fluids, including bone, teeth, blood, hair, and whiskers, and can be measured by methods which are both specific and reliable (Blanchard et al. 1969; Clemente et al. 1984; Gotchy and Schiager 1969). Although the presence of radon progeny in these tissues and fluids indicate exposure to radon, exposure to uranium or radium may also result in the presence of these decay products. Polonium-210 may also be found in tissues after exposure to cigarette smoke. Levels of lead-210 in teeth have been associated with levels of radon in the environment in an area with high natural background levels of radon and radon daughters (Clemente et al. 1984). In addition, Black et al. (1968) reported correlation of radiation exposure and lead-210 levels in bone from uranium miners. However, cumulative exposure to these individuals was estimated. Biomarkers of radon or radon progeny exposure may be present after any exposure duration (e.g., acute, intermediate, chronic). Because of the relatively short half-lives of most radon progeny, with respect to a human lifetime, the time at which the biological sample is taken related to time of exposure may be important. However, for the longer lived progeny the time factor is less critical.

Models are available which estimate exposure to radon-222 from levels of stable radon daughter products, lead-210 and polonium-210, in bone, teeth, and blood (Blanchard et al. 1969; Clemente et al. 1982, 1984; Eisenbud et al. 1969; Gotchy and Schiager 1969; Weissbuch et al. 1980). However, these models make numerous assumptions, and uncertainties inherent in all models are

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involved in these estimates. Therefore, at present, these estimated levels of biomarkers of exposure are not useful for quantifying exposure to radon and progeny. Quantification of exposure to radon is further complicated by the fact that radon is an ubiquitous substance and background levels of radon and radon progeny are needed to quantify higher than "average" exposures.

2.5.2 Biomarkers Used to Characterize Effects Caused by Radon

The principal target organ identified in both human and animal studies following exposure to radon and progeny is the lung. Alterations in sputum cytology have been evaluated as an early indicator of radiation damage to lung tissue. The frequency of abnormalities in sputum cytology, which may indicate potential lung cancer development, increased with increasing cumulative exposures to radon and radon daughters (Band et al. 1980; Saccomanno et al. 1974). Although abnormal sputum cytology may be observed following radon exposure, this effect is also seen following exposure to other xenobiotics such as cigarette smoke. In addition, even though increases in the frequency of abnormal sputum cytology can be measured, they may not provide a reliable correlation between levels in human tissues or fluids with health effects in exposed individuals.

A dose-response relationship between chromosome aberrations and increased environmental levels of radon has been reported (Pohl-Rtilling and Fischer 1983; Pohl-Riiling et al. 1976, 1987). Although the presence of chromosome aberrations is a biomarker of effect, the potential range of chemicals which could cause this effect is so great that it would not necessarily be considered radon-specific.

Additional biomarkers of effect for radon and radon progeny exposure may exist; however, these were not located in the reviewed literature. For more information on biomarkers for effects of the immune, renal, and hepatic systems see ATSDR, CDC Subcommittee Report on Biological Indicators of Organ Damage (1990c) and for biomarkers of effect for the neurological system see OTA (1990). For more information on health effects following exposure to radon and radon daughters see Section 2.2.

2.6 INTERACTIONS WITH OTHER CHEMICALS

The interaction of cigarette smoke with radon and the possible effect on radon-induced toxicity is a complex one and is still an issue under consideration. Cigarette smoke appears to interact with radon and radon daughters to potentiate their effects. In general, epidemiological studies have reported synergistic, multiplicative, or additive effects of cigarette smoke in lung cancer induction among miners exposed to radon and radon daughters (US DHHS 1985). Studies by Lundin et al. (1969, 1971) reported 10 times more lung cancer among United States uranium miners who smoked. In a case-control study of United States uranium miners, Archer (1985) reported that smoking miners with lung cancer had significantly reduced latency induction periods than nonsmokers. Cigarette smoking also appeared to shorten

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the latency period for lung cancer among Swedish lead-zinc miners (Axelson and Sundell 1978), and Swedish iron miners (Damber and Larsson 1982). Miners who smoke cigarettes may be at higher risk because of the possible synergistic or additive effect between radon and radon daughters and cigarette smoking (Klassen et al. 1986). However, an antagonistic relationship between cigarette smoking and lung cancer in humans may exist according to Sterling (1983). His hypothesis is that smokers may have a lower potential retention of deposited radon daughter particles due to enhanced mucociliary clearance. Other investigators have reported that nonsmoking miners exhibited a higher incidence of lung cancer than smokers, although the latency of cancer induction was shorter in nonsmokers than for smokers (Axelson 1980; Axelson and Sundell 1978). Again, the theories put forth to explain this phenomenon include increased mucus production and alterations in mucociliary clearance in smokers resulting in the increased mucus thickness.

Some animal studies support the theory that cigarette smoke potentiates the effects of radon and radon daughters alone or in conjunction with uranium ore dust. A study by Chameaud et al. (1982b) reported an increase in the incidence of lung cancer, as well as a decrease in the cancer latency period in rats exposed to radon and then to cigarette smoke, compared to rats exposed to radon and radon daughters alone. This study did not include untreated controls. Alterations in normal blood parameters, including carboxyhemoglobin levels and leukocyte counts, were observed in dogs exposed to cigarette smoke followed by exposure to radon daughters plus uranium ore dust, compared to animals exposed to only radon daughters plus uranium ore (Filipy et al. 1974). In contrast, some studies suggest an antagonistic interaction between smoking and radon daughter-induced lung cancer. Dogs exposed daily to cigarette smoke followed immediately by exposure to radon and radon daughters and uranium ore dust exhibited a decrease in the incidence of lung tumors, compared to dogs exposed to radon and radon daughters plus uranium ore dust (Cross et al. 1982b). Cross (1988) reported that this was possibly due to a thickening of the mucus layer as a result of smoking and, to a lesser extent, a stimulatory effect of cigarette smoke on mucociliary clearance, although no empirical evidence was collected during the experiment to test these possibilities.

In rats administration of chemicals present in cigarette smoke after exposure to radon and radon daughters resulted in a decrease in the lung cancer latency period when compared to the time-to-tumor induction in animals treated with radon alone. This effect was seen with 5,6-benzoflavon (Queval et al. 1979) and with cerium hydroxide (Chameaud et al. 1974).

Other airborne irritants, as well as ore dust and diesel exhaust, may act synergistically with radon and radon daughters to increase the incidence of adverse health effects. Epidemiological studies report the presence of other airborne irritants in mining environments, including arsenic, hexavalent chromium, nickel, cobalt (Sevc et al. 1984), serpentine (Radford and Renard 1984), iron ore dust (Damber and Larsson 1982; Edling and Axelson 1983; Radford and Renard 1984), and diesel exhaust (Damber and Larsson 1982; Sevc et al. 1984).

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Cross and colleagues at Pacific Northwest Laboratory have conducted extensive experiments involving exposure of dogs, mice, rats, and hamsters to radon and its progeny in conjunction with uranium ore dust and/or diesel exhaust (Cross 1988; Cross et al. 1981, 1982b, 1984; Pacific Northwest Laboratory 1978; Palmer et al. 1973). Studies in hamsters, mice, and rats have shown that exposure to uranium ore dust and/or diesel exhaust increases the pulmonary effects of radon. Radon and combinations of uranium ore dust and/or diesel exhaust produced greater incidences of pulmonary emphysema and fibrosis in hamsters than radon and radon daughters alone (Cross 1988). Exposure to uranium ore dust or diesel exhaust alone caused significant bronchial hyperplasia, but not as great an effect as combining either of these with radon and radon daughters. The incidence of severe lesions of the upper respiratory tract (nasal passages and trachea) of mice and rats was increased following exposure to radon and uranium ore dust, compared to animals exposed to radon and radon daughters alone (Palmer et al. 1973). An increased incidence of thoracic cancer (40%) was observed in rats treated with asbestos (mineral dust) after inhalation of radon and radon daughters, compared with animals exposed to radon alone (Bignon et al. 1983). However, these tumors may have been due to asbestos rather than to an interaction between agents. This experiment did not include a group exposed only to mineral dusts. Inhalation exposure to radon and radon daughters in conjunction with silicon dioxide increased the incidence of nodular fibrosis of the lungs in rats (Kushneva 1959).

2.7 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

Children may be more susceptible to the effects of radon and radon daughters. Differences in lung morphometry and breathing rates in children result in higher estimated doses that may make children more susceptible to the effects of radon than adults (Samet et al. 1989). In calculating the inhaled dose of radon, Hofmann et al. (1979) reported that dose was strongly dependent on age, with a maximum value reached at about the age of 6 years. Risk of cancer from exposure to low levels of ionizing radiation during childhood are estimated to be twice that of adults (BEIR V 1990). Risk of lung cancer in children resulting from exposure to radon may be almost twice as high as the risk to adults exposed to the same amount of radon (NCRP 1984a).

Populations that may be more susceptible to the respiratory effects of radon and radon daughters are people who have chronic respiratory disease, such as asthma, emphysema, or fibrosis. People with chronic respiratory disease often have reduced expiration efficiency and increased residual volume; i.e., greater than normal amounts of air left in the lungs after normal expiration (Guyton 1977). Therefore, radon and its progeny would be resident in the lungs for longer periods of time, increasing the risk of damage to the lung tissue. In addition, persons who have existing lung lesions may be more susceptible to the tumor-causing effects of radon (Morken 1973).

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2.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA 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 radon 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 radon.

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 or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

2.8.1 Existing Information on Health Effects of Radon

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to radon and radon daughters are summarized in Figure 2-2. The purpose of this figure is to illustrate the existing information concerning the health effects of radon. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs" information.

Figure 2-2 graphically describes whether a particular health effect end point has been studied for a specific route and duration of exposure. Most of the information on health effects in humans caused by exposure to radon and radon progeny was obtained from epidemiological studies of uranium and other hard rock miners. These studies of chronic occupational exposure to radon via inhalation provide information on cancer and lethality, and limited insight into reproductive and genetic effects. Limited information is also available regarding cancer following dermal exposure to radon and radon daughters. No information on the health effects of radon and radon daughters in humans was available following acute or intermediate exposure by any route. No information on the health effects of radon and radon daughters in animals following acute, intermediate, or chronic oral or dermal exposure was located. The only information available from animal studies was by the inhalation route of exposure which provides data on systemic and genetic effects, as well as cancer.

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		SYSTEMIC									
		Death	Acute	Intermed.	Chronic	Immunologic	Neurologic	Developmental	Reproductive	Genotoxic	Cancer
Inhalation		●			●				●	●	●
Oral											
Dermal											

HUMAN

		SYSTEMIC									
		Death	Acute	Intermed.	Chronic	Immunologic	Neurologic	Developmental	Reproductive	Genotoxic	Cancer
Inhalation		●	●	●	●					●	●
Oral											
Dermal											

ANIMAL

● Existing Studies

FIGURE 2-2. Existing Information on Health Effects of Radon

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2.8.2 Identification of Data Needs

Acute-Duration Exposure. No information exists regarding the health effects to humans following their acute exposure to radon and radon daughters by any route. Single dose studies are available for laboratory animals that have been exposed by the inhalation and parenteral routes. No information is available on acute oral exposure in laboratory animals. Information is available on lethality following acute inhalation exposure to high doses. However, this study did not provide information on target organs, sensitive tissues, or cause of death. No information is available on effects in humans or animals following acute exposure to lower levels of radon. This information is needed in order to assess the toxicity of radon.

Intermediate-Duration Exposure. No information regarding health effects following intermediate-duration exposure to humans by any route was clearly identified in the available literature. Epidemiological studies in general focused on cohorts exposed to radon and radon daughters for durations longer than one year. Animal studies demonstrate that intermediate exposure to high levels of radon and radon daughters resulted in chronic respiratory toxicity and lung cancers. This is an indication of the potential for such effects in exposed human populations. The relationship between the nature and severity of the respiratory toxicity and the amount of radon exposure is not clearly defined; nor is there any information on toxicity to other organs, other than the respiratory tract following intermediate-duration exposure. Additional research on the dose-duration-response relationship between radon exposure and the type and permanence of resulting toxicity would provide pertinent information. Carefully designed studies in which laboratory animals are exposed to levels that are similar to high environmental levels for partial lifetime and observed for life could provide important information. These studies would facilitate the estimation of cancer risk to persons living in an area with high natural levels for only a portion of their life. These animal studies should address both the effect of total dose and dose-rate on development of adverse health effects. This information may also be useful in situations in which the time lapse between identifying the presence of radon and any remediation effort is of an intermediate duration.

Chronic-Duration Exposure and Cancer. Knowledge of the adverse health effects in humans following chronic radon and radon daughter exposure is based primarily on studies in adult male underground miners. These studies describe predominantly respiratory end points, such as emphysema, fibrosis, and cancer. To a large extent other health effects have not been studied. Epidemiological studies in general report only the cause of death for each member of the cohort; therefore, there is insufficient information on whether other adverse effects were identified other than the ones listed as cause of death. Little or no information exists on cardiovascular, gastrointestinal, renal, musculoskeletal, immunological, or dermal/ocular effects in humans or animals. In addition, these miners also may have been simultaneously exposed to other pollutants (e.g., long-lived radioactive dusts (uranium), diesel-engine

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exhaust, cigarette smoke, and external gamma radiation). Several of these factors have been implicated independently as causative agents of lung cancer and respiratory diseases and the excess lung cancers in cigarette smokers have been well documented. Thus, the data currently used to characterize a human health hazard with regard to respiratory toxicity represent a composite response to other factors as well as to radon daughters.

Chronic exposure to radon and radon daughters in laboratory animals also results in respiratory lesions. In laboratory animals exposed to radon and radon daughters in combination with uranium ore dust, pulmonary fibrosis and emphysema have resulted. Further research of the interaction of radon and radon daughters with other environmental pollutants, especially cigarette smoke, is needed. This information could be used to clarify uncertainties in the extrapolation of the data in miners to describe the potential hazard to human health from environmental radon daughter exposures. Well-defined studies that examine both pathological and functional changes in other organ systems are necessary to clarify these issues.

Radon dissolved in drinking water is a source of human exposure. Studies are needed which describe the absorption and translocation of radon gas and the effects of alpha radiation emitted by radon daughters at the site of entry, the gastrointestinal tract. While translocation of radon daughters from the portal of entry to other sites in the body may be limited (due primarily to the short half-life of most alpha emitting radon daughters), radon gas may distribute to other organs and, thereby, provide an internal source of radon daughter alpha radiation.

Epidemiological studies have demonstrated a causal association between exposure to radon and radon daughters and lung cancer mortality. The number of lung cancer mortality cases in these cohorts was influenced by the total cumulative radiation exposure, by the age at first exposure, and by the time course of the exposure duration. Significant increases in lung cancer that were demonstrated in chronic studies in mice, rats, and dogs resulted from exposure of these animals to radon and radon daughters in combination with one or more other pollutants, such as uranium ore dust, diesel-engine exhaust, or cigarette smoke. Chronic studies in hamsters (Pacific Northwest Laboratories 1978) in which animals were exposed to radon and radon daughters alone did not demonstrate a significant carcinogenic response; however, the hamster may be resistant to radiation-induced lung cancer. Hamsters did not develop lung tumors when exposed to another alpha-emitter, plutonium (ATSDR 1990b). Evidence from animal studies indicates that factors such as the unattached fraction and disequilibrium of radon daughters influence lung cancer production. Other air pollutants may interact synergistically with radon daughters in lung tumor induction. Long-term studies designed to evaluate the potential interaction of radon daughters with other pollutants would provide information necessary to determine the toxicity of radon and radon daughters. Factorial studies, i.e., studies that test radon and radon daughters alone and radon and radon daughters with only one other confounding factor are needed because much of the cancer information to date is from studies with several

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confounding factors. These studies could help elucidate the extent of interaction between radon and each confounding factor.

Genotoxicity. Studies of miners and other populations exposed to radon and radon daughters showed an increased occurrence of chromosomal abnormalities. However, because exposure-effect relationships have not yet been established and the biological significance of these chromosomal effects is uncertain, further studies should be performed. In vitro studies using human cell lines could help determine a dose-response for exposure to radon and radon daughters and increased chromosomal abnormalities. Such relationships may be difficult to establish because of possible interactions with other substances, i.e., uranium ore dust. There are no in vivo animal data to support the observed increase in chromosomal abnormalities in human populations. Further observations in laboratory animals are needed to explain these effects.

Reproductive Toxicity. Recent epidemiological studies have suggested that exposure to radon and radon daughters during uranium mining may be associated with adverse reproductive outcomes (Dean 1981; Muller et al. 1967; Wiese and Skipper 1986). While the evidence of the possible reproductive effects of uranium mining is largely descriptive, reports of alterations in the secondary sex ratio among offspring of uranium miners merits further study. Currently there are no experimental data that evaluate the reproductive toxicity of radon and radon progeny exposure by any route. Controlled experiments that are designed to evaluate reproductive toxicity and that attempt to correlate the amount of alpha radiation to germ cells could provide an explanation of the effects that have been observed in the epidemiology studies.

Developmental Toxicity. Recent data indicate that mental retardation may result from low-level exposure of children to radiation during their development in utero (Otake and Schull 1984). While this effect may have resulted from external radiation rather than internally delivered radiation dose, the potential of ionizing radiation to induce developmental toxicity is generally accepted. No experimental data currently exist that evaluate the developmental toxicity of radon and radon progeny by any route. Controlled experiments that are designed to evaluate developmental toxicity and that attempt to correlate the amount of alpha radiation available to the fetus could show whether the effects observed following exposure to other forms of radiation also may occur following exposure to radon and progeny.

Immunotoxicity. No information currently exists on humans or laboratory animals regarding adverse effects on the immune system following exposure by any route to radon or radon progeny. However, data indicate that acute exposure to radon in laboratory animals results in a transient decrease in lymphocytes. Although these effects were transient, it is possible that the immune system may be compromised during this time. In addition, some epidemiological studies have reported increased chromosomal abnormalities

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following exposure to radon and radon daughters. Depending upon the target cells in which these chromosomal changes occurred, adverse effects on the immune system could result. A battery of immunological tests administered to members of a nonminer cohort, such as radon spa workers or people exposed to high background levels, is needed to clarify whether immunological effects occur following exposure to radon or radon progeny. Animal studies designed to evaluate immune competence also are necessary to provide information on subtle alterations in immune function. In addition, lymphocytes and lymphatic tissues are sensitive to the radiation-induced damage caused by other alpha-emitting radionuclides (ATSDR 1990b). Although lymphocytopenia observed in dogs exposed to plutonium is not seen following exposure to radon and radon daughters or uranium ore dust, other tests for immunocompetence have not been conducted (ATSDR 1990a, 1990b).

Neurotoxicity. Cells and tissues in the nervous system may be less radiosensitive, due to a lack of cell turnover or cellular regeneration, than faster regenerating cells of the gastrointestinal tract or pulmonary epithelium. Consequently, neuronal impairment as a result of radon alpha emissions is not expected. Therefore, studies which specifically or directly measure either pathological or functional damage to the nervous system following exposure to radon do not appear to be necessary at this time.

Epidemiological and Human Dosimetry Studies. Epidemiological studies of uranium and hardrock miner cohorts in the United States, Czechoslovakia, and Canada have demonstrated an increase in lung cancer deaths. A similar increase in lung cancer deaths also has been reported in epidemiological studies of iron ore, zinc-lead, tin, phosphate, niobium, and fluorspar miners. Many of the persons included in the various mining cohorts began work in underground mines prior to 1969 when recommendations for the maximum radon daughter levels were established in United States mines or prior to 1972 when yearly exposure levels (4 WLM) for United States miners were proposed (MSHA 1989). (The WLM represents a cumulative exposure; see Section 2.1, Introduction or Appendix B.) Since institution of these guidelines, radon daughter levels in United States mines have decreased. For example, the average radon and radon daughter levels in United States uranium mines were as high as 10,000 pCi/L of air (3.7×10^5 Bq/m³) in the early 1950s but dropped to less than 100 pCi radon-222/L of air (3.7×10^2 Bq/m³) by 1968 (Lundin et al. 1971). Among the Colorado uranium miner study group, only a relatively small number of persons who have been exposed to low levels of radiation have had a long follow-up (Archer 1980). A continuation of the follow-up on this group is needed to contribute to the evaluation of health hazards at levels at or below the current exposure standard for radon daughters or at the levels present in the environment. Continuation of the follow-up of epidemiological studies of New Mexico uranium miners is also necessary because smoking is less frequent in this group than in other groups studied. Continuation of studies of underground miners exposed to radon daughters to cover the full lifetimes of the cohort members would generate useful information. Additional information on the smoking habits of these cohorts is required to provide some

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insights on the complex interaction between radon daughters and cigarette smoking with regard to the induction of lung cancer. If exposure warrants, new population studies could be initiated or additional information could be gathered on previously defined populations.

The exact duration and level of exposure in human studies involving underground miners are not adequately characterized. Generally, approximate exposure is used based on environmental measurements of radon and radon daughters in the mines and individual work histories. The relationship between WLM and dose to the respiratory tract can differ in the occupational and environmental settings primarily due to differences in type and quantity of dust levels or ventilation rates. Additional evaluation of radon daughter dosimetry in various settings is needed to provide a better basis for estimating adverse health effects and correlating these effects with environmental exposures.

As with some of the chronic animal studies, exposures in most of the occupational miner cohorts consist of exposure to radon and radon progeny in the presence of other contaminants such as uranium ore dust, diesel-engine exhaust, or other mine pollutants. Only a few studies of lung cancer associated with environmental exposures to radon and radon daughters have been reported. These studies are primarily case-control or case-referent studies that involve a small number of subjects and have exposure estimates that are based on either surrogates for measurements or limited measurements. Additional studies of the extent of the hazard associated with environmental radon daughter exposures would provide useful information since radon is an ubiquitous substance, especially as they compare to estimates of the human health hazard based on the occupational setting.

Biomarkers of Exposure and Effect. Potential biomarkers of exposure may include the presence of radon progeny in urine, blood, bone, teeth, or hair. Although the detection of radon progeny in these media is not a direct measurement of an exposure level, estimates may be derived from mathematical models. Quantification of exposure to radon is further complicated by the fact that radon is an ubiquitous substance and background levels of radon and radon progeny are needed to quantify higher than "average" exposures. It has been reported (Brandom et al. 1978; Pohl Ruling et al. 1976) that chromosome aberrations in the peripheral blood lymphocytes may be a biological doseresponse indicator of radiation exposure. In addition, the frequency of abnormalities in sputum cytology has been utilized as an early indicator of radiation damage to lung tissue (Band et al. 1980). However, more extensive research is needed in order to correlate these effects with radon exposure levels and subsequent development of lung cancer or other adverse effects.

Absorption, Distribution, Metabolism, and Excretion. Some quantitative information is available on the absorption, distribution, and excretion of radon and radon daughters following inhalation and oral exposure, but information following dermal exposure is inadequate. Additional information

2. HEALTH EFFECTS

on the deposition patterns in airways for radon daughters and the relationship of these deposition patterns to the onset of respiratory disease is needed to enhance understanding of the disease process and delineate health protective measures to reduce deposition. In particular, further study of the role of ultra fine particles on lung doses is needed. More information on chronic exposure to low levels of radon in air and water is also necessary since this is the most common type of exposure for the majority of people who are exposed environmentally. Although absorption of radon via the oral route is known to occur, dosimetry of the gastrointestinal tract wall and the radiosensitivity of the wall is poorly understood. This information would be important in assessing the impact of oral exposure. Information on the storage of radon and radon daughters in fat tissue, especially following chronic exposure, is necessary to determine whether steady-state conditions can be achieved and the possibility of long-term bioaccumulation of radon daughters in body tissues. No information is available on the rate or extent of bioaccumulation of the long-lived radon daughter products such as lead-210 or polonium-210. This information is needed so that past exposures to radon may be quantified.

Comparative Toxicokinetics. Very little information is known about the comparative toxicokinetics of radon and radon daughters among animals and humans. However, similar target organs have been identified in both humans and laboratory animals exposed to radon and radon progeny. More information on respiratory physiology, target cells, lung deposition, and absorption of radon and radon daughters in different animal species is needed to clarify observed differences in species-sensitivity and tumor types. For example, rats generally develop lung tumors in the bronchioalveolar region of the lung while humans develop lung tumors in higher regions (tracheobronchial area). These studies could identify the appropriate animal model for further study of radon-induced adverse effects, although differences in anatomy and physiology of the respiratory system between animals and humans require careful consideration. Most of the information available on the toxicokinetics of radon and progeny has been obtained from studies of inhalation exposure. Studies on the transport of radon and progeny following oral and dermal exposures are needed to compare different routes of exposure.

2.8.3 On-going Studies

In recent years, concern over exposure to radon in both occupational and residential settings has increased. Consequently, numerous institutions have become involved in radon-related activities, partly to investigate the adverse health effects of radon. The following discussion is intended to be a representative sample of on-going research and is not an exhaustive list of the work in this area.

Several epidemiological studies pertaining to radon in homes and lung cancer incidence are underway. Comprehensive case-control studies of lung cancer among nonsmoking women are under investigation by M. Alavanja (NCI) in Missouri, Z. Hrubec (NCI) in Stockholm, Sweden, and New Jersey, J. Boice (NCI)

2. HEALTH EFFECTS

in Shenyang, China, J.H. Stebbings (Argonne National Laboratory), and G.W. Collman (National Institute of Environmental Health Sciences). All studies involve residential exposure to radon. Epidemiological studies of New Mexico uranium miners and tin miners are being conducted by J.M. Samet (University of New Mexico School of Medicine) and J.H. Lubin (NCI), respectively. C. Eheman (Centers for Disease Control) has been working with the National Park Service in assessing past and current radon exposure of employees who work in caves for the possibility of an epidemiology study of park service employees exposed to radon at home and in caves.

F.T. Cross (Pacific Northwest Laboratories) is studying the exposure-rate effect in radon daughter-induced carcinogenesis, and the role of oncogenes and the involvement of growth factors and receptors in radon-induced carcinogenesis. Similar studies on the influence of dose and dose-rate on carcinogenesis and other biological effects are being conducted by M. Terzaghi-Howe (Oak Ridge National Laboratories). F.T. Cross (Pacific Northwest Laboratories) is also continuing a series of animal experiments, in particular studies in rats with exposure to low cumulative doses of radon (more than 20 WLM). R.S. Caswell (National Institute for Standards and Technology) is developing a mechanistic model of the interaction of the alpha particles of radon and its daughters with the cells at risk in the lung.

L.A. Braby (Pacific Northwest Laboratories) is studying the malignant transformation of mammalian cells exposed to alpha particles that pass through the cell nuclei in an attempt to elucidate the mechanisms of action of radiation. The mechanisms of cell killing by alpha particles (M. Raju, Los Alamos Laboratories), cell neoplastic transformation from alpha particles (S.B. Curtis, Lawrence Berkeley Laboratory), and pulmonary tissue injury from radon/radon daughter exposure (T.M. Seed, Argonne National Laboratory) are also under investigation.

Radon-induced genotoxicity is another subject of interest under investigation. D.J. Chen (Los Alamos National Laboratories) is investigating the mechanistic basis for gene mutation induced by ionizing radiation in normal human fibroblasts. J.E. Turner (Oak Ridge National Laboratories) is examining the early physical and chemical changes produced by energetic alpha particles to elucidate the mechanisms involved in DNA damage. F.T. Cross (Pacific Northwest Laboratories) is studying the effects of exposure to radon on DNA and DNA-repair processes. M.N. Cornforth (Los Alamos National Laboratory) is attempting to provide quantitative data concerning both doseresponse and reparability of cytogenetic damage to human cells caused by ultra low doses of ionizing radiation. The types and yields of damage produced in mammalian-cell DNA by radon (J.F. Ward, University of California, La Jolla); radon-induced mutation in mammalian cells, utilizing a recombinant shuttle plasmid containing a target gene (S. Mitra, Oak Ridge National Laboratories); and cytotoxic, mutagenic, and molecular lesions induced in mammalian cells differing in DNA repair capabilities by low rates of radon and radon daughters (H.H. Evans, Case Western Reserve University) are under investigation. The direct effect of radon progeny and other high-LET alpha

2. HEALTH EFFECTS

radiation on DNA damage in respiratory epithelial cells (D.G. Thomassen, Lovelace Inhalation Toxicology Research Institute) and the biological consequences of high-LET alphas from radon on chromosomal and episomal DNA in human cells (J.E. Cleaver, University of California, San Francisco) are under investigation. Alteration in the DNA content of critical cells in the respiratory tract following exposure to radon and other aspects of radiation-induced damage to DNA is the current topic of study by many other investigators, such as N.F. Johnson (Lovelace Inhalation Toxicology Research Institute) and J.L. Schwarz (University of Chicago Medical Center).

Interaction of radon and radon progeny with other pollutants is another area of investigation. J.M. Daisey (University of California, Berkeley) and Y-S. Cheng (Los Alamos National Laboratories) are independently studying the complex interactions between radon and its progeny with other gaseous indoor pollutants. Further, F.J. Burns (New York University Medical Center) also is conducting experiments on rats to study lung cancer risk from inhalation of radon alone or in combination with other pollutants commonly found in the home environment. Interaction of radon and cigarette smoke in causing lung tumors in rats is being studied by S.H. Moolgavkar (Fred Hutchinson Cancer Research Center). The induction/promotion relationships associated with radon and cigarette smoke mixtures also are being studied by F.T. Cross (Pacific Northwest Laboratories).

Another factor that influences radon toxicity is the toxicokinetics of radon and radon progeny. Target regions of the lung for inhaled radon and radon progeny are being studied independently by R.R. Mercer (Duke University) and R.G. Cuddihy (Lovelace Inhalation Toxicology Institute) to determine the sensitivity of cell types located in the target regions. H-C. Yeh (Lovelace Inhalation Toxicology Research Institute) is quantifying radon deposition in the respiratory tract of humans, based on the mode of breathing, body size, and aerosol characteristics. B.S. Cohen (New York University Medical Center) is also conducting a similar study on humans and laboratory animals. A comparative morphometric study between dogs and humans is being conducted by E.S. Robbins (New York University Medical Center). W. Castleman, Jr. (Pennsylvania State University) is investigating the chemical and physical processes associated with radon distribution and effects. This would aid in assessing the mechanisms governing distribution, fate, and pathways of entry into biological systems. More studies related to the above topics are in progress by R.G. Cuddihy (Lovelace Inhalation Toxicology Research Institute), D.R. Fisher (Pacific Northwest Laboratory), N.H. Harley (New York University Medical Center), and D.L. Swift (Johns Hopkins University).

3. CHEMICAL AND PHYSICAL INFORMATION

3.1 CHEMICAL IDENTITY

The chemical formula and identification numbers for radon are listed in Table 3-1.

3.2 PHYSICAL AND CHEMICAL PROPERTIES

Important physical and chemical properties of radon are listed in Table 3-2. The radioactive properties of the important, short-lived daughters of radon-222 are listed in Table 3-3. The radon-222 decay series is depicted in Figure 3-1.

3. CHEMICAL AND PHYSICAL INFORMATION

TABLE 3-1. Chemical Identity of Radon

	Value	Reference
Chemical name	Radon	Windholz 1983
Isotopes	Radon-222 (Radon) Radon-220 (Thoron) Radon-219 (Actinon)	Cothorn 1987a
Trade name	No data	
Chemical formula	Rn	
Chemical structure	Not applicable	
Identification numbers:		
CAS Registry	14859-67-7 (radon-222) 22481-48-7 (radon-220) 14835-02-0 (radon-219)	EPA 1989
NIOSH RTECS	No data	
EPA Hazardous Waste	No data	
OHM/TADS	No data	
DOT/UN/NA/IMCO Shipping	No data	
HSDB	No data	
NCI	No data	

CAS = Chemical Abstract Service; NIOSH = National Institute for Occupational Safety and Health; EPA = Environmental Protection Agency; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; HSDB = Hazardous Substance Data Base; NCI = National Cancer Institute

3. CHEMICAL AND PHYSICAL INFORMATION

TABLE 3-2. Chemical and Physical Properties of Radon

Property	Value	Reference
Molecular weight	222(radon), 220(thoron), 219(actinon)	Cothorn 1987a
Color	Colorless	Cothorn 1987a
Physical state	Gas	Cothorn 1987a
Melting point	-71°C	Cothorn 1987a
Boiling point	-61.8°C	Cothorn 1987a
Density at 20°C	9.96×10^{-3} gm/cm ³	Cothorn 1987a
Odor	Odorless	Cothorn 1987a
Odor threshold	No data	
Solubility:		
Water at 20°C	230 cm ³ /L	NCRP 1988
Organic solvents	Organic liquid, slightly soluble in alcohol	Weast 1980
Partition coefficients:		
Log octanol/water	No data	
Log K _{oc}	No data	
Vapor pressure at -71°C	395.2 mmHg	Cothorn 1987a
Henry's Law constant	No data	
Autoignition temperature	No data	
Flash point	No data	
Flammability limits	No data	
Half-life		
Radon-222	3.823 days	Cothorn 1987a
Radon-220	55 seconds	Cothorn 1987a
Radon-219	4 seconds	Cothorn 1987a
Decay modes and energy, MeV		
Radon-222	α , 5.4897 γ , 0.512	US DHEW 1970
Radon-220	α , 6.29	US DHEW 1970
Radon-219	α , 6.42 α , 6.55 α , 6.82	US DHEW 1970
Specific activity (Ci/gm)		
Radon-222	3.6×10^4	US DHEW 1970
Radon-220	9.3×10^8	US DHEW 1970
Radon-219	1.3×10^{10}	US DHEW 1970

3. CHEMICAL AND PHYSICAL INFORMATION

TABLE 3-2 (Continued)

Property	Value	Reference
Decay products		
Radon-222	Polonium-218 Lead-214 Bismuth-214 Polonium-214 Lead-210 Bismuth-210 Polonium-210 Lead-206	Cothorn 1987a
Radon-220	Polonium-216 Lead-212 Bismuth-212 Polonium-212 Thallium-208 Lead-208	Cothorn 1987a
Radon-219	Polonium-215 Lead-211 Bismuth-211 Thallium-207 Lead-207	Cothorn 1987a

MeV = Million electron volts

3. CHEMICAL AND PHYSICAL INFORMATION

TABLE 3-3. Radioactive Properties of Radon-222 and Its Short-lived Progeny^a

Element	Historical Symbol	Principal Radiation(s)	Decay Energies (MeV)	Half-Life	Specific Activity (Ci/gm)
Radon-222	Rn	α	5.5	3.82 days	3.6×10^4
Polonium-218 ^b	RaA	α	6.0	3.05 min	2.8×10^8
Lead-214	RaB	γ, β	1.0	26.8 min	3.3×10^7
Bismuth-214	RaC	γ, β	3.3	19.7 min	4.5×10^7
Polonium-214 ^b	RaC'	α	7.7	164 μ sec	3.2×10^{14}

^aSource: BEIR IV 1988; US DHEW 1970.

^bIsotopes of primary radiological interest due to the potential for retention in the lung and subsequent alpha decay.

MeV = million electron volts

min = minutes

max = maximum

μ sec = microseconds

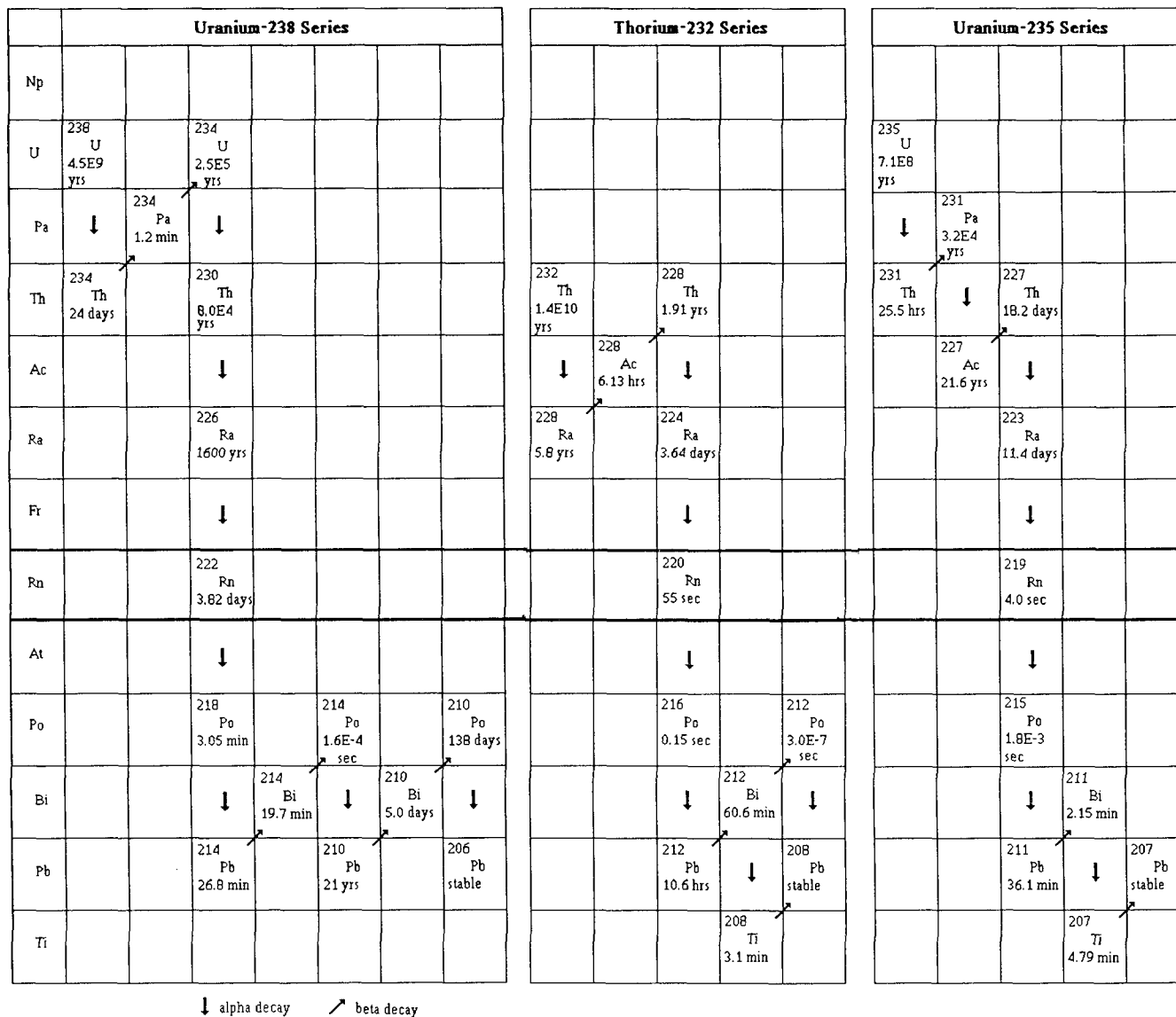


FIGURE 3-1. Uranium and Thorium Isotope Decay Series Showing the Sources and Decay Products of the Three Naturally-Occurring Isotopes of Uranium

Adapted from Aieta et al. 1987

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Radon is a naturally occurring radionuclide. The largest source of radon in the environment is due to the ambient levels produced by the widespread distribution of uranium and its decay products in the soil. Radon is a decay product of radium and part of the uranium decay chain (see Figure 3-1). Every square mile of surface soil, to a depth of 6 inches, contains approximately 1 gram of radium, which releases radon in small amounts to the atmosphere (Weast 1980). The ambient outdoor radon level goes through a daily cycle of concentrations ranging from 0.03 to 3.50 pCi radon-222/L (1.11 to 130 Bq/m³) of air with the average level in the United States being about 0.3 pCi radon-222/L (11.1 Bq/m³) of outdoor air (Martin and Mills 1973).

The amount of naturally occurring radon released to the atmosphere is increased in areas with uranium and thorium ore deposits and granite formations, which have a high concentration of natural uranium. It is the presence of granite formations that has greatly increased radon concentrations in eastern Pennsylvania and parts of New York and New Jersey. Sources of radon in the global atmosphere include natural emanation from radium in soil and water, uranium tailings, phosphate residues, coal, and building materials (NCRP 1984a). In a few locations, tailings have been used for landfills and were subsequently built on, resulting in possible increased exposure to radon (Eichholz 1987). There is also an increased radon concentration in spring water due to the deposition of radium isotopes in the sinter areas about hot springs, where it is coprecipitated with calcium carbonate or silica (NCRP 1975).

Radon has been produced commercially for use in radiation therapy but for the most part has been replaced by radionuclides made in accelerators and nuclear reactors. Radiopharmaceutical companies and a few hospitals pump the radon from a radium source into tubes called "seeds" or "needles" which may be implanted in patients (Cohen 1979). Research laboratories and universities produce radon for experimental studies.

4.2 IMPORT

Radon is not imported into the United States.

4.3 USE

Medical uses of radon in the United States began as early as 1914. Treatments were primarily for malignant tumors. The radon was encapsulated in gold seeds and then implanted into the site of malignancy. During the period of 1930 to 1950, radon seeds were used for dermatological disorders, including acne.

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

Radon therapy is still being studied and applied (Morken 1980). In many places in the world, water or air containing naturally high levels of radon-222 is used for therapeutic treatment of various diseases (Pohl- Ruling et al. 1982). These diseases include obliterative arteritis and atherosclerosis of lower extremities. In a few places, "radon mines" (caves with a high radon concentration in the air) are used as a health treatment . By law , these facilities cannot advertise; therefore , the number of people involved is quite small (Cohen 1979). A few of these caves are located in old Montana mines. Thousands of people seek medical cures through exposure to radon gas for ailments ranging from arthritis, asthma, and allergies to diabetes, ulcers, and cancer (Dobbin 1987). Radon "spas" are used in Europe for the treatment of hypertension and a number of other disorders. In the U.S.S.R. , about 25,000 radon baths are prescribed daily by the National Health System, and in Badgastein, Austria, every year 1 million radon thermal baths are taken (Usunov et al. 1981).

The prediction of earthquakes is fairly new technology that uses radon (Cothorn 1987b). The emanation of radon from soil and the concentration measured in groundwater appear to be good indicators of crustal activity. Other uses of radon include the study of atmospheric transport, and the exploration for petroleum or uranium (Cothorn 1987b).

4.4 DISPOSAL

Disposal of radon would only be applicable to those facilities producing and/or using it for medical or experimental purposes where its release may be controlled. Regulations regarding the land disposal of radionuclides are set forth in 10 CFR 61 (NRC 1988); however, there appear to be no regulations specific to radon. See Chapter 7 for a listing of regulations concerning radon. Radioactive effluents from facilities operating under a Nuclear Regulatory Commission (NRC) license are regulated by 10 CFR 20 (NRC 1988). The NRC effluent regulations and also disposal regulations regarding uranium tailings are listed in Table 7-1. Since radon is relatively short lived, it may be compressed and stored in tanks until it decays or, if the quantity is small , it may be absorbed on activated charcoal (Cember 1983). Particulate matter may be removed from the gas by a variety of different devices including detention chambers, adsorbent beds, and liquefaction columns. After filtration, the remaining radioactive particulates are discharged into the atmosphere for dispersion of the nonfilterable low levels of activity (Cember 1983).

Low-level radioactive waste produced as a result of using radon medically or experimental include paper towels, protective clothing, rags, animal excreta, and animal carcasses. This waste is often accumulated in containers. Combustible waste is incinerated and the activity is concentrated by burning away the substrate in which activity is held. The ashes are then either dispersed to the atmosphere or packaged for disposal into the sea or into the ground.

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Radon is a product of the natural radioactive decay of uranium, which occurs naturally in the earth's crust, to radium and then to radon. As radium decays, radon is formed and is released into small air or water-containing pores between soil and rock particles. If this occurs near the soil surface, the radon may be released to ambient air. Radon may also be released into groundwater. If this groundwater reaches the surface, most of the radon gas will quickly be released to ambient air, but small amounts may remain in the water. By far, the major source of radon is its formation in and release from soil and groundwater, with soil contributing the greater amount. Smaller amounts of radon are released from the near surface water of oceans, tailings from mines (particularly uranium and phosphate mines), coal residues and combustion products, natural gas, and building products, such as concrete and brick.

The ultimate fate of radon is transformation through radioactive decay. Radon decays only by normal radioactive processes, that is, an atom of radon emits an alpha particle resulting in an atom of polonium, which itself undergoes radioactive decay to other radon progeny. There are no sinks for radon; therefore, small amounts of radon are lost to the stratosphere.

In soil, radon is transported primarily by alpha recoil and mechanical flow of air and water in the soil. Alpha recoil is the process by which radon, when it is formed by radium emitting an alpha particle, actually recoils in the opposite direction from the path of particle ejection. After radon is released into the pore spaces, its ultimate release to ambient air is a function of the soil porosity and meteorological factors, such as precipitation and atmospheric pressure. Once radon is released to ambient air, its dispersion is primarily determined by atmospheric stability, including vertical temperature gradients and effects of wind.

Transport of radon in indoor air is almost entirely controlled by the ventilation rate in the enclosure. Generally, the indoor radon concentrations increase as ventilation rates decrease.

In groundwater, radon moves by diffusion and, primarily, by the mechanical flow of the water. Radon solubility in water is relatively low and, with its short radioactive half-life of 3.8 days, much of it will decay before it can be released from groundwater.

Radon levels in ambient air vary with the type of soil and underlying bedrock of the area. Available measurements indicate that the mean value for atmospheric radon in the contiguous United States is approximately 0.25 pCi radon-222/L of air (9 Bq/m³). However, measurements of air from the Colorado Plateau show radon levels up to 0.75 pCi radon-222/L of air (30 Bq/m³). Studies of indoor radon levels indicate an average concentration of from 1.5

5. POTENTIAL FOR HUMAN EXPOSURE

to 4.2 pCi radon-222/L of air (55 to 157 Bq/m³) (Alter and Oswald 1987; Nero et al 1986).

Groundwater supplies in the United States have been surveyed for radon levels. In larger aquifers, average radon concentrations were reported to be 240 pCi (8.8 Bq) radon-222/L of water, while in smaller aquifers and wells average levels were considerably higher (780 pCi radon-222/L of water; 28.9 Bq/L) (Cothorn et al. 1986). These differences in radon levels between large and small groundwater supplies are a reflection of the type of rock which surrounds them.

Measurements of radon in soil are expressed in terms of levels in soilgas. However, these measurements do not directly relate to rates of radon released to the atmosphere. Factors which affect radon soil-gas levels include radium content, soil porosity, moisture content, and density. Technically, measurement of soil-gas is difficult and there are few studies which report such data.

Delivered dose of radon and its progeny can only be estimated by complex mathematical models. Therefore, exposure, both occupational and environmental, will be discussed, primarily in terms of radon levels in the air. However, some estimates of daily intake have been made. Daily intake of radon originating outdoors is estimated to be 970 pCi (36 Bq) radon-222/day (Cothorn et al. 1986). Exposure from indoor radon is higher due to concentration of levels from lack of ventilation and other factors. Total daily intake of radon originating indoors is estimated as 8,100 pCi (300 Bq) radon-222/day, assuming a breathing rate of 20 m³/day. However, daily intake is dependent on time spent in and outdoors and on breathing rate (Cothorn et al. 1986).

Radon releases to the environment (primarily indoor levels) from groundwater also contribute to environmental exposures. The daily intake of radon originating from drinking water only is estimated at 100 to 600 pCi (3.7 to 22.2 Bq) radon-222/day both from ingestion of drinking water and inhalation of radon released from drinking water (Cothorn et al. 1986). Radon releases from building materials contribute little to potential exposure.

Occupational exposure to radon results from employment in uranium and other hard rock mining, or in phosphate mining. Persons engaged in uranium mining are believed to receive the largest exposures, although the number of persons employed in uranium mining has steadily decreased in the past 9 years. Measurements of radon progeny in these mines from 1976 to 1985 showed annual mean concentrations of 0.11 to 0.36 WL (22 to 72 pCi radon-222/L of air; 800 to 2,664 Bq/m³) (NIOSH 1987). However, levels in phosphate mines measured during the same period showed a larger range of mean levels (0.12 to 1.20 WL; 24 to 240 pCi radon-222/L of air; 888 to 8,880 Bq/m³). Radon exposure in underground mines is continually being reduced due to improved engineering controls (NIOSH 1987).

5. POTENTIAL FOR HUMAN EXPOSURE

5.2 RELEASES TO THE ENVIRONMENT

5.2.1 Air

Because of the extended half-lives of uranium and radium and their abundance in the earth's surface, radon is continually being formed in soil and released to air. This normal emanation of radon from radium-226 in soils is the largest single source of radon in the global atmosphere (NCRP 1984a). Using average emanation rates from available measurements, Harley (1973) estimated soil emanation of radon to be on the order of 2×10^3 Ci (7.4×10^{19} Bq) radon-222/year. This estimation is equivalent to 1,600 pCi (60 Bq/cm²) radon/cm² soil/year (Harley 1973). The emanation rate at a particular location is highly variable and is affected by many factors, including barometric pressure, composition of soil, and soil moisture and temperature. Usually, less than 10% of radon in upper soil layers is released to the atmosphere (Vilenskiy 1969). Some radon is released by plants through evapotranspiration. However, the amount released has not been estimated (Taskayev et al. 1986).

Groundwater that is in contact with radium-containing rock and soil will be a receptor of radon emanating from the surroundings. When the groundwater reaches the surface by natural or man-made forces, this radon will be released to air. Although most of the radon present in groundwater will decay before reaching the surface, groundwater is still considered to be the second largest source of environmental radon and is estimated to contribute 5×10^8 Ci (1.85×10^{19} Bq) radon-222/year to the global atmosphere (NCRP 1984a). Radon is also released from oceans, but only from the near surface water, and in amounts that are an order of magnitude less than that from groundwater. Radium in oceans is largely restricted to the sediments where it cannot affect atmospheric levels of radon (Harley 1973).

Tailings from uranium mines and residues from phosphate mines both contribute to global radon in the approximate amount of 2 to 3×10^6 Ci (7.4×10^{16} to 1.11×10^{17} Bq) radon-222/year. Although these sites are not numerous (in 1984 there were 50 sites containing uranium tailings), emanation rates to air may be substantial. It is estimated that 20% of the radon formed in tailings is released and that emanation rates can be as high as 1,000 pCi (37 Bq) radon/m²/second (NCRP 1984a).

Coal residues and combustion products, as well as natural gas, each contribute to atmospheric radon levels to a minor extent (NCRP 1984a). Coal and natural gas, at the time of combustion, release radon to air. Coal residues, such as fly ash, contribute very small amounts to atmospheric radon.

Some building materials release very small amounts of radon. However, the major source of radon in single family dwellings is the soil directly under the building (NCRP 1984b).

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According to the VIEW database, 14 NPL sites reportedly contain radon above background levels (VIEW 1989). The frequency of these sites within the United States can be seen in Figure 5-1. Quantification of the levels found is not available. However, the majority of the radon released would be to air.

5.2.2 Water

The amount of radon released to groundwater is a function of the chemical concentration of radium-226 in the surrounding soil or rock and in the water itself. High radon activity is associated with groundwater surrounded by granitic rock. The physical characteristics of the rock matrix are important also since it is believed that much of the radon released diffuses along microcrystalline imperfections in the rock matrix (Hess et al. 1985). Radon is rarely found in surface water due to the fact that it is rapidly released to the air when the water reaches surface levels (Michel 1987).

In a reanalysis of published data, Hess et al. (1985) reported a geometric population average of 187 pCi (6.9 Bq) radon-222/L of water in over 6,000 samples of groundwater supplies for public use. In contrast, samples of surface water supplies indicated that the average level of radon was 1 pCi (0.037 Bq) radon-222/L of water.

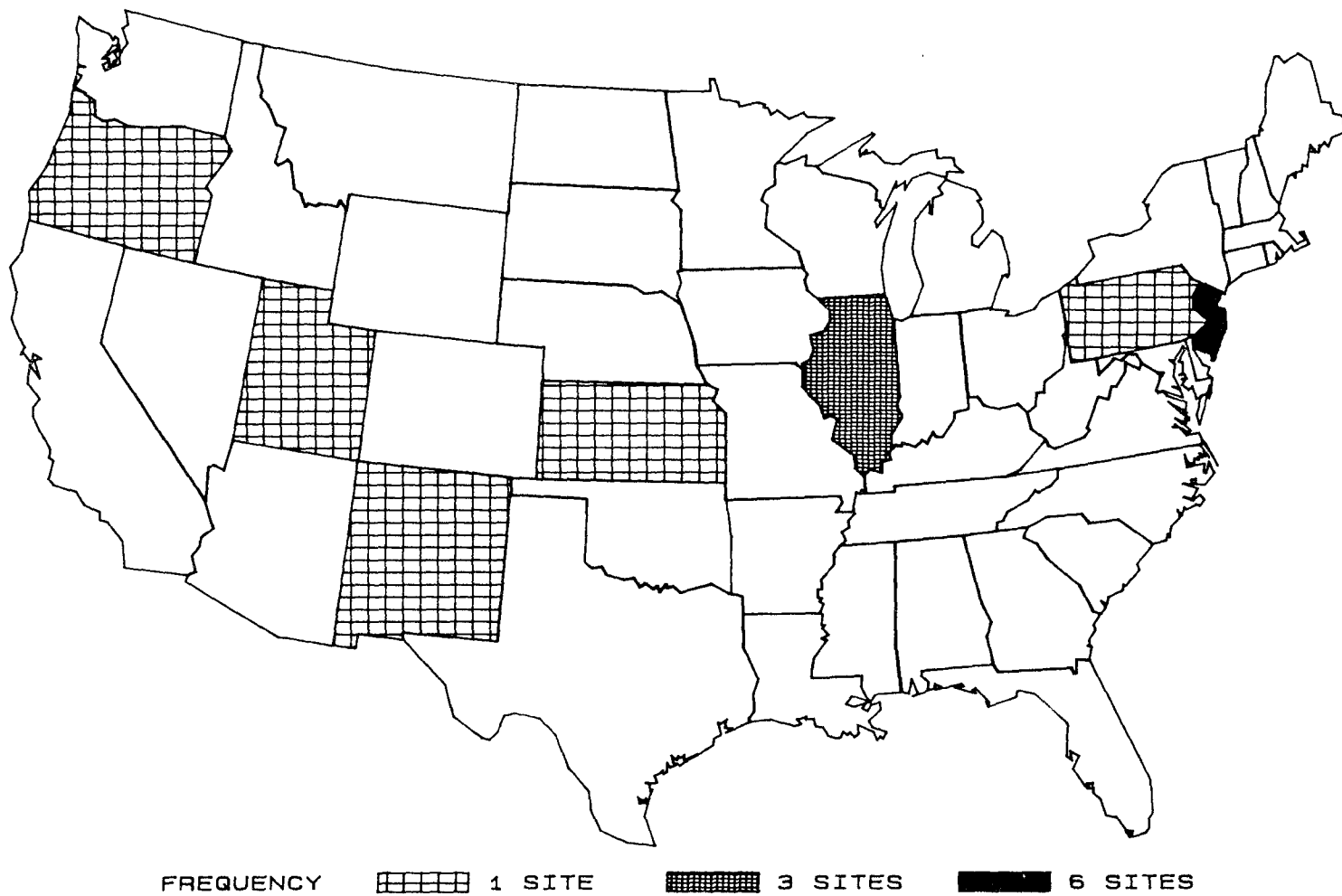
5.2.3 Soil

As stated in Section 5.2.1, soil is the primary source of radon. As such, radon is not released to soil but is the result of radioactive decay of radium-226 within the soil. The radon concentration in the soil is a function of the radium concentration, the soil moisture content, the soil particle size, and the rate of exchange of air with the atmosphere (Hopke 1987). Hopke (1987) states that normal soil-gas radon measurements are in the range of 270 to 675 pCi radon-222/L of air (10,000 to 25,000 Bq/m³). However, levels exceeding 10,000 pCi radon-222/L of air (370,000 Bq/m³) have been documented.

5.3 ENVIRONMENTAL FATE

5.3.1 Transport and Partitioning

Emanation is the process by which radon is transported from a solid to a gas or liquid medium. At the soil particle level, radon gas is transferred from soil particles into pore spaces (gas- or liquid-filled spaces between soil particles) primarily by alpha recoil. Alpha recoil occurs after radium decays by emitting an alpha particle. After the particle is ejected, the resulting radon atom actually recoils in the opposite direction. Alpha recoil results in breaking of chemical bonds in the solid, physically moving the atom to a different position, and damaging the crystal structure. The radon atom may recoil to a position from which it will not be released (embedded in the same particle or in another particle) or may recoil into the pore space from



FREQUENCY  1 SITE  3 SITES  6 SITES

FIGURE 5-1. FREQUENCY OF SITES WITH RADON CONTAMINATION

5. POTENTIAL FOR HUMAN EXPOSURE

which it may move by diffusion or convection toward the soil surface. If the pore space is filled with liquid, any radon atoms which recoil into it will travel slower than those that recoil into air-filled spaces (Michel 1987). Although alpha recoil is believed to be the major process of radon release from solids, diffusion from very small pores near the particle surfaces and along imperfections of the crystalline structure of the particle also occurs.

Once radon enters the pore space, it is transported by diffusion, convection, and flow of rain and groundwater. The diffusion constant for radon is approximately 10^{-2} cm² per second in air and 10^{-5} cm² per second in water (WHO 1983). These constants indicate that diffusion of radon is a relatively slow process and that its movement is, therefore, primarily accomplished by mechanical transport of air and water in the pore space.

The actual release of radon from the pore space or soil-gas to ambient air is called exhalation. The rate of this process is a function of many variables including the concentration of radon in the soil-gas, the soil porosity, and meteorological factors such as precipitation and variations in atmospheric pressure (WHO 1983).

Behavior of radon at the interface between soil and ambient air is not well understood. However, once radon reaches a height of approximately 1 meter above the soil surface, its dispersion is predominantly determined by atmospheric stability (Cohen 1979). This stability is a function of vertical temperature gradient, direction and force of the wind, and turbulence. Temperature inversions in the early morning act to produce a stable atmosphere which keeps radon concentrations near the ground. Solar radiation breaks up the inversion, leading to upward dispersion of radon which reverses with radiant cooling in late afternoon (Gesell 1983). In addition, general trends in air turbulence lead to maximum levels in air in the early autumn and early winter (when turbulence is generally less) and lower levels in air in the spring due to increased turbulence (Michel 1987). In the absence of these factors, radon levels in air decrease exponentially with altitude (Cohen 1979). This phenomenon has been studied by sampling and many models have been derived to fit the data (WHO 1983).

Sources of indoor radon include entry of amounts released beneath the structure? entry in utilities such as water and natural gas, and release from building materials. The greatest contribution is that from radon released from soil or rock (Nero 1987). Entry occurs primarily by bulk flow of soilgas driven by small pressure differences between the lower part of the house interior and the outdoors. The pressure differences are primarily due to differences in indoor/outdoor temperature and the effects of wind (Nero 1987).

Transport of radon in indoor air is primarily a function of the ventilation rate of the enclosure. Under most conditions, the indoor radon concentration increases in direct proportion to the decrease in ventilation rates (WHO 1983). However, in some indoor radon studies, radon concentrations showed greater variability than could be accounted for by ventilation rates.

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This was said to suggest that the strength of the radon source was the main cause of the wide range in observed indoor radon levels (Nero 1987). Behavior of radon in enclosed areas has also been extensively studied and predicted by modeling (Eichholz 1987; Jonassen 1975).

Transport of radon daughters indoors has also been extensively modeled. Transport is primarily a function of the rate of attachment of radon daughters to particles, the concentration and size of the particles, and the rate of deposition. A major complication of modeling both radon and radon daughter transport indoors is that the ventilation rate acts both to increase flow of radon into the structure and to remove radon and radon daughters from the structure (Nero 1987). Ventilation rate also acts on the movement of air indoors causing variations in radon concentrations from room to room, as well as within a room.

Mechanisms for transport of radon in groundwater are much less complex than those for other media. In fact, transport of radon in groundwater is accomplished by diffusion and, primarily, by the mechanical flow of groundwater. As previously stated, the diffusion coefficient of radon in water is sufficiently low so that diffusion is only important for movement in very small spaces (such as pore spaces). The solubility of radon in water is relatively low (230 cm³ radon-222/L of water at 20°C) and, due to radon's relatively short half-life, much of it will have decayed before the groundwater reaches the surface. However, that remaining in solution will be quickly released to ambient air once it is encountered. In areas where groundwater has high levels of radon, release from groundwater may significantly affect ambient air levels.

5.3.2 Transformation and Degradation

5.3.2.1 Air

Regardless of the surrounding media, radon is transformed or degrades only by radioactive decay. There are no sinks for radon, and it is estimated that only negligible amounts escape to the stratosphere (Harley 1973). Therefore, degradation proceeds by alpha-emission to form polonium-218. As stated in Table 3-2, the half-life of radon is 3.82 days. The half-lives of the progeny are much shorter, ranging from approximately 0.0002 seconds for polonium-214 to 30 minutes for lead-214.

5.3.2.2 Water

See Section 5.3.2.1.

5.3.2.3 Soil

See Section 5.3.2.1.

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5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

5.4.1 Air

The most comprehensive compilation of data on radon levels in outdoor air was reported by Gesell (1983). Measurements were taken over the continental United States, Hawaii, and Alaska. The highest concentrations were found in the Colorado Plateau, which is a region containing high levels of uranium as well as mines and uranium tailings. Measurements in this region ranged from 0.5 to 0.75 pCi radon-222/L of air (18.5 to 30 Bq/m³). Average values from the continental United States ranged from 0.12 to 0.3 pCi radon-222/L of air (4.4 to 11 Bq/m³). Based on these and other data, Michel (1987) states that the mean value for atmospheric radon in normal geological areas of the contiguous United States is approximately 0.25 pCi radon-222/L of air (9 Bq/m³) with a range of 0.1 to 0.4 pCi radon-222/L of air (4 to 15 Bq/m³).

Data reported by Fisenne (1987) indicate variability of radon levels with time. In continuous data (9 years of hourly measurements), both diurnal and seasonal patterns were observed. Diurnal variations showed an early morning peak and a drop in the afternoon. Seasonally, levels were highest in early autumn and lowest in early spring.

Radon concentrations in air decrease with height from the soil surface. Several investigators have measured radon levels in the troposphere. Machta and Lucas (1962) measured 0.007 pCi radon-222/L of air (0.26 Bq/m³) at 25,000 feet. Comparable measurements have been taken over Alaska and the southwestern United States (Harley 1973).

Although there are many studies which undertake to quantify radon in indoor air, the work of Nero et al. (1986) is the most comprehensive and the most often cited. This study reanalyzed up to 38 small data sets, of which 22 were considered unbiased. Biased data were those collected from areas where high radon concentrations were expected. On the basis of the unbiased data, the geometric mean of indoor radon levels was reported to be approximately 0.9 pCi radon-222/L of air (33 Bq/m³). These data implied an arithmetic average concentration of 1.5 pCi radon-222/L of air (56 Bq/m³). Distribution studies of household levels indicated that from 1% to 3% of single-family houses may exceed 8 pCi radon-222/L of air (296 Bq/m³). In this study many of the measurements were made in main-floor living rooms or average living areas (Nero et al. 1986).

Indoor radon levels were measured in homes located in the Reading Prong area of Pennsylvania. This area has an unusual abundance of homes with high radon concentrations that is presumed to be from geologically produced emanation of radon. Indoor levels of radon in this area ranged from 4 to 20 pCi/L (150 to 740 Bq/m³) in 29% of the homes to >80 pCi/L (3,000 Bq/m³) in 1% of the homes (Fleischer 1986).

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Other studies of indoor radon levels were summarized in NCRP (1987). The median levels ranged up to 18.9 pCi radon-222/L of air (700 Bq/m³) in homes in Butte, Montana (Israeli 1985). A study by Cohen (1986) reported results from 453 indoor sites in 42 states and showed a mean of 1.62 pCi radon-222/L of air (60 Bq/m³) with a median of 1.08 pCi radon-222/L of air (40 Bq/m³).

5.4.2 Water

In a nationwide survey by the EPA, almost 2,500 public drinking water supplies were sampled (nonrandom) with most of these serving greater than 1,000 people. Results of this survey were used to estimate the mean population-weighted radon levels in public groundwater systems by state (Cothorn et al. 1986). Average concentrations for United States groundwater were estimated to be 240 pCi radon-222/L of water (8.8 Bq/L) for larger systems (>1,000 persons served), and for smaller systems 780 pCi radon-222/L of water (28.9 Bq/L). The nationwide average for all groundwater samples tested was 351 pCi radon-222/L of water (13 Bq/L). When surface water supplies were taken into consideration, due to the fact that their radon levels are essentially zero, the average radon concentration in all community water supplies was estimated to range from 54 to 270 pCi radon-222/L of water (2 to 10 Bq/L) (Michel 1987). The highest levels reported were in smaller groundwater systems in Maine which averaged 10,000 pCi radon-222/L of water (370 Bq/L); lowest average levels were found in larger systems in Tennessee with levels of 24 pCi radon-222/L (8.9 Bq/L).

This same relationship, i.e., radon concentrations in groundwater increasing with decreasing system size, was previously reported by Hess et al. (1985). This correlation is believed to reflect a relationship between system size and aquifer composition. Those rock types that are associated with high radon levels (granitic rock) do not form aquifers large enough to support large systems. However, smaller systems may tap into such aquifers.

Crystalline aquifers of igneous and metamorphic rocks generally have higher radon levels than other aquifer types with granites consistently showing the highest levels. Average radon levels in water from granite aquifers are usually 2,703 pCi radon-222/L of water (100 Bq/L) or greater (Michel 1987). This is indicated in the data of Cothorn et al. (1986) which report the following trends in groundwater radon levels: in New England and the Piedmont and Appalachian Mountain Provinces, where igneous and metamorphic rocks form the aquifers, concentrations are in the range of 1,000 to 10,000 pCi radon-222/L of water (37 to 370 Bq/L); in the sandstone and sand aquifers which extend from the Appalachian Mountains west to the Plains, concentrations are generally less than 1,000 pCi radon-222/L of water (37 Bq/L).

5.4.3 Soil

Because radon is a gas, its occurrence in soil is most appropriately referred to as its occurrence in "soil-gas", which is in the gas or waterfilled space between individual particles of soil. Factors that affect radon

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soil-gas levels include radium content and distribution, soil porosity, moisture, and density. However, soil as a source of radon is seldom characterized by radon levels in soil-gas, but is usually characterized directly by emanation measurements or indirectly by measurements of members of the uranium-238 series (National Research Council 1981). Radon content is not a direct function of the radium concentration of the soil, but radium concentration is an important indicator of the potential for radon production in soils and bedrock. However, Michel (1987) states that average radium content cannot be used to estimate radon soil-gas levels, primarily due to differences in soil porosity.

Despite such caveats, theoretical rates of radon formation in soil have been estimated as demonstrated by the following:

"Consider a cube which is 1 meter in each dimension. Using rounded numbers, if the average density of the soil is 2.0 grams per cubiccentimeter and the average radium-226 concentration is 1.0 pCi/g (0.037 M/g), the cube will contain 2 million grams of soil and 2×10^{-6} Ci (7.4×10^4 Bq) of radium-226. This corresponds to the production of 7.4×10^4 radon atoms per cubic-meter per second and the escape of 7,400 atoms per square meter per second, in rough correspondence to the average measured value." (Nevissi and Bodansky 1987).

For a discussion of uranium-238 and radium-226 levels in soil, see the ATSDR Toxicological Profiles for Uranium and Radium (ATSDR 1990a, 1990d).

Only two soil-gas measurements for United States locations were found in the literature: one from Spokane, Washington, with soil-gas radon from 189 to 1,000 pCi radon-222/L of air (7,000 to 37,000 Bq/m³) in soils formed from coarse glacial outwash deposits with 2.3 ppm uranium, and the other from Reading Prong, New Jersey, with soil-gas radon levels from 1,081 to 27,027 pCi radon-222/L of air (40,000 to 1,000,000 Bq/m³) (Michel 1987). Hopke (1987) states that normal soil-gas radon measurements are in the range of 270 to 675 pCi radon-222/L of air (10,000 to 25,000 Bq/m³). It is reported that radon-222 levels increase with soil depth, reaching a probable maximum at about 800 cm below ground level (Jaki and Hess 1958).

5.4.4 Other Media

Limited information exists to indicate that plants absorb both radium-226 and radon-222 from the soil layer and that these compounds are translocated to above ground plant parts (Taskayev et al. 1986). However, there is little information on the quantitative contribution of this process to exposure from ingestion of plant crops or of emanation rates from these plants. One measurement of emanation rates from field corn was located in the literature. Radon-222 release from leaves was reported to be 2.47×10^{-4} pCi (9.15×10^{-6} Bq)/cm²/sec. This emanation rate was 1.8 times greater than the emanation rate from local soil (Pearson 1967).

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5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

In the following section, exposure to radon is discussed in terms of environmental levels not in terms of actual or estimated dose. The estimation of whole body or target tissue dose of radionuclides is extremely complex and must be accomplished by mathematical models for the specific radionuclide. Although such models are available to estimate whole body and target tissue dose for radon, discussion of these lies outside the scope of this document. For a discussion of these models the reader is referred to NCRP (1984a) or BEIR IV (1988).

The general population is exposed to radon by inhalation both outdoors and indoors. Outdoor levels, also referred to as ambient or background levels, are the result of radon emanating from soil. These levels vary widely with geographical location, depending on factors such as the radium content of soil and soil porosity and moisture content. However, a reasonable average for near ground level is suggested by Eichholz (1987) to be on the order of 0.150 pCi radon-222/L of air (5.55 Bq/m³). Michel (1987) states that the mean value for atmospheric radon in the contiguous United States is approximately 0.24 pCi radon-222/L of air (8.88 Bq/m³) with a range of 0.11 to 0.41 pCi radon-222/L of air (4.07 to 15.2 Bq/m³). Cothorn et al. (1986) report a daily intake of radon originating outdoors of approximately 1,000 pCi (36 Bq) radon-222/day based on data derived from the United Nations Scientific Committee on the Effects of Atomic Radiation (1982) and assuming an inhalation rate of 20 m³/day of air containing 0.05 pCi/L (1.8 Bq/m³) radon-222. Because of the gaseous nature of radon, radon levels will decrease with increasing height from the soil surface. Studies of this vertical gradient indicate that a child who is 0.5 m tall would be exposed to 16% more radon than an adult who is 1.5 m tall (Michel 1987).

In contrast to the average ambient levels of radon, which are usually quite low, levels indoors are found to be greater than ambient outdoor levels. This is due to enhancement and it is believed to be a function of the following: movement of radon from underlying soil and rock through the foundation of the building, release of radon from water and utility use, radon emanation from radium-containing structural materials, and rate of ventilation (NCRP 1984b). The contribution of each of these to the overall indoor radon level is difficult to assess, except qualitatively. It has been determined that elevated indoor radon levels are primarily due to radon emanation from underlying soil (Eichholz 1987). The actual indoor levels are greatly affected by other parameters such as composition of the foundation materials and the ventilation rate of the enclosed area. Two of the largest indoor monitoring efforts in the United States reported arithmetic mean levels ranging from 1.5 to 4.2 pCi radon-222/L of air (55 to 157 Bq/m³) (Alter and Oswald 1987; Nero et al. 1986). The data from Alter and Oswald (1987) are limited in that the dwellings do not represent a random sample and individual measurement values were reported rather than average concentrations from a residence. Cothorn et al. (1986) report daily intake of radon originating indoors of 8,100 pCi (300 Bq) radon-222/day based on data derived from the

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United Nations Scientific Committee on the Effects of Atomic Radiation (1982) and assuming indoor radon levels of 0.4 pCi radon-222/L of air (15 Bq/m³).

Although the primary source of indoor radon is emanation from soil, release of radon from the water supply may contribute to indoor levels. Nazaroff et al. (1987) performed an analysis which combined information on water use, efficiency of radon release from water, house volumes, and ventilation rates to determine the impact on indoor radon levels. Their analysis estimated that use of groundwater contributes an average of 2% to the mean indoor radon concentration in houses. As with levels in other media, levels of radon in groundwater vary greatly. In areas with high groundwater levels, the relative contribution to indoor radon levels will increase accordingly. Cothorn et al. (1986) report a daily intake of radon originating from drinking water of 100 to 600 pCi (3.7 to 22.2 Bq) radon-222/day assuming that consumption was 2 L/day of groundwater.

Radon ingestion from drinking water has been of less concern relative to the dose received from inhalation. Due to the short residence time in the stomach, ingested radon contributes a small dose to the stomach when compared to that delivered to the lungs from inhalation of radon released from water. Although the dose to the stomach is small, it is not significant (Eichholz 1987).

The contribution of building materials to indoor radon is estimated to be low in comparison with amounts which emanate directly from soil and rock. In general, among common building materials, concrete releases more radon than other materials. The potential of radon release from building materials is expressed by the radium content and the radon emanation rate, which is a function of pressure, temperature, porosity, and radon concentration. The radon emanation rate of concrete utilized in the United States is estimated to be in the range of 1.2 to 3.4x10⁻⁴ pCi (4.3 to 12.6x10⁻⁶ Bq) radon-222/kg/second (Michel 1987).

Persons who are occupationally exposed to radon are those employed in mining, primarily mining of uranium and hard rock (NIOSH 1987). NIOSH reports that in 1986, 22,499 workers were employed in metal and nonmetal mines in the United States. However, the number of underground uranium mines has steadily decreased from 300 in 1980 to 16 in 1984. In turn, the number of employees in underground uranium mines has decreased from 9,000 in 1979 (3,400 of whom worked underground 1,500 hours or more) to 448 in 1986 (NIOSH 1987). Measurements of radon progeny concentrations in these mines from 1976 to 1985 showed annual geometric mean concentrations in uranium mines of 0.11 to 0.36 WL (equivalent to 22 to 72 pCi radon-222/L of air [800 to 2,664 Bq/m³] assuming an equilibrium factor of 0.5), with 95th percentile levels ranging up to 2.73 WL (546 pCi radon-222/L of air; 20,202 Bq/m³). Annual geometric mean levels in phosphate mines for the same period were 0.12 to 1.20 WL (24 to 240 pCi radon-222/L of air [888 to 8,880 Bq/m³]) with 95th percentile levels as high as 1.69 WL (338 pCi radon-222/L of air; 12,506 Bq/m³). Measurements in uranium/vanadium mines showed annual geometric mean concentrations similar to

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those in uranium mines. However, 95th percentile levels ranged up to 4.80 WL (960 pCi radon-222/L of air [3.6×10^4 Bq/m³]), which was the highest annual concentration reported among the different types of mines (NIOSH 1987). Estimates of annual cumulative radon progeny exposures indicated that of the 1,405 underground uranium miners working in 1984, 28% had exposures greater than 1.0 WL (200 pCi radon-222/L of air; 7,400 Bq/m³).

Radon exposure in underground mines has been vastly reduced by installation of improved engineering controls. In New Mexico mines the median annual exposure in 1967 of 5.4 WLM was reduced to 0.5 WLM by 1980 due to this technique (Eichholz 1987).

Several researchers have attempted to correlate levels of lead-210 in bone with cumulative radon daughter exposure. Eisenbud et al. (1969) employed in vivo techniques to measure lead-210 in the skull of nonoccupationally exposed and occupationally exposed individuals. Exposure for miners was derived from mine records and compared to that estimated from a model. Their results showed that the amount of lead-210 deposited, regardless of temporal considerations, will be within a factor of two of that deposited if exposure is assumed to be uniform over time. In addition, they reported that a burden of 2,000 pCi (74 Bq) is equivalent to a calculated cumulative exposure of approximately 800 WLM.

Blanchard et al. (1969) reported a positive correlation between the log of lead-210 concentration in post-mortem derived bone and the log of estimated miners' cumulative exposure. However, more lead-210 was observed in bone than was predicted by the model utilized. Furthermore, a linear correlation was observed between lead-210 levels in blood and that in bone; however, for both of these analyses sample numbers were small (n=11 to 22). Another study (Clemente et al. 1984) has analyzed the correlation between lead-210 in human teeth and environmental radon levels in various countries. This analysis reported that for the incremental increase in lead-210 in teeth, a value of 3.24×10^{-3} pCi (1.2×10^{-4} Bq) radon-222/gm of tissue has been associated with a lifetime exposure to 1 WLM. All of these studies are limited by the difficulty in estimating exposure to individuals on the basis of mine levels and worker histories (often related by next of kin). Such estimates, although unavoidable, introduce considerable uncertainty into these analyses. In addition, lead-210 can be introduced in cigarette smoke, food, and ambient air, thus confounding results of studies (NCRP 1984b).

5.6 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Populations with potentially high exposures include those occupationally exposed as previously described (see Section 5.5). In addition, certain populations are exposed to elevated environmental levels, such as those resulting from emanation from soil in the Reading Prong area of Pennsylvania (soil-gas of up to 27,000 pCi [1.0×10^3 Bq] radon-222/L soil-gas) and from release from groundwater in certain areas in Maine (levels up to 180,000 pCi [6.7×10^3 Bq] radon-222/L of water) (Hess et al. 1983). Communities that are

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very near uranium or phosphate mill tailing piles may have increased environmental radon levels. In addition, in some areas mill tailings have been used for landfills and were subsequently developed (for example, Grand Junction, Colorado). Persons in these communities could be exposed to levels of radon exceeding normal background levels.

5.7 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA 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 radon is available. Where adequate information is not available, ATSDR, in conjunction with the 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 radon.

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 or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.7.1 Identification of Data Needs

Physical and Chemical Properties. Information is available on the physical and chemical properties of radon, and parameters that influence the behavior of radon in the environment have been determined. Therefore, no data needs are identified concerning physical and chemical properties of radon.

Production, Use, Release, and Disposal. The production of radon occurs directly from a radium source either in the environment or in a laboratory environment. The disposal of gaseous radioactive effluents has been documented. Increased radon concentrations have been detected in waste generated by uranium and phosphate mining; therefore, these sites should be monitored on a continual basis. Although there are regulations for disposal of radionuclides in general, there are none that specifically address disposal of radon contaminated materials. Further research on the disposal of radon attached to charcoal, which is used in radon monitoring indoors, would be beneficial.

Environmental Fate. Information is available on the environmental fate of radon in air and water and on the transport of radon in environmental media. Factors which affect the partitioning of radon from soil or water to air have been identified. However, rates of flux from one media to another are rarely reported. The emanation rate of radon from soil is uncertain. Additional information on the behavior of radon at the soil-air interface, as well as soil-gas measurements, would facilitate a better understanding of the

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emanation rate of radon from soil. Movement of radon into and within homes and the influence of meteorological conditions on this movement should be investigated. Study of radon movement would enhance understanding of potential indoor exposures. Transformation of radon has been adequately characterized. There is limited information on the uptake of radon by plants. Additional research of this phenomenon is needed in order to determine the effects of exposures which might be incurred from ingestion of food.

Bioavailability from Environmental Media. Radon and radon progeny are known to be absorbed from air and water and information is available which characterizes the relative contribution of various media to levels of radon in air and water. Further studies of bioavailability are not necessary at this time.

Food Chain Bioaccumulation. Information on bioaccumulation of radon and radon daughters in the food chain is not available. Therefore, the potential for bioconcentration in plants, aquatic organisms or animals, or for biomagnification in the food chain is unknown. However, due to the short half-life of radon, it would not tend to bioaccumulate. Studies of the bioaccumulation of radon in the food chain are not necessary at this time.

Exposure Levels in Environmental Media. Some information is available on exposure levels in environmental media, however, most of this information is from areas with higher than average levels of radon. Although levels in groundwater, primarily for public water supplies, have been more comprehensively reported than levels in ambient air, on-going monitoring efforts for both media are necessary for quantification of human exposure. Comprehensive data on levels of radon in ambient air are needed in order to assess potential human exposure.

Exposure Levels in Humans. There is a lack of comprehensive information associating radon and radon progeny levels monitored in the environment and exposure of the general population. Although levels of radon may be measured in exhaled air, the relationship of that amount exhaled to the exposure level can be estimated only by use of mathematical models. Concentrations of radon progeny are measurable in urine, blood, bone, teeth, and hair; however, these levels are not direct measurements of levels of exposure. These estimates also may be derived through use of mathematical models. Studies are needed to characterize the utility of these biomarkers of exposure.

Exposure Registries. No exposure registries for radon were located. This compound is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The compound will be considered in the future when chemical selection is made for subregistries to be established. 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 the exposure to this compound. The Hanford Environmental Foundation in Richland, Washington, maintains a registry

5. POTENTIAL FOR HUMAN EXPOSURE

of United States uranium miners and millers. The data in the registry are derived from autopsy material and include exposure information. Since uranium decays to radon, this exposure registry on miners and millers may provide information on radon exposure.

5.7.2 On-going Studies

S.D. Schery (New Mexico Institute of Mining and Technology) is studying the fundamental processes influencing release of radon isotopes from porous media and the physical properties of radon isotopes which affect their behavior in enclosed environments. The hypothesis that plant functions increase soil-radon flow to the atmosphere, thus measurably reducing the flow into subsurface areas is under investigation by F.W. Whicker (Colorado State University). A.B. Tanner (U.S. Department of Interior) is completing a qualitative study on the range and variability of diffusive and advective/convective transport of radon and its controlling factors at selected areas. Further, a study designed to provide information on the transport pathway of radon and radon progeny, their charge state, and the effect of clustering on decay products is being conducted by M.G. Payne (Oak Ridge National Laboratories). Computer models are also being developed in an attempt to simplify studies on radon transport within and from soils into the atmosphere and structures (P.C. Owczarski, Pacific Northwest Laboratories) and to unify theories of radon emanation and transport in the soil (K.K. Nielson, Rogers and Associates Engineering Corporation).

Investigations of factors which influence transport or mobility of radon and its progeny from rocks/soils to the environment or homes are underway by K.K. Turekian (Yale University), D. Thomas (University of Hawaii at Manoa), R.H. Socolow (Center for Energy and Environmental Studies, Princeton University), and C.S. Dudney (Oak Ridge National Laboratories). The study by Dudney included New Jersey and the Tennessee Valley areas with high background levels. The influence of season, heating fuel, tobacco smoking, and building characteristics on indoor air pollutant levels is being studied by R.H. Rainey (Office of Power, Tennessee Valley Authority). Research Triangle Institute (Research Triangle Park, North Carolina) is presently studying both modeling and measurement of radon in houses.

One aspect of radon mobility, in relation to groundwater, is being studied by O.S. Zepezca (U.S. Geological Survey). He is determining the factors which control radionuclide transport and fate in groundwater in the Newark basin and southern coastal plains of New Jersey, and the mechanism of release of radionuclides to groundwater or retention in aquifer solids.

G. Harbottle (Brookhaven National Laboratories) is studying the mobility and chemical behavior of radium in the soil and the processes involved in the emanation of radon. The dynamic behavior of radon and radon daughters will be studied in controlled laboratory environments (J.S. Johnson, Lawrence Livermore National Laboratory).

6. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting and/or measuring and monitoring radon in environmental media and in biological samples. The intent is not to provide an exhaustive list of analytical methods that could be used to detect and quantify radon. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used to detect radon in environmental samples are the methods approved by federal agencies 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 a trade association such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that refine previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

6.1 BIOLOGICAL MATERIALS

Urine analysis and whole body counting are most frequently performed to monitor exposure to radon. Tooth enamel and bone are also used as indicators of radon exposure. The longer-lived radioactive isotopes, lead-210 and polonium-210, are generally used as a means of estimating exposure to the short-lived radon-222 decay products. It is generally known that lead-210 is deposited primarily in bone with a relatively long biological half-life, which enables it to reach transient radioactive equilibrium conditions with its descendant, polonium-210 (Clemente et al. 1984). The short half-lives of radon and the daughters, polonium-218 through polonium-214, preclude their detection through normal bioassay techniques which typically require a day or more after the sample has been collected before counting can commence (Gotchy and Schiager 1969).

Direct measurements of emerging gamma rays typically use the gamma rays from lead-210 and rely on decays occurring in lung or bone tissues. This method utilizes a system of either sodium iodide or germanium detectors placed over the body in a well-shielded room (Crawford-Brown and Michel 1987). For past exposures, the lead-210 and polonium-210 concentrations in the urine are determined by counting the number of decays on a sodium iodide system or by use of liquid scintillation.

Applying these concentrations to estimate the exposure an individual might have received introduces large uncertainties. Pharmacokinetic metabolic models are used to detail the movement of the radionuclides within the organs and tissues of the body (EPA 1988a; ICRP 1978). Several additional models are described in BEIR IV (1988). The uncertainties involved make it unlikely that these approaches can yield estimates of exposure to within better than a factor of four to five, particularly when values specific to individuals (rather than populations) are required (Crawford-Brown and Michel 1987). Analytical methods for determining radon in biological samples are given in

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Table 6-1. These methods provide indirect measurements of radon; i.e., the activity emitted from radon and radon progeny is detected and quantified.

6.2 ENVIRONMENTAL SAMPLES

Radon has been recognized as a health hazard for many years, primarily for uranium miners. Recently, unusually high radon concentrations have been found in several areas of the country, particularly Northeast Pennsylvania. This has prompted nationwide concern and interest in the measurement of radon.

To aid in the effort in standardizing procedures for making accurate and reproducible measurements and to ensure consistency, the EPA has issued two reports recommending measurement techniques and strategies. The 1986 report, "Interim Radon and Radon Decay Product Measurement Protocols," provides procedures for measuring radon-222 concentrations with continuous monitors, charcoal canisters, alpha-track detectors and grab techniques (EPA 1986). The second report, "Interim Protocols for Screening and Follow-up Radon and Radon Decay Product Measurements" (EPA 1987a), outlines the recommendations for making reliable, cost effective radon measurements in homes (Ronca-Battista et al. 1988).

There are several generalizations about the measurement of radon which apply regardless of the specific measurement technique used. Radon concentrations in the same location may differ by a factor of two over a period of 1 hour. Also, the concentration in one room of a building may be significantly different than the concentration in an adjoining room. Therefore, the absolute accuracy of a single measurement is not critical, but improvements in sampling methodology would be helpful. Since radon concentrations vary substantially from day to day, single grab-type measurements are generally not very useful, except as a means of identifying a potential problem area, and indicating a need for more sophisticated testing.

An initial screening for radon can be made with activated charcoal. After a potential problem is identified, more accurate measurements can be made using additional techniques.

There are three main methods of determining the air concentration of radon: an instantaneous measurement, or grab sample, a continuous readout monitor which continually registers the concentration, and a time averaged concentration where the sample is obtained over a relatively long period of time and yields a single, average concentration for an extended time period from a few days to a week or more.

Several techniques to measure air concentrations are outlined by Breslin (1980). Most of the techniques for measuring radon use the fact that both radon-222 and the short-lived daughters are alpha-emitting nuclides. The sample is collected and taken back to the laboratory for "alpha-counting" or an alpha-detector is taken to the field for on-site measurement. There are

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TABLE 6-1. Analytical Methods for Determining Radon in Biological Materials

Sample Matrix	Sample Preparation	Analytical Method	Sample Detection Limit	Accuracy	Reference
Tooth	Clean and dry tooth; dry overnight and grind to fine powder; separate enamel from dentin and compress into pellets; coat with titanium nitride	PIXE for PB-210 content	No data	0.5 ppm	Anttila 1987
Urine	Wet ash in HNO ₃ -NClO ₄ , electrostatic precipitation	Alpha spectrometry	0.1 pCi (3.7x10 ⁻³ Bq)	85%	Gotchy and Schiager 1969
Blood	Wet ash	Alpha spectrometry	0.1 pCi (3.7x10 ⁻³ Bq)	85%	Gotchy and Schiager 1969
Blood	Wet ash and plate on disk	Autoradiography of tracks, using nuclear emulsion	No data	No data	Weissbuch et al. 1980
Bone	Extract fat with anhydrous benzene; wet ash	Scintillation counter	No data	No data	Blanchard et al. 1969
Bone	<u>In vivo</u>	Whole body counting gamma spectroscopy	No data	No data	Eisenbud et al. 1969
Tissue	Place in tared test tube	Scintillation counter	No data	No data	Nussbaum and Hursh 1957

PIXE = proton induced X-ray emission analysis

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several ways to measure alpha decay. A scintillation flask is one of the oldest and most commonly used methods. The flask is equipped with valves which are lined with a phosphor (silver-activated zinc sulfide) and emit light flashes when bombarded with alpha particles. Other methods draw the air through a filter (or filters) for a variety of time intervals and then count the number of alpha-decays occurring on the filter. EPA (1986) and NCRP (1988) reports provide more in-depth discussions of these methods.

EPA (1986) outlines the most common procedures for making measurements and describes conditions that should exist at the time of the measurement. The simplest, least expensive method of radon measurement is with charcoaladsorption. One side of the container is fitted with a screen to keep the charcoal in and allow the radon to diffuse into the charcoal. The adsorbed radon subsequently decays, depositing decay products in the charcoal. After exposure for 3 to 7 days the canister is sealed and sent to the laboratory where the charcoal is placed directly into a gamma detector.

For continuous monitoring of an indoor environment, a common method is the scintillation cell method. The monitor pumps air into a scintillation cell after passing it through a particulate filter that removes dust and radon decay products. As the radon in the cell decays, the decay products plate out on the interior surface of the scintillation cell. The alpha particles emitted by radon and radon daughters strike the coating on the inside of the cell causing scintillations to occur. These scintillations are detected by a photomultiplier tube in the detector and an electrical signal is generated.

Another widely used method is solid state nuclear track detection. In the case of radon, an alpha track detector is used. It consists of a small piece of plastic enclosed in a container with a filter-covered opening. Alpha particles in the air strike the plastic and produce submicroscopic damage tracks. At the end of the measurement period the plastic is placed in a caustic solution that accentuates the damage tracks. The tracks are then counted using a microscope or automated counting system.

Radon daughter aerosols may also be measured by using electrets. These are uniformly charged surfaces which provide a collection medium with a builtin electrostatic force to attract the aerosols, therefore avoiding use of a pump (Khan and Phillips 1984). Deposition is quantified with an alpha counter.

There are two primary methods for measuring radon in aqueous samples, the radon bubbler/alpha scintillation cell method and the liquid scintillation counting method. There are problems associated with sample collection for the radon bubbler/alpha scintillation method. One problem is that the sample in the field must be collected in a glass bubbler flask that must then be transported to the lab. Due to transport and handling problems, sample results may be compromised. Therefore, the liquid scintillation counting method is more commonly used. A description of the liquid scintillation counting method is given in Table 6-2. The greatest analytical uncertainty of

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TABLE 6-2. Analytical Methods for Determining Radon in Environmental Samples

Sample Matrix	Sample Preparation	Analytical Method	Sample Detection Limit	Accuracy	Reference
<u>Air</u>					
Radon					
	Adsorb onto activated charcoal, 2 to 7 days	Gamma spectroscopy	1.3 pCi/m ³ (0.048 Bq/m ³)	No data	Cohen and Nason 1986
	Adsorb onto activated charcoal; extract with toluene; gently shake	Liquid scintillation	0.21-0.37 pCi/m ³ (0.007-0.014 Bq/m ³)	0.094 of true concentration	Prichard and Marlen 1983
	<u>Scintillation Cell</u> Allow air to enter detection chamber through millipore filter until equilibrated, or collect sample in bag (Mylar or Tedlar), transfer to chamber ASAP	ZnS(Ag) scintillation/photomultiplier tube	0.1 pCi/m ³ (0.004 Bq/m ³)	No data	Crawford-Brown and Michel 1987
	Diffuse through filter into detector housing; collections with electret	TLD chip	89 pCi/m ³ (3.30 Bq/m ³)	0.95-1.08 of true concentration	Maiello and Harley 1987
	<u>Two-Filter Method</u> Draw air into fixed length tube with entry and exit filters; monitor exit filter activity	ZnS(Ag) scintillation/photomultiplier tube	0.011 pCi/m ³ (<0.001 Bq/m ³)	90%	Schery et al. 1980

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TABLE 6-2 (Continued)

Sample Matrix	Sample Preparation	Analytical Method	Sample Detection Limit	Accuracy	Reference
<u>Solid State Nuclear Track Detector</u>					
	Diffuse through a filter into a cup containing alpha track material (cellulose nitrate film) for up to 1 year; etch in acidic or basic solution operated upon by an alternating electric field	Microscopic examination of damaged material	14 pCi/m ³ (0.519 Bq/m ³)	No data	NCRP 1988
Radon progeny					
	Draw air through filter for a sampling time of 5 to 10 minutes	Gross alpha counting	1.1 pCi/m ³ (0.041 Bq/m ³)	No data	NCRP 1988
	Draw air through filter at a known flow rate for specified time (10 m to 1 hr)	Alpha spectrometry	1.1 pCi/m ³ (0.041 Bq/m ³)	70%	NCRP 1988
	Charge surface electrostatically to attract aerosols	Alpha spectrometry	1.1 pCi/m ³ (0.041 Bq/m ³)	70%	NCRP 1988
<u>Soil</u>					
	Dry in 55°C oven for 24 hours; place 5 grams in 20 ml borosilicate glass scintillation. Cover with 10 ml distilled water; allow soil to become wet; add 5 ml high-efficiency mineral oil; allow to age 30 days	Scintillation counter	No data	No data	Rangarajan and Eapen 1987; Wadach and Hess 1985

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TABLE 6-2 (Continued)

Sample Matrix	Sample Preparation	Analytical Method	Sample Detection Limit	Accuracy	Reference
	None	Track etch detector buried 30 cm deep	No data	No data	Rangarajan and Eapen 1987
<u>Water</u>					
Radon					
	Pass carrier gas through samples in a bubbler flask to purge out dissolved radon; transfer radon to evacuated scintillation cell	Scintillation counter	1.4 pCi/L (52 Bq/m ³)	90%	Crawford-Brown and Michel 1987
	Inject into glass vial containing liquid scintillation solution; shake vigorously	Liquid scintillation counter	10 pCi/L (370 Bq/m ³)	No data	Crawford-Brown and Michel 1987
	Direct measurement	Gamma ray spectroscopy	10 pCi/L for 1 L sample (370 Bq/m ³)	No data	Yang 1987

TLD = Thermoluminescent Dosimeter

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these methods is due to sampling. Since radon is a gas, care must be taken to prevent its escape from the sample (Crawford-Brown and Michel 1987). A discussion of measurement techniques in water may be found in the report by Crawford-Brown and Michel (1987).

There has been little attempt to standardize a method for measuring radon in soil. However, a method which utilizes liquid scintillation counting for determining concentration is given by Wadach and Hess (1985). A description of this method may be found in Table 6-2.

The accuracy of any measurement will depend upon the calibration of the instrument used. The calibration of an instrument determines its response to a known amount or concentration of radioactivity. This allows a correlation to be made between the instrument reading and the actual amount or concentration present. A range of activities of radium-226 standard reference materials (SRM) is available from the U.S. Department of Commerce, National Bureau of Standards (NBS) as solutions for calibrating detection systems. Also, an elevated radon atmosphere may be produced in a chamber, and samples drawn and measured in systems previously calibrated by radon emanation from an NBS radium-226 SRM. Other radon detectors may then be filled from or exposed in the chamber and standardized based on this "secondary" standard (NCRP 1988). Analytical methods for measuring radon in environmental samples are given in Table 6-2. These methods provide indirect measurements of radon; i.e., the activity emitted from radon and radon progeny is detected and quantified.

6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA 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 radon is available. Where adequate information is not available, ATSDR, in conjunction with the 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 radon.

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 or eliminate the uncertainties of human health assessment. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Methods are available to measure the presence of radon progeny in urine, blood, bone, teeth, and hair. However, the accuracy and the sample detection limits for

6. ANALYTICAL METHODS

the majority of these methods are unknown and should be determined so that exposure to radon may be quantified. In addition, measurement of radon gas in expired air should be possible by methods such as gas chromatography. However, descriptions of any such methods have not been found in the literature.

The frequency of abnormalities in sputum cytology has been utilized as a possible early indicator of radiation damage to lung tissue (Band et al. 1980; Brandom et al. 1978; Saccomanno et al. 1974). The accuracy and precision of this measurement is not known.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods are available which allow for the quantification of radon in air, water, and soil. However, methods for the measurement of radon concentrations in soil-gas are limited. The ability to accurately measure soil-gas is needed to provide a better understanding of the emanation rate of radon gas from soil.

6.3.2 On-going Studies

Although several analytical methods for measuring and determining radon and radon progeny from environmental media or biological tissues exist, several on-going studies have been identified in the Federal Radon Activities Inventory. There are a number of animal studies underway. Occupationally exposed individuals are continually monitored in order to obtain more accurate models and better measurement techniques.

R. Cole (National Institute for Standards and Technology (NIST)) is currently upgrading the primary radon measurement system which constitutes the national radon measurement standard. D.R. Fisher (Pacific Northwest Laboratories) is attempting to develop analytical methods which will aid in calculating microdosimetry within the tracheobronchial epithelium after inhalation of radon and radon progeny. Also, R.S. Caswell (NIST) is working on a related investigation but with cells at risk in other parts of the lung and adjacent areas.

J.R. Duray (Chem Nuclear Geotech, formerly United Nuclear Corporation Geotech) is testing instruments and devices in order to develop accurate and reliable measurements of annual indoor and outdoor levels of radon and radon daughters. I. Pomerantz (EPA) is investigating analytical techniques to measure certain radionuclides, which would aid in monitoring radon levels in drinking water; whereas K. Fox (EPA) is working on radon removal techniques for community water supplies in New Hampshire. Another area of concern is the development of analytical methods for measuring radon in buildings. C. Arnolts (Department of Housing and Urban Development) is investigating techniques builders can use to identify the presence of radon in a given building, and T. Peake (EPA) is working on methodology which would identify areas with a high potential for radon exposure. M. Ronca-Battista (EPA)

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reports the steps that are being taken to revise EPA radon measurement protocols and includes a new method for measuring indoor radon and radon progeny concentrations.

7. REGULATIONS AND ADVISORIES

International and national regulations and guidelines pertinent to human exposure to radon are summarized in Table 7-1. Recommendations for radiation protection for people in the general population as a result of exposure to radiation in the environment are found in the Federal Radiation Guidance (FRC 1960) and ICRP No. 26 (ICRP 1977). National guidelines for occupational radiation protection are found in the "Federal Radiation Protection Guidance for Occupational Exposure" (EPA 1987b). This guidance for occupational exposure supersedes recommendations of the Federal Radiation Council for occupational exposure (FRC 1960). The new guidance presents general principles for the radiation protection of workers and specifies the numerical primary guides for limiting occupational exposure. These recommendations are consistent with the ICRP (ICRP 1977).

The basic philosophy of radiation protection is the concept of ALARA (As Low As Reasonably Achievable). As a rule, all exposure should be kept as low as reasonably achievable and the regulations and guidelines are meant to give an upper limit to exposure. Based on the primary guides, guides for Annual Limits on Intake (ALIs) have been calculated (EPA 1988a; ICRP 1979). The ALI is defined as "that activity of a radionuclide which, if inhaled or ingested by Reference Man (ICRP 1975), will result in a dose equal to the most limiting primary guide for committed dose" (EPA 1988a) (see Appendix B).

7. REGULATIONS AND ADVISORIES

TABLE 7-1. Regulations and Guidelines Applicable to Radon-222

Agency	Description	Value	Reference
<u>International</u>			
<u>Guidelines</u>			
WHO	Remedial action should be considered if exceeded in building	2,700 pCi/L (99,900 Bq/m ³) EER	Suess 1988
WHO	Remedial action should be considered without long delay if exceeded in building	10,800 pCi/L (399,600 Bq/m ³) EER	Suess 1988
WHO	Should not be exceeded before remedial action	5.4x10 ⁴ pCi yr/L (2.00x10 ⁶ Bq yr/m ³) EER	Suess 1988
ICRP	Maximum cumulative occupational exposure	4.8 WLM/yr	Bodansky et al. 1987
ICRP	Annual limit for intake by inhalation	0.02 Joules/yr	ICRP 1977
<u>National</u>			
<u>Regulations</u>			
a. Air			
Environmental and indoor			
EPA	Average annual atmospheric release rate from residual radioactive material from inactive uranium processing sites	20 pCi/m ² /sec (0.74 Bq/m ² / sec)	EPA 1988b (40 CFR 190 192.02)
EPA	Annual average concentration should not be increased by more than this due to inactive uranium processing sites	0.5 pCi/L (18.5 Bq/m ³)	EPA 1988b (40 CFR 190 192.02)

7. REGULATIONS AND ADVISORIES

TABLE 7-1 (Continued)

Agency	Description	Value	Reference
EPA	Maximum average annual radon decay product concentration (including background) as a result of inactive uranium processing sites, in any occupied or habitable building	0.02 WL	EPA 1988b (40 CFR 190 192.12)
EPA	Maximum radon decay product concentration (including background) as a result of inactive uranium processing sites, in any occupied or habitable building	0.03 WL	EPA 1988b (40 CFR 190 192.12)
NRC	Maximum permissible concentration in air released to unrestricted areas	3×10^{-9} $\mu\text{Ci}/\text{cm}^3$ (1.1×10^{-4} Bq/cm ³)	NRC 1988 ^a (10 CFR 20)
Mine and cave			
OSHA	Individual exposure limit	4.0 WLM/yr	OSHA 1988 (41 CFR 57.5038)
OSHA	Monitor workspace at least once yearly	0.1 WL	OSHA 1988 (41 CFR 57.5087)
OSHA	Monitor workspace quarterly	0.1 - 0.3 WL	OSHA 1988 (41 CFR 57.5037)
OSHA	Monitor workspace weekly and maintain exposure records on all exposed employees	> 3.0 WL	OSHA 1988 (41 CFR 57.5037)
OSHA	Immediate corrective action to lower the concentration	1.0 WL	OSHA 1988 (41 CFR 57.5041)
MSHA	Maximal cumulative dose	4.0 WLM/yr	MSHA 1989 (30 CFR 57)

7. REGULATIONS AND ADVISORIES

TABLE 7-1 (Continued)

Agency	Description	Value	Reference
MSHA	Instantaneous maximum	1.0 WL	MSHA 1989 (30 CFR 57)
b. Drinking water			
NRC	Maximum permissible concentration in water released to unrestricted areas	No data	
c. Food			
		No data	
d. Nonspecific media			
EPA	Reportable quantity	<u>Ci (Bq)</u>	EPA 1989b
	Radon-220	0.1 (3.7x10 ⁹)	40 CFR 302
	Radon-222	0.1 (3.7x10 ⁹)	
<u>Guidelines</u>			
a. Air			
ANSI/ ASHRAE	Annual average concentration of indoor radon	0.01 WL	Natl. Res. Council 1981
EPA	Upper level of exposure in home	4 pCi radon-222/L of air (148 Bq/m ³)	Deluca and Castronovo 1988
EPA	Desired target concentration in the home	0.02 WL	Bodansky et al. 1987
EPA	Action within several months	0.1 WL	Bodansky et al. 1987
EPA	Remedial action must be undertaken	8 pCi radon-222/L of air (300 Bq/m ³)	Deluca and Castronovo 1988
EPA	Occupational ALI for inhalation ^b	4 WLM	EPA 1988a
NCRP	Remedial action level	2 WLM/yr	NCRP 1984b
NIOSH	Recommended exposure limit	1.0 WLM/yr	NIOSH 1987

7. REGULATIONS AND ADVISORIES

TABLE 7-1 (Continued)

Agency	Description	Value	Reference
NIOSH	Average work shift concentration limit	0.083 WL	NIOSH 1987
b.	Drinking water	No data	
c.	Food	No data	
	<u>State</u>		
	<u>Regulations and Guidelines</u>		
a.	Air		
Alaska	Regulated hazardous substance	No data	Alaska 1988
New Mexico	Immediate corrective action or withdraw workers	1.0 - 1.4 WL	New Mexico 1981 (NMMSC 11)
New Mexico	Withdraw workers until corrective action is taken or until reduced to 1.0 WL or less	> 1.4 WL	New Mexico 1981 (NMMSC 11)
New Mexico	Maximal cumulative exposure to workers	4.0 WLM/yr	New Mexico 1981 (SIM Rule 76-1)
New Mexico	Instantaneous maximum to workers	1.0 WL	New Mexico 1981 (SIM Rule 76-1)
New Mexico	Exposure records should be kept for employees entering areas with this concentration	0.3 WL	New Mexico 1981 (SIM Rule 71-2)
New Mexico	Respiratory devices to prevent inhalation of radon daughters should be worn by workers	1.0 WL	New Mexico 1981 (SIM Rule 78-1(2a))
New Mexico	Respiratory devices to prevent inhalation of radon gas and daughters should be worn by workers	10 WL	New Mexico 1981 (SIM Rule 78-1(2a))

7. REGULATIONS AND ADVISORIES

TABLE 7-1 (Continued)

Agency	Description	Value	Reference
b. Water/Drinking water			
Maine		10,000 pCi/L (3.7×10^5 Bq/m ³)	FSTRAC 1988
Rhode Island		10,000 pCi/L (3.7×10^5 Bq/m ³)	FSTRAC 1988

^aThe Nuclear Regulatory Commission limits in 10 CFR 20 are in the process of revision.

^bThe ALI recommended by the EPA is numerically identical to that recommended by the ICRP Publication 30 (ICRP 1979).

ALI = Annual Limit of Intake

ANSI/ASHRAE = American National Standards Institute/American Society of Heating, Refrigerating and Air Conditioning

EER = Equilibrium Equivalent Radon

EPA = Environmental Protection Agency

FSTRAC = Federal-State Toxicology and Regulatory Alliance Committee

ICRP = International Commission on Radiological Protection

MSHA = Mine Safety and Health Administration

NCRP = National Council for Radiation Protection and Measurements

NRC = Nuclear Regulatory Commission

NIOSH = National Institute for Occupational Safety and Health

NMMSA = New Mexico Mine Safety Code

OSHA = Occupational Safety and Health Administration

SIM = State Inspector of Mines, New Mexico

WHO = World Health Organization

WL = Working Level

WLM = Working Level Month

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9. GLOSSARY

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

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 radiation imparts some or all of its energy to any material through which it passes.

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.

Absorption Coefficient -- Fractional decrease in the intensity 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 deposition of energy in the absorber. The total absorption coefficient is the sum of individual energy absorption processes. (See Compton Effect, Photoelectric Effect, and Pair Production.)

Linear Absorption Coefficient -- A factor expressing the fraction of a beam of x or gamma radiation absorbed in a unit thickness of material. In the expression $I = I_0 e^{-\mu x}$, I_0 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.

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

Activity -- The number of nuclear transformations occurring in a given quantity of material per unit time. (See Curie.)

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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 particulate whose activity is the median for the entire aerosol.

Acute Exposure -- Exposure to a chemical for a duration of 14 days or less, as specified in the toxicological profiles.

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

Adsorption Coefficient (Koc) -- The ratio of the amount of a chemical adsorbed per unit weight of organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (Kd) -- The amount of a chemical adsorbed by a sediment or soil (i.e., the solid phase) divided by the amount of chemical in the solution phase, which is in equilibrium with the solid phase, at a fixed solid/solution ratio. It is generally expressed in micrograms of chemical sorbed per gram of soil or sediment.

Alpha Particle -- A charged particle emitted from the nucleus of an atom. An alpha particle has a mass charge equal in magnitude to that of a helium nucleus; i.e., two protons and two neutrons and has a charge of +2.

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

Atomic Mass -- 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} gm. (Symbol: u)

Atomic Number -- The number of protons in the nucleus of a neutral atom of a nuclide. 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 atoms of an element expressed in atomic mass units.

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 -- Radiation arising from radioactive material other than that under consideration. Background radiation due to cosmic rays and natural

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radioactivity is always present. There may also be background radiation due to the presence of radioactive substances in building materials.

Becquerel (Bq) -- International System of Units unit of activity and equals one transformation (disintegration) per second. (See Units.)

Beta Particle -- Charged particle emitted from the nucleus of an atom. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 or -1.

Biologic Effectiveness of Radiation -- (See Relative Biological Effectiveness.)

Bone Seeker -- Any compound or ion which migrates in the body preferentially into bone.

Branching -- The occurrence of two or more modes by which a radionuclide can undergo radioactive decay. For example, radium C can undergo α or β^- decay, ^{64}Cu can undergo β^- , β^+ , 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 -- The production of electromagnetic radiation (photons) by the negative acceleration that a fast, charged particle (usually an electron) undergoes from the effect of an electric or magnetic field, for instance, from the field of another charged particle (usually a nucleus).

Cancer Effect Level (CEL) -- The lowest dose of chemical 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 is designated as "K-electron capture," "L-electron capture," etc.

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 capable of inducing cancer.

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

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

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Ceiling Value (DL) -- A concentration of a substance that should not be exceeded, even instantaneously.

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

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 of energy less than the incident photon.

Containment -- The confinement of radioactive material in such a way that it is prevented from being dispersed into the environment or is released only at a specified rate.

Contamination, Radioactive -- Deposition of radioactive material in any place where it is not desired, particularly where its presence may be harmful.

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

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

Counter, Geiger-Mueller -- Highly sensitive, gas-filled radiation-measuring device. It operates at voltages sufficiently high to produce avalanche ionization.

Counter, Scintillation -- The combination of phosphor, photomultiplier tube, and associated circuits for counting light emissions produced in the phosphors by ionizing radiation.

Curie -- A unit of activity. One curie equals 3.7×10^{10} nuclear transformations per second. (Abbreviated Ci.) Several fractions of the curie are in common usage.

Megacurie -- One million curies. Abbreviated MCi.

Microcurie -- One-millionth of a curie (3.7×10^4 disintegrations per set). Abbreviated μ Ci.

Millicurie -- One-thousandth of a curie (3.7×10^7 disintegrations per set). Abbreviated mCi.

Nanocurie -- One-billionth of a curie. Abbreviated nCi.

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Picocurie -- One-millionth of a microcurie (3.7×10^{-2} disintegrations per second or 2.22 disintegrations per minute). Abbreviated pCi; replaces the term μc .

Decay, Radioactive -- Transformation of the nucleus of an unstable nuclide by spontaneous emission of charged particles and/or photons.

Decay Chain or Decay Series -- A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus, the parent, decays into a daughter 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. Sometimes, to distinguish the sequence, the daughter of the first daughter is called the granddaughter, etc.

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

Decay Product, Daughter Product -- A new isotope 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) may be either radioactive or stable.

Delta Ray -- Energetic or swiftly moving electrons ejected from an atom during the process of ionization. Delta rays cause a track of secondary ionizations along their path.

Developmental Toxicity -- The occurrence of adverse effects on the developing organism that may result from exposure to a chemical 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 lifespan of the organism.

Disintegration Constant -- The fraction of the number of atoms of a radioactive nuclide which decay in unit time; constant in the equation $N = N_0 e^{-\lambda t}$, is the symbol for the decay where N_0 is the initial number of atoms present, and N is the number of atoms present after some time, t . (See Decay Constant.)

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

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

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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 protection. It expresses all radiations on a common scale for calculating the effective absorbed dose. It is defined as the product of the absorbed dose in rad and certain modifying factors. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

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

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

Median Lethal Dose (MLD) -- Dose of radiation required to kill, within a specified period, 50 percent of the individuals in a large group of animals or organisms. Also called the LD₅₀.

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 and Rad.)

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

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

Dose-distribution Factor -- A factor which accounts for modification of the dose effectiveness in cases in which the radionuclide distribution is nonuniform.

Dose Rate -- Absorbed dose delivered per unit time.

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Dosimetry -- Quantification of radiation doses to individuals or populations resulting from specified exposures.

Early Effects (of radiation exposure) -- Effects which 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. (Abbreviated: eV, 1 eV = 1.6×10^{-12} erg.)

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

Energy -- Capacity for doing work. "Potential energy" is the energy inherent in a mass because of its spatial relation to other masses. "Kinetic energy" is the energy possessed by a mass because of its motion; MKSA unit: $\text{kg}\cdot\text{m}^2/\text{sec}^2$ or joules.

Binding Energy -- The energy represented by the difference in mass between the sum of the component parts and the actual mass of the nucleus.

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 average energy lost by ionizing radiation in producing an ion pair in a gas. For air, it is about 33.73 eV.

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

Enriched Material -- (1) Material in which the relative amount of one or more isotopes of a constituent has been increased. (2) Uranium in which the abundance of the ^{235}U isotope is increased above normal.

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.

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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 radium and its series to Radium D. The half-life of radium is about 1,600 years; of radon, 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.

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. An example of this is radon (half-life of approximately 3.82 days) and successive members of the series to Radium D.

Equilibrium, Radiation -- The condition in a radiation field where the energy of the radiations entering a volume equals the energy of the radiations leaving that volume.

Equilibrium Fraction (F) -- In radon-radon daughter equilibrium, the parents and daughters have equal radioactivity, that is, as many decay into a specific nuclide as decay out. However, if fresh radon is continually entering a volume of air or if daughters are lost by processes other than radioactive decay, e.g., plate out or migration out of the volume, a disequilibrium develops. The equilibrium fraction is a measure of the degree of equilibrium/disequilibrium. The working-level definition of radon does not take into account the amount of equilibrium, The equilibrium fraction is used to estimate working levels based on measurement of radon only.

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 a metastable state and will return to ground state by radiation of the excess energy.

Exposure -- A measure of the ionization produced in air by x or gamma radiation. It is the sum of the electrical charges on all ions of one sign produced in air when all electrons liberated by photons in a volume element of

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air are completely stopped in air, divided by the mass of the air in the volume element. The special unit of exposure is the roentgen.

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

Gamma Ray -- Short wavelength electromagnetic radiation of nuclear origin (range of energy from 10 keV to 9 MeV).

Genetic Effect of Radiation -- Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells. On the basis of present knowledge these effects are purely additive; there is no recovery.

Gray (Gy) -- SI unit of absorbed dose. One gray equals 100 rad. (See Units.)

Half-Life, Biological -- The time required for the body to eliminate one-half of any absorbed substance by regular processes of elimination. Approximately the same for both stable and radioactive isotopes of a particular element. This is sometimes referred to as half-time.

Half-Life, Effective -- Time required for a radioactive element in an animal body to be diminished 50% as a result of the combined action of radioactive decay and biological elimination.

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

Half-life, Radioactive -- Time required for a radioactive substance to lose 50% of its activity by decay, Each radionuclide has a unique 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.

In Vitro -- Isolated from the living organism and artificially maintained, as in a test tube.

In Vivo -- Occurring within the living organism.

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 to 364 days as specified in the Toxicological Profiles.

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Internal Conversion -- One of the possible mechanisms of decay from the metastable state (isomeric transition) in which the transition energy is transferred to an orbital electron, causing its ejection from the atom. 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 radiation energy.

Ionization Density -- Number of ion pairs per unit volume.

Ionization Path (Track) -- The trail of ion pairs produced by ionizing radiation in its passage through matter.

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.

Isotones -- Nuclides having the same number of neutrons in their nuclei.

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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. Almost identical chemical properties exist between isotopes of a particular element. The term should not be used as a synonym for nuclide.

Stable Isotope -- A nonradioactive isotope of an element.

Joule -- The unit for work and energy, equal to one newton expended along a distance of one meter ($1\text{J} = 1\text{N} \times 1\text{m}$).

Labeled Compound -- A compound consisting, in part, of labeled molecules. That is molecules including radionuclides in their 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.

Lethal Concentration_(LO) (LC_{LO}) -- The lowest concentration of a chemical in air which has been reported to have caused death in humans or animals.

Lethal Concentration₍₅₀₎ (LC₅₀) -- The 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 laboratory animal population.

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.

Lethal Dose₍₅₀₎ (LD₅₀) -- The dose of a chemical which has been calculated to cause death in 50% of a defined laboratory animal population.

Lethal Time₍₅₀₎ (LT₅₀) -- A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined laboratory animal population.

Linear Energy Transfer (LET) -- The average amount of energy transferred locally to the medium per unit of particle track length.

Low-LET -- Radiation characteristic of electrons, x-rays, and gamma rays.

High-LET -- Radiation characteristic of protons or fast neutrons.

Average LET -- is specified to even out the effect of a particle that is slowing down near the end of its path and to allow for the fact that secondary particles from photon or fast-neutron beams are not all of the same energy.

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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.

Linear Hypothesis -- The assumption that a dose-effect 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.

Malformations -- Permanent structural changes in an organism that may adversely affect survival, development, or function.

Mass Numbers -- The number of nucleons (protons and neutrons) in the nucleus of an atom. (Symbol: A)

Minimal Risk Level -- An estimate of daily human exposure to a chemical that is likely to be without an appreciable risk of deleterious effects (noncancerous) over a specified duration of exposure.

Mutagen -- A substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutation can lead to birth defects, miscarriages, or cancer.

Neurotoxicity -- The occurrence of adverse effects on the nervous system following exposure to chemical.

Neutrino -- A neutral particle of very small rest mass originally postulated to account for the continuous distribution of energy among particles in the beta-decay process.

No-Observed-Adverse-Effect Level (NOAEL) -- The dose of chemical 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.

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.

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Octanol-Water Partition Coefficient (K_{ow}) -- The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Pair Production -- An absorption process for x and gamma radiation in which the incident photon is annihilated in the vicinity of the nucleus of the absorbing atom, with subsequent production of an electron and positron pair. This reaction only occurs for incident photon energies exceeding 1.02 MeV.

Parent -- A radionuclide which, upon disintegration, yields a specified nuclide--either directly or as a later member of a radioactive series.
Photon -- A quantity of electromagnetic energy (E) whose value in joules is the product of its frequency (ν) in hertz and Planck constant (h). The equation is: $E=h\nu$.

Photoelectric Effect -- An attenuation process observed for x- and gamma radiation in which an incident photon interacts with an orbital electron of an atom delivering all of its energy to produce a recoil electron, but with no scattered photon.

Positron -- Particle equal in mass to the electron (9.1091×10^{-31} kg) and having an equal but positive charge ($+1.60210 \times 10^{-19}$ Coulombs). (See Electron).

Potential Ionization -- The potential necessary to separate one electron from an atom, resulting in the formation of an ion pair.

Power, Stopping -- A measure of the effect of a substance upon the kinetic energy of a charged particle passing through it.

Progeny -- The decay products resulting after a series of radioactive decays, 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 1.007277 mass units.

q₁* -- The upper-bound estimate of the low-dose slope of the dose-response curve as determined by the multistage procedure. 3232 q₁* can be used to calculate an estimate of carcinogenic potency, the incremental excess cancer risk per unit of exposure (usually $\mu\text{g/L}$ for water, mg/kg/day for food, and $\mu\text{g/m}^3$ for air).

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."

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Quality Factor (QF) -- 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 effectiveness of the absorbed dose.

Rad -- The unit of absorbed dose equal to 0.01 J/kg in any medium. (See Absorbed Dose.)

Radiation -- (1) The emission and propagation of energy through space or through a material medium in the form of waves; for instance, the emission and propagation of electromagnetic waves, or of sound and elastic waves. (2) The energy propagated through space or through a material medium as waves; for example, energy in the form of electromagnetic waves or of elastic waves. The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation commonly is classified, according to frequency, as Hertzian, infra-red, visible (light), ultra-violet, X-ray and gamma ray. (See Photon.) (3) By extension, corpuscular emission, such as alpha and beta radiation, or rays of mixed or unknown type, as cosmic radiation.

Annihilation Radiation -- 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.

Background Radiation -- Radiation arising from radioactive material other than the one directly under consideration. Background radiation due to cosmic rays and natural radioactivity is always present. There may also be background radiation due to the presence of radioactive substances in other parts of the building, in the building material itself, etc.

Characteristic (Discrete) Radiation -- Radiation originating from an atom after removal of an electron or excitation of the nucleus. The wavelength of the emitted radiation is specific, depending only on the nuclide and particular energy levels involved.

External Radiation -- Radiation from a source outside the body -- the radiation must penetrate the skin.

Internal Radiation -- Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

Ionizing Radiation -- Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter.

Monoenergetic Radiation -- Radiation of a given type (alpha, beta, neutron, gamma, etc.) in which all particles or photons originate with and have the same energy.

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Scattered Radiation -- Radiation which during its passage through a substance, has been deviated in direction. It may also have been modified by a decrease in energy.

Secondary Radiation -- Radiation that results from absorption of other radiation in matter. It may be either electromagnetic or particulate.

Radioactivity -- The property of certain nuclides to spontaneously emit particles or gamma radiation or x radiation following orbital electron capture or after undergoing spontaneous fission.

Artificial Radioactivity -- Man-made radioactivity produced by particle bombardment or electromagnetic irradiation, as opposed to natural radioactivity.

Induced Radioactivity -- 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.

Natural Radioactivity -- The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

Radioisotopes -- A radioactive atomic species of an element with the same atomic number and usually identical chemical properties.

Radionuclide -- A radioactive species of an atom characterized by the constitution of its nucleus.

Radiosensitivity -- Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are currently used in a comparative sense, rather than in an absolute one.

Reaction (Nuclear) -- An induced nuclear disintegration, i.e., a process occurring when a nucleus comes in contact with a photon, an elementary particle, or another nucleus. In many cases the reaction can be represented by the symbolic equation: $X+a-Y+b$ or, in abbreviated form, $X(a,b) Y$. X is the target nucleus, a is the incident particle or photon, b is an emitted particle or photon, and Y is the product nucleus.

Reference Dose (RfD) -- An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate REDS and an additional modifying factor, which is based on a professional judgment of the entire

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database on the chemical. The RfDs are not applicable to nonthreshold 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 required to produce an identical biological effect in a particular experimental organism or tissue. NOTE: This term should not be used in radiation protection. (See Quality Factor.)

Rem -- A unit of dose equivalent. The dose equivalent in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor, the distribution factor, and any other necessary modifying factors.

Reportable Quantity (RQ) -- The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 lb or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Section 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 for photon radiation. One roentgen equals 2.58×10^{-4} Coulomb per kilogram of air.

Short-Term Exposure Limit (STEL) -- The maximum concentration to which workers can be exposed continually for up to 15 minutes. 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, Radiation -- (Radiation Therapy): A self-limited syndrome characterized by nausea, vomiting, diarrhea, and psychic depression following exposure to appreciable doses of ionizing radiation, particularly to the abdominal region. Its mechanism is unknown and there is no satisfactory remedy. It usually appears a few hours after irradiation and may subside within a day. It may be sufficiently severe to necessitate interrupting the treatment series or to incapacitate the patient. (General): The syndrome

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associated with intense acute exposure to ionizing radiations. The rapidity with which symptoms develop is a rough measure of the level of exposure.

Sievert -- The SI unit of radiation dose equivalent. It is equal to dose in grays times a quality factor times other modifying factors, for example, a distribution factor; 1 sievert equals 100 rem.

Specific Activity -- Total activity of a given nuclide per gram of an element.

Specific Energy -- The actual energy per unit mass deposited per unit volume in a given event. This is a stochastic quantity as opposed to the average value over a large number of instance (i.e., the absorbed dose).

Standard Mortality Ratio (SMR) -- Standard mortality ratio is the ratio of the disease or accident mortality rate in a certain specific population compared with that in a standard population. The ratio is based on 200 for the standard so that an SMR of 100 means that the test population has twice the mortality from that particular cause of death.

Stopping Power -- The average rate of energy loss of a charged particle per unit thickness of a material or per unit mass of material traversed.

Surface-seeking Radionuclide -- A bone-seeking internal emitter that is deposited and remains on the surface for a long period of time. 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 structural defects that affect the development of a fetus.

Threshold Limit Value (TLV) -- An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

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Toxic Dose (TD₅₀) -- 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 laboratory animal population.

Transformation, Nuclear -- The process by which a nuclide is transformed into a different nuclide by absorbing or emitting a particle.

Transition, Isomeric -- The process by which a nuclide decays to an isomeric nuclide (i.e., one of the same mass number and atomic number) of lower quantum energy. Isomeric transitions, often abbreviated I.T., proceed by gamma ray and/or internal conversion electron emission.

Tritium -- The hydrogen isotopes with one proton and two neutrons in the nucleus (Symbol: ³H or T).

Unattached Fraction -- That fraction of the radon daughters, usually ²¹⁸Po (Radium A), which has not yet attached to a particle. As a free atom, it has a high probability of being retained within the lung and depositing alpha energy when it decays.

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.

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Units, Radiological --

Units	Equivalents
Becquerel*	1 Bq = 1 disintegration per second = 2.7×10^{-11} Ci
Curie	1 Ci = 3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq
Gray*	1 Gy = 1 J/kg = 100 rad
Rad	1 Rad = 100 erg/g = 0.01 Gy
Rem	1 Rem = 0.01 Sievert
Sievert*	1 Sv = 100 rem

*International Units are 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) -- 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 shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. In nuclear reaction, it is customary to refer to photons originating in the extranuclear part of the atom as X-rays. These rays are sometimes called roentgen rays after their discoverer, W.C. Roentgen.

APPENDIX A**PEER REVIEW**

A peer review panel was assembled for radon. The panel consisted of the following members: Dr. Victor E. Archer, University of Utah Medical Center; Dr. Douglas J. Crawford-Brown, University of North Carolina; Dr. Richard Gerstle, private consultant; and Dr. John Spengler, Harvard School of Public Health. These experts collectively have knowledge of radon'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 the Section 104(i)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

A joint panel of scientists from ATSDR and EPA has 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 Agency for Toxic Substances and Disease Registry.

APPENDIX B**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), and Early et al. (1979).

B.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 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 atom is said to be stable; however, if there are too few or too many neutrons, the nucleus of the atom is unstable. Unstable nuclides undergo a process referred to as radioactive transformation in which energy is emitted. 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 man-made. Naturally-occurring radionuclides 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-

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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 man-made sources of ionizing radiation.

Man-made radioactive atoms are produced either as a by-product of fission of uranium atoms in a nuclear reactor or by bombarding stable atoms with particles, such as neutrons, 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 man-made 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.

B.2 RADIOACTIVE DECAY

B. 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 (electrons, neutrons, neutrinos, alpha particles, beta particles, protons, and fission products) or electromagnetic radiation (gamma rays and X-rays). Table B-1 summarizes the basic characteristics of the more common types of radiation encountered.

B.2.2 Half-Life and Activity

For any given radionuclide, the rate of decay is a first-order process that depends on the number of 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

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TABLE B-1. Characteristics of Nuclear Radiations

Radiation	Rest Mass	Charge	Typical Energy Range	Path Length (Order of Magnitude)		General Comments
				Air	Solid	
α	4.00 amu	2+	4-10 MeV	5-10 cm	25-40 μ m	Identical to ionized He nucleus
β (negatron)	5.48x10 ⁻⁴ amu 0.51 MeV	-	0-4 MeV	0-1 m	0-1 cm	Identical to electron
Positron (β positive)	5.48x10 ⁻⁴ amu 0.51 MeV	+	-	0-1 m	0-1 cm	Identical to electron except for charge
Proton	938.26 MeV 1.0073 amu	+	-	-	-	-
Neutron	1.0086 amu 939.55 MeV	0	0-15 MeV	0-100 m	0-100 cm	Free half life: 16 min
X (e.m. photon)	-	0	eV-100 keV	0.1-10 m ^a	0-1 m ^a	Photons from electron transitions
γ (e.m. photon)	-	0	10 KeV-3 MeV	0.1-10 m ^a	1 mm-1 m	Photons from nuclear transitions

^aExponential attenuation in the case of electromagnetic radiation.

α = alpha

β = beta

X = X-ray

γ = gamma

amu = atomic mass unit

MeV = Mega electron volts

KeV = Kiloelectron volts

cm = centimeter

m = meter

μ m = micrometer

mm = millimeter

e.m. = electromagnetic

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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 the average, how many atoms will transform during any interval of time.

The source strength is a measure of the rate of emission of radiation. For these radioactive materials it is customary to describe the source strength in terms of the source activity, which is defined as the number of disintegrations (transformations) per unit time occurring in a given quantity of this material. 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:

$$1 \text{ curie (Ci)} = 3.7 \times 10^{10} \text{ disintegrations (transformations)/second (dps) or} \\ 2.22 \times 10^{12} \text{ disintegrations (transformations)/minute (dpm).}$$

The SI unit of activity is the becquerel (Bq); 1 Bq = 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 rate of transformation 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 activity of a radionuclide at time t may be calculated by:

$$A = A_0 e^{-0.693t/T_{rad}}$$

where A is the activity in dps, A_0 is the activity at time zero, t is the time at which measured, and T_{rad} is the radiological half-life of the radionuclide. It is apparent that activity exponentially decays with 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.

The specific activity is the radioactivity per unit weight of material. This activity is usually expressed in curies per gram and may be calculated by

$$\text{curies/gram} = 1.3 \times 10^8 / (T_{rad}) \text{ (atomic weight)}$$

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_{bio1}) which is the time required for biological

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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 half-life:

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

Table B-2 presents representative effective half-lives of particular interest.

B.2.3 Interaction of Radiation with Matter

Both ionizing and nonionizing radiation will interact with materials, that is, it 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 secondarily may react with a target molecule. Charged particles, in contrast, strike the tissue or medium and directly react with target molecules, such as oxygen or water. These particulate radiations are directly ionizing radiations. Examples of directly ionizing particles include alpha and beta particles. Indirectly ionizing radiations are always more penetrating than directly ionizing particulate radiations.

Mass, charge, and velocity of a particle all affect the rate at which ionization occurs. The higher the charge of the particle and the lower the velocity, the greater the propensity to cause ionization. Heavy, highly charged particles, such as alpha particles, lose energy rapidly with distance and, therefore, do not penetrate deeply. The result of these interaction processes is a gradual slowing down of any incident particle until it is brought to rest or "stopped" at the end of its range,

B.2.4 Characteristics of Emitted Radiation

B.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 by two, thereby changing the parent to a different element. The alpha particle is identical

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TABLE B-2. Half-Lives of Some Radionuclides in Adult Body Organs

Radionuclide	Critical Organ	Half-Life ^a		
		Physical	Biological	Effective
Hydrogen-3 ^b (Tritium)	Whole body	12.3 y	12 d	11.97d
Iodine-131	Thyroid	8 d	138 d	7.6 d
Strontium-90	Bone	28 y	50 y	18 y
Plutonium-239	Bone	24,400 y	200 y	198 y
	Lung	24,400 y	500 d	500 d
Cobalt-60	Whole body	5.3 y	99.5 d	9.5 d
Iron-55	Spleen	2.7 y	600 d	388 d
Iron-59	Spleen	45.1 d	600 d	41.9 d
Manganese-54	Liver	303 d	25 d	23 d
Cesium-137	Whole body	30 y	70 d	70 d

^ad = days, y = years.

^bMixed in body water as tritiated water.

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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. Alpha particles have a large mass as compared to electrons. Decay of alpha-emitting radionuclides may result in the emission of several different alpha particles. A radionuclide has an alpha emission with a discrete alpha energy and characteristic pattern of alpha energy emitted.

The alpha particle has an electrical charge of +2. Because of this double positive charge, alpha particles have great ionizing power, but their large size results in very little penetrating power. In fact, an alpha particle cannot penetrate a sheet of paper. The range of an alpha particle, that is, 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 ingested, inhaled, or otherwise absorbed).

B.2.4.2. Beta Emission. Nuclei which are excessively neutron rich decay by B-decay. A beta particle (B) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (B-) or a positively charged electron, termed a positron (B+). Although the precise definition of "beta emission" refers to both B- and B+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the B+ particle.

B.2.4.2.1 Beta Negative Emission. Beta particle (B-) emission is another process by which a radionuclide, usually those 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 remains unchanged. This transformation results in the formation of a different element. The energy spectrum of beta particle emission ranges from a certain maximum down to zero with the mean energy of the spectrum being about one-third of the maximum. The range in tissue is much less. Beta negative emitting radionuclides can cause injury to the skin and superficial body tissues but mostly present an internal contamination hazard.

B.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 (B+) is emitted, accompanied by a neutrino (see glossary). This increases the number of neutrons by one, decreases the number of protons by one, and again leaves the atomic mass 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 B emitters of equal energy.

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B.2.4.2.3 Gamma Emission. Radioactive decay by alpha, beta, positron emission or electron capture often leaves some of the energy resulting from these changes in the nucleus. As a result, the nucleus is raised to an excited level. None of these excited nuclei can remain in this high-energy state. Nuclei release this energy returning to ground state or to the lowest possible stable energy level. The energy released is in the form of gamma radiation (high energy photons) and has an energy equal to the change in the energy state of the nucleus. Gamma and X-rays behave similarly but differ in their origin; gamma emissions originate in the nucleus while X-rays originate in the orbital electron structure.

B.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.

B.3.1 Dose Units

B.3.1.1 Roentgen. The roentgen (R) is a unit of exposure related to the amount of ionization caused in air by gamma or x-radiation. One roentgen equals 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.

B.3.1.2 Absorbed Dose and Absorbed Dose Rate. Since different types of radiation interact differently with any material through which they pass, any

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attempt to assess their effect on humans or animals should take into account these differences. The absorbed dose is defined as the energy imparted by the incident radiation to a unit mass of the tissue or organ. The unit of absorbed dose is the rad; 1 rad = 100 erg/gram = 0.01 J/kg in any medium. 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.

B.3.1.3 Working Levels and Working Level Months. Working levels are units that have been used to describe the radon decay-product activities in air in terms of potential alpha energy. It 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.

B.3.2 Dosimetry Models

Dosimetry models are used to estimate the internally deposited dose from exposure to radioactive substances. The models for internal dosimetry consider the quantity 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 models for external dosimetry consider only the photon doses to organs of individuals who are immersed in air or are exposed to a contaminated ground surface. 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 man-produced radionuclides from nuclear weapons testing.

B.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

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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.

B.3.2.2 Inhalation. The inhalation route of exposure has long been recognized as being of major importance 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. Giliary 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 particulates are absorbed into the blood and translocated to other organs and tissues. Dosimetric lung models are reviewed by James (1987) and James and Roy (1987).

B.3.3 Internal Emitters

The absorbed dose from internally deposited radioisotopes is the energy absorbed by the surrounding tissue. For a radioisotope distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the isotope. An infinitely large medium may be approximated by a tissue mass whose dimensions exceed the range of the particle. All alpha and most beta radiation will be absorbed in the organ (or tissue) of reference. Gamma-emitting isotope emissions are penetrating radiation and a substantial fraction may travel great distances within tissue, leaving the tissue without interacting. 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.

B.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

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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 (Hobbs and McClellan 1986; ICRP 1984; Mettber and Moseley 1985; Rubin and Casarett 1968).

B.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 to 500 rad (0.5 to 5 Gy), irreparable double-stranded DNA breaks are likely, resulting in cellular reproductive death after one or more divisions of the irradiated parent cell. At large doses of radiation, usually greater than 500 rad (5 Gy), direct cell death before division (interphase death) may occur from the direct interaction of free-radicals with essentially cellular macromolecules. Morphological changes at the cellular level, the severity of which are dose-dependent, may also be observed.

The sensitivity of various cell types varies. According to the Bergonie-Tribondeau law, the sensitivity of cell lines is directly proportional to their mitotic rate and inversely proportional to the degree of differentiation (Mettler and Moseley 1985). Rubin and Casarett (1968) devised a classification system that categorized cells according to type, function, and mitotic activity. The categories range from the most sensitive type, "vegetative intermitotic cells," found in the stem cells of the bone marrow and the gastrointestinal tract, to the least sensitive cell type, "fixed postmitotic cells," found in striated muscles or long-lived neural tissues.

Cellular changes may result in cell death, which if extensive, may produce irreversible damage to an organ or tissue or may result in the death of the individual. If the cell recovers, altered metabolism and function may still occur, which may be repaired or may result in the manifestation of clinical symptoms. These changes may also be expressed at a later time as tumors or mutations.

B.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.

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Injury may also result from constriction of the microcirculation and from edema and inflammation of the basement membrane (designated as the histohematic barrier - HHB), which may progress to fibrosis. In slow renewal and nonrenewal systems, the radiation may have little effect on the parenchymal cells, but ultimate parenchymal atrophy and death over several months result from HHB fibrosis and occlusion of the microcirculation.

B.4.3 Acute and Chronic Somatic Effects

B.4.3.1 Acute Effects. The result of acute exposure to radiation is commonly referred to as acute radiation syndrome. This effect is seen only after exposures to relatively high doses (>50 rad), which would only be expected to occur in the event of a serious nuclear accident. The four stages of acute radiation syndrome are prodrome, latent stage, manifest illness stage, recovery or death. The initial phase is characterized by nausea, vomiting, malaise and fatigue, increased temperature, and blood changes. The latent stage is similar to an incubation period. Subjective symptoms may subside, but changes may be taking place within the blood-forming organs and elsewhere which will subsequently give rise to the next stage. The manifest illness stage gives rise to symptoms specifically associated with the radiation injury. Among these symptoms are hair loss, fever, infection, hemorrhage, severe diarrhea, prostration, disorientation, and cardiovascular collapse. The symptoms and their severity depend upon the radiation dose received.

B.4.3.2 Delayed Effects. The level of exposure to radioactive pollutants that may be encountered in the environment is expected to be too low to result in the acute effects described above. When one is exposed to radiation in the environment, the amount of radiation absorbed is more likely to produce long-term effects, which manifest themselves years after the original exposure, and may be due to a single large over-exposure or continuing low-level exposure.

Sufficient evidence exists in both human populations and laboratory animals to establish that radiation can cause cancer and that the incidence of cancer increases with increasing radiation dose. Human data are extensive and include epidemiological studies of atomic bomb survivors, many types of radiation-treated patients, underground miners, and radium dial painters. Reports on the survivors of the atomic bomb explosions at Hiroshima and Nagasaki, Japan (with whole-body external radiation doses of 0 to more than 200 rad) indicate that cancer mortality has increased (Kato and Schull 1982). Use of X-rays (at doses of approximately 100 rad) in medical treatment for ankylosing spondylitis or other benign conditions or diagnostic purposes, such as breast conditions, has resulted in excess cancers in irradiated organs (BEIR 1980, 1990; UNSCEAR 1977, 1988). Cancers, such as leukemia, have been observed in children exposed in utero to doses of 0.2 to 20 rad (BEIR, 1980, 1990; UNSCRAR 1977, 1988). Medical use of Thorotrast (colloidal thorium dioxide) resulted in increases in the incidence of cancers of the liver, bone,

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and lung (ATSDR 1990a; BEIR 1980, 1990; UNSCEAR 1977, 1988). Occupational exposure to radiation provides further evidence of the ability of radiation to cause cancer. Numerous studies of underground miners exposed to radon and radon daughters, which are alpha emitters, in uranium and other hard rock mines have demonstrated increases in lung cancer in exposed workers (ATSDR 1990b). Workers who ingested radium-226 while painting watch dials had an increased incidence of leukemia and bone cancer (ATSDR 1990c). These studies indicate that depending on radiation dose and the exposure schedule, ionizing radiation can induce cancer in nearly any tissue or organ in the body. Radiation-induced cancers in humans are found to occur in the hemopoietic system, the lung, the thyroid, the liver, the bone, the skin, and other tissues.

Laboratory animal data indicate that ionizing radiation is carcinogenic and mutagenic at relatively high doses usually delivered at high dose rates. However, due to the uncertainty regarding the shape of the dose-response curve, especially at low doses, the commonly held conservative position is that the cancer may occur at dose rates that extend down to doses that could be received from environmental exposures. Estimates of cancer risk are based on the absorbed dose of radiation in an organ or tissue. The cancer risk at a particular dose is the same regardless of the source of the radiation. A comprehensive discussion of radiation-induced cancer is found in BEIR IV (1988), BEIR V (1990), and UNSCEAR (1982, 1988).

B.4.4 Genetic Effects

Radiation can induce genetic damage, such as gene mutations or chromosomal aberrations, by causing changes in the structure, number, or genetic content of chromosomes in the nucleus. The evidence for the mutagenicity of radiation is derived from studies in laboratory animals, mostly mice (BEIR 1980, 1988, 1990; UNSCEAR 1982, 1986, 1988). Evidence for genetic effects in humans is derived from tissue cultures of human lymphocytes from persons exposed to ingested or inhaled radionuclides (ATSDR 1990c, 1990d). Evidence for mutagenesis in human germ cells (cells of the ovaries or testis) is not conclusive (BEIR 1980, 1988, 1990; UNSCFAR 1977, 1986, 1988). Chromosome aberrations following radiation exposure have been demonstrated in man and in experimental animals (BEIR 1980, 1988, 1990; UNSCEAR 1982, 1986, 1988).

B.4.5 Teratogenic Effects

There is evidence that radiation produces teratogenicity in animals. It appears that the developing fetus is more sensitive to radiation than the mother and is most sensitive to radiation-induced damage during the early stages of organ development. The type of malformation depends on the stage of development and the cells that are undergoing the most rapid differentiation at the time. Studies of mental retardation in children exposed in utero to radiation from the atomic bomb provide evidence that radiation may produce

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teratogenic effects in human fetuses (Otake and Schull 1984). The damage to the child was found to be related to the dose that the fetus received.

B.5 UNITS IN RADIATION PROTECTION AND REGULATION

B.5.1 Dose Equivalent and Dose Equivalent Rate. Dose equivalent or rem is a special radiation protection quantity that is used to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. The ICRU has defined the dose equivalent, H , as the product of the absorbed dose, D , the quality factor, Q , and all other modifying factors, N , at the point of interest in biological tissue. This relationship is expressed as follows:

$$H = D \times Q \times N.$$

The quality factor is a dimensionless quantity that depends in part on the stopping power for charged particles, and it accounts for the differences in biological effectiveness found among the types of radiation. By definition it is independent of tissue and biological end point and, therefore, of little use in risk assessment now. Originally Relative Biological Effectiveness (RBE) was used rather than Q to define the quantity, rem, which was of use in risk assessment. The generally accepted values for quality factors for various radiation types are provided in Table B-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.

B.5.2 Relative Biological Effectiveness. The term relative biologic 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 to 250 KeV X-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor used in calculations of dose equivalents for radiation protection purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). The generally accepted values for RBE are provided in Table B-4.

B.5.3 Effective Dose Equivalent and Effective Dose Equivalent Rate. The absorbed dose is usually defined as the mean absorbed dose within an organ or tissue. This represents a simplification of the actual problem. Normally when an individual ingests or inhales a radionuclide or is exposed to external radiation that enters the body (gamma), the dose is not uniform throughout the whole body. The simplifying assumption is that the detriment will be the same whether the body is uniformly or nonuniformly 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.

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TABLE B-3. Quality Factors (QF)

-
1. X-rays, electrons, and positrons of any specific ionization

$$QF = 1.$$

2. Heavy ionizing particles

Average LET in Water (MeV/cm)	QF
35 or less	1
35 to 70	1 to 2
70 to 230	2 to 5
230 to 530	5 to 10
530 to 1750	10 to 20

For practical purposes, a QF of 10 is often used for alpha particles^a and fast neutrons and protons up to 10 MeV. A QF of 20 is used for heavy recoil nuclei.

^aThe ICRP (1977) recommended a quality factor of 20 for alpha particles.

LET = Linear energy transfer

MeV/cm = Megaelectron volts per centimeter

MeV = Megaelectron volts

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TABLE B-4. Representative LET and RBE Values*

Radiation	Energy (MeV)	Av. LET (keV/μ)	RBE	Quality Factor
X-rays, 200 kVp	0.01-0.2	3.0	1.00	1
Gamma rays	1.25	0.3	0.7	1
	4	0.3	0.6	1
Electrons (β)	0.1	0.42	1.0	1
	0.6	0.3	1.3	1
	1.0	0.25	1.4	--
Protons	0.1	90.0	--	6
	2.0	16.0	2	10
	5.0	8.0	2	10
Alpha particle	0.1	260.0	--	--
	5.0	95.0	10-20	10
Heavy ions	10-30	~150.0	~25	20
Neutrons	thermal		4-5	3
	1.0	20.0	2-10	10

*These values are general and approximate. RBE and QF values vary widely with different measures of biological injury.

MeV = Megaelectron volts

KeV/μ = Kiloelectron volts per micron

RBE = Relative biological effectiveness

kVp = Kilovolt potential

LET = Linear energy transfer

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The effective dose equivalent, H_e is

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

where H_t is the dose equivalent in the tissue, W_t is the weighting factor, which represents the estimated proportion of the stochastic risk resulting from tissue, T, to the stochastic risk when the whole body is uniformly irradiated for occupational exposures under certain conditions (ICRP 1977). Weighting factors for selected tissues are listed in Table B-5.

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 B-6.

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TABLE B-5. Weighting Factors for Calculating Effective Dose Equivalent for Selected Tissues

Tissue	Weighting Factor
Gonads	0.25
Breast	0.15
Red bone marrow	0.12
Lung	0.12
Thyroid	0.03
Bone surface	0.03
Remainder	0.30

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TABLE B-6. Comparison of Common and SI Units for Radiation Quantities

Quantity	Customary Units	Definition	SI Units	Definition
Activity (A)	Curie (Ci)	3.7×10^{10} transformations s^{-1}	becquerel (Bq)	s^{-1}
Absorbed Dose (D)		rad (rad)	10^{-2}Jkg^{-1}	gray (Gy) Jkg^{-1}
Absorbed Dose Rate (D)	rad per second (rad s^{-1})	$10^{-2} \text{Jkg}^{-1} \text{s}^{-1}$	gray per second (Gy s^{-1})	$\text{Jkg}^{-1} \text{s}^{-1}$
Dose Equivalent (H)	rem (rem)	10^{-2}Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose Equivalent Rate (H)	rem per second (rem s^{-1})	$10^{-2} \text{Jkg}^{-1} \text{s}^{-1}$	sievert per second (Sv s^{-1})	$\text{Jkg}^{-1} \text{s}^{-1}$
Linear Energy Transfer (L_w)	kiloelectron volts per micrometer ($\text{keV}\mu\text{m}^{-1}$)	$1.602 \times 10^{-10} \text{Jm}^{-1}$	kiloelectron volts per micrometer ($\text{keV}\mu\text{m}^{-1}$)	$1.602 \times 10^{-10} \text{Jm}^{-1}$

S^{-1} = per second

Jkg^{-1} = Joules per kilogram

$\text{Jkg}^{-1} \text{s}^{-1}$ = Joules per kilogram per second

Jm^{-1} = Joules per meter

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