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RADON

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Radon (radon-222), an inert gas at usual temperatures, is a naturally occurring decay product of radium-226, the fifth daughter of uranium-238. Both uranium-238 and radium-226 are present in most soils and rocks although the concentrations vary widely (National Council on Radiation Protection and Measurements [NCRP] 1984a). As radon forms from the decay of radium-226, some of the molecules leave the soil or rock and enter the surrounding air or water. As a result, radon is ubiquitous in indoor and outdoor air, and its concentration is increased by the presence of a rich source and by low ventilation of the air in contact with that source.

Radon decays with a half-life of 3.82 days into a series of solid, short-lived radioisotopes that are collectively referred to as *radon daughters*, *radon progeny*, or *radon decay products* (Figure 15.1). Two of the decay products, polonium-218 and polonium-214, emit α-particles, high-energy and high-mass particles consisting of two protons and two neutrons. When these emissions take place within the lung as inhaled radon progeny decay, the cells lining the airways may be damaged, and lung cancer may ultimately result.

The mining of radioactive ores that release radon was the first occupation to be associated with an increased risk of lung cancer. More than one hundred years ago, Harting and Hesse (1879) described autopsy findings in Eastern European miners which documented an occupational hazard of lung cancer. Excess occurrences of lung cancer have subsequently been found in uranium miners in the United States, Czechoslovakia, France, and Canada, and in other underground miners exposed to radon progeny, including Newfoundland fluorspar miners, Swedish and U.S. metal miners, British and French iron miners, and Chinese and British tin miners (National Research Council [NRC] 1988). In recent years, the exposure of animals

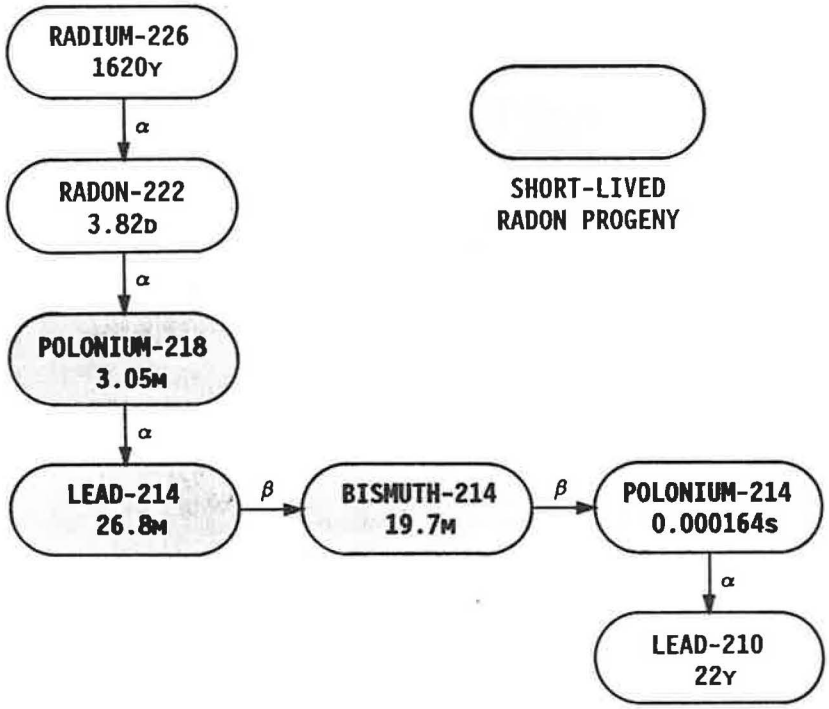

Figure 15.1. Decay pathway from radium-226 to lead-210. y, year; d, day; m, minute; s, second.

to radon and its progeny has confirmed that radon progeny cause lung cancer (Cross 1988; NRC 1988).

As information on air quality in indoor environments accumulated in the late 1970s, it became apparent that radon and its decay products are invariably present in indoor environments and that concentrations may reach unacceptably high levels in some dwellings. The well-documented excess of lung cancer among underground miners exposed to radon progeny raised concern that exposure to radon progeny might also be a cause of lung cancer in smokers and nonsmokers in the general population. During the 1970s, the Scandinavian and other European countries and the United States developed research programs on radon in homes. However, the problem of radon in indoor environments did not receive great attention in the United States until a widely publicized incident in 1984. During routine monitoring, a Pennsylvania nuclear power plant worker was found to be contaminated with radioactivity. This contamination was subsequently traced to a high concentration of radon in his home, which was located on a geologic formation named the Reading Prong. High levels of radon have now been documented in

numerous homes on the Reading Prong (Logue and Fox 1985) and in other locations throughout the United States.

This dramatic incident and the subsequent finding of high levels of radon in many homes have resulted in substantial media coverage of radon in the indoor environment and intense public concern about the problem. Several books on radon have been published for the general public (Cohen and Nelson 1987; Lafavore 1987), and the subject has been covered in numerous magazine articles. The Environmental Protection Agency (U.S. EPA), although without direct authority to regulate indoor air quality, has published "action guidelines" for acceptable levels of radon indoors in its pamphlet "A Citizen's Guide to Radon" (U.S. EPA 1986). In September 1988 the EPA and the Centers for Disease Control issued an advisory urging that most houses in the United States be tested for radon. A 1988 amendment to the Toxic Substances Control Act sets a national long-term goal of making the air in buildings "as free of radon as the ambient air outside of buildings."

New research initiatives on radon have been implemented in the United States by the Department of Energy, the EPA, and the National Institutes of Health, by state agencies, and by other governments throughout the world. This research addresses not only biologic aspects of radon exposure, but also geologic aspects, the movement of radon into homes, and control measures. Recent monographs (Cothern and Smith 1987; Nazaroff and Nero 1988) cover these areas comprehensively.

Other isotopes of radon, radon-219 (actinon) and radon-220 (thoron), occur naturally, and their progeny also include α-emitters. Radon-219, which has a half-life of only 3.9 seconds, is present in extremely low concentrations and contributes little to human exposure. Because of its short half-life (56 seconds), the concentration of radon-220 is also usually low. Further, the dosimetry of thoron progeny in the respiratory tract implies much lower doses to the target tissues from thoron daughters than from radon daughters. Accordingly, this chapter does not consider either radon-219 or radon-220.

EXPOSURE TO RADON

For historical reasons, the concentration of radon progeny is generally expressed as working levels (WL), where 1 WL is any combination of radon progeny in 1 liter of air which ultimately releases 1.3×10^5 MeV of α-energy during decay (Holaday et al. 1957). Concentrations of radon are also frequently expressed as picocuries (pCi) per liter; a concentration of 1 pCi/liter translates to about 0.005 WL under usual conditions in a home. Exposure to 1 WL for 170 hours equals one working level month (WLM) of exposure. The WLM was developed to describe exposure sustained by miners during the average number of hours spent underground. Because most persons spend much more than 170 hours in their home each month, a concentration of 1 WL in a residence results in an exposure much greater

than 1 WLM on a monthly basis. Thus, if one assumes that 70 percent of one's time is spent at home, a 1-WL concentration (200 pCi/liter at 50 percent equilibrium) would yield an exposure of 3.0 WLM monthly or 36 WLM annually. The approximate average concentration in U.S. homes (1.5 pCi/liter) (Nero 1988), under the same occupancy assumptions, results in an exposure of about 0.02 WLM monthly, about 0.3 WLM annually, and about 20 WLM over a seventy-year lifetime.

These units are now frequently replaced by Système International (SI) units. In SI units, the concentration of radon in air is expressed as Becquerels per cubic meter (Bq/m^3); at radioactive equilibrium between radon and its decay products, 1 WL corresponds to 3.7×10^3 Bq/m^3. Cumulative exposure in SI units is expressed in Joule-hours per cubic meter (Jh/m^3), and 1 WLM is 3.5×10^{-3} Jh/m^3.

The predominant source of radon in indoor air is the soil beneath structures, but building materials, water used within the home, and utility natural gas may also contribute (NCRP 1984a; Nero and Nazaroff 1984; Nazaroff, Moed, and Sextro 1988; Nazaroff et al. 1988; Stranden 1988) (Figure 15.2). The concentrations of radium in soil and in rock vary over several orders of magnitude; this variation in source strength, rather than a variation in ventilation rate, underlies most of the variation in radon concentration among dwellings (Nero and Nazaroff 1984; Nazaroff, Moed, and Sextro 1988). The rate of exchange of indoor air with outdoor air, the "tightness" of the home, also determines the indoor radon concentration. However, air exchange rate is a less important determinant of variation of radon concentrations among homes than is the source strength of the soil (Nero and Nazaroff 1984; Nero 1988).

For the most part, building materials make only a small contribution to indoor radon. Certain materials, however, have high concentrations of radium and are strong sources of radon: granites, Swedish concrete made with alum shale, and building materials made with wastes from industrial processes, such as phosphogypsum, phosphate slag, and fly ash (Nero and Nazaroff 1984; Stranden 1988). Drinking water from most sources contributes little to indoor radon, but water from some private wells may add significant amounts of radon to indoor air (Nazaroff et al. 1988).

The movement of radon through building materials and soil is a complex process that involves molecular diffusion, gas flow, and transport via water (Nero and Nazaroff 1984; Nazaroff, Moed, and Sextro 1988). The entry of radon into a building is determined by the structural characteristics and the flow of radon-containing air into the building, as the flow is influenced by wind, temperature, barometric pressure, and soil moisture (see Chapter 3). Portals of entry include cracks and sump holes in basements, crawlspaces, and cracks in concrete slabs. Short-term variation in the concentration of radon within a home results from changes in air exchange rates, varying meteorologic conditions, and the use of water and natural gas.

Radon concentrations have not yet been measured within a large random sample of U.S. homes although surveys have been undertaken in other countries (see

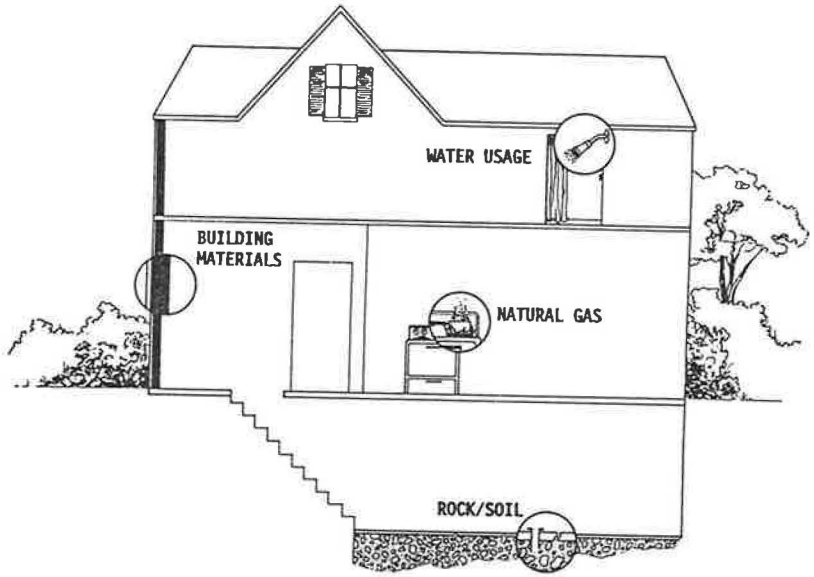

Figure 15.2. Sources of radon in indoor air.

Nazaroff and Nero [1988] for a review of these data). Three nationwide data bases and surveys of states conducted by the EPA provide descriptions of concentrations within U.S. homes. Nero and colleagues (1986) examined thirty-five data sets of radon measurements taken in the United States and identified twenty-two considered to provide unbiased data on concentrations in single-family homes. The data followed a log normal distribution (Figure 15.3). The average concentration was approximately 1.5 pCi/liter; 1–3 percent of the homes exceeded 8 pCi/liter. Based on this analysis, Nero et al. (1986) suggested that more than one million U.S. homes may have annual average radon concentrations exceeding 8 pCi/liter.

Cohen (1986) conducted a survey of radon levels in homes of 453 physics professors from forty-two states. One-year measurements were made with nuclear track detectors. The distribution of the measurements was log normal, with a geometric mean of 1.0 pCi/liter and an arithmetic mean of 1.5 pCi/liter.

Alter and Oswald (1987) reported the results of more than sixty thousand indoor radon measurements made with Track Etch radon detectors. The data are limited because the dwellings do not represent a random sample, and measurement values rather than concentrations within individual residences were reported. The measurements document that most states have homes with concentrations above the action limit of 8 pCi/liter set by the EPA. After the removal of the measurements most likely to introduce bias, the geometric mean concentration was approxi-

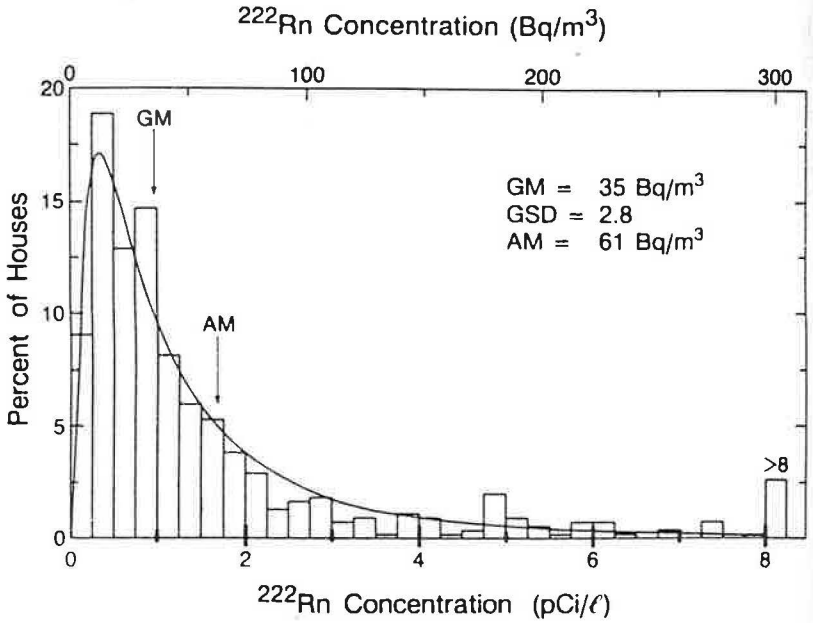

Figure 15.3. Probability distribution of radon in U.S. homes. The distribution represents data from 552 U.S. homes taken from nineteen sets of data. GM, geometric mean; GSD, geometric standard deviation; AM, arithmetic mean. *Source:* Nazaroff W. W. and Nero A. V. Jr., *Radon and Its Decay Products in Indoor Air* (New York: John Wiley and Sons, 1988). Reprinted with permission.

mately 1.5 pCi/liter and the arithmetic mean concentration about 4 pCi/liter. However, as pointed out by Nero et al. (1986), this result may be misleading since approximate adjustment of the average to take account of the sampling protocols yields a result similar to the 1.5 pCi/liter cited above.

Through 1988, the EPA had surveyed homes in seventeen states with the use of charcoal canisters to make short-term measurements. The measurement protocol used by the EPA for screening homes specifies conditions that tend to bias concentrations upward in relation to the annual average concentration (Samet and Nero 1989); the agency's protocol specifies that measurements should be made in the lowest potentially habitable space under closed-house conditions. In the initial survey of ten states, more than one-fifth of the homes tested had levels above the action guideline of 4 pCi/liter. In a September 1988 press conference, the agency reported that nearly one in three homes surveyed in seven additional states had a concentration above the guideline, as assessed with the screening protocol. The EPA projected that more than three million homes in the seventeen states surveyed have screening concentrations above 4 pCi/liter.

Although radon measurements have not yet been made within a nationwide

sample of homes, the available data provide important insights into the population's exposure. Most of the collective exposure results from homes with low concentrations, well below the action limit of 4 pCi/liter set by the EPA (Figure 15.3). Thus, the population's total burden of exposure and of radon-related lung cancer reflects primarily the average concentration in all homes rather than in the small proportion of homes with extremely high levels. Such homes, however, place long-term occupants at particularly high risk.

RESPIRATORY DOSIMETRY OF RADON PROGENY

OVERVIEW

The relationship between exposure to radon decay products, measured as WLM or Jh/m^3, and dose to target tissues in the respiratory tract is extremely complex and is dependent on both biologic and nonbiologic factors, including the physical characteristics of the inhaled air, the amount of air inhaled, breathing patterns, and the biologic characteristics of the lung (Table 15.1) (NCRP 1984b; James 1988). The factors influencing the relation between exposure and dose could plausibly differ for the circumstances of exposure in homes and in mines; it cannot be assumed that the same exposures in a home and in a mine lead to the same doses of α-radiation to target cells in the lung and hence to the same lung cancer risk. Thus, if the epidemiologic evidence from studies of miners is to be used to estimate the risk of indoor radon, the comparative dosimetry of radon decay products in the mining and indoor environments must be assessed.

Certain aspects of lung structure and function are important determinants of the dosimetry of radon decay products (Murray 1986; James 1988). Inhaled air flows through the nasal and oral airways to the trachea; at rest, the nasal route predominates, but flow through the oral route increases with exercise. The lung comprises the airways, a dichotomously branching system of tubes, and the alveoli, the saccular structures in which gas exchange takes place. Gas flow is turbulent in

Table 15.1 Physical and Biologic Factors Influencing the Dose to Target Cells in the Respiratory Tract from Radon Exposure

Physical Factors
Fraction of daughters unattached to particles
Aerosol size distribution
Equilibrium of radon with its progeny
Biologic Factors
Tidal volume and respiratory frequency
Partitioning of breathing between the oral and nasal routes
Bronchial morphometry
Mucociliary clearance rate
Mucus thickness
Location of target cells

the larger airways and laminar in the smaller airways; gases move by diffusion in the alveolar spaces. The respiratory system has multiple defense mechanisms for handling inhaled particles such as radon decay products and gases. The nose efficiently removes soluble gases, large particles, and charged particles, such as the unattached fraction of radon decay products. In the lung, particles in the size range of 2–10 μm tend to deposit in the airways and are cleared by the mucociliary apparatus, which moves mucus toward the larynx, where it is coughed or swallowed. Submicron particles also deposit in the airways with increasingly high deposition fractions as the particle diameter decreases.

Most human lung cancers arise at the level of segmental and subsegmental airways, at about the third through the fifth airways generations (Fraser et al. 1989). Relatively few human lung cancers occur peripherally. The airways where most lung cancers develop have a cartilaginous structure and are lined by a pseudostratified ciliated columnar epithelium; that is, the superficial layer includes cells with cilia, which beat in an organized fashion to propel mucus toward the trachea, and the cells appear to be in multiple strata although only one layer is present (Murray 1986). The cellular components of the airways epithelium include the ciliated epithelial cells, mucus-secreting cells, basal reserve cells, and other types. Although the cellular origins of human lung cancer are controversial, all of the principal cell types of the airways epithelium are considered to have the potential to undergo malignant transformation (McDowell and Trump 1983). The relevant target for carcinogenesis by α-particles is assumed to be the entire epithelium and not just the basal cells.

The dose of α-energy delivered to target cells in the lungs cannot be measured directly; modeling approaches are used to simulate the complex sequence of events from inhalation of radon decay products to cellular injury by α-particles. The models incorporate the biologic processes that follow inhalation as well as the physical state of the inhaled radon decay products, also an important determinant of the exposure-dose relationship for radon decay products.

Radon is an inert gas, but its decay products are solid, charged particles. Although most of the decay products attach to aerosols immediately after formation, a variable proportion of the atoms remains unattached and is referred to as the unattached fraction (Phillips, Kim, and Lucas 1992). The fraction of unattached radon decay products in inhaled air is an important determinant of the dose received by target cells at a particular concentration in inhaled air; as the unattached fraction increases, the dose also increases because of the efficient deposition of the unattached decay products in the larger airways (James 1988). The size distribution of particles in the inhaled air also influences the dose to the airways because particles of different sizes deposit preferentially in different generations of airways (James 1988). The specific mixture of radon decay products also affects the dose to target cells, although to a lesser extent.

The amount of inhaled radon decay products varies directly with the minute ventilation, the total volume of air inhaled each minute. The increased ventilation associated with activity increases the inhaled burden of radon decay products. The deposition of radon decay products within the lung, however, does not vary in a simple fashion with the minute ventilation but varies with the flow rates in each airway generation (James 1988). The dose changes approximately with the square root of the breathing rate. The proportions of oral and of nasal breathing also influence the relationship between exposure and dose (James 1988). A substantial proportion of the unattached radon decay products deposits in the nose with nasal breathing whereas unattached decay products do not deposit in the mouth with oral breathing but rather on the bronchial epithelium, where target cells are located.

Characteristics of the lung also influence the relationship between exposure and dose (Table 15.1). The sizes and branching patterns of the airways affect deposition, and these aspects of airways configuration may differ between children and adults and between males and females. At a given level of exposure, the dose to target cells may be higher for children (International Commission on Radiological Protection [ICRP] 1987; James 1988). Once deposited in the airways, radon decay products are cleared by the mucociliary apparatus. Thus, the rate of mucociliary clearance and the thickness of the mucous layer in the airways also enter into dose calculations. The dose increases as the mucociliary clearance slows and diminishes with increasing thickness of the mucous layer. Cigarette smoking tends to reduce the rate of clearance and to increase the thickness of the mucous layer.

The cells of the airways absorb α-energy as α-particles released in the decay of polonium-218 and polonium-214 on the epithelium's surface move through the epithelial layer. These particles have a short range in tissue but can penetrate to the basal layer. Cellular doses can be calculated (James 1988).

Computer models have been developed to describe the relationship between exposure to radon decay products and the dose of α-radiation to target tissues and to assess the consequences on this relationship of the physical and biologic factors listed in Table 15.1. These complex models generally incorporate biologic factors including airways geometry, mucociliary deposition, particle deposition, ventilation pattern, and location of the target cells, and physical factors including the unattached fraction and the aerosol size distribution (Nuclear Energy Agency 1983; NCRP 1984b; James 1988). Using such models, factors for converting exposure to an absorbed radiation dose can be calculated, but the range of published dose conversion factors is wide (James 1988). As summarized by James (1988), the values span from 0.8 rad/WLM (0.8 mGy/WLM) to about 10 rad/WLM (100 mGy/WLM). For the attached and unattached fractions specifically, the dose conversion factors cover a narrower range. Recent estimates for the attached fraction are about 0.2–1.3 rad/WLM (2–13 mGy/WLM); and for the unattached fraction, from about 10 to 20 rad/WLM (100–200 mGy/WLM). To convert absorbed dose to tissue dose equivalent in units of rem or sieverts in the SI system, the absorbed dose in rads or grays is multiplied by twenty, the quality factor for α-radiation.

COMPARISONS OF DOSIMETRY IN THE INDOOR AND MINING ENVIRONMENTS

Dosimetric models have proved useful for evaluation of uncertainties in extrapolating from the mining to the general indoor environment. Using dosimetry models, the α-dose to the respiratory tract has been compared under the circumstances of exposure in homes and in mines (James 1988). In comparison with mines, the unattached fraction is higher in homes, and the aerosol size distributions may differ in the two environments. The ventilation rates of working miners are higher on average than those of the general population during usual home activities. In addition, the airways geometry of children differs from that of adults. These comparative analyses indicate that exposures to radon decay products in homes and in mines yield essentially comparable doses of α-energy to the respiratory tracts of adults (NCRP 1984b; James 1988); for children, the estimated doses are higher than for adult miners and nonminers because of the morphometric differences between the lungs of children and adults. For adults, the equivalence of the exposure-dose relationship holds in each of the currently prominent dosimetric models (James 1984).

The Committee on the Biological Effects of Ionizing Radiation (BEIR IV) (NRC 1988) used a descriptive approach and also concluded that exposure-dose relationships were similar for exposure in homes and in mines. The committee reviewed the likely range dose conversion factors for particle size, unattached fraction, equilibrium factor, and minute ventilation in homes and in mines. The committee's estimates for the ratios of these factors in homes to mines were 1.4, 1.2, 1, and 0.56, respectively. When considered together, the product of these ratios is near unity. The committee's approach assumed that the remaining biologic determinants of the exposure-dose relationship were comparable in miners and in the general population. A separate analysis was not performed for children.

RADON AND LUNG CANCER

INTRODUCTION

The causal association of exposure to radon and decay products with lung cancer has been amply documented through epidemiologic investigations of underground miners (NRC 1988). Studies of miners have shown rising lung cancer risk as cumulative exposure to radon decay products increases and have provided some insights into the combined effects of cigarette smoking and exposure to radon decay products. Quantitative exposure-response relationships have been described with the use of data from several of the cohort studies (Lubin 1988; Samet 1989). The range of excess relative risk coefficients, from 0.5 to 3.0/100 WLM, is remarkably narrow in view of the differing assessments of exposure and analytic methods among the investigations. These studies have been less informative concerning the temporal expression of the excess risk and the effect of varying exposure rates.

Animal experiments have also provided data on exposure-response relationships and on the modifying effects of exposure rate and the physical characteristics of the inhaled radon decay products (NRC 1988). Animal models have proved less useful for studying the interaction of radon decay products with cigarette smoking because of the difficulties of replicating smoking patterns of humans with animals.

The lung cancer risk associated with exposure to radon decay products must be considered in the context of the extensive literature on cigarette smoking and lung cancer. This malignancy, uncommon at the start of the century, has become the leading cause of cancer death in the United States (U.S. Department of Health and Human Services [DHHS] 1982). Most lung cancers are caused by cigarette smoking, and only 5–10 percent of the total occurs in lifelong nonsmokers (U.S. DHHS 1982; World Health Organization 1986). In cigarette smokers, the risk of developing lung cancer increases with the number of cigarettes smoked daily and with the number of years smoked (U.S. DHHS 1982; Doll and Peto 1978). The risk of lung cancer for a smoker compared with a nonsmoker is increased approximately tenfold on average but reaches twentyfold or higher in heavier smokers.

Because cigarette smoking predominates as the cause of lung cancer, the risk from exposure to radon decay products must be addressed separately for smokers and for nonsmokers. When one agent (cigarette smoke, for example) modifies the effect of another (radon, for example), interaction is present. Interactions between two agents may be either synergistic or antagonistic; *synergism* refers to an increased effect of the independent exposures when both are present whereas *antagonism* refers to a reduced effect. Synergism is considered to be present if the joint effect of the two exposures exceeds the sum of the independent effects. If the combined effect equals the product of the independent risks, then the interaction is considered to be multiplicative; the interaction is considered additive if the combined effect equals the sum of the independent risks.

If two agents interact in a synergistic fashion, then some cases can be attributed to the two factors acting alone and some to their joint action. The cases having shared causation can in theory be prevented by removing either of the two interacting agents. Estimates of the numbers or proportions of preventable cases may thus exceed the total number of cases or 100 percent. For agents interacting synergistically with cigarette smoking, the cases attributable to the agent are not only those in never smokers, but those in smokers that are caused by the interaction. For radon decay products, which appear to interact synergistically with cigarette smoking, the proportion or number of attributable lung cancer cases exceeds the approximately 10 percent or fifteen thousand cases estimated to occur in never smokers.

Cigarette smoking has well-described effects on both the airways and the lung parenchyma (U.S. DHHS 1984); these effects may plausibly modify the relationship between exposure to radon and dose of α-energy to cells. In comparison with that in nonsmokers, the dose in smokers might be increased by the greater central deposition, the increased airways permeability, and the slowed mucociliary transport that have been demonstrated to result from smoking. The dose in smokers might be reduced by mucosal edema and by the increased mucus thickness, on average, secondary to the heightened mucus production in the airways of smokers.

Components of tobacco smoke might also interact with α-particles in the process of carcinogenesis itself. At present, a conclusion cannot be reached through biologically based arguments alone concerning the net consequence of interaction between cigarette smoking and exposure to radon decay products. Thus, the determination of the form of interaction between exposure to radon decay products and cigarette smoking has been based primarily on the epidemiologic studies of underground miners.

Additional insight into the interaction between exposure to radon and cigarette smoking can be gained from those epidemiologic studies of miners which documented both of these exposures; unfortunately, such information is not available for all of the study groups. Small case numbers in some of the studies also limit the statistical precision with which the interaction can be described. Although the smaller investigations have yielded inconsistent results, the largest investigation, that of Colorado Plateau uranium miners, indicates a multiplicative or somewhat submultiplicative interaction (Whittemore and McMillan 1983; Hornung and Meinhardt 1987; NRC 1988). Analyses of data from the New Mexico uranium miners, at present the only other large cohort with smoking information for all cohort members, also indicate a multiplicative interaction (Samet et al. 1989). A multiplicative interaction yields the same level of relative risk in smokers and nonsmokers for a particular exposure, but the higher background risk of the smokers is multiplied by that resulting from radon.

EPIDEMIOLOGIC INVESTIGATIONS

To date, epidemiologic investigations of indoor exposure to radon as a risk factor for lung cancer have been limited by the methodologic difficulties of studying this exposure. Both descriptive and analytical approaches have been used to examine the association between exposure to radon in the home and lung cancer. Techniques for accurately estimating lifetime exposure of individuals to radon in indoor air are not yet available, and surrogates for exposure based on residence type, geology, or limited measurements have of necessity been used in the case-control and cohort studies. The principal published reports are reviewed; Borak and Johnson (1988) summarized the relevant literature, including several unpublished investigations.

In the descriptive studies, incidence or mortality rates for lung cancer within geographic units were correlated with measures of exposure for inhabitants of these units (Table 15.2). In spite of crude exposure measures, most of these studies showed associations between exposure to radon and incidence or mortality from lung cancer. Two studies of counties in the Reading Prong are of particular interest because of the number of homes in this region with high radon concentrations (Fleischer 1986; Archer 1987). Both studies indicated increased mortality from lung cancer in residents of the counties with the highest exposures. However, these descriptive studies, which did not consider the exposures of individuals to radon decay products and other agents, can provide only suggestive evidence that exposure to radon in the home increases the risk of lung cancer.

Table 15.2 Descriptive Studies of Exposure to Radon and Lung Cancer

Location (Reference)	Outcome Measure	Exposure Measure	Findings
U.S. (Fleischer 1981)	Lung cancer mortality for U.S. counties, 1950–69	Presence of a phosphate deposit, mine, or processing plant in the county	Significant excess of high lung cancer rates in counties with phosphate mills
Iowa, U.S. (Bean et al. 1982)	Lung cancer incidence for municipalities of 1,000–10,000 residents for years 1969–79	Mean level of radium-226 in the water supply	Significantly increasing cancer incidence for males with exposure; increase not significant for females
Sweden (Edling et al. 1982)	Lung cancer mortality rates by county, 1969–78	Estimated background γ-radiation, assumed to correlate with radon	Significant correlations for lung cancer rates in males and females with exposure
Canada (Letourneau et al. 1983)	Lung cancer mortality rates for 18 cities for 1966–79	Geometric mean WL from a survey of 14,000 homes done 1978–80	No association of lung cancer mortality rates with radon daughter levels
Maine, U.S. (Hess, Weiffenbach, and Norton 1983)	Lung cancer mortality rates by county, 1950–69	Estimated county average for radon concentration in water	Significant associations in males and females of lung cancer mortality with exposure
Central Italy (Forastiere et al. 1985)	Lung cancer mortality rates for 31 towns, 1969–78	Soil geologic features	Nonsignificant increase for males and females in higher exposure area
Guangdong Province, China (Hofmann, Katz, and Zhang 1985)	Lung cancer mortality rates for two areas, 1970–83	By area: "control" and "high background"	Similar lung cancer mortality rates in the two areas
Limousin and Poitou-Charentes, France (Dousset and Jammet 1985)	Lung cancer mortality rates for the two regions, 1968–75	By area: from geology indoor radon estimated three to four times higher in Limousin region	Similar lung cancer mortality rates in the two regions
Reading Prong, U.S. (Fleischer 1986)	Lung cancer mortality rates by county, 1950–69)	By county, based on the proportion within the Reading Prong	For the three counties mostly within the Reading Prong, lung cancer mortality significantly elevated in all three for men and in two for women
Reading Prong, U.S. (Archer 1987)	Lung cancer mortality rates by county, 1950–79	By county, based on geology; three levels of exposure	For both sexes combined, lung cancer mortality follows a gradient consistent with exposure
U.S. (Cohen 1988)	Lung cancer mortality rates for 411 U.S. counties, 1950–69	By county, geometric mean concentration measured in 10 or more homes	For males and females, lung cancer mortality rates were inversely associated with county-average radon levels

The association of exposure to radon and lung cancer has been more directly tested in case-control and cohort studies (Table 15.3). In the first of these investigations, Axelson, Edling, and Kling (1979) conducted a case-control study in a rural area of Sweden. Those subjects who lived in stone houses were assumed to be most exposed, and those who lived in wooden houses were assumed to be least exposed; other types of dwellings were considered to be a source of intermediate exposure. In spite of this crude exposure classification, the study showed that residence in stone houses was associated with a significantly increased relative risk compared with residency in wooden houses (age- and sex-adjusted relative risk = 5.4). The study did not consider data on cigarette smoking or lifetime residence history.

In several later case-control studies performed in Sweden (Table 15.3), surrogate exposure indexes were validated against measurements of radon with satisfactory agreement (Edling, Kling, and Axelson 1984; Edling, Wingren, and Axelson 1984; Svensson, Eklund, and Pershagen 1987). The findings of these case-control studies were mixed; some showed significantly increased risk associated with exposure whereas others did not. However, this may be due to the small number of cases in several of the studies and the general use of surrogate measures of exposure. Reliance on surrogate measures may introduce misclassification; that is, some subjects may be assigned higher or lower exposures than they actually received. If misclassification occurs randomly in cases and controls alike, the relative risk estimates will be biased toward unity, and an effect of exposure may not be found.

The more recent studies in Sweden have included larger numbers of cases and controls than those reported initially, and some have incorporated measurement of radon for large numbers of dwellings (Axelson et al. 1988; Svensson, Pershagen, and Klominek 1989). Two investigations in Stockholm have shown approximately doubled lung cancer risk for more exposed compared with less exposed subjects (Svensson, Eklund, and Pershagen 1987; Svensson, Pershagen, and Klominek 1989). A study in northern Sweden which assumed exposure from type of residence found no increased risk overall (Damber and Larsson 1987). In a study in southern Sweden, Axelson and colleagues (1988) used measurement data and information on residence type and geology to estimate exposure to radon; association was found in rural but not urban dwellers. This variation in the effect of exposure to radon with residence location could not be readily explained by the investigators.

In the United States, Simpson and Comstock (1983) examined the relationship between the incidence of lung cancer and housing characteristics. During a twelve-year period in Washington County, Maryland, the incidence of lung cancer in the county's residents was not significantly affected by the type of basement construction or building materials. Without specific validation, the dwelling characteristics were assumed to be surrogates for exposure to radon.

In New Jersey, Klotz and colleagues (Klotz, Petix, and Zagraniski 1989) evaluated mortality of 752 persons who had resided in forty-five homes contaminated by radon from radium processing waste. Overall, lung cancer mortality was not elevated. The standardized mortality ratio for white males was increased, but the excess was not statistically significant. In another recent study in New Jersey, radon exposures for the ten to thirty years before diagnosis were estimated for 433 cases and 402 controls drawn from a previously completed study of 994 cases and 995 controls (New Jersey State Department of Health 1989). Overall, the risk of lung cancer tended to increase at higher exposures, but the association of radon with lung cancer was not statistically significant in most analyses. Inexplicably, the risk from radon exposure was less among heavier cigarette smokers.

More recently, a case-control study was conducted in Port Hope, Ontario, where some homes had been constructed with contaminated building materials (Lees, Steele, and Roberts 1987). Exposures were estimated for the period of residence in Port Hope on the basis of earlier measurement data. The analyses indicated an increased risk for subjects with higher exposure, which persisted when cigarette smoking was controlled. However, the number of subjects was small, and the results were not statistically significant.

Many new case-control studies are now in progress throughout the world. Most incorporate measurement of radon concentrations in current and former residences. The sample sizes of most of the investigations are substantially greater than many of the completed studies (Table 15.3). Results of most of the newer studies will not be forthcoming for several years, however.

RISK ASSESSMENTS

Because only scant epidemiologic data on domestic exposure are available, the hazard posed by exposure to radon in indoor air has been addressed primarily with risk assessment procedures (Table 15.4). Information on the population distribution of exposure in dwellings is used in a risk-projection equation or "model" that describes the increment in the occurrence of lung cancer per unit of exposure. For the United States, however, the needed data on the concentrations of radon in homes have not yet been collected from large population samples. The selection of risk coefficients to describe the excess lung cancer risk associated with exposure to radon decay products is also problematic; the studies of miners include only males, much of the exposure of miners was at concentrations higher than generally occur in homes, and none of the miner populations has yet been followed throughout the full lifetime of the subjects. Furthermore, the various factors that affect the dosimetry of radon decay products may differ substantially in homes and in mines (Table 15.1). As discussed previously, analyses based on dosimetric models of the respiratory tract suggest, however, that exposures to radon decay products in homes and in mines have approximately equivalent potency in causing lung cancer.

To accomplish the risk estimation, a mathematical model is used to project the occurrence of cases of lung cancer caused by exposure. These risk projection models require assumptions concerning the temporal pattern of the occurrence of lung cancer after exposure and the effects of such potentially important cofactors as age at exposure, age at risk, and cigarette smoking. The two most widely

Table 15.3 Epidemiologic Studies of Domestic Exposure to Radon and Lung Cancer

Location (Reference)	Study Design	Subjects	Exposure Measure	Findings
Southern Sweden (Axelson, Edling, and Kling 1979)	Case-control	37 cases and 178 controls	Residence type: wood, "mixed," or stone	RR[a] = 1.8 ($p < .05$) for stone and mixed versus wood
Oeland, Sweden (Edling, Kling, and Axelson 1984)	Case-control	23 cases and 202 controls	Residence type and 4 months' measurements	RR = 4.3 (90% CI[b] 1.7–10.6) for low versus high by home type; RR = 2.7 (90% CI 1.4–18.5) low versus high by measurement
Southern Sweden (Edling, Wingren, and Axelson 1984)	Case-control	23 cases and 202 controls	Measurement with α-sensitive film	RR increased for higher versus lowest exposure categories; multiplicative interaction with smoking
Northern Sweden (Pershagen, Damber, and Falk 1984)	Case-control	15 nonsmoker and 15 smoker case-control pairs	Construction characteristics	Estimated mean exposure significantly higher for smoking cases than controls; exposure not different for nonsmokers
Sweden (Pershagen, Damber, and Falk 1984)	Case-control	11 nonsmoker and 12 smoker case-control pairs	Construction characteristics	Estimated mean exposures comparable for cases and controls regardless of smoking
Northern Sweden (Damber and Larsson 1986, 1987)	Case-control	589 male cases, 582 deceased controls, 453 living controls	Residence type: wood or nonwood	RR not increased, with or without smoking adjustment; RR increased for those never employed in occupations not associated with lung cancer
Stockholm, Sweden (Svensson, Eklund, and Pershagen 1987)	Case-control	292 female cases and 584 controls	Geology and living near ground level	RR = 2.2 (95% CI 1.2–4.0) for exposed versus nonexposed; exposure-response relationship not found
Southern Sweden (Axelson et al. 1988)	Case-control	177 cases and 677 controls	Residence type and geology, all homes; 2-month measurement, some homes	Exposure associated with increased risk for rural but not urban dwellers
Stockholm, Sweden (Svensson, Pershagen, and Klominek 1989)	Case-control	210 female cases, and 209 population and 191 hospital	2-week measurement and assumed values	RR = 1.8 (95% CI 1.2–2.9) comparing high and intermediate with low; RR highest for small-cell cancer
Maryland, U.S. (Simpson and Comstock 1983)	Cohort	298 cases over a 12-year period	Housing characteristics	No associations of incidence rates with housing characteristics
New Jersey, U.S. (Klotz, Petix, and Zagraniski 1989)	Cohort	752 persons who had resided in 45 homes contaminated by radium waste	Residence for at least 1 year in one of the homes	SMR[c] = 1.7 (95% CI 0.8–3.2) for lung cancer in white males; no excess for females
New Jersey, U.S. (N.J. State Dept. of Health 1989)	Case-control	433 female cases and 402 controls	Year-long α-track measurements, some estimates	RR = 1.9 (95% CI 1.0–3.4) comparing ≥2 pCi/liter with lower values
Ontario, Canada (Lees, Steele, and Roberts 1987)	Case-control	27 cases and 49 controls	Reconstructed exposures based on measurements	RR = 2.4 (95% CI 0.8–7.1) with smoking adjustment for exposed versus nonexposed

[a]RR, relative risk.
[b]CI, confidence interval.
[c]SMR, standardized mortality ratio.

Table 15.4 Recent Risk Projection Models for Radon and Lung Cancer

Agency	Type of Model	Source of Risk Estimate
National Council on Radiation Protection and Measurements (NCRP 1984b)	Attributable risk, time dependent	Average risk coefficient from principal studies of miners
International Commission on Radiological Protection (ICRP 1987)	Constant relative risk	Adjusted risk coefficient from three studies of miners
Environmental Protection Agency (U.S. EPA 1987)	Constant relative risk	Range of coefficients based on studies of miners
National Institute for Occupational Safety and Health (NIOSH 1987)	Relative risk, time dependent	Risk based on Colorado Plateau uranium miners
National Research Council, Biological Effects of Ionizing Radiation (BEIR) IV Committee (NRC 1988)	Relative risk, time dependent	Risk based on analysis of four studies of miners
Environmental Protection Agency (Puskin and Nelson 1989)	Relative risk, time dependent	Combines the ICRP and BEIR IV models

applied models are the relative risk and attributable risk models; the relative risk model assumes that the background risk is multiplied by the risk from radon decay products whereas the attributable risk model assumes that the excess risk is additive to the background risk. Two models, those of the BEIR IV committee (NRC 1988) and of the (NCRP 1984b), describe the risk as varying with the time since the exposure.

The manner in which exposure to radon decay products and cigarette smoking are assumed to interact strongly influences the results of risk estimation models for radon-associated lung cancer. If a multiplicative interaction is assumed, then the risk for smokers, already much greater than that for nonsmokers, is multiplied by the risk from exposure to radon decay products. If an additive interaction is assumed, then the same excess risk is added to the background rates for smokers and for nonsmokers. The interaction between the two agents might plausibly take some form other than purely additive or purely multiplicative.

Diverse risk projection models have been developed; Table 15.4 describes the most recent and widely used models (see NRC [1988] for a review of earlier models). Each of the recent models estimates lung cancer risk on the basis of the epidemiologic evidence from underground miners, but the biologic assumptions underlying the models and their resulting risk projections differ substantially. Table 15.5 provides additional description of the most prominent risk models: those of the NCRP (1984b), the ICRP (1987), and the BEIR IV committee of the NRC (1988).

The NCRP model generally projects the lowest excess risk because it is an additive model, and the radon-associated excess declines over time (Table 15.6). The ICRP model, a constant relative risk model, projects the highest risks. Exposures received by age twenty years lead to a particularly large excess because of

Table 15.5 Features of Selected Risk Projection Models for Radon and Lung Cancer

	NCRP	ICRP	BEIR IV
Form of model	Attributable risk	Relative risk	Relative risk
Time dependent	Yes; risk declines exponentially after exposure	No	Yes; risk declines as time since exposure lengthens
Lag interval	5 years	10 years	5 years
Age at exposure	No effect of age at exposure	Three-fold increased risk for exposures before age 20 years	No effect of age at exposure
Age at risk	Risk commences at age 40 years	Constant relative risk with age	Lower risks for ages 55 years and older
Dosimetry adjustment	Increased risk for indoor exposure	Decreased risk for indoor exposure	No adjustment
Risk coefficient	10×10^{-6}/year/WLM	Excess relative risks: 1.9%/WLM at ages 0–20 years and 0.64%/WLM for ages 21 years and above	Excess relative risk of 2.5%/WLM but modified by time since exposure

Source: Data from Samet and Hornung (1990).

Table 15.6 Increments in Lung Cancer Risks for One WLM[a], Projected by *NCRP*, *ICRP*, and *BEIR IV* Models

Increment at Age (years)	Exposure at Age 15 Years (%)			
	NCRP[b]			
	Male	Female	ICRP	BEIR IV
35	0	0	1.9	1.5
50	0.3	0.7	1.9	1.5
65	0.05	0.2	1.9	0.5
85	0.02	0.1	1.9	0.5
	Exposure at Age 35 Years (%)			
50	0.6	1.4	0.6	3.0
65	0.1	0.4	0.6	0.5
85	0.05	0.2	0.6	0.5

Source: Data from Samet and Hornung (1990).

[a]An annual exposure of 1 WLM would be received in a home with a concentration of 6 pCi/liter, assuming 70 percent occupancy.

[b]The excess is additive for the NCRP model. The percent excess relative risk was calculated for illustration using sex-specific lung cancer mortality rates for the United States, 1980–84. The additive increments are 3.0×10^{-6}, 1.8×10^{-6} and 0.9×10^{-6} for ages 50, 65, and 85 years, respectively, for exposure at age 15 years; and 6.0×10^{-6}, 3.5×10^{-6}, and 1.8×10^{-6}, respectively, for exposure at age 35 years.

Table 15.7 Lung Cancer Mortality Rates Per 100,000 Projected for Nonsmoking and Smoking Males at Age 65 Years by *NCRP*, *ICRP*, and *BEIR IV* Models[a]

	NCRP	ICRP	BEIR IV
Exposure to 10 WLM at age 15 years			
Nonsmoking	59.8	69.0	60.9
Smoking	698.3	828.8	731.3
Exposure to 10 WLM at age 35 years			
Nonsmoking	61.5	61.5	60.9
Smoking	700.0	738.3	731.3

Source: Data from Samet and Hornung (1990).
[a]Background lung cancer mortality rates estimated as 58.0×10^{-5} for nonsmokers and 696.5×10^{-5} for smokers (NRC 1988).

the threefold higher risk assumed up to age twenty years than at subsequent ages. In the BEIR IV model, the percent of excess risk varies with both age and time since exposure.

When smokers and nonsmokers are considered separately, the substantial difference between assuming an additive or a multiplicative interaction between smoking and radon exposure is evident (Table 15.7). The additive NCRP model projects small increments for smokers in comparison with the multiplicative ICRP and BEIR IV models. Lifetime excess lung cancer risks for smokers estimated by the three models are markedly different. Land (1988) has calculated the excess lung cancer risk per one hundred thousand smokers exposed to 1 WLM at age fifteen as follows: NCRP, 7.4; ICRP, 278.7; BEIR IV, 114.5; for exposure to 1 WLM at age thirty-five, the corresponding projections are 15.5, 94.3, and 129.4, respectively.

These models have been used to project the lung cancer burden associated with exposure to indoor radon. For exposure at 0.2 WLM/year (approximately equivalent to residence in a home at 1.5 pCi/liter), the approximate average annual exposure, the NCRP model projects lifetime lung cancer risk as 0.18 percent; the NCRP report estimates that nine thousand lung cancer deaths annually in the United States can be attributed to indoor radon. For an annual exposure of about 0.16 WLM, the ICRP model estimates lifetime risk of lung cancer as 0.42 percent for males and as 0.09 percent for females. The BEIR IV report describes risk for exposures received above background; for an exposure rate of 0.20 WLM/year, the model projects attributable lifetime risks of 0.7 percent for males and 0.3 percent for females. Using the BEIR IV model, Lubin and Boice (1989) estimated that approximately 13,300 lung cancer deaths annually can be attributed to indoor radon exposure. Using the current model of the EPA, Puskin and Nelson (1989) calculated that radon exposure in single-family homes may cause twenty thousand lung cancer deaths annually in the United States.

Thus, in spite of the differing underlying assumptions and risk projections, each of the models indicates that radon must be considered as an important cause of lung

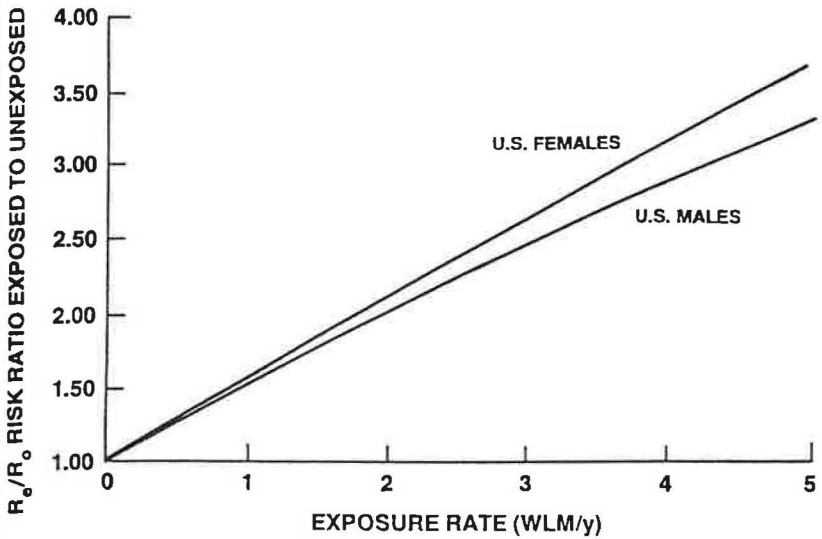

Figure 15.4. Risk ratio (R_e/R_o) of lung cancer mortality for lifetime exposure to radon decay products at constant rates of annual exposure, as estimated by the BEIR IV committee model. *Source: Health Risks of Radon and Other Internally Deposited Alpha-Emitters*, ©1988, by the National Academy of Sciences, National Academy Press, Washington, D.C. Reprinted with permission.

cancer for the general population. Each also demonstrates that unacceptable levels of risk are associated with higher levels of exposure. For example, in the BEIR IV model, exposure at 4 WLM/year above background leads to a tripling of the lifetime risk of lung cancer for males and females (Figure 15.4); this level of exposure would be received from residing in a home with a concentration of about 25 pCi/liter. As a basis for policy decisions, these risk projection models can be used to estimate the risks associated with levels of exposure that might be designated as guidelines or standards. The models can also be used to estimate the reduction in lung cancer occurrence which would follow reduction of exposure.

SUMMARY

Radon and its decay products are invariably present in indoor environments; most homes have concentrations of only a few pCi/liter, but concentrations in some homes are as high as those measured in uranium and other underground mines. Exposure to radon decay products has been shown to increase the mortality from lung cancer of underground miners working in mines with high concentrations. An increased risk of lung cancer must also be presumed to result from domestic exposure although the epidemiologic data are scant and preliminary at present. Risk assessments have been performed to evaluate the magnitude of the problem of

lung cancer associated with domestic exposure to radon. The most recent, by the BEIR IV committee, showed a doubling exposure rate of 2.0 WLM/year for males and 1.8 WLM/year for females.

REFERENCES

Alter, H. W., and Oswald, R. A. 1987. Nationwide distribution of indoor radon measurements: A preliminary data base. *J. Air Pollut. Control Assoc.* 37:227–31.

Archer, V. E. 1987. Association of lung cancer mortality with precambrian granite. *Arch. Environ. Health* 42:87–91.

Axelson, O.; Edling, C.; and Kling, H. 1979. Lung cancer and residency: A case-referent study on the possible impact of exposure to radon and its daughters in dwellings. *Scand. J. Work Environ. Health* 5:10–15.

Axelson, O., et al. 1988. Indoor radon exposure and active and passive smoking in relation to the occurrence of cancer. *Scand. J. Work Environ. Health* 14:286–92.

Bean, J. A., et al. 1982. Drinking water and cancer incidence in Iowa, II: Radioactivity in drinking water. *Am. J. Epidemiol.* 116:924–32.

Borak, T. D., and Johnson, J. A. 1988. Estimating the risk of lung cancer from inhalation of radon daughters indoors: Review and evaluation. Las Vegas, Nev.: EPA Publication no. EPA/600/6-88/008.

Cohen, B. L. 1986. A national survey of ^{222}Rn in U.S. homes and correlating factors. *Health Phys.* 51:175–83.

Cohen, B. L. 1988. An experimental test of the linear–no threshold theory of radiation carcinogenesis. Presented at the U.S. Environmental Protection Agency 1988 symposium on radon and radon reduction technology, October, Denver, Colo.

Cohen, B. L., and Nelson, D. 1987. Radon, a homeowner's guide to detection and control: A consumer report book. Mount Vernon, N.Y.: Consumers' Union.

Cothern, C. R., and Smith, J. E., Jr., eds. 1987. *Environmental radon*. New York: Plenum.

Cross, F. T. 1988. Evidence of lung cancer from animal studies. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 373–406. New York: John Wiley & Sons.

Damber, L., and Larsson, L.-G. 1986. Lung cancer in males: An epidemiological study in northern Sweden with special regard to smoking and occupation. Umeå University medical dissertation, Department of Oncology. Umeå, Sweden.

Damber, L. A., and Larsson, L.-G. 1987. Lung cancer in males and type of dwelling: An epidemiologic pilot study. *Acta Oncol.* 26:211–15.

Doll, R., and Peto, R. 1978. Cigarette smoking and bronchial carcinoma: Dose and time relationships among regular smokers and life-long non-smokers. *J. Epidemiol. Community Health* 32:303–13.

Dousset, M., and Jammet, H. 1985. Comparaison de la mortalite par cancer dans la Limousin et le Poitou-Charentes: (Etude preliminaire 1968–1975). *Radioprotection GEDIM* 20:61–67.

Edling, C.; Kling, H.; and Axelson, O. 1984. Radon in homes: A possible cause of lung cancer. *Scand. J. Work Environ. Health* 10:25–34.

Edling, C.; Wingren, G.; and Axelson, O. 1984. Radon daughter exposure in dwellings and lung cancer. In *Indoor air, Vol. 2: Radon, passive smoking, particulates and housing epidemiology*. Ed. B. Berglund, T. Lindvall, and J. Sundell, 29–34. Stockholm: Swedish Council for Building Research.

Edling, C. et al. 1982. Effects of low-dose radiation: A correlation study. *Scand. J. Work Environ. Health* 8(Suppl. 1):59–64.

Fleischer, R. L. 1981. A possible association between lung cancer and phosphate mining and processing. *Health Phys.* 41:171–75.

Fleischer, R. L. 1986. A possible association between lung cancer and a geological outcrop. *Health Phys.* 50:823–27.

Forastiere, F., et al. 1985. Lung cancer and natural radiation in an Italian province. *Sci. Total Environ.* 45:519–26.

Fraser, R. G., et al. 1989. *Diagnosis of diseases of the chest*, Vol. 2, 1367–68. Philadelphia: W. B. Saunders.

Harting, F. H., and Hesse, W. 1879. Der lungenkrebs, die bergkrankheit in den Schneeberger gruben. *Vjschr. Gerichtl. Med. Offentl. Gesundheitswesen* 31:102–32, 313–37.

Hess, C. T.; Weiffenbach, C. V.; and Norton, S. A. 1983. Environmental radon and cancer correlations in Maine. *Health Phys.* 45:339–48.

Hofmann, W.; Katz, R.; and Zhang, C. X. 1985. Lung cancer in a Chinese high background area: Epidemiological results and theoretical interpretation. *Sci. Total Environ.* 45:527–34.

Holaday, D. A., et al. 1957. *Control of radon and daughters in uranium mines and calculations on biologic effects*. Washington, D.C.: Government Printing Office. Public Health Service Publication no. 494.

Hornung, R. W. and Meinhardt, T. J. 1987. Quantitative risk assessment of lung cancer in U.S. uranium miners. *Health Phys.* 52:417–30.

International Commission on Radiological Protection. 1987. *Lung cancer risk from indoor exposures to radon daughters*. Oxford, England: Pergamon Press. ICRP Publication no. 50.

James, A. C. 1984. Dosimetric approaches to risk assessment for indoor exposure to radon daughters. *Radiat. Prot. Dosim.* 7:353–56.

James, A. C. 1988. Lung dosimetry. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 259–309. New York: John Wiley & Sons.

Klotz, J. B.; Petix, J. R.; and Zagraniski, R. T. 1989. Mortality of a residential cohort exposed to radon from industrially contaminated soil. *Am. J. Epidemiol.* 129:1179–86.

Lafavore, M. 1987. Radon: The invisible threat. Emmaus, Pa.: Rodale Press.

Land, C. E. 1988. The ICRP 50 model. In *Radon: Proceedings of the twenty-fourth annual meeting of the National Council on Radiation Protection and Measurements*. Ed. N. H. Harley, 115–26. Washington, D.C., 30–31 March.

Lees, R. E. M.; Steele, R.; and Roberts, J. H. 1987. A case-control study of lung cancer relative to domestic radon exposure. *Int. J. Epidemiol.* 16:7–12.

Letourneau, E. G., et al. 1983. *Lung cancer mortality and indoor radon concentrations in 18 Canadian cities: Proceedings of the sixteenth midyear topical meeting of the Health Physics Society*. Albuquerque, N.M. National Technical Information Service CONF-830101, distribution category UC-41.

Logue, J., and Fox, J. 1985. Health hazards associated with elevated levels of indoor radon: 1985. Pennsylvania. *MMWR* 34(43):657–58.

Lubin, J. H. 1988. Methods for the analysis of radon-exposed populations. *Yale J. Biol. Med.* 61:195–214.

Lubin, J. H., and Boice, J. D. 1989. Estimating Rn-induced lung cancer in the U.S. *Health Phys.*, 57:417–27.

McDowell, E. M., and Trump, B. F. 1983. Histogenesis of preneoplastic and neoplastic lesions in tracheobronchial epithelium. In *Survey and synthesis of pathology research*, 235–79. Basel: S. Karger.

Murray, J. F. 1986. *The normal lung*. Philadelphia: W. B. Saunders.

National Council on Radiation Protection and Measurements. 1984a. *Exposure from the uranium series with emphasis on radon and its daughters*. Bethesda, Md.: NCRP Report no. 77.

National Council on Radiation Protection and Measurements. 1984b. *Evaluation of occupational and environmental exposure to radon and radon daughters in the United States*. Bethesda, Md.: NCRP Report no. 78.

National Institute for Occupational Safety and Health. 1987. *Radon progeny in underground mines: A recommended standard for occupational exposure*. Washington, D.C.: Government Printing Office. DHHS Publication no. NIOSH/88-101.

National Research Council (committee on the biological effects of ionizing radiation) 1988. *Health risks of radon and other internally deposited alpha-emitters: BEIR IV*. Washington, D.C.: National Academy Press.

Nazaroff, W. W., and Nero, A. V., Jr., eds. 1988. *Radon and its decay products in indoor air*. New York: John Wiley & Sons.

Nazaroff, W. W.; Moed, B. A.; and Sextro, R. G. 1988. Soil as a source of indoor radon: Generation, migration, and entry. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 57–112. New York: John Wiley & Sons.

Nazaroff, W. W., et al. 1988. Radon entry via potable water. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 131–57. New York: John Wiley & Sons.

Nero, A. V., Jr. 1988. Radon and its decay products in indoor air: An overview. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 1–53. New York: John Wiley & Sons.

Nero, A. V., and Nazaroff, W. W. 1984. Characterizing the source of radon indoors. *Radiat. Prot. Dosim.* 7:23–39.

Nero, A. V., et al. 1986. Distribution of airborne radon-222 concentrations in U.S. homes. *Science* 234:992–97.

Nero, A. V.; Revzan, K. L.; and Sextro, R. G. 1987. Appraisal of the *U.S. data on indoor radon concentrations*. Presented at the fourth international symposium of the natural radiation environment, 30–31 December, Lisbon, Berkeley, Calif.: Lawrence Berkeley Laboratories. Report LBL 243-45.

New Jersey State Department of Health. 1989. *A case-control study of radon and lung cancer among New Jersey women:* Technical report, phase 1.

Nuclear Energy Agency (Organization for Economic Cooperation and Development). 1983. *Dosimetry aspects of exposure to radon and thoron daughter products*. Washington, D.C.: OECD.

Pershagen, G.; Damber, L.; and Falk, R. 1984. Exposure to radon in dwellings and lung cancer: A pilot study. In *Indoor air, Vol. 2: Radon, passive smoking, particulates and housing epidemiology*. Ed. B. Berglund, T. Lindvall, and J. Sundell, 73–78. Stockholm: Swedish Council for Building Research.

Phillips, C. R.; Khan, A.; and Leung, H. M. Y. 1988. The nature and determination of the unattached fraction of radon and thoron progeny. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 203–56. New York: John Wiley & Sons.

Puskin, J. S., and Nelson, C. B. 1989. EPA's perspective on risks from residential radon exposure. *J. Air Pollut. Control Assoc.* 39:915–20.

Samet, J. M. 1989. Radon and lung cancer. *J. Natl. Cancer Inst.* 81:745–57.

Samet, J. M., and Hornung, R. W. 1990. Review of radon and lung cancer risk. *Risk Analysis* 1:65–75.

Samet, J. M., and Nero, A. V., Jr. 1989. Indoor radon and lung cancer. *N. Engl. J. Med.* 320:591–94.

Samet, J. M., et al. 1989. Radon progeny exposure and lung cancer risk in New Mexico uranium miners: A case-control study. *Health Phys.*, 56:415–21.

Simpson, S. G., and Comstock, G. W. 1983. Lung cancer and housing characteristics. *Arch. Environ. Health* 38:248–51.

Stranden, E. 1988. Building materials as a source of indoor radon. In *Radon and its decay products in indoor air*. Ed. W. W. Nazaroff and A. V. Nero, Jr., 113–30. New York: John Wiley & Sons.

Svensson, C.; Eklund, G.; and Pershagen, G. 1987. Indoor exposure to radon from the ground and bronchial cancer in women. *Int. Arch. Occup. Environ. Health* 59:123–31.

Svensson, C.; Pershagen, G.; and Klominek, J. 1989. Lung cancer in women and type of dwelling in relation to radon exposure. *Cancer Res.* 49:1861–65.

U.S. Department of Health and Human Services. 1982. *The health consequences of smoking: Cancer. A report of the surgeon general*. Washington, D.C.: Government Printing Office. DHHS Publication no. PHS 82-50179.

U.S. Department of Health and Human Services. 1984. *The health consequences of smoking: Chronic obstructive lung disease. A report of the surgeon general*. Washington, D.C.: Public Health Service, Office of Smoking and Health, Government Printing Office. DHHS Publication no. PHS 84-50525.

U.S. Environmental Protection Agency. 1986. *A citizen's guide to radon: What it is and what to do about it*. Washington, D.C.: Government Printing Office. DHHS Publication no. OPA/86/004.

U.S. Environmental Protection Agency. 1987. *Radon reference manual*. Washington, D.C.: Office of Radiation Programs, U.S. Environmental Protection Agency. Publication no. EPA/520/1-87-20.

Whittemore, A. S., and McMillan, A. 1983. Lung cancer mortality among U.S. uranium miners: A reappraisal. *J. Natl. Cancer Inst.* 71:489–99.

World Health Organization (International Agency for Research on Cancer). 1986. *IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans: Tobacco smoking*, Vol. 38. Lyon, France: IARC.