# 23

# Radon

*Nirmalla Barros and R. William Field*

## Introduction

Radon-222 (radon) is a radioactive noble gas that occurs naturally in soil and rock where radium is present as well as in many groundwater supplies. It is responsible for about 68 percent of our natural radiation and approximately 37 percent of our overall radiation exposure in the United States.[1] Radon is fairly ubiquitous outdoors and can reach high concentrations in enclosed areas such as homes. Levels of radon tend to be highest in the lower levels of buildings.

Radon is the leading environmental cause of cancer mortality. Studies of radon-exposed underground miners and direct evidence from large-scale pooled analyses from numerous residential radon studies demonstrate radon's potency as a lung carcinogen. According to the U.S. Environmental Protection Agency (EPA), radon is the second leading cause of lung cancer, contributing an estimated 21,000 deaths each year in the United States, and the primary cause of lung cancer among individuals who have never smoked.[2] Smokers with radon exposures are at greater risk of developing lung cancer compared to individuals who never smoked with the same radon exposure, due to the synergistic effect (submultiplicative) between tobacco and radon.

## Background

Radon-222 (radon) is a noble gas with an atomic number of 86 (the number of protons in an atom's nucleus). Like other noble gases, radon is colorless,

odorless, and tasteless. In 1898 Marie Curie isolated a radioactive element, which she named radium. A year later, Ernest Rutherford identified the types of radiation emitted from uranium through radioactive decay. Both Curie's and Rutherford's findings were preceded by experiments with uranium by Henri Becquerel.[3] "Radium emanation" (radon) was discovered in 1900 by a German physicist, Friedrich Dorn.[4] Historically, radon has been used as a treatment for certain diseases, to approximate radium body burden by measuring the gas in exhaled breath, as a tracer for the presence of uranium and radium, to predict earthquakes, and to track the movement of air masses as well as groundwater flow.

## Radioactive Decay

Among the more than thirty known isotopes of radon, only three are found naturally.[5] These isotopes are Rn-219 (actinon), Rn-220 (thoron), and Rn-222 (radon). Rn-222 has the longest half-life, 3.8 days (i.e., it takes around four days for the gas to decay spontaneously to half of its initial activity), while the other two have half-lives of less than a minute. Radon is formed from the radioactive decay of uranium's most plentiful isotope, uranium-238, whereas actinon and thoron are associated with the uranium-235 and thorium-232 decay series, respectively. Uranium-238 goes through a series of radioactive decays, breaking down to radium (Ra-226), which in turn decays to radon (Rn-222), resulting in the eventual decay of polonium (Po-218, Po-214, Po-210), lead (Pb-214, Pb-210), and bismuth (Bi-214, Bi-210) isotopes, ending with a stable nonradioactive lead atom (Pb-206) (see Figure 23.1). Radon decays to both short- and long-lived decay products with half-lives as short as 160 microseconds (Po-214) to as long as 22 years (Pb-210).

During the decay of radon and its decay products, ionizing radiation is released. Ionizing radiation emits energy in the form of alpha, beta, and gamma radiation. Alpha radiation releases energy as particles, equivalent to a helium nucleus, containing two protons and two neutrons. Alpha particles have the heaviest mass and deliver the greatest radiation dose to the lungs from the radon decay products. Like alpha radiation, beta radiation emits energy in the form of particles. Beta particles have very little mass, allowing them to move faster and penetrate deeper than alpha particles. Gamma radiation, on the other hand, releases energy as a ray, has no mass, and moves at


**Figure 23.1** Radon decay chain.

the speed of light. From the decay of radon and its decay products (see Figure 23.1), Po-218 and Po-214 deliver the greatest radiation dose to the lungs.

### Radioactivity Units

The radioactivity of a substance is measured by the rate that it decays. The International System of Units (SI) adopted the becquerel (Bq) in 1960 as a standard unit to measure radioactivity.[6] One Bq is equivalent to one

disintegration per second. The curie is an older, non SI, unit of measurement. A curie (Ci) equals 3.7 x $10^{10}$ becquerels or 3.7 x $10^{10}$ disintegrations per second. Radon measurements in air are typically expressed in picocuries per liter of air (pCi/L) in the United States, but in becquerels per cubic meter of air (Bq/$m^3$) elsewhere. One pCi/L is equivalent to 37 Bq/$m^3$.

To measure occupational exposures to radon, the Working Level (WL) is used. One WL is characterized as the concentration of decay products of radon in one liter of air from the release of 1.3 x $10^5$ million electron volts (MeV) of potential alpha radiation. This translates to 100 pCi/L (3,700 Bq/$m^3$) each of Rn-222, Po-218, Bi-214, and Po-214. Cumulative radon exposure in the workplace is measured by a working level month (WLM). WLM is the product of the WL exposure and the number of hours of work exposure over the total hours worked in a month (equivalent to 170 hours).

## Sources of Exposure

Radon has long been present in the earth because of the extremely long, 4.5-billion-year, half-life of uranium-238. Uranium-238 has been present in soils, rocks, and water since the earth was formed. From the decay of uranium-238, and subsequently radium-226, radon gas is released and can move through the earth into the atmosphere, where it easily dilutes with outdoor air. However, because most homes are not built radon resistant, the gas moves up through the soil and can enter a building, where it accumulates. From this buildup, radon can reach high concentrations, and breathing its decay products over an extended period of time can pose a significant health risk. Another source of exposure is the emanation of radon from groundwater supplies. This is due to radon off-gassing from the water when used for dishwashing, showering, and other uses in the home. Yet another source of radon exposure is from radon emanations of building materials.

As mentioned above, radon contributes about 37 percent of the average individual's total radiation dose in the United States. Although the percent contribution of radon to our overall radiation exposure has decreased over the past thirty years, it is not because we are getting less exposure from radon, but because an increasing percent of our overall radiation exposure is coming from computed topography (CT) scans and nuclear medicine procedures (see Figure 23.2).

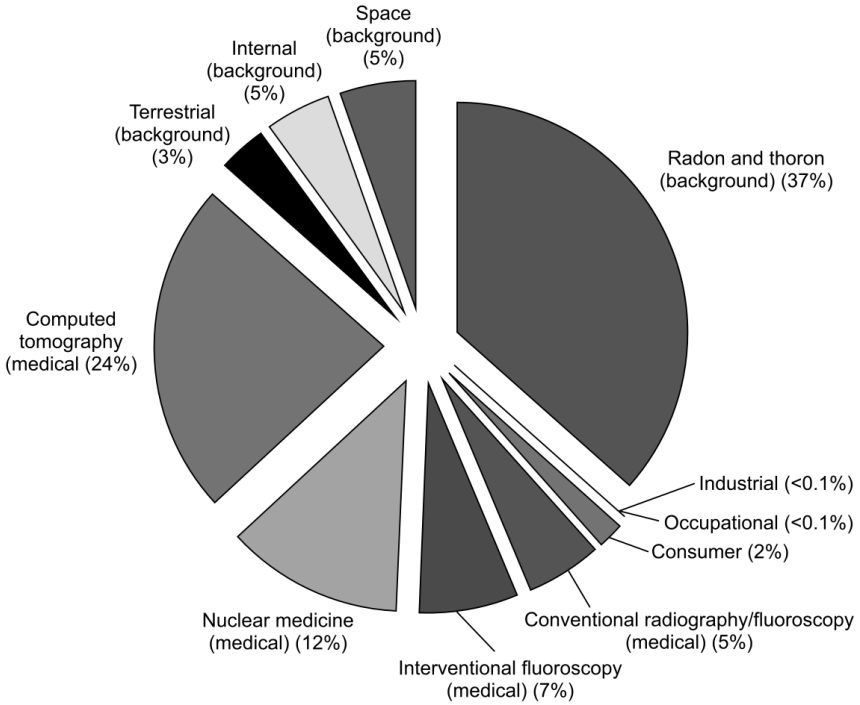


**Figure 23.2** Ionizing radiation exposure in the United States. (Reprinted with permission of National Council on Radiation Protection and Measurements, http://NCRPonline.org [1])

## Soil

The greatest source of indoor radon exposure is from inhalation of radon gas and the resulting decay products from the soil and rocks below the foundation of a building. Soil is composed of a mixture of grains in the form of clay, silt, or sand, as well as water and air. Sand is the largest grain, and soil with larger grains tends to be more permeable to air and fluids, whereas soil made up of smaller grains like clay is less permeable. The soil's permeability affects the distance radon can travel. Radon can travel several meters per day in dry or slightly moist soils compared to limited movement in water-saturated soil.

## Rock

Over time, soil is formed from the breakdown of rock. Rock types with a low melting point such as granite and shale generally have higher uranium

concentrations than those with high melting points like diorites and basalt. Uranium enriches in volatile phases as molten or partially melted rocks cool, which are processes associated with the formation of igneous and metamorphic rocks. Building materials containing these types of rock have contributed to increased radon concentrations in the indoor environment.

## Water

Radon in water involves two routes of exposure, inhalation and ingestion, with inhalation as the predominant mode of radiation exposure.[7] Radon from surface water (i.e., rivers, lakes) is less of a concern because most of the radon is lost rapidly into the atmosphere. Groundwater, on the other hand, often travels through radium-rich soil and rock, and since radon is soluble in water, it is able to dissolve and become a radioactive source. Radon is also a concern within water distribution systems that have a history of high radium levels. Radium can become adsorbed to iron pipe scales,[8] which releases radon into the water distribution system, increasing concentrations at the point-of-use. Small public groundwater suppliers and private wells tend to have the highest radon concentrations due to the short water processing time before radon can decay. Larger water suppliers often have longer transit times, which increases the likelihood that radon will decay prior to reaching the point-of-use. These suppliers also aerate the water or mix well water with surface water supplies to reduce the waterborne radon concentrations prior to it entering the water distribution system. Activities such as showering and washing dishes allow radon to out-gas and contribute to higher radon concentrations in indoor air. A general rule of thumb is that a waterborne radon concentration of 10,000 pCi/L entering a home will increase the indoor air radon concentration by 1 pCi/L (37 $Bq/m^3$).[9]

## Radon Surveys

The U.S. Geological Survey and EPA have developed a geologic map to assess indoor radon potential in counties within the United States, as advised by the Indoor Radon Abatement Act of 1988 (see Figure 23.3). Estimates of indoor radon potential were made from the EPA's National Residential Radon Survey and other sources utilizing bedrock and surficial

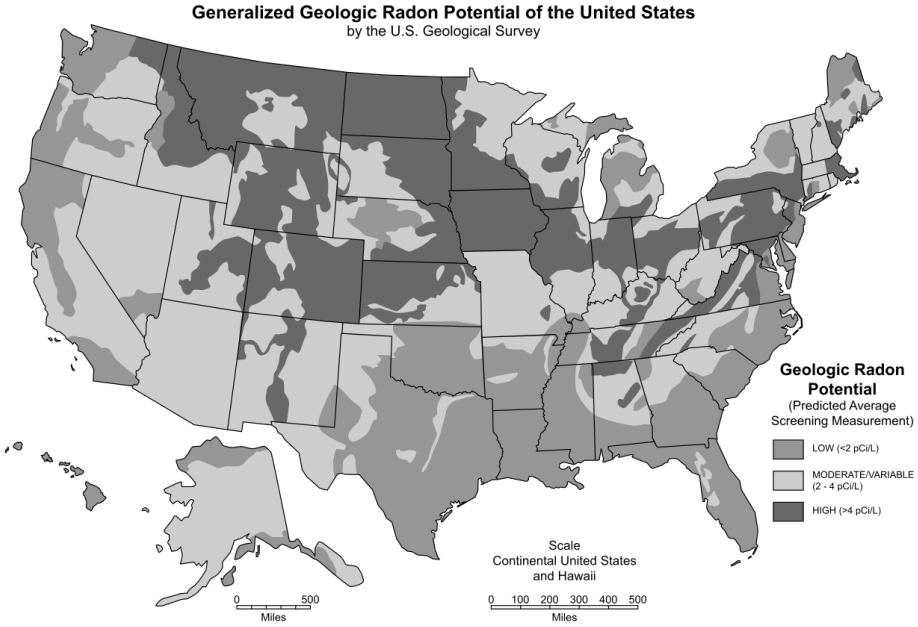


**Figure 23.3** Radon potential in the United States.

*Source:* R. R. Schumann, Geologic radon potential of the United States, U.S. Geological Survey open-file report 93-292, parts A–J (Washington, DC: U.S. Geological Survey, 1993).

geology, radiochemistry, aerial radioactivity, soil properties, and foundation type.[10]

Radon concentrations vary across geographical regions in the United States based on the radium content in the soil, underlying geology, and soil porosity. The radon potential map is intended to provide general guidance for areas that may be at higher risk from exposure. It is important to note that elevated residential radon concentrations can also be found in areas that are mapped as having a low radon potential. Therefore, the radon potential map should not be used as a reference for determining where not to test. Studies conducted by the EPA have found that Iowa has both the highest mean radon concentrations and the greatest percentage (71 percent) of screening (i.e., short term) radon measurements greater than the EPA's action level of 4 pCi/L (150 $Bq/m^3$) compared to any other state surveyed in the United States. According to the U.S. Geological Survey, the high radon potential in the upper Midwest compared to other regions can be explained by continental glacial deposits.[11] Glaciers are effective in

disseminating uranium-rich rocks from their origins to other regions, making it possible to spread the radon potential. The characteristics of soils formed from glacial deposits differ by the type of bedrock source and the type of glaciers from which they were formed. However, in most parts of the country, like Pennsylvania, the underlying geology, rather than glacial tills, provides the source for the elevated radon concentrations. The EPA has noted that the counties surrounding the Three Mile Island Nuclear Generating Plant in Pennsylvania have the highest regional radon concentrations in the United States, but these elevated radon concentrations have no relationship to the nearby power plant.[12]

Elevated radon concentrations may be observed in any region of the country, even in areas expected to have low radon potential. The only way to know if a home has an elevated radon concentration is to test the home. Radon concentrations within the home can exhibit significant temporal and spatial variability. Meteorological conditions, influencing wind speed and direction, atmospheric pressure, and temperature, can affect the driving forces of radon transport. Indoor radon concentrations are also affected by the daily activities of the occupant of the dwelling (e.g., opening windows, level of ventilation).

Based on a nationwide study that placed one radon detector outdoors in each state, the EPA estimated that the average outdoor radon concentration in the United States is 0.4 pCi/L (15 Bq/$m^3$). However, researchers have noted[13] that in some regions of the country, annual outdoor radon concentrations can be as high as the national average indoor radon concentration. The EPA's National Residential Radon Survey, conducted from 1989 through 1990, estimated that the U.S. indoor average annual radon concentration is 1.25 ± 0.06 pCi/L (46 ± 2 Bq/$m^3$), with a median value of 0.67 pCi/L (25 Bq/$m^3$).[14] This survey placed two to four alpha track detectors in 7,100 homes in 125 counties over a year and gathered information about smoking history, occupant living activities, house construction, weatherproofing, heating, ventilation, and air conditioning patterns. In general, the worldwide average indoor radon concentration is estimated to be 1.1 pCi/L (39 Bq/$m^3$).[15]

As for other buildings such as schools, where people also spend a great deal of time, a national survey of radon levels in schools conducted by the EPA found that close to 19 percent of U.S. schools had at least one frequently occupied room with short-term radon concentration at or above

4 pCi/L (150 Bq/m$^3$).[16] Another potentially important contributor to a person's overall radon exposure is radon exposure at the workplace. There have been few studies examining radon exposure in places of work other than mines, but based on the studies that have been performed, radon concentrations were generally found to be lower than in residences, in part due to differences in building ventilation, structure, and volume.[17]

## Exposure Guidelines

The EPA currently advises mitigation of radon levels in the home at 4 pCi/L (150 Bq/m$^3$) or greater.[18] The EPA's radon action level of 4 pCi/L is not a health-based guideline. In fact, the EPA notes in the *Citizen's Guide to Radon*[18] that exposure to radon concentrations below 4 pCi/L still poses a risk. Radon concentrations in most homes can be reduced to 2 pCi/L or below. In 2009 the World Health Organization adopted a radon reference level of 2.7 pCi/L (100 Bq/m$^3$). The WHO radon reference level represents the maximum accepted radon concentration for a residential dwelling and is intended to minimize health hazards due to indoor radon exposure.[15]

An amendment to the 1986 Safe Drinking Water Act (SDWA) directed the EPA to promulgate regulatory standards for waterborne radon for large water systems. The EPA proposed a Maximum Contaminant Level (MCL) of 300 pCi/L in 1991 for large water systems. In 1996 a congressional amendment to the SDWA allowed the EPA to establish an Alternative Maximum Contaminant Level (AMCL) of 4,000 pCi/L for large public water suppliers who agreed to implement a multimedia mitigation (MMM) plan, such as supporting efforts to reduce indoor airborne radon concentrations to levels that would provide an equivalent risk reduction to the MCL. The proposed AMCL of 4,000 pCi/L would yield an equivalent risk of exposure at the average outdoor radon concentration (0.4 pCi/L), assuming the transfer coefficient of 10,000 pCi/L of radon in water to 1 pCi/L (37 Bq/m$^3$) of radon in indoor air. The water to air transfer coefficient of 10,000 to 1 indicates that a waterborne radon concentration of 10,000 pCi/L would increase the radon concentration in indoor air by approximately 1 pCi/L. While some states have passed requirements limiting the radon concentration for large water systems, the EPA has not promulgated its proposed limits. In addition, waterborne radon concentrations for smaller utilities and private wells are not regulated.[19]

## Health Effects

As radon gas decays, it produces a series of solid decay products (see Figure 23.1). Some of the decay products attach to existing airborne aerosols in the room air, others remain unattached, and some attached particles deposit on room surfaces. The rate of attachment to aerosols depends on the size and amount of aerosols in the air as well as air movement in the room. The likelihood of radon decay product deposition in the lung after attachment is influenced by a number of factors, including aerosol size, breathing frequency, nasal versus oral breathing, lung tidal volume, and lung architecture. The inhalation of the short-lived particles, especially the unattached ultrafine particles, contributes the majority of the dose to the lung. Although alpha particles cannot penetrate as far into the tissue or even penetrate the dead layer of one's skin as other types of radiation can, the release of the energy imparted to lung tissues is capable of causing disruptions to DNA, including gene mutations, chromosomal damage, induction of micronuclei, and sister chromatid exchanges. Alpha particles are somewhat unique among environmental carcinogens in that they can cause double strand DNA breaks. Defects of the DNA can progress to lung cancer. Among radon's decay products, polonium-218 and polonium-214 (see Figure 23.1) have the shortest half-lives, but deliver the most significant radiation dose to the lung's epithelial tissue. The primary hazards of radon, therefore, come from its decay products rather than from radon itself. Since even a single alpha particle can cause significant biological damage to a cell, and because cancer is considered monoclonal in origin, it is unlikely that a threshold exists for radon-induced lung cancer.

### Lung Cancer

Lung cancer is by far the most established adverse health outcome associated with protracted exposure to radon and its decay products. Radon and its decay products have been classified as a Group 1 carcinogen by the WHO's International Agency for Research on Cancer (IARC) since 1988.[20] Group 1 is the highest ranking risk potential for a carcinogen (e.g., tobacco is also classified as a Group 1 carcinogen). A Group 1 classification is given when there is sufficient evidence of carcinogenicity in humans

gathered from studies in experimental animals and humans exposed to the agent. Among the various types of cancers, lung cancer causes the most deaths in the United States in both males and females, with tobacco smoking (e.g., cigarettes, cigars, pipes) as the primary risk factor.[21] Among individuals who have never used tobacco products, radon is the leading cause of lung cancer.

### *Lung Cancer Risk from Miner Studies*

As early as the 1500s, Agricola reported on the occurrence of high mortality of pulmonary disease among underground metal miners at Schneeberg in the Erz Mountains of Central Europe. Several hundred years later, in 1879, Härting and Hesse linked the high mortality rates of pulmonary disease in miners with lung cancer. While Friedrich Dorn is credited with discovering "radium emanations" in 1900, which were eventually named radon in 1923, it was not until the early 1900s that H. E. Müeller, in 1913, and Margaret Uhlig, in 1921, causally associated lung cancer occurrence with "radium emanations." Radon progeny, as opposed to radon gas, was discovered to be the primary causative agent for lung cancer by the 1950s, and by the 1970s, quantitative risk estimates for lung cancer in radon-exposed miners were finally starting to be calculated.

National media spread the news about the potential for residential radon exposure in December 1984 after a construction engineer named Stanley Watras set off radiation monitors at the newly constructed Limerick Nuclear Power Plant in Pennsylvania. When a source of radiation exposure that was responsible for Mr. Watras's contamination was not found at the plant, a team of scientists surveyed his home and discovered radon concentrations as high as 2,700 pCi/L. The radiation that was setting off the radiation monitors was traced back to the radon decay products that attached to his clothing before he left home. Fortunately Mr. Watras and his family had only lived at the home for a relatively short time. Because of the discovery of elevated radon concentrations in homes, the EPA created a National Radon Program in 1985.

In order to assess the risk of lung cancer from radon exposure, epidemiological studies involving miners were conducted. The National Research Council's Committee on Health Risks of Exposure to Radon (BEIR VI) evaluated the lung cancer risk posed by radon among workers mining uranium and other metals, such as tin, ore, and fluorspar in Asia, Australia,

Europe, and North America.[17] Besides the type of mining environments, the cohorts also differed by demographics, smoking patterns, data collection techniques, and degree of exposure to silica and diesel fumes. The miner-based cohort studies were conducted as early as the 1950s and consisted of approximately 68,000 men, 1.2M person-years of follow-up, and approximately 2,800 lung cancer deaths.

Overall, the studies found that lung cancer mortality increased with increasing cumulative radon exposure. Due to the extent of the variation in the magnitude of risk between the studies, the BEIR VI Committee pooled the data from the eleven cohorts to conduct additional analyses. The risk noted from the pooled analysis varied by time since exposure and age. Specifically, a greater risk of lung cancer was observed among individuals who had been exposed five to fourteen years prior to lung cancer and among miners exposed at a younger age. On the whole, the pooled analysis of the miners' studies found that 39 percent of the lung cancer mortality among smokers was likely attributed to occupational radon exposure. Among those who never smoked, 73 percent of the lung cancer deaths were thought to be radon-induced.

In order to develop radon risk estimates for the general population, the results from the miner studies were extrapolated to predict the lung cancer risk posed by radon in the residential setting. The committee proposed two models, an exposure-age-concentration model and an exposure-age-duration model, to predict the number of lung cancer deaths from protracted radon exposure in the general population. Based on the model used, the BEIR VI committee estimated that residential radon causes approximately 15,400 to 21,800 lung cancer deaths each year in the United States.[17] The EPA extended the BEIR VI risk estimates in 2003 by constructing a single model midway between the two BEIR VI preferred models. The newer EPA model estimated that of the 146,400 lung cancer deaths that occurred in the United States in 1995, 21,100 (14.4 percent) were caused by protracted radon exposure.[2]

However, many scientists remained skeptical that the results of the miner-based studies could be used to indirectly predict the lung cancer risk for the residential setting, in part because of 1) sex and age differences between workers and the public; 2) higher concentrations of airborne dust in the mines versus the home that affect the activity size distribution of radon's

decay products and the rate of attachment; 3) the presence of other potential toxicants like silica and diesel fumes in the mining atmosphere, which may act as confounders; 4) a higher level of physical activity among the miners, resulting in increased respiration rates; 5) exposure rate and duration of exposure differences; 6) the equilibrium ratio between radon and its decay products; and 6) more oral versus nasal breathing in miners, which tends to increase the deposition of larger-sized particles into the lung.[22]

### *Lung Cancer Risk from Residential Studies*

Initial epidemiological study designs such as ecological studies (i.e., geographic correlations) were used to provide insights into the relationship between radon exposure and lung cancer. Early ecological studies used either surrogate measures for radon (e.g., building materials, underlying geology) or a geographic measure of radon (e.g., average county radon concentrations) with comparisons to lung cancer mortality at the geographic level (e.g., county lung cancer rates). However, the inability of ecological studies to control for confounding, effect modification, and bias limited their usefulness to only hypotheses generation.

Because of the limitations of ecological studies and the uncertainty regarding the validity of extrapolations of the findings from miner studies, investigators started to directly assess the risk posed by protracted residential radon exposure by use of case-control studies. Globally, more than twenty-five case-control studies have been performed in North America, Europe, and China since the early 1980s. Investigators from twenty-two of the more rigorous case-control studies started meeting in the 1980s to develop plans to pool the studies to increase the overall sample size. Seven of the studies were conducted in North America, thirteen in Europe, and two in China. The three collaborative pooled studies from Europe, North America, and China reported increased lung cancer risks of about 8 percent (95% CI: 3–16%), 11 percent (95% CI: 0–28%), and 13 percent (95% CI: 1–36%) at a radon concentration of 2.7 pCi/L (100 Bq/$m^3$), respectively.[23–25] The risk estimates from the three pooled analyses were in close agreement with the increased risk of 12 percent at 2.7 pCi/L (100 Bq/$m^3$) predicted from extrapolations from the miner-based data. It should be noted that higher risk estimates were found when the pooled analyses used more rigorous methods to model radon exposure. In fact, empirical models

have indicated that studies utilizing more sophisticated retrospective radon exposure assessment techniques had higher risk estimates than studies that used more simplistic radon exposure estimates.[26]

### Interaction between Smoking and Radon

The findings from the miner-based data suggested a synergistic effect (i.e., submultiplicative) between radon and smoking and the subsequent development of lung cancer. The attributable risks for lung cancer in the miner population were 9 to 13 percent for individuals who ever smoked and 19 to 26 percent for miners who never smoked. Radon-attributable lung cancer mortality estimates among miners ranged from 4,400 to 6,100 in never smokers and 12,500 to 18,000 for those who have smoked.[27] Direct observations from the pooled residential radon studies also supported the finding that while the attributable risk for protracted radon exposure is greater for never smokers, the number of radon-attributable deaths for ever smokers is several-fold higher. Studies have suggested that in addition to smoking, individuals who have exposure to other lung carcinogens (e.g., nickel, radiation from medical procedures) are also likely at greater risk for developing lung cancer. Furthermore, exposure to radon also increases the risk for individuals with certain genetic predispositions.[28]

### Other Adverse Health Outcomes Related to Radon Exposure

The BEIR VI committee explored increases in cause-specific mortality, other than lung cancer, using the miner data, and no compelling dose-response related increases in mortality were noted. However, many adverse health outcomes are not readily observable in mortality studies, because cancer and other chronic conditions are often not included on death certificates. A case-cohort cancer incidence study performed by Řeřicha and colleagues[29, 30] detected a positive association between protracted radon exposure and leukemia, including chronic lymphocytic leukemia. In addition, a Bayesian study using a hierarchical risk model and data from the Iowa Cancer Registry reported a slightly increased, nonsignificant risk for residential radon exposure and both chronic lymphocytic leukemia and chronic myelocytic leukemia.[30] Additional studies, especially for women,

would be helpful to assess the risk posed by radon in relation to cancer incidence and other adverse health outcomes.

## Measurement

Radon is likely the most measured radionuclide and carcinogen in the environment. Protocols have been established for conducting radon measurements for various situations.[15] The majority of radon testing protocols provide guidelines for testing in homes, schools, and other buildings. A home or business owner can either test for radon by purchasing a detector at a local retailer (e.g., hardware store, department store) or hiring a qualified tester. Names of qualified testers are available at the radon office located in the department of health in most states. However, licensed radon testers are generally hired if the test is being conducted as part of a real estate transaction.

Short-term radon measurements, or screening radon measurements, have measurement periods ranging from two to ninety days, depending on detector type. Because of the need for rapid testing during real estate transactions, measurements for real estate purposes usually last for two to five days in the United States. Alternatively, the testing period for long-term measurements is greater than ninety days. Long-term measurements, especially when performed for one year, help account for seasonal radon variation and provide a better estimate of a building's average year-long radon concentration than do the short-term measurements. The EPA has provided recommendations for measuring radon concentrations.[18] If the measured radon concentration is 4 pCi/L (150 Bq/$m^3$) or greater after performing a short-term test, the EPA recommends follow-up testing, which can take the form of either a long-term measurement or another short-term measurement if results are needed quickly.

### Testing Conditions and Placement of Detector

The EPA recommends that prior to performing a short-term test and during testing, "closed-house" conditions should be maintained.[18] For "closed-house" conditions, the windows and outside doors should be kept shut as much as possible. Heating or air-conditioning system fans may continue

to be operated during the testing. However, fans that bring air into the home from the outside should not be operated. If the short-term tests will last just two or three days, the EPA suggests closing up the house for at least twelve hours prior to the start of testing.[18] Closed-house conditions are suggested so that the measured radon concentrations will in most cases represent a worst case scenario.

Detectors, especially charcoal-based detectors, should not be placed in areas of high humidity and moisture such as bathrooms. The EPA recommends that either short- or long-term detectors be placed in an occupied area of the lowest lived-in level of the home. The detector should be placed at least twenty inches from the floor, three feet away from windows or doors, four inches from other objects, and not above eight feet from the floor if possible.

## Detectors

Radon measurement devices can be classified in several ways: 1) by measurement of radon or radon decay products, 2) as passive (i.e., no pump or electricity required) or active, 3) by duration of measurement, or 4) by type of detector. Devices measuring radon decay products are primarily used by radon professionals and researchers. Overall, radon gas concentrations are considered a good indicator of radon decay product concentrations. Some of the more frequently used radon measurement devices are listed in Table 23.1.

Passive devices require no energy to operate. Two of the more popular passive devices are the activated charcoal and alpha track detectors. The

**TABLE 23.1**
Radon Gas Measurement Devices and Their Characteristics

| *Detector Type* | *Passive/Active* | *Typical Sampling Period* | *Cost* |
|---|---|---|---|
| Activated Charcoal Detector (ACD) | Passive | 2–7 days | Low |
| Alpha Track Detector (ATD) | Passive | 1–12 months | Low |
| Electret Ion Chamber (EIC) | Passive | 2 days–1 year | Medium |
| Electronic Integrating Device (EID) | Active | 2 days–year(s) | Medium |
| Continuous Radon Monitor (CRM) | Active | 2 hours–year(s) | High |

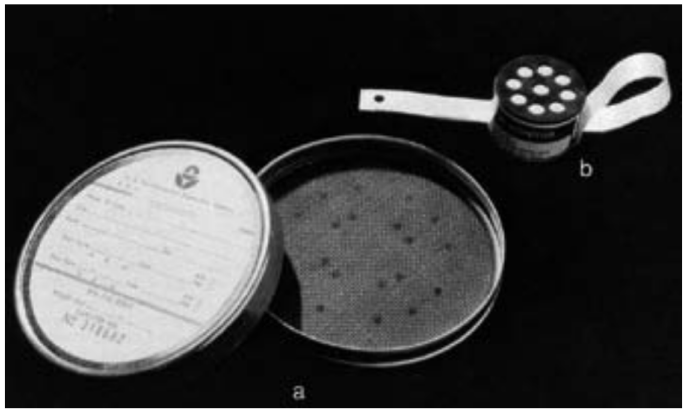


**Figure 23.4** Photo of activated charcoal detector (a) and alpha track detector (b).

activated charcoal detector is often a circular device containing activated charcoal. The detector can be open face (i.e., no diffusion barrier) or covered with a diffusion barrier that helps provide a more integrated average radon concentration (see Figure 23.4a). When the detector covering is removed, radon can enter the detector, where it adsorbs to the activated charcoal. After the testing period ends, the detector is closed and sent to a laboratory for analysis. Activated charcoal detectors also come in other configurations, such as bag or tray type detectors. The radon concentration is obtained by measuring the amount of gamma decay from the adsorbed radon.

Another passive device is the alpha track detector (see Figure 23.4b), which is commonly used for long-term measurements. These devices contain a small piece of plastic that records the alpha decay of radon. After the device is opened and exposed to air, radon gas enters the detector through the filter in either the top or side of the detector, depending on the vendor. After the radon enters the detector, it undergoes radioactive decay and some of the emitted alpha particles strike the plastic chip inside the detector, forming small pits. After chemical etching of the plastic chip is performed, the pits can be counted using a light microscope to determine the number of tracks per unit of area. The detectors are calibrated so that the radon concentration can be calculated given the duration of exposure and number of tracks. A major advantage of this detector is that it can be placed in the home for as long as a year to obtain an average integrated year-long radon concentration. Another advantage of this detector over the charcoal-based detector is that it is not influenced by humidity or temperature.[15]

Other types of radon measurement devices, used by professional radon testers and researchers, include the electret ion chamber (sometimes called an E-PERM), continuous radon monitor, and radon progeny detector. A detailed description of these and other devices (e.g., electronic radon detector) as well as information on the measurement of radon in water are available in the WHO *Handbook on Indoor Radon*.[15] The handbook also contains important information on quality assurance and quality control methods for performing radon measurements that should be followed to obtain reliable measurements.

## Radon Mitigation

Radon reduction control systems have been effectively developed that can reduce indoor radon concentrations to less than 2 pCi/L (75 Bq/$m^3$) in over 85 percent of cases. Certified radon mitigators are trained to install radon-reduction systems. Contact information for certified radon mitigators in the United States is available to the public from state health departments or by calling 1-800-SOS-RADON. The cost of mitigating a home depends on the type of property (e.g., difficulty in installing a system) and geographic area within the United States.

Common entry points for radon in the substructure of a home or other building include locations around drains, foundation cracks, sump pumps, pipe penetrations in walls, and areas under toilets or bathtubs if installed on the ground level. Sealing openings in the basement or floor in contact with the ground is generally not sufficient alone to reduce radon concentrations, but is often carried out to improve the effectiveness of radon reduction systems.[30] Ventilation can also be used to dilute the radon in indoor air with outdoor air. But the expense of continuously ventilating a home may result in high energy costs.

Active soil depressurization (ASD) is often the most efficient, reliable, and cost-effective radon mitigation technique.[31] It is also one of the most common methods for radon mitigation in Europe.[32] ASD is applied in existing homes by creating a vacuum below the foundation of a building and removing the radon away from the lowest level before it enters the building. A subslab suction is established by creating a hole in the foundation, digging the area beneath the hole to form a small pit, inserting a four-inch polyvinyl chloride (PVC) pipe into the hole, running the

PVC pipe above the roof line, and installing a radon mitigation fan. The fan can be located in an area of the building away from the main occupied spaces, such as a garage, attic, or even outside. A discharge point for the PVC pipe above the eaves of the roof is recommended to help prevent the radon from reentering the home. The expected life of the fan is about ten to fifteen years. The radon reduction system should have a performance indicator installed for the fan to ensure it is functioning properly. Testing of radon should also be performed at least every two years to confirm that the mitigation system is still reducing radon concentrations.

For newly constructed buildings, radon control systems can be installed during construction that use passive soil depressurization. Passive systems operate in a similar manner to ASD systems, but without the fan. However, the homeowner should perform a radon test in the home prior to or at the time of residency, to make sure the ASD system is reducing the radon concentrations to acceptable concentrations. If the passive system does not reduce the radon concentration to below 2 pCi/L (75 Bq/m$^3$), a fan can be installed to make the system active (i.e., ASD). See the WHO *Handbook on Indoor Radon* for additional information on radon measurement devices and radon mitigation techniques.[15]

## Notes


1. National Council on Radiation Protection and Measurements, *Ionizing radiation exposure of the population of the United States*, Report no. 160 (Bethesda, MD: NCRP, 2009).
2. United States Environmental Protection Agency, *EPA assessment of risks from radon in homes*, EPA 402-R-03-003 (Washington, DC: U.S. Environmental Protection Agency, Office of Radiation and Indoor Air, 2003).
3. H. Becquerel, Discovery of radioactivity, *Nature* 161 (1948): 609–612.
4. J. R. Partington, Discovery of radon, *Nature* 179 (1957): 912.
5. Agency for Toxic Substances and Disease Registry, *Toxicological profile for radon* (Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, 2009).
6. H. O. Wyckoff, International system of units (SI), *American Journal of Roentgenology* 131 (1978): 536–538.

7. National Research Council, Committee on Risk Assessment of Exposure to Radon in Drinking Water, Board on Radiation Effects Research, Commission on Life Sciences, *Risk assessment of radon in drinking water* (Washington, DC: National Academy Press, 1999).
8. R. W. Field, E. L. Fisher, R. L. Valentine, et al., Radium-bearing pipe scale deposits: Implications for national waterborne radon sampling methods, *American Journal of Public Health* 85 (1995): 567–570.
9. United States Environmental Protection Agency, Office of Radiation Programs, *Radon reference manual*, EPA 520/1-87-20 (Washington, DC: U.S. Environmental Protection Agency, 1987).
10. S. W. White, L. C. S. Gundersen, and R. R. Schumann, Development of EPA's map of radon zones, EPA 600/R-93/083b (The 1992 International Symposium on Radon and Radon Reduction Technology, Vol. 2, oral papers, technical sessions VII-XII, Research Triangle Park, NC, 1992).
11. United States Geological Survey, *Radon potential of the upper Midwest* (Washington, DC: U.S. Geological Survey, 1995).
12. B. Alexander, N. Rodman, S. B. White, et al., Areas of the United States with elevated screening levels of 222Rn, *Health Physics* 66 (1994): 50–54.
13. D. J. Steck, R. W. Field, and C. F. Lynch, Exposure to atmospheric radon (222Rn) in central North America, *Environmental Health Perspectives* 1072 (1999): 123–127.
14. F. Marcinowski, Nationwide survey of residential radon levels in the US, *Radiation Protection Dosimetry* 45 (1992): 419–424.
15. World Health Organization, *WHO handbook on indoor radon: A public health perspective* (Geneva, Switzerland: WHO, 2009).
16. United States Environmental Protection Agency, Indoor air quality tools for schools reference guide: Appendix G, EPA 402-K-07-008 (Washington, DC: U.S. Environmental Protection Agency, 2009).
17. National Research Council, Committee on Health Risks of Exposure to Radon (BEIR VI), Board on Radiation Effects Research, Commission on Life Sciences, *Health effects of exposure to radon, BEIR VI* (Washington, DC: National Academy Press, 1999).
18. United States Environmental Protection Agency, Indoor Environments Division, *Citizen's guide to radon: The guide to protecting yourself and your family from radon*, EPA 402-K-09-001 (Washington, DC: U.S. Environmental Protection Agency, 2009).

19. United States Environmental Protection Agency, *Technical fact sheet: Proposed radon in drinking water rule*, EPA 815-F-99-006 (Washington, DC: U.S. Environmental Protection Agency, 1999).
20. International Agency for Research on Cancer, *Man-made mineral fibres and radon*, IARC Monographs on the evaluation of carcinogenic risks to humans, vol. 43 (Lyon, France: IARC, 1988).
21. American Cancer Society, *Cancer facts & figures 2010* (Atlanta, GA: American Cancer Society, 2010).
22. National Research Council, Panel on Dosimetric Assumptions Affecting the Application of Radon Risk Estimates, Board on Radiation Effects Research, Commission on Life Sciences, *Comparative dosimetry of radon in mines and homes* (Washington, DC: National Academy Press, 1991).
23. S. Darby, D. Hill, A. Auvinen, et al., Radon in homes and risk of lung cancer: Collaborative analysis of individual data from 13 European case-control studies, *British Medical Journal* 330 (2005): 223–226.
24. D. Krewski, J. H. Lubin, J. M. Zielinski, et al., Residential radon and risk of lung cancer: A combined analysis of 7 North American case-control studies, *Epidemiology* 16 (2005): 137–145.
25. J. H. Lubin, Z. Y. Wang, J. D. J. Boice, et al., Risk of lung cancer and residential radon in China: Pooled results of two studies, *International Journal of Cancer* 109 (2004): 132–137.
26. R. W. Field, B. J. Smith, D. J. Steck, et al., Residential radon exposure and lung cancer: Variation in risk estimates using alternative exposure scenarios, *Journal of Exposure Analysis and Environmental Epidemiology* 12 (2002): 197–203.
27. J. H. Lubin, Environmental factors in cancer: Radon, *Reviews on Environmental Health* 25 (2010): 33–38.
28. M. R. Bonner, W. P. Bennett, W. Xiong, et al., Radon, secondhand smoke, glutathione-S-transferase M1 and lung cancer among women, *International Journal of Cancer* 119 (2006): 1462–1467.
29. V. Řeřicha, M. Kulich, R. Řeřicha, et al., Incidence of leukemia, lymphoma, and multiple myeloma in Czech uranium miners: A case-cohort study, *Environmental Health Perspectives* 114 (2006): 818–822.
30. B. J. Smith, L. Zhang, and R. W. Field, Iowa radon leukaemia study: A hierarchical population risk model for spatially correlated exposure measured with error, *Statistics in Medicine* 26 (2007): 4619–4642.

31. United States Environmental Protection Agency, *Radon reduction techniques for existing detached houses: Technical guidance for active soil depressurization*, 3rd ed., EPA 625-R-93-011 (Washington, DC: U.S. Environmental Protection Agency, 1993).
32. World Health Organization, *International radon project: Survey on radon guidelines, programmes, and activities* (Geneva, Switzerland: WHO, 2007).