

Radon Update: Facts Concerning Environmental Radon: Levels, Mitigation Strategies, Dosimetry, Effects and Guidelines

Prepared by A. Bertrand Brill, David V. Becker, Kevin Donahoe, Stanley J. Goldsmith, Bennett Greenspan, Ken Kase, Henry Royal, Edward B. Silberstein and Edward W. Webster on behalf of the SNM Committee on Radiobiological Effects of Ionizing Radiation

- The risk from environmental radon levels is not higher now than in the past, when residential exposures were not considered to be a significant health hazard.
- The majority of the radon dose is not from radon itself, but from short-lived alpha-emitting radon daughters, most notably ^{218}Po ($T_{1/2}$ 3 min) and ^{214}Po ($T_{1/2}$ 0.164 msec) along with beta particles from ^{214}Bi ($T_{1/2}$ 19.7 min).
- Radon gas can penetrate homes from many sources and in various fashions. Measuring radon in homes is simple and relatively inexpensive and may be accomplished in a variety of ways. Although it is not possible to radon-proof a house, it is possible to reduce the level. In high radon areas, if the average level is higher than 4–8 pCi/liter (NCRP recommended level is 8 pCi/liter; EPA recommended level is 4 pCi/liter), appropriate action is advised.
- The shape of the dose response curves for miners exposed to alpha-emitting particles in the workplace is consistent with current biologic knowledge. It is linear in the low dose range and saturates in the high dose range. No detectable increase in lung cancer frequency is seen in the lowest exposed miners (those with exposures <120 WLM, the relevant dose interval for most homes).
- Evidence for a health effect from radon exposure is based on data from animal studies and epidemiologic studies of mines. Extensive radiobiologic data predict a linear dose-response curve in the low dose region due to poor biological repair mechanisms for the high density of ionizing events that alpha particles create. However, no compelling evidence for increased cancer risks has yet been demonstrated from "acceptable" levels (<4–8 pCi/liter).

Key Words: radon; environmental radiation

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The potential hazard of radiation exposure to radon gas and its daughter products from natural background has been highlighted in the press and has become a matter of

concern and a source of confusion to the public. Home-owners are besieged with devices to measure radon levels and may not know what to do about the results they obtain. The Environmental Protection Agency (EPA), the National Council on Radiation Protection and Measurement (NCRP), the International Commission on Radiation Protection (ICRP) and other groups concerned with radiation protection matters have all issued guidelines (1–7). Although they differ in detail, these guidelines and recommended actions are in general agreement. A matter of concern is that the media have chosen the lowest level of the guidelines, which the public translates into the upper limit of "safe dose." It is not surprising that there is widespread confusion regarding the nature and severity of the problem, the risk magnitude, the steps that should be taken to cope with different circumstances and the costs associated with different actions. This report provides information needed to understand these issues and to provide a compilation of the relevant facts for those individuals interested in the potential health effects of environmental radon.

Many articles have been published in the scientific literature and by the mainstream press dealing with the issue of human risk from radon exposure. Many of these appear in publications by the National Academy of Sciences (NAS), NCRP, EPA, Department of Energy (DOE) (8–15).

RADON CHARACTERISTICS

Radon, ^{222}Rn ($T_{1/2}$ = 3.82 days), is a daughter product of radium, ^{226}Ra , which in turn is derived from the longer-lived antecedent, ^{238}U . Thoron, ^{220}Rn ($T_{1/2}$ = 56 sec) is a daughter of thorium, ^{232}Th , which is present in larger amounts in the earth's crust than radon. Because of thoron's short half-life, it is essentially all gone before it leaves the ground and is of no significant radiobiologic consequence. These radionuclide series are present in slowly decreasing amounts in the environment (geologic time scale) due to radioactive decay of their parents which has been known and understood since the end of the last century.

Widely varying radon levels exist in different regions related to geological circumstances. New concern regard-

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For correspondence or reprints contact: A.B. Brill, University of Massachusetts Medical Center, 55 Lake Ave. North, Worcester, MA 01655.

ing radon exposures is traceable to the discovery that there are more houses with high radon levels than previously realized and to the use of a new method of expressing and summing doses from partial body exposures, such as the lung dose from radon daughters (7-16). This method of dose expression was promulgated by the ICRP and the NCRP based on defined weighting factors which make it possible to sum partial body doses and thereby estimate a total body dose that would have a quantifiable risk. This quantity is defined as the effective dose (16). Thus, the previously estimated partial body environmental radon dose to the tracheobronchial epithelium (TBE) (2500 mrem/yr) was not included in whole-body dose calculations because that exposure was limited to a small fraction of the body.

The new method of calculation multiplies the 2500-mrem/yr dose to the TBE by a weighting factor which allows the dose to the TBE to be included in the effective dose from environmental radiation exposure. Different weighting factors have been proposed, ranging from 0.06 to 0.12 with 0.12 currently used by the EPA, NCRP and ICRP. This tissue weighting is performed in order to estimate the overall risk from exposure to only a small part of the body and this raises the radon contribution to the whole body from 0 mrem to 300 mrem. NCRP quotes an uncertainty of $\pm 50\%$ in these numbers. Based on these estimates, radon in equilibrium with its daughters delivers two times more dose than previously accepted as the total dose received from all sources of natural background exposure (~ 100 mrem/yr on the average in the United States) (Table 1). Thus, it is not surprising that adoption of the effective dose notion by many radiation protection groups, including the NCRP and the EPA in the United States, has led to increased concern regarding the potential health effects of radon. It should be noted that lung cancer risk coefficients from radon are not increased as there are no new cases of lung cancer that led to the increased dose estimate. In fact, the new estimates of radiation dose imply a lower risk coefficient. That is, when the same number of lung cancer cases that occur are attributed to the higher doses (effective doses), the risk per unit exposure is decreased. The effective dose concept is discussed at greater length in NCRP Reports nos. 93 (17) and 100 (18) and ICRP no. 60 (7).

UNITS OF MEASUREMENT FOR RADON LEVELS

Almost all measurements of radon levels in the home or outdoors are expressed as the concentration of radon in units of picocuries per liter of air (pCi/liter), or in SI units as Becquerels per cubic meter (Bq/m^3), and radon daughters are expressed in working levels (WL). A working level month (WLM) is defined as 170 hr (21.25 working days/month \times 8 hr/day) in a workplace at one WL. Thus, a 12-hr/day exposure in the home at one WL, corresponds to ~ 26 working level months per year i.e., $2.1 \times$ the occupational exposure, assuming equal radon levels at home and in the

workplace, other things being equal. Exposure rate is typically given in working level months per year (WLM/yr).

The WL unit was developed for use in radon occupational exposure assessment since often there was incomplete information on the degree of equilibrium with daughter products. Dosimetrically, it corresponds to the dose delivered in 1 liter of air that results in the emission of 1.3×10^5 MeV of potential alpha energy (19). The amount of time spent in the mine or in the home determines the number of WLM associated with a particular exposure level, but because most people spend more time at home than at work, the WLM could be higher than from a comparable mine radon daughter concentration. Typical outdoor levels in the U.S. are given by NCRP no. 78 as 0.2 pCi/liter (11).

The correspondence between WLs and radon concentration in air in pCi/liters depends on the extent to which radon daughters (which impart dose to the tracheobronchial epithelium dose) are in equilibrium with the parent radon. At complete equilibrium, 1 pCi/liter results in an exposure equal to 0.01 working levels. The assumption is generally made that inside buildings the radon decay product/radon equilibrium is 50%. Thus, inside buildings, 1 pCi/liter = 0.005 WL, or 1 WL = 200 pCi/liter. (Note: Consideration must also be given to radionuclide attachment and distribution) (Table 2).

DOSIMETRY

Radon-222 is a decay product from the ^{238}U decay chain, illustrated in Figure 1. The external dose from ^{222}Rn and its airborne progeny is a very small fraction of the natural external radiation dose received by individuals. However, inhalation of radon and its daughters, may be followed by deposition of potentially large amounts of energy, i.e., absorbed dose in the tracheobronchial epithelium from the short-lived alpha and beta particle-emitting decay products (primarily ^{218}Po , ^{214}Pb , ^{214}Bi and ^{214}Po).

The radiation dose from these densely ionizing alpha radiations to the bronchial mucosa depends on radionuclide deposition and residence time. Particle deposition depends on three mechanisms: impaction, sedimentation and diffusion. Deposition and residence time depend on whether the radioactivity is attached to airborne dust particles or is unattached (following inhalation, unattached daughters are able to deposit deeper in the lung than dust particle-attached radon daughters). Respiratory factors (breathing rate and depth, mucociliary clearance, and site of impaction in the bronchial tree) influence depth of penetration into the lung with deeper particles having a longer residence time. Dose to the TBE from radon per se is negligible, since its intrapulmonary residence time is short with respect to its half-life. The high absorbed dose is from the decay of radon daughters attached to the TBE.

Although the location of the critical target for lung cancer induction is not known, it is assumed to be the basal cell at the fourth generation of the tracheobronchial tree

TABLE 1
Average Annual Radiation Exposure Rate (mrem/yr) in the US

Source	Dose equivalent* (mrem/yr)	Effective dose	
		(mrem/yr)	% total
Natural			
Cosmic	27	27	8
Terrestrial	28	28	8
Radon†	2400	200	55
Internal	39	39	11
Total	—	294	82
Artificial			
Medical: x-ray Dx	39	39	11
—nuclear medicine	14	14	4
Consumer products	10	10	4
Other	<0.01	<0.01	<0.3
Total	—	63	19
Total (Natural + Artificial)	—	360	100

*To soft tissues. Modified from reference 9.

†Dose equivalent to bronchi from radon daughter products. Assumed weighting factor for effective dose = 0.08.

and beyond and dose delivered to the mucous-covered cell is calculated to the basal cell nucleus at this location. The depth of mucous covering the critical target strongly influences the dose received from the short-range energetic alpha emissions as does the integrity and activity of the muco-ciliary escalator that carries particles in a retrograde fashion out of the lung. Alpha particles contribute more than 85% of the TBE dose which will be deposited within 30 μm of the decay site.

Dose calculations depend on the airborne radiation levels and concentration of radon and its progeny and on the modeling assumptions noted above (20). The radiation levels can now be measured with reasonable accuracy and

precision. Present calculations for an average indoor and outdoor exposure (0.75 pCi/liter) to a cell 22 μm deep, in a fourth generation airway, range from 140–340 mrad/yr, with the highest doses to 10-yr-old children. (A continuous exposure to radon at a concentration of 1 pCi/liter would result in an annual exposure to radon progeny of 0.25 WLM/yr, which corresponds to 188 mrad/yr or 3750 mrem/yr for an adult, assuming a quality factor of 20 for alpha particles (1)).

RADON MEASUREMENTS

There are three classes of measurement techniques that are used: (1) grab sampling, (2) continuous active sampling,

TABLE 2
Conversion Factors

SI unit	Traditional unit conversion
Activity (Bq)	1 Ci = 3.7×10^{10} Bq (1 pCi = 0.037 Bq)
Concentration (Bq/m ³)	1 pCi/liter = 37 Bq/m ³
Potential alpha energy (conc.(PAEC))	1 WL* = 1.3×10^5 MeV/liter = 2.08×10^{-5} J/m ³
Exposure (Jm ⁻³ s)	1 WLM = 12.97 Jm ⁻³ s
Exposure (Bqm ⁻³ yr)	1 WLM = 74.0 Bqm ⁻³ yr (for ²²² Rn series)
Exposure rate	1 WLM/yr = 4.11×10^{-7} Jm ⁻³
Exposure rate	1 WLM/yr = 74.0 Bqm ⁻³ (for ²²² Rn series)

1 WL = 200 pCi/liter (50% equil.) (from reference 14)

*1 WL (occup. exposure) \times 12 M/yr (i.e., 8 hr/day, 5 days/wk) = 12 WLM/yr and 1 WL in the home conveys a higher dose due to occupancy time: 1 WL (home) \times 51.6/2 M/yr (i.e., 12 hr/day, 7 days/wk occupancy) = 25.8 WLM/yr, (0.2 pCi/liter = Typical outside level = 120 mrad/yr (TBE)).

Assuming 8 hr outdoors (33%) at 0.2 pCi/liter and 16 hr indoors (67%) at 1 pCi/liter, NCRP estimates: average radon level = 0.75 pCi/liter = 0.004 WL which corresponds to $51.6 \times 0.004 = 0.2$ WLM/yr. TBE dose from environmental radon depends mostly on indoor levels.

If home levels are at the NCRP action guideline (8 pCi/liter), then total radon exposure = 0.029 WL (i.e., 7.25 times above the average, i.e., 0.004 WL).

120 mrad/yr (TBE) \times 20 (RBE) = 2400 mrem/yr (TBE).

2400 mrem/yr \times 0.12 (WF) = 300 mrem/yr (ED).

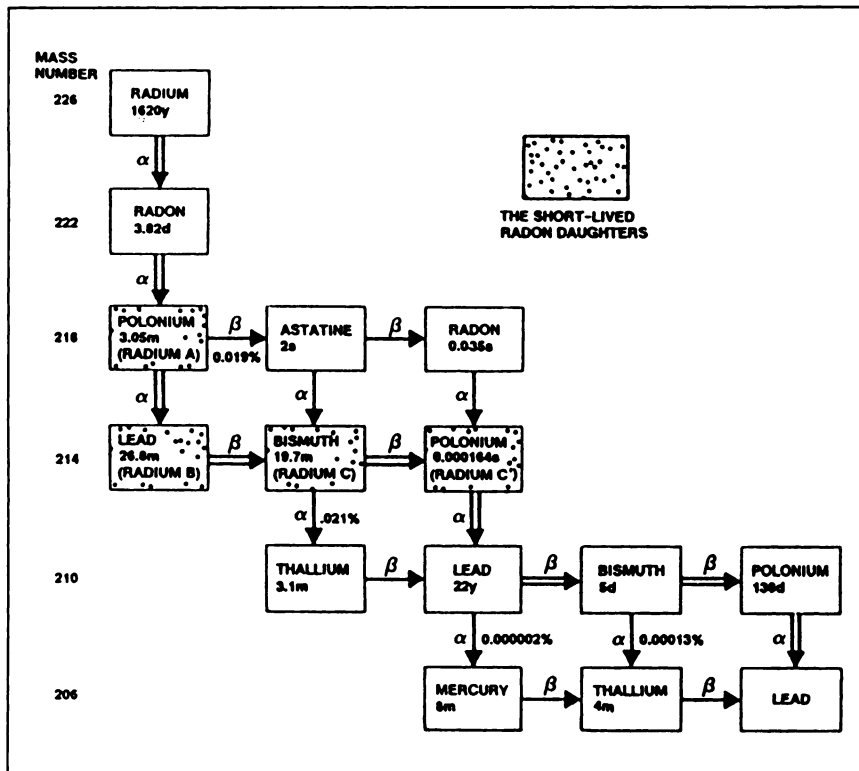


FIGURE 1. The radon decay chain (10).

and (3) integrative sampling (21). Grab sampling provides instantaneous measures of radon or radon progeny in air. Since values fluctuate widely depending on various factors, grab sampling techniques are used in industrial monitoring. Continuous active sampling involves multiple measurements at closely spaced time intervals over a long period. These are costly and only recommended when other measures indicate a problem and the source of radon entry needs to be precisely pinpointed. Integrative sampling devices are passive, and collect data on radon levels over a fixed period of time.

Typical integrative devices are charcoal canisters, or alpha track film dosimeters. The charcoal devices (Fig. 2) come in a canister, which is opened and placed in selected locations. Radon in air diffuses into the canister and is adsorbed onto charcoal. Following exposure for 2–7 days, depending on the particular device and the instructions for its use, it is sent back to the supplier who assays it by counting the gamma rays from the daughter nuclide (e.g., ^{214}Pb). If the canisters are exposed for several weeks or longer, the results will be primarily indicative of the activity sampled toward the end of the exposure interval since ^{222}Rn has a half-life of 3.8 days. Some of the canisters have an additional filter that affects the integration period, and make the canister insensitive to thoron (^{220}Rn).

A second type of integrative sampling detector is the alpha particle track etch detector (Fig. 3). This device can be used to average data over longer periods of time, as the track etch evidence of exposure does not decay. However, dust and electrostatics make them less reliable and they are only sensitive to radon gas activity.

Ordinarily, charcoal canisters are used to measure activity in the area where occupants spend the most time. Indoor radon levels are normally highest in winter. If levels are not elevated at that time, additional measurements should not be necessary. If high activity levels are found, then additional measurements should be made throughout the year in other parts of the house, especially the basement which usually has the most activity. Resources recommended by state or local health or environmental protection agencies are available if more intensive programs are needed to pinpoint and remedy high levels. These agencies also advise on testing methods and can provide lists of radon testing laboratories that performed successfully in the EPA proficiency testing program.

A new method of estimating the long-term integrated radon exposures was recently developed in Sweden and measures the amount of ^{210}Po in vitreous glass found in the home (23–25). Short-lived radon daughters plate out on the glass and undergo alpha decay leading to the formation of ^{214}Pb which decays to ^{210}Pb (22 yr $T_{1/2}$). The activity of ^{210}Pb or its daughter product ^{210}Po can be used to estimate cumulative exposures to residents from radon daughter concentration in the home. The activity of the glass is measured in the home using large surface area ionization chambers or surface barrier detectors which assay the amount of the 5.3 MeV alpha energy emitted (Fig. 4). The phenomenon is based on the fact that when the alpha particle is emitted, the daughter nucleus (^{210}Pb) recoils in the opposite direction and gets embedded in the glass close to the surface. One would presume that 50% of the recoils would result in deposited activity in the glass, but the ratio

is closer to 30%. Factors such as heat circulation patterns in the room and the frequency with which surface grime is washed from the window does not appear to seriously affect the estimated dose (26).

Another new technique promises to be useful for estimating cumulative in vivo absorbed dose from radon. The technique measures the ^{210}Pb content in the skull. Lead-210 emits a 47-keV gamma ray (4% abundance), which can easily penetrate the soft tissue that intervenes between the skull and the five large-area, thin-crystal NaI scintillator detectors placed about the subject's head. Assuming 14% of the bone mass is contained in the skull (27) and the effective half-life of ^{210}Pb in the body (12–18 yr), the cumulative dose from radon in measured subjects can be estimated (28). These calculations require knowledge of the mechanisms and the rate of transfer of radon daughters from the lung to the skeleton. These factors have yet to be established.

The alpha recoil method makes it possible to estimate the dose from radon daughters accumulated over the lifetime of window or picture frame glass in a particular residence, as well as in a miner's lamp. Residential measurements should make it possible to rank houses on an exposure index and miner's lamp readings could be important in ranking mines. The measurement of ^{210}Pb levels in vivo is also likely to be useful for ranking individuals into dose groups. However, ^{210}Pb levels are complicated by other factors including radium in the diet and one cannot differentiate recent from old exposures which will make it difficult to estimate person-years (no. of persons exposed \times no. of yr exposed) at risk for individual subjects. Nonetheless, all these new methods should be useful in epidemiology studies, but it is likely that it will still be difficult to estimate TBE dose accurately.

GEOGRAPHICAL DISTRIBUTION OF HIGH RADON LEVELS

The use of average values of dose from natural background radon suggests that dose is rather uniform, whereas in fact radon levels vary markedly in different regions of

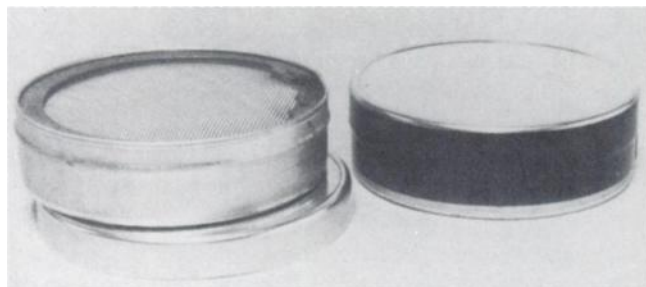


FIGURE 2. Charcoal canisters can be manufactured simply using a small can covered by a screen. The charcoal is contained in the space below the screen, which is held in place by a ring. A top is fitted over this arrangement until exposure, at which time it is removed. The top is replaced and sealed at the end of exposure, and the entire can is placed on a NaI gamma counting system for analysis (22).

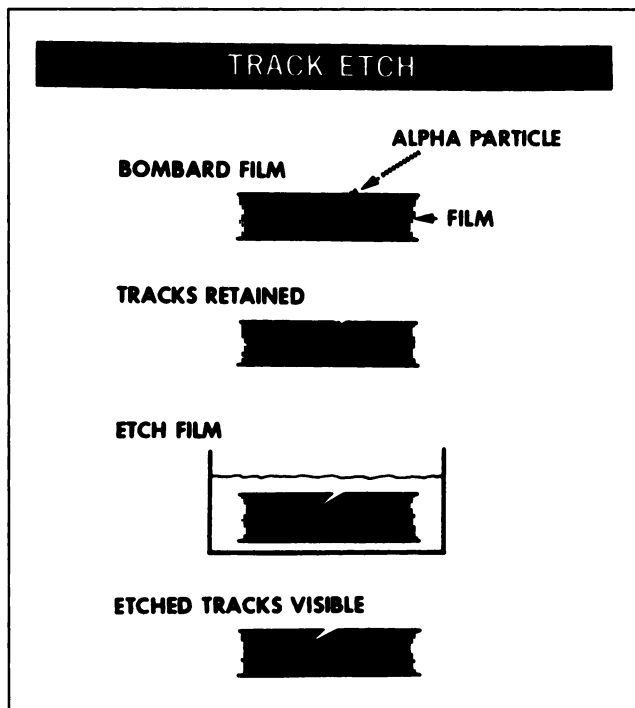


FIGURE 3. Alpha-track devices consist of material, such as film, which sustains damage along the track of an alpha particle. The material is then placed into etching fluid, which enlarges the track by extending the region of damage. Once the tracks have been sufficiently enlarged to become visible, their density at the surface of the material is determined and related to dose (22). (Photo courtesy of Terradex).

the country based on geologic factors, relation to mines and mine tailings, as well as levels of radium and radon in water supplies (29). In general, high levels of radon are associated with granite igneous rocks, shale and dirty quartz sedimentary rocks, phosphate deposits and some beach sands, which may contain high levels of radon progenitors, i.e., uranium or thorium. Basalt has relatively little uranium, i.e., half of the average value found in rocks of all kinds, whereas the granite strata contain upwards of twofold increases above average values (0.7 pCi/g). Figure 5 shows a map of locations with potentially high radon levels based on geologic formations in the United States.

Rock types that are high radon sources in the U.S. include (29):

1. Uraniferous metamorphic rocks and granites: Sheared faults in these formations cause some of the highest indoor levels in the U.S., particularly in the Rocky and Appalachian Mountain ranges and the Sierra Nevadas.
2. Marine black shales: Sources of high radon throughout the U.S., especially the central region from Ohio to Colorado.
3. Glacial deposits derived from uranium-bearing rock and sediment. Major components of glacial deposits in the northern midwest. They have high radon em-

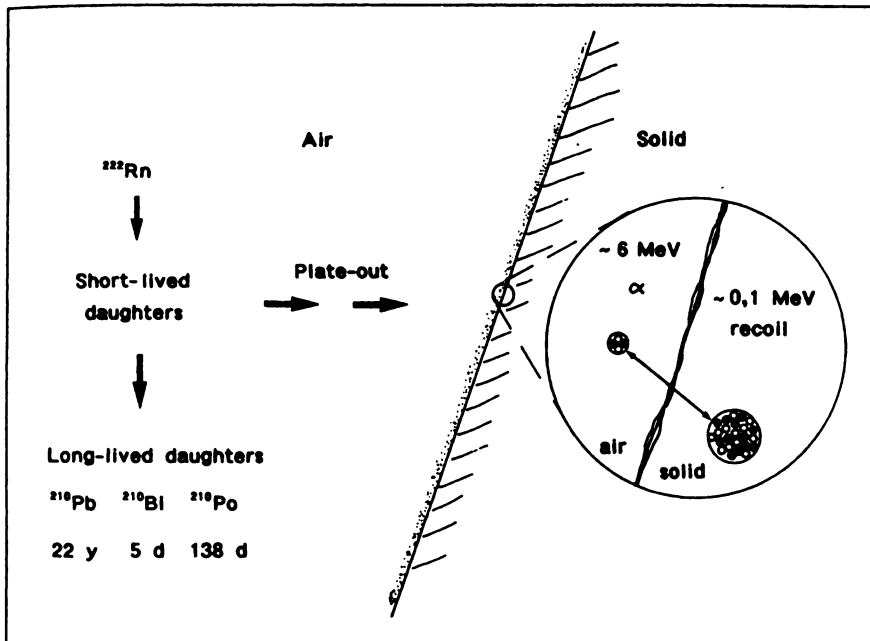


FIGURE 4. Surface plate out followed by alpha recoils embeds long-lived daughter activity in a surface. Range of recoiling nuclei in glass is about 50 nm (24).

anation due to large surface area, and high permeability due to cracking when dry.

4. Soils derived from carbonate, especially karstic terrain which is high in uranium and radium.
5. Uraniferous fluvial, deltaic, marine and lacustrine deposits, which provide most of the U.S. uranium, and are located in the western U.S.

Typically, the maximum ^{226}Ra concentration in phosphate ores is about 40 pCi/g (about 50 times greater than average concentration in soil). Thus, ore that is close to the surface, or residues from mining that are left on the surface, can give rise to very high local concentrations. In the U.S., this problem is mostly localized to Polk County, Florida and although not a great contributor to global levels, there is concern within those communities and local abatement efforts are underway. In some mining communities in Colorado, local releases from uranium mining residues and mine tailings can be significant sources of atmospheric radon. Typical emanation rates may exceed 300 pCi/m²-s (30). In 1983, the EPA established regulations that average releases from tailing sites may not exceed 20 pCi/m²-s (which is 40 times greater than the average from soil). Releases from coal residues and the burning of natural gas and coal complete the list of major contributors to atmospheric radon.

It should be noted that indoor levels of radon are not related simply to geologic factors but depend on many factors, including degree of fracturing of the bedrock and on the intervening pathway. Radon mobility through soil may vary by up to 10⁶-fold depending on soil porosity (30). Rock permeability is now recognized as a key factor influencing radon availability at the surface, even in low-uranium-containing rock types, such as limestone (31).

Another potentially important source of radon expo-

sure is from radon outgassing from high levels in water (Fig. 6). Radon concentrations in surface waters are usually very low. Since municipal water supplies are typically aerated, this results in diminished radon levels. Rural household wells are a potentially bigger problem. Deep aquifers have highly variable radon levels. Levels depend on uranium content of the rock and distribution of the aquifer relative to the rock, and on groundwater flow patterns. Thus, areas with granite-based aquifers may have highly variable levels, as noted in Table 3.

SOURCES OF ATMOSPHERIC RADON

The major source of ^{222}Rn in the atmosphere (at least 80%) is from emanations from soil from rock formations close to the ground surface (11), from the decay of ^{238}U through ^{226}Ra to ^{222}Rn (Table 4).

Radon dissolved in ground water is the second most important potential source of atmospheric radon. Nonetheless, in most locations it is a minor source of human exposure in view of the small absorbed dose following oral ingestion. In some locations where water from highly radioactive deep wells is used, it can be a significant contributor. Thus, in Maine, New Hampshire, some regions of the Appalachian mountains and Florida, concentrations found in some private wells exceed 10,000 pCi/liter. When water use is high in the home, air levels are found to be elevated due to outgassing from the water (32). Typically, a radon concentration of 10,000 pCi/liter in water would result in air concentration of 1 pCi/liter.

Turbulent or heated water (flowing in wash basins, showers, washing machines, flush toilets, etc.) is a source of elevated radon levels in the home, as these activities liberate dissolved radon into the home atmosphere. The amount released depends on the radon content of the water

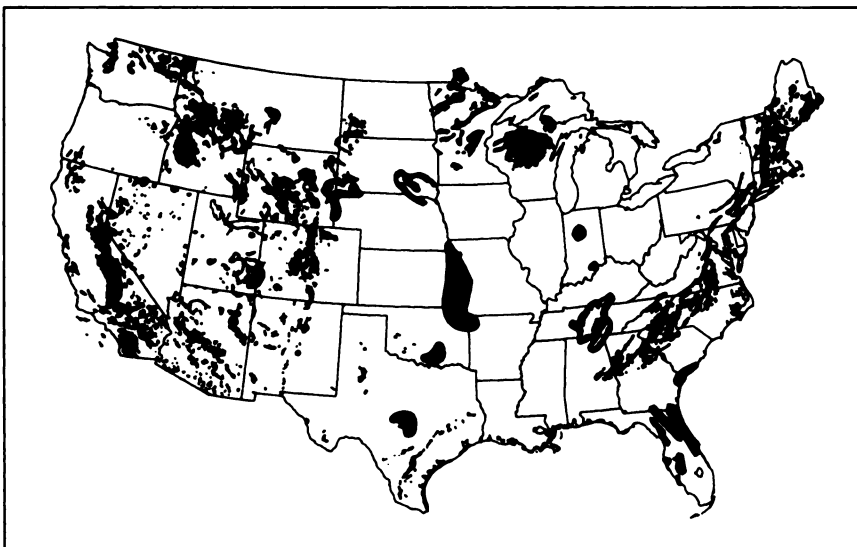


FIGURE 5. Map of the United States showing areas with potentially high radon levels in soil gas, based primarily on geological reports and modification of national uranium resource evaluation data (22).

(which varies widely between regions) and the amount used (70–250 gal in a typical household per day). On the average, 70% of radon contained in household water is released into indoor air (35).

The effect of human inhabitation on home radon levels is illustrated graphically in Figure 7 which is a record of radon levels in a Houston apartment during a two-day period. Radon concentration in air increased 3–5-fold during times the apartment was occupied.

NCRP Commentary No. 6 discusses the main sources of indoor radon and gives specific geographic areas in the U.S. where high levels exist (5). The EPA stratified survey was conducted in 125 counties in all 50 states. An average level of 1.25 pCi/liter (46 Bq/m³) was found, with 6% of the housing units exceeding the EPA action limit of 150 Bq/m³ or 4 pCi/liter. (13)

MECHANISMS OF RADON ENTRY INTO BUILDINGS

Since radon is constantly escaping from the ground, it is always present in the air, but under certain circumstances the concentration of radon in a building can be increased significantly over its normal outdoor level. Most buildings have a confined air space with limited air movement and only a slow exchange with outside air. Consequently, the concentration of any particulates or gases released into the building atmosphere will tend to increase above the concentration normally found in outside air. Radon can enter a building in a number of ways and once inside, the concentration of its particulate progeny will increase as the radon decays. Thus, high concentrations of radon in soils with high transport efficiency (i.e., loose, porous, dry soil) can lead to elevated radon concentrations in buildings.

Soil is the major source of radon. Studies underway by

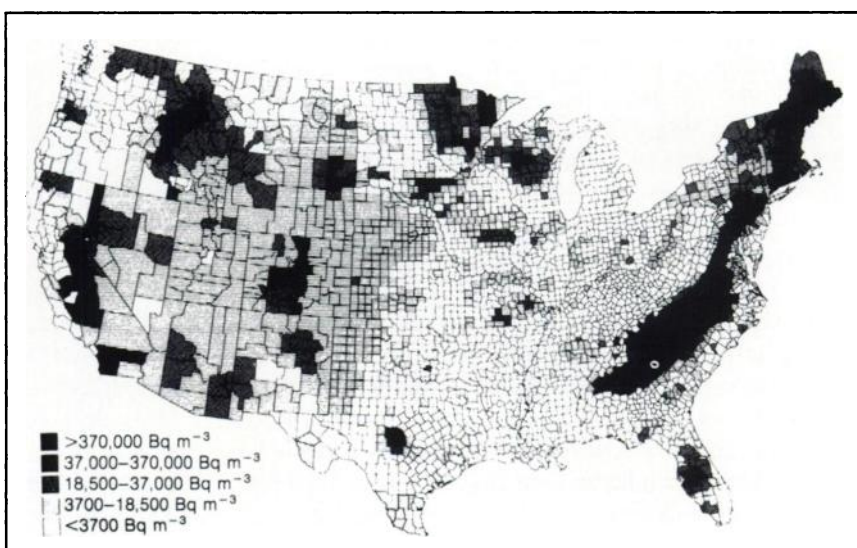


FIGURE 6. Map of the United States showing the distribution of radon in ground water supplies by county (22).

TABLE 3
Average Radon Concentrations in Groundwater by Aquifer Type*

Aquifer type	No. of samples	²²² Rn (Bq m ⁻³)
Granites		
Maine	136	817,700
North Carolina	24	390,800
South Carolina	22	298,800
Sweden	14	92,000
Metamorphic rocks		
Maine		
Sillimanite zone	35	503,300
Chlorite zone	56	41,000
North Carolina		
Gneiss/schist	71	83,000
Metavolcanic	21	49,900
South Carolina		
High-grade—Monazite belt	12	53,400
Medium-grade	11	118,100
Low-grade	7	274,700
Sweden		
Gneiss	8	26,000
Limestone		
Florida		
Florida	165	550
South Carolina		
South Carolina	15	1,300
North Carolina		
North Carolina	22	3,440
Sweden		
Sweden	12	24,000
Unconsolidated sand aquifers		
North Carolina coastal plain		
North Carolina coastal plain	139	15,760
Minnesota (glacial drift)		
Minnesota (glacial drift)	350	11,470
South Carolina		
Lower coastal plain	15	6,950
Middle coastal plain	34	9,470
Upper coastal plain	29	17,340

*From reference 22.

the U.S. Geological Survey show that soil-gas radon levels vary widely in small areas (within a housing lot) and are not well correlated with the radium content of the soil. Pressure-driven flow is the major means of transport from soil into buildings because the pressure inside buildings is usually lower than that in the soil, especially in the winter. Houses with no barrier between the soil and the interior (e.g., with a dirt floor in the basement or crawl space) are especially vulnerable. Houses with porous foundations (e.g., concrete block or fieldstone) present only a minimal barrier to flow. Even houses with poured concrete basement floors and foundations usually have routes of entry for soil gas through joints, penetrations, cracks, sumps and drains. Radon can enter a house from soil gas through ground level drainage systems, flaws in a concrete floor slab and concrete block walls (33).

The water supply can be a route of entry if there is a significant amount of radon in the ground water and if the water supply is derived directly from deep wells (34). Differences in water usage patterns, ventilation and air flow can cause significant temporal variations in radon levels indoors. However, soil gas radon content may be the great-

est determinant of home levels. Table 5 shows the variations in contributions to radon in the home (35).

Radon concentrations indoors will generally be highest in the basement or on the ground level since the major source is influx from the soil under and around the house. First floor concentrations will be lower by about a factor of two. Indoor radon concentrations are typically a factor of two to three times higher than outdoor levels. The radon concentration in the upper levels and in apartments above the first floor are usually of no concern.

In addition to soil and water sources of indoor radon, home construction materials can be a significant contributor. Table 6 indicates the emission rate measured from various building materials. Clearly, the concrete used in a building depending on its origin can be a major contributor, and in all cases, concrete is a more significant radon source than other building materials.

The frequency of homes with elevated radon levels varies in different regions of the country. The shape of the measured distribution is log normal. The distribution is highly skewed with most homes in the low-dose region. Based on measurements in 552 homes from 19 studies conducted in regions without unusually high radon concentrations, average levels measured in single-family homes by Nero (36) were found to be 0.96–1.66 pCi/liter based on geometric and arithmetic mean calculations. In Nero's data, 2.5% of the houses were above 8 pCi/liter, which is the action level recommended by NCRP (Fig. 8).

An EPA survey of 11,000 homes from 125 counties nationwide found the average annual radon concentration in U.S. housing units is 1.25 ± 0.06 pCi/liter, with a median value of 0.67 pCi/liter. They estimate that $6.01\% \pm 0.58\%$ of housing units (6 million homes) exceed the EPA action level of 4 pCi/liter (37).

The distribution of population dose to residents in the Reading Prong region is given in Table 7. The levels in Pennsylvania are higher than in New Jersey which is significantly higher than the U.S. average (unpublished data). The difference is most notable in the portion of the population exposed at levels above 8 and 20 pCi/liter. It is in the

TABLE 4
Sources of Global Atmospheric Radon*

Source	Input to atmosphere (million Ci/yr)
Emanation from soil	2000.0
Ground water (potential)	500.0
Emanation from oceans	30.0
Phosphate residues	3.0
Uranium mill tailings	2.0
Coal residues	0.02
Natural gas	0.01
Coal combustion	0.0009
Human exhalation	0.00001

*From reference 11.

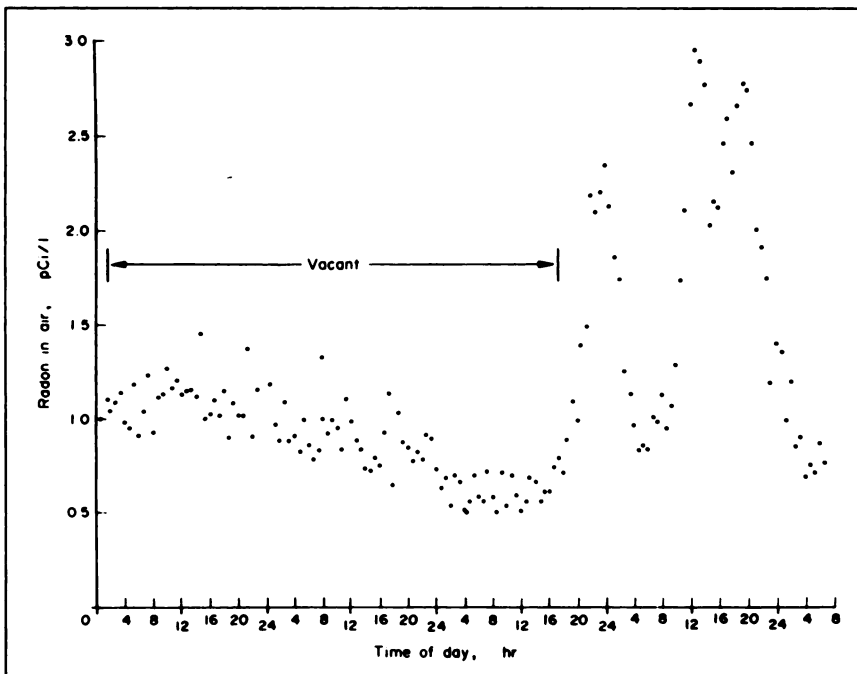


FIGURE 7. Continuous radon monitor record from Houston apartment indicating influence of occupancy on radon concentration (32).

high-dose regions of the country that greatest attention to measurement and remediation needs to be focused.

On rare occasions, radon levels have been found in houses which exceed those measured in uranium mines. This situation has occurred where the house is above a deep fissure in a granite shelf containing higher than normal levels of radium. The radon outgasses from the fissure through the soil under and around the home and enters it. The high pressure underground relative to the pressure in the home forces radon into the building. This is especially true in the winter when hot, low density air is vented from chimneys and other openings and is replaced by cold radon-containing air from the environment. When the ground adjacent to the house is frozen, soil permeability is diminished. A high access channel for radon entry into the home is found under the house, where the ground is warmer.

When a house is located on a shelf of granite with a deep fault, the surface area from which ^{222}Rn escapes includes the depth of the fault with granite on both sides, as well as the ground surface. A similar situation was noted where a granite shelf lay below an empty large salt dome through which the radon migrated rapidly, and reached the surface

with less time for radioactive decay. Clearly, such rare, very local circumstances justify quick remedial action. Public concern about radon has been raised and remedial action has been recommended at levels below which increased risk has been documented. It is not surprising that little in the way of remedial action has been taken at levels in the 4–8-pCi/liter interval. The EPA is concerned, however, that testing even in high radon areas has not been widely carried out.

MITIGATION STRATEGIES

The EPA has issued a series of documents on radon that provide useful guidance to the homeowner. A report which provides detailed and practical information on mitigation strategies for existing homes is found in the *Citizen's Guides* (1,3). Practical information concerning methods for reducing radon levels in new construction are also given by

TABLE 5
Approximate Contributions from Sources of Radon in Houses*

Source	Estimated contribution (activity/sec)	
Soil gas transport†	0–6 Bq	(0–150 pCi)
Release from potable H ₂ O	0–2 Bq	(0–60 pCi)
Soil gas diffusion	0.1–0.2 Bq	(3–6 pCi)
Diffusion from building materials	0.01–1 Bq	(0.3–30 pCi)

*From reference 35.

†May be a factor of 10–100 times higher in certain regions (34).

TABLE 6
Estimates of ^{226}Ra Concentration in Building Materials*

Material	^{226}Ra concentration (pCi/g)†
Wood	0.03
Concrete	0.43–1.65
Brick	1.1–2.6
Tile	2.1
Wall board	
Natural gypsum	0.11–0.27
Phosphogypsum	0.73
Insulating material (glass wool)‡	0.35–1.1

*From reference 33.

†Concentrations of solid material from which the home is made.

‡The impervious (glassy) nature of these products retards radon release.

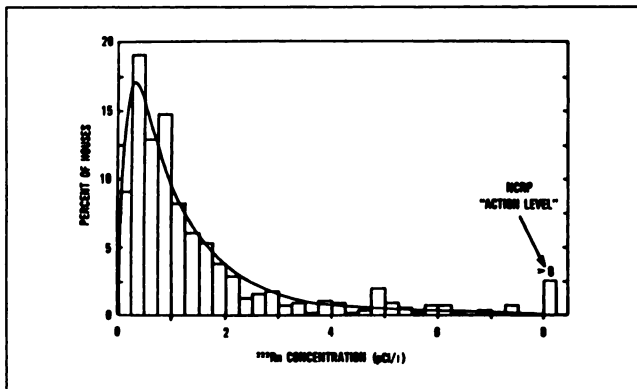


FIGURE 8. Percent of single-family homes with different values of ^{222}Rn concentration (36).

the EPA as are specifically outlined abatement methods (2,4).

Water purification systems and aeration techniques can be useful in areas with high levels of radon in the home water supply. Typically these are charcoal filter systems, but the filter itself presents potential difficulty in disposal and is a potential source of elevated external radiation dose. Air cleaning systems are not recommended because they have not been found to be effective.

Major attention is given to methods in which natural or forced ventilation is increased to diminish indoor levels of radon gas. These range from simply opening windows to forced ventilation systems when higher levels need to be abated. Twofold reductions can be obtained by the use of simple rotating household fans commonly used for summer ventilation during the winter months (38).

Covering exposed earth reduces ingress of radon, as does sealing cracks and openings in ground level walls and floors. Drain tiles can be placed surrounding the foundation and vented away from the house (drain tile suction method). This method is designed to pull radon from the soil surrounding the house and vent it away from the house. Sub-slab suction is more difficult to accomplish as it involves placing pipes under the house laterally through side walls or by drilling holes in the concrete slab. A fan is used to vent these pipes away from the house. The walls of

concrete-block houses can be vented by sucking air from the hollow spaces in the wall and venting away from the house to prevent radon from entering from this route. Lastly, methods are described for decreasing negative pressures within the house by bringing air into the house in proportion to losses from chimneys, dryers, etc., or by positive pressure including basement pressurization by blowing air from upper floors into the sealed basement. A comparison of the features of different systems is given in Table 8.

HEALTH EFFECTS AND HEALTH RISK

The major health risk from exposure to radon progeny is bronchogenic carcinoma. There are two major sources of human data: (1) Miners (old, poorly chronicled exposures with large numbers of person-years at risk, plus newer, better monitored, but incomplete studies); and (2) Epidemiology studies (high background areas; large populations, lower exposures).

Almost all large epidemiologic studies of lung cancer in miners indicate an excess mortality in groups receiving cumulative exposures of >120 WLM. However, dosimetric measurements made in working mines in different countries many years ago (especially prior to 1950) are subject to considerable uncertainty (8). Moreover, interpretation of dose-response curves for alpha particles is complicated. Evidence derived from radiobiology indicates that densely ionizing radiations, such as those from alpha particles, show dose-response curves which increase linearly from low doses to a maximum value, above which cancer induction rates fall due to wasted radiation, i.e., an additional dose to transformed cells is less efficient since the affected cells are already damaged; hence further doses either have no additional effect or result in cell killing (14).

Figure 9 shows representative data from studies of underground miners. A positive linear high slope region is seen following exposures below 200–300 WLM, which falls off and becomes negative at higher doses presumably due to cell killing.

It is assumed that the increased lung cancer risk to miners is due to ^{222}Rn and its daughters, but the cofactor role of the other dusts they breath in the mine has long been debated (39).

A retrospective cohort study conducted in southern China in collaboration with the U.S. National Cancer Institute involved 175,143 person-years of observation of workers in a tin mine (40). Eighty percent of the workers were employed underground and were exposed to radon and arsenic-containing dusts. Death was attributed to lung cancer in 981 individuals. This is the largest study reported to date, and is the first in which these detailed relationships could be tested with a reasonable statistical power. In addition to lung cancer, statistically significant increases in mortality were also observed for leukemia (12 deaths), lymphoma (5 deaths), pneumoconiosis (32 deaths), other respiratory diseases (63 deaths), coronary heart disease (47 deaths), cerebral vascular disease (302 deaths) and accidents (81 deaths).

TABLE 7
Radon Distribution (U.S. Average Versus Reading Prong Region)*

Radon Conc. (pCi/liter)	% Population NJ	Reading Prong PA	U.S.
0–2	40.1	14.7	83
2–4	26.3	24.4	11
4–8	19.3	29	4
8–20	10.6	19.5	2
20–100	3.4	10.9	<1
>100	0.3	1.4	<1

*From unpublished data.

TABLE 8
Mitigation Strategies: A Comparison of Features

Technique	Typical radon reductions	Typical range of installation costs* (contractor)	Typical operating cost range for fan electricity and heated/cooled air loss* (annual)	Comments
Sub-slab suction (sub-slab depressurization)	80%–99%	\$800–2,500	\$75–175	Works best if air can move easily in the material under the floor slab.
Drain-tile suction	90%–99%	\$800–1,700	\$75–175	Works best if drain tiles form complete loop around the house.
Block-wall suction	50%–99%	\$1,500–3,000	\$150–300	Only in houses with hollow block walls; requires sealing job of major openings.
Sump hole suction	90%–99%	\$800–2,500	\$100–225	Works best if air can move easily to sump under slab or if drain tiles form complete loop.
Sub-membrane depressurization in crawlspace	80%–99%	\$1,000–2,500	\$50–175	Less heat loss than natural ventilation in cold winter climates.
Natural ventilation in a crawlspace	0%–50%	\$200–500 if additional vents are installed; \$0 with no additional vents	May be some energy penalties	Costs are variable.
Sealing of radon entry routes	0%–50%	\$100–2,000	None	Normally used in combination with other techniques. Requires proper materials and careful installation.
House (basement) pressurization	50%–99%	\$500–1,500	\$150–500	Works best with tight basement that can be isolated from outdoors and upper floors.
Natural ventilation	Variable	\$200–500 if additional vents installed; \$0 with no additional vents	\$100–700	Significant heat and conditioned air loss; operating cost dependent upon utility rates and amount of ventilation.
Heat recovery ventilation	25%–50% if used for full house; 25%–75% if used for basement	\$1,200–2,500	\$75–500 for continuous operation	Limited use; works best in a tight house and when used for basement; less conditioned air loss than natural ventilation.

*The costs provided in this exhibit represent the range of typical costs for reducing radon levels in homes above 4 pCi/liter down to radon levels below 4 pCi/liter. In most cases, homes are reduced to an average of about 2 pCi/liter. Adapted from reference 13.

Table 9 shows age-adjusted relative risk in relation to exposure. Level 0 is nonexposed and increasing levels are graded by quartiles. The excess relative risk (ER) of lung cancer per WLM (ER/WLM) from radon fell from 0.6% to 0.2% when adjusted for arsenic exposures. The increase in relative risk with increasing levels of arsenic exposure is much stronger than the increase with level of radon exposure. Radon exposures ranged to greater than 800 WLM with the majority of exposures exceeding 400 WLM, and arsenic exposures ranged to greater than 10 mg-yr m⁻³ with the average exposure in the 3–5 mg-yr m⁻³ interval. The study is the largest of its kind and permits analysis of several other important factors. The ER/WLM declined significantly with (1) increasing exposure rate (cumulative WLM/duration of exposure); (2) years since last exposure,

and (3) increasing attained age. These effects only became significant after adjustment for the exposure effect from arsenic. In this cohort, 41% of the underground workers were <15 yr old when they started mining, however, lung cancer risk did not vary consistently with age at first radon exposure.

Figure 10 shows the relative risk estimates for different groups of miners, which indicate a wide uncertainty in cancer induction rates observed (41). Great variation in the ER/WLM from lung cancer has been seen in the different miner studies with the lowest risk observed in the Port Radium and American uranium miners and the highest risk observed in the Swedish and Beaverlodge miners. Whether the differences are due to errors in dose estimation, failure to correct for smoking and other lifestyle cofactors, or to

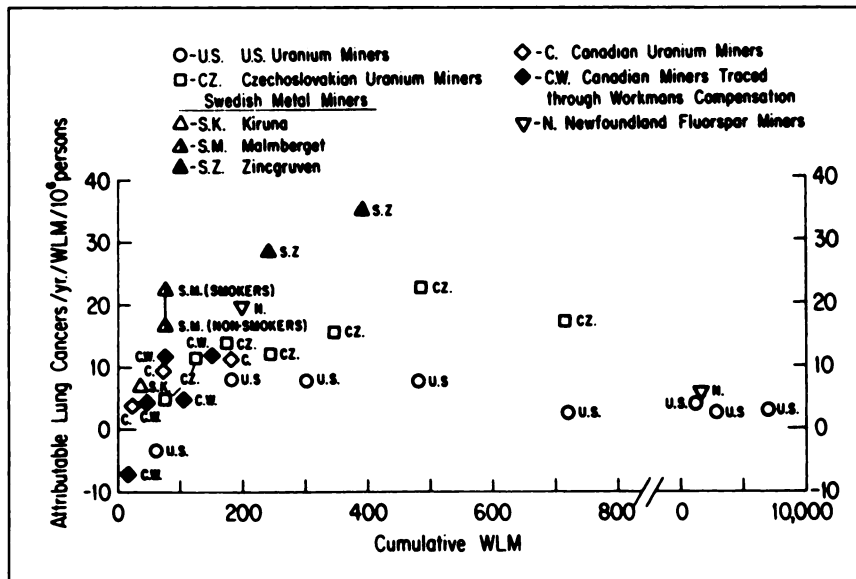


FIGURE 9. Lung cancer risk per WLM as a function of cumulative exposure. The risk is expressed as the attributable annual risk per WLM per million persons (11).

other exposures received in the mines cannot be assessed at this time. Studies are going on in these mines to establish and corroborate dosimetry estimates and to measure other materials, such as from arsenic, to which the miners may have been exposed. Continuing follow-up is proceeding and more definitive information may be forthcoming from these studies, although the uncertainties in doses received by miners many years ago will be very hard to overcome.

The effect of smoking as a cofactor in these studies is well accepted. Tumors appear earlier in smoking miners, and smoking is a significant cofactor (it is estimated that smokers have a 10 times higher risk per unit absorbed dose than nonsmokers) (8). It is also true that exposure to passive smoking has not been controlled in any of the miner studies and this may be as important as the radon exposures themselves.

The major uncertainties in the miner studies arise from uncertainties in dosimetry and in exposure to other carcinogens and/or promoters in the mine, as well as difficulty in controlling for smoking. The studies being conducted in China (28,40) point out the importance of controlling the other exposures in the mines (arsenic in this case), and the use of ²¹⁰Pb skull measurements provides a potential means of improving the dosimetry which has been a problem in all radon epidemiology studies.

EPIDEMIOLOGY STUDIES: ENVIRONMENTAL EXPOSURES

A second source of data on radon risk comes from epidemiologic studies of persons living in homes with increased radon levels. A large Canadian study was con-

TABLE 9
Lung Cancer Mortality by Levels of Exposure to Arsenic and Radon*

Arsenic exposure	Entry type	Cumulative radon exposure					Total
		0	I	II	III	IV	
0	Cases	41	14	1	2	1	59
	RRs	1.0	1.3	0.4	1.1	0.8	1.0
I	Cases	2	124	70	23	12	231
	RRs	4.7	2.0	3.9	3.6	2.7	2.5
II	Cases	0	63	68	58	44	233
	RRs	—	3.4	4.3	6.5	7.9	4.0
III	Cases	0	18	60	86	64	228
	RRs	—	5.6	5.5	8.2	11.3	5.5
IV	Cases	0	14	36	66	114	230
	RRs	—	6.0	6.3	8.0	10.9	5.7
Total	Cases	43	233	235	235	235	981
	RRs	1.00	0.8	1.1	1.5	1.9	—

*From reference 40.

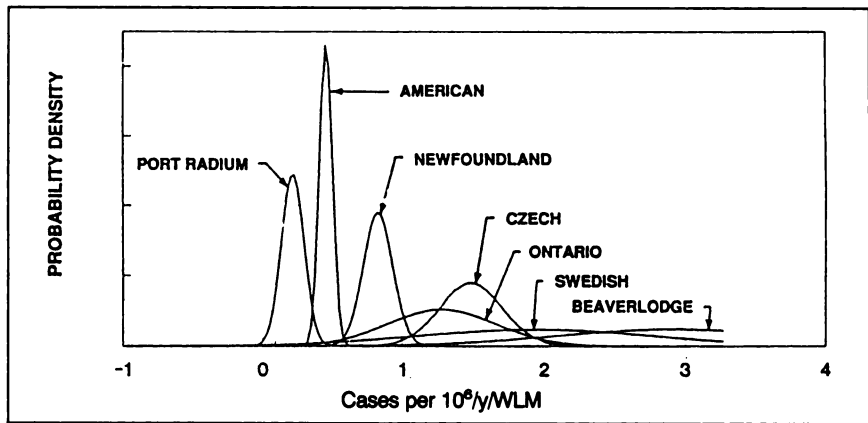


FIGURE 10. Lung cancer risk distributions for different uranium mines (41).

ducted in 18 cities involving 14,000 homes (42). A statistically significant correlation was found for smoking and lung cancer mortality in males, but the correlation was negative for mortality on measures of radon daughter concentration for males and positive for females, neither of which were statistically significant. Multiple linear regression analysis revealed that radon daughter concentrations did not add significantly to the effect of smoking on lung cancer rates. The authors concluded that any effect of radon, if present, was so small in comparison to the effect due to smoking, that it could not be detected in this type or size of study.

Case-control studies are ongoing in 10 countries attempting to relate radon exposure and lung cancer risk. Features of the different studies including the prevalence of homes with exposures greater than 4 pCi/liter are enumerated by Neuberger (43). The studies range in size from 32 to 3200 lung cancer cases with equal or greater numbers of controls in each study. The total number of subjects in the study include 12,273 lung cancer cases and 19,082 controls. Sample sizes needed to reach statistical significance at different exposure levels are calculated and suggest that many of the studies have adequate statistical power to reject the null hypothesis at high doses. But Neuberger believes that radon health effect studies at low doses could provide an opportunity to test the linear hypothesis and assist in deciding whether and in what circumstances the costs of radon remediation could be justified. He notes that through 1990, only about 25% of radon studies found statistically significant associations (44). A large number of the studies found lower than expected hazards from low doses, but these effects are almost never statistically significant. These studies conclude that deleterious effects of low doses, if present, are too low to detect in human population studies.

Letourneau has recently completed a large case-control study in Winnipeg involving 750 histologically confirmed lung cancer cases, age- and sex-matched against 750 controls. Winnipeg was studied because it has the highest radon levels in urban Canada. Over 80% of the residences were measured with alpha-track detectors. They adjusted for occupational factors, active smoking and ethnicity and

found no evidence of a correlation between lung cancer and residential radon levels (Letourneau E, *personal communication*).

An NIH-sponsored case-control study in Sweden investigated the correlation between radon exposure and lung cancer in 210 women with lung cancer and 400 control subjects (45). Smoking and residential history were obtained by interviews and radon measurements were made in a small fraction of the homes lived in by the subjects over their lifetime. Time-weighted radon measurements were made using either alpha-track detectors (1 yr average level per household measured), or thermoluminescent dosimeters which recorded radon levels during a 2-wk period in the heating season.

The authors indicate that lung cancer risk tended to increase with estimated radon exposure, reaching a relative risk of 1.7 (1.0–2.9) in women exposed to average radon levels, some of which greatly exceeded 4 pCi/liter. They note that these risk estimates are within the range reported for radon-exposed miners. The risk was 14 times higher in smokers than nonsmokers in the lowest exposure group (<2 pCi/liter), while in the higher exposure group, it was 6 times higher. In none of the smoking groups was there a significant trend relating level of radon exposure to cancer risk. They found an increased trend with exposure in young women based on five cases in the low-dose group and 11 in the high-dose group. The group aged under 55 yr was the only one in which a significant trend was noted. They report a stepwise increase in relative risk for lung cancer in nonsmokers ($p = 0.04$). This correlation was strongly dependent on the dose intervals chosen because no significant correlation was obtained when the dose was a continuous variable ($p = 0.5$) (Lubin JH, *personal communication*). A problem common to all residential radon studies is the difficulty of locating homes in which cases and controls lived, especially in the remote past (46). Three methods of assigning dose to missing time periods were used, and in only one case did they find a significant correlation. In neither of the other two methods of adjustment was a significant trend noted. This study does not provide strong support for a positive association between residential radon exposure and

an increased risk of lung cancer in Sweden with its high residential levels.

A more recent report was released by the Swedish group at a press conference in February 1993 (47). They conducted a large case-control study based on 1360 lung cancer cases diagnosed between 1980 and 1985 and 2857 matched controls. Track-etch dosimetry was obtained during the winter season in approximately 70% of their residences. Regression analysis included smoking as a variable along with radon exposure, age, degree of urbanization and occupation. They found a relative risk of 1.3 (1.1–1.6) at 4–11 pCi/liter and 1.8 (1.1–2.9) at exposures >11 pCi/liter, and attributed 15% of the lung cancer cases to radon. They also found a greater than multiplicative role for smoking.

A large study is being conducted in the high background region of China in the Guangdong Province and an adjacent control region (48). The study involves 2 million person-years of observation equally divided between the two regions. The ^{222}Rn levels differ by a factor of 3 in the two areas, but the rates of lung cancer mortality were reversed in relation to radon dose. There were 25 lung cancer deaths in the high-background area (average lung and treobronchial dose = 300–400 mrem), and 35 in the control region (average lung and treobronchial dose = 100 mrem), i.e., a 25% higher lung cancer mortality rate in the low-background region.

Levels of radon in the Reading Prong region are very high in certain areas of Pennsylvania and New Jersey. A case-control study was carried out in New Jersey in 433 women with lung cancer and 402 controls (49). They reported a statistically significant positive trend, compatible with increasing risk of elevated radon residential exposures. Only a small fraction of the cases and their residences could be located and radon levels measured. The study revealed a high relative risk associated with the highest exposed individuals. The authors urged caution in interpretation because of selection biases and the small numbers of subjects in the high exposure group.

Umhausen, Austria is a small village (2600 inhabitants) in the West Tyrol in which very high radon concentrations (median 50 pCi/liter) are found in an area between two rivers. In the rest of the town, radon levels are lower (median = 5 pCi/liter). The median lifetime radon exposure in these two areas is 242 and 23 WLM, with relative risks of 6.1(4.4–8.4) and 1.43 (0.7–2.7), respectively. The rates in the very high exposure group are comparable to those observed in uranium miners, whereas the rate in the lower exposed group is not significantly elevated (50).

In the United States, Cohen (51) has studied lung cancer rates in 965 counties in all states. He found a strong negative slope, which is highly significantly different from the slope predicted using linear/nonthreshold models and BEIR IV data (Fig. 11).

An extension of that study includes data from 1600 U.S. counties and compares mortality rates for various cancers to average radon levels. The strongest correlations are found with lung cancer, and the sign of the correlation is negative (52).

All of the environmental radon epidemiology studies have serious methodological problems. One problem is uncertain dosimetry. Uncertainties arise from difficulty in locating former residences and determining the cumulative dose to assign to each individual in case-control studies, as well as to what dose to assign to the TBE cell from which radon-induced lung cancer is thought to arise. The new methods being used for radon measurements should provide some help on the data collection aspect. Remaining major problems common to all epidemiology studies are the difficulty in identifying and controlling for the presence of confounding variables, such as smoking (active and passive), along with the problems in identifying and correcting for various selection and ascertainment biases.

Because of these uncertainties, the size of the study needed to establish statistical confidence is very large and the power of the statistical tests is often too weak to identify a significant difference between no risk from residential radon and increased risk at the level found in miner studies. A reasonable conclusion from these studies is that deleterious effects from average levels or natural background, if present, are too small to detect in most residential radon epidemiology studies. Evidence derived from ecologic studies has recently been critically reviewed with special relevance to radon. The authors conclude that the 15 largest ecologic studies they surveyed did not contribute to better understanding the quantitative risks of indoor radon (53).

The American Cancer Society estimated that there were 136,000 deaths from lung cancer in the U.S. in 1987, and that about 113,000 of these were the direct result from cigarette smoking. This assumption would leave 23,000 lung cancer deaths that may arise from all other causes. Using the average continuous radon exposure of 0.75 pCi/liter (0.19 WLM/yr) and the NAS-BEIR IV risk estimates, the number of radon-induced lung cancer deaths expected annually can be computed. Assuming a population of 240,000,000 in the U.S., between 4500 and 23,000 lung cancer deaths could be attributed to radon exposure annually (54). The average risk from NAS-BEIR IV (3.5×10^{-4} /WLM) would predict 16,000 deaths. Since there must be other causes of lung cancer besides cigarette smoking and radon progeny, many scientists involved in radiation protection matters believe that the hazards of radon exposure are significantly overestimated. In any event, the cheapest and most effective way of diminishing the lung cancer risk is to decrease or eliminate cigarette smoking.

Based upon the results of studies in miners, the estimated risk of lung cancer from exposure to radon progeny from ICRP, NCRP and BEIR IV are shown in Table 10. The estimates average 3.5×10^{-4} /WLM.

The Second International Workshop on Residential Radon (46) discussed the various ongoing case-control studies of residential radon exposure and lung cancer risk. Over 10,000 lung cancer cases are included in these investigations. A tabular summary of these studies is given by the EPA (13). Given the large number of studies now being conducted, and the difficulties in establishing meaningful

dosimetry, correcting for confounders and in pooling data, the DOE report concluded that it was unlikely that meaningful low dose risk estimates could be derived from additional radon epidemiology studies.

GUIDELINES FOR MITIGATING HIGH LEVELS OF RADON IN THE HOME

Currently there are no U.S. statutory limits covering naturally occurring radioactive materials such as radon and its progeny. However, both the NCRP and EPA have published guidelines for acceptable levels of radon in the home (1,16). The NCRP recommends that in single-family homes, remedial action should be taken to reduce radon levels if the average annual exposure exceeds 2 WLM/yr (equal to 8 pCi/liter assuming radon daughters are in 50% equilibrium with ²²²Rn).

EPA recommendations are based on average airborne radon levels in the home, and they recommend a graded scale of actions as presented in Table 11. Their recommendations suggest action at a lower dose (factor of 2) than the NCRP, but otherwise there is no major difference. The recently passed radon act (55) poses a long-term goal of remediation to outdoor levels of 0.2–0.7 pCi/liter which would require many billions of dollars to accomplish. The

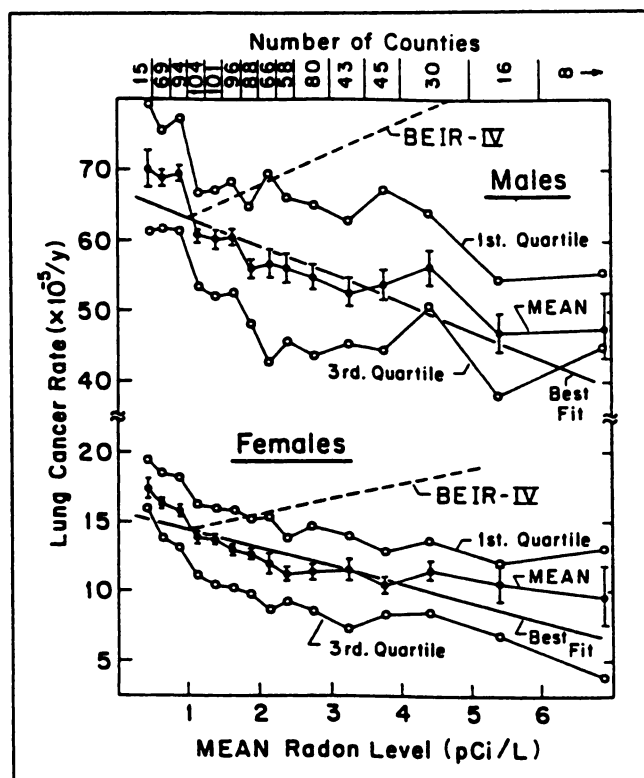


FIGURE 11. Age-adjusted lung cancer rates are plotted versus radon levels in counties in the study data base. The abscissa is divided into ranges shown at the top which also shows the number of counties in each range. Lung cancer rates for each set of counties are plotted along with mean, standard deviation and first and third quartiles. Least-squares best fit lines are plotted for the mean values (52).

TABLE 10
Comparison of Estimates of Lifetime Risk of Lung Cancer Mortality Due to a Lifetime Exposure to Radon Progeny*

Study	Year	Excess lifetime lung cancer mortality (deaths/10 ⁶ person WLM)
BEIR IV	1988	350
ICRP	1987	170–230 [†] 360 [‡]
NCRP	1984	130
BEIR III	1980	730
UNSCEAR	1977	200–450

*Adapted from references 8 and 6.
[†]Relative risk with ICRP population.
[‡]Relative risk with 1980 U.S. population as in BEIR IV.

urgency of recommended actions depends on the average radon levels in the living areas of individual homes and not simply on the highest level in an uninhabited portion of the house. The amount of time spent in the home and where one spends most of that time needs to be considered when making decisions on corrective actions. If high levels are found in high occupancy areas, remedial action should be considered and advice obtained from experts. Radiation control officials at the state or local level can suggest additional kinds of measurements, as well as recommend remedial actions, if indicated.

The EPA estimates approximately 22,000 lung cancer deaths per year may be related to radon exposure in the U.S. (56). Over a period of 70 yr, with 75% of an individual's day spent in the home, they calculate that an indoor level of 4 pCi/liter, with a 50% equilibrium between radon and its daughters would result in 54 WLM cumulative exposure, assuming 0.25 WLM/yr and 240 million people results in 60-million-person WLM. They then assume 360 deaths per million WLM (an average between the lower BEIR IV, and higher EPA estimates), from lung cancer (age-averaged rate for the U.S. population), and compute 21,600 deaths due to lung cancer due to radon per year. The ICRP gives a range of 8,600–25,900 to these estimates. The EPA evaluation of the risks from radon relative to other causes of lung cancer is given in Table 12.

Much controversy surrounds the true magnitude of health risks from radon, and the appropriate actions to be taken at different measured levels in the home or workplace. The issue boils down to understanding the magnitude of the health and economic risks and the costs and benefits of different responses. The ICRP (7) recommends that “proposed interventions should... be sufficiently (beneficial) to justify the harm and the costs, including social costs, of the intervention. The form, scale and duration of the intervention should be chosen so that the net benefit of the reduction of dose, i.e., the benefit of the reduction in radiation detriment, less the detriment associated with the intervention, should be maximized” (57).

The issue comes down to cost and benefit. The EPA has

TABLE 11
EPA Recommendations*

How quickly should action be taken?

In considering whether and how quickly to take action based on test results, the following guidelines may prove to be useful. The EPA believes that radon levels should try to be permanently reduced as much as possible. Based on currently available information, the EPA believes that levels in most homes can be reduced to about 0.02 WL (4 pCi/liter).

If results are about 1.0 WL or higher or about 200 pCi/liter or higher:

Exposures in this range are among the highest observed in homes. Residents should undertake action to reduce levels as far below 1.0 WL (200 pCi/liter) as possible. It is recommended that action should be taken within several weeks. If this is not possible, consultation with appropriate state or local health or radiation protection officials can determine if temporary relocation is appropriate until the levels can be reduced.

If results are about 0.1 to about 1.0 WL or about 20 to about 200 pCi/liter:

Exposures in this range are considered greatly above average for residential structures. Action should be undertaken to reduce levels as far below 0.1 WL (20 pCi/liter) as possible within several months.

If results are about 0.02 to about 0.1 WL, or about 4 pCi/liter to about 20 pCi/liter:

Exposures in this range are considered above average for residential structures. Action should be undertaken to lower levels to about 0.02 WL (4 pCi/liter) or below within a few years, sooner if levels are at the upper end of this range.

If results are about 0.02 WL or lower, or about 4 pCi/liter or lower:

Exposures in this range are considered average or slightly above average for residential structures. Although exposures in this range do present some risk of lung cancer, reductions of levels this low may be difficult, and sometimes impossible, to achieve.

NOTE: There is increasing urgency for action at higher concentrations of radon. The higher the radon level in a home, the faster action should be taken to reduce exposure.

*From reference 1.

estimated the cost per life saved (by averting a predicted lung cancer from radon) for various action levels that might be chosen. The numbers range from \$1.1 million dollars at 2.0 pCi/liter to \$0.7 million dollars at 4 pCi/liter and \$0.4 million dollars at the NCRP level of 8 pCi/liter (13). The cost per life saved from other nonradiological risks can reach the 0.4-million dollar figure (57).

SUMMARY STATEMENT BY REIR COMMITTEE

Radon is a naturally occurring element which has been shown to cause lung cancer in high doses. Miners exposed to high doses have an increased lung cancer risk which is significantly enhanced by smoking. Radiobiology data reveal a linear dose response following exposures to alpha

TABLE 12
Radon Risk Evaluation Chart*

pCi/liter	WL	Estimated number of lung cancer deaths due to radon exposure (out of 1000)	Comparable exposure levels	Comparable risk
200	1	440-770	1000 times average outdoor level	More than 60 times nonsmoker risk 4 pack-a-day smoker
100	0.5	270-630	100 times average indoor level	20,000 chest x-rays per yr
40	0.2	120-380		2 pack-a-day smoker
20	0.1	60-210	100 times average outdoor level	1 pack-a-day smoker
10	0.05	30-120	10 times average indoor level	5 times nonsmoker risk
4	0.02	13-50		200 chest x-rays per yr
2	0.01	7-30	10 times average outdoor level	Nonsmoker risk of dying from lung cancer
1	0.005	3-13	Average indoor level	20 chest x-rays per yr
0.2	0.001	1-3	Average outdoor level	

*From reference 1.

particle emitters in the low-dose region with saturation at high exposure levels (>200–400 WLM). A resident of a 4-pCi/liter house (0.04 WL) could be exposed at a rate of 0.5 WLM/yr. A small fraction of homes have much higher radon concentrations, exceeding levels in mines in some cases. It is clear that these homes need to be identified and their levels reduced. The cost of remediation of an individual dwelling is reasonably inexpensive at low radon levels, but there are many such houses. Very high-level houses are more difficult and expensive to mitigate, but they are relatively rare. The estimated costs are very high even at the 4-pCi/liter level, but are consistent with the costs society has and does spend for various health and safety problems.

To date, the EPA has had little success in stimulating homeowners to measure radon levels in their homes which would be the first step in the process of deciding on a course of action if a high radon level is found. This is partly because it is difficult to get people concerned that their home, a place that one looks to for security, is a potential source of hidden danger. Also, it has not yet been possible to generate convincing data on increased risk at or below levels <4–8 pCi/liter. However, it is prudent for people living in areas in which high levels do exist and are well known and publicized should they choose to test their home. Then, based on individual attitudes toward acceptable risks, appropriate action can be taken depending upon their resources and competing needs. This is also the position that society, faced with a multitude of costly options, must take. The cost-to-benefit ratio for radon abatement needs to be carefully considered in that context. Based on currently available data, the committee concludes that the costs of remediation exceeded the anticipated potential benefits for radon levels less than 8 pCi/liter.

REFERENCES

- EPA. *A citizen's guide to radon*. Washington, DC: U.S. Environmental Protection Agency; 1986.
- EPA. *Radon reduction methods, a homeowner's guide, second edition*. Washington, DC: U.S. Environmental Protection Agency; 1987.
- EPA. *The guide to protecting yourself and your family from radon. A citizen's guide to radon, second edition*, ANR-464, Washington D.C.: U.S. Environmental Protection Agency; 1992.
- EPA. *Consumer's guide to radon reduction*. 402-K92-003. Washington D.C.: U.S. Environmental Protection Agency; 1992.
- NCRP. *Commentary no. 6*. Radon exposure of the U.S. population—status of the problem. Washington, DC: NCRP; 1991.
- NCRP. *Report No. 103*. Control of radon in houses. Washington D.C.: NCRP; 1989.
- ICRP 60. *Recommendations of the international commission on radiological protection*. New York: Pergamon Press; 1991.
- NAS-BEIR IV. *Health risks of radon and other internationally deposited alpha-emitters*. Committee on the Biological Effects of Ionizing Radiations. Washington DC: National Academy of Sciences Press; 1988.
- NAS-BEIR V. *Health effects of exposure to low levels of ionizing radiation*. Committee on the Biological Effects of Ionizing Radiations. Washington DC: National Academy of Sciences Press; 1990.
- NAS. *Comparative dosimetry of radon in mines and homes. Companion to health risks of radon and other internationally deposited alpha-emitters*. Committee on the Biological Effects of Ionizing Radiations (BEIR IV). Washington DC: National Academy of Sciences Press; 1991.
- NCRP. *Report no. 78*. Evaluation of occupational and environmental exposures to radon and radon daughters in the United States. Washington D.C.: NCRP; 1984.
- NCRP. *Proceedings no. 10 of 24th annual meeting, March 30–31, 1988*. Washington, DC: NCRP; 1988.
- EPA. *Technical support document for the 1992 citizen's guide to radon*. EPA 400-R-92-011, Washington D.C.: U.S. Environmental Protection Agency; 1992.
- DOE. *Indoor radon and decay products: concentrations, causes and control strategies*. DOE/ER-0480P. Washington D.C.: DOE; 1990.
- DOE. *Radon research program, FY 1992*. DOE/ER-0588 1993. U.S. Office of Health and Environmental Research, Washington D.C.: DOE 1993; 20585.
- NCRP. *Report no. 116*. Limitation of exposure to ionizing radiation. Washington D.C.: NCRP; 1993.
- NCRP. *Report no. 93*. Ionizing radiation exposure of the population of the United States. Washington D.C.: NCRP; 1987.
- NCRP. *Report no. 100*. Exposure of the U.S. population to diagnostic medical radiation. Washington D.C.: NCRP; 1989.
- Rock RL, Walker DK, Dalzell RW, Harris EJ. Controlling employee exposure to alpha radiation in underground uranium mines. *U.S. Bureau of Mines handbook, volumes 1 and 2*. Washington D.C.: U.S. Bureau of Mines; 1970, 1971.
- James AC. Dosimetry of radon and thoron exposures: implications from risks from indoor radon. In: Cross FT, ed. *Indoor radon and lung cancer: reality or myth*. Columbus: Batelle Press; 1992:167–188.
- NCRP. Measurement of radon and radon daughters in air. *NCRP report no. 97*. Washington DC: NCRP; 1988.
- Cothern CR, Smith JE, Jr. ed. *Environmental radon. Environmental science research series, Vol. 35*. NY: Plenum Press; 1987.
- Samuelsson C. Retrospective determination of radon in houses. *Nature* 1988;334:338–340.
- Samuelsson C. Recoil deposited ^{210}Po in radon-exposed dwellings. In: Cross FT, ed. *Indoor radon and lung cancer: reality or myth*. Columbus: Batelle Press. 1992;89–100.
- Cornelis J, Landsheere C, Poffijn A, Vanmarcke H. Experimental and theoretical study of the fraction of ^{210}Po absorbed in glass. In: Cross FT, ed. *Indoor radon and lung cancer: reality or myth*. Columbus: Batelle Press. 1992;101–111.
- Lively RS, Steck DJ. Long-term radon concentrations estimated from ^{210}Po embedded in glass. *Health Phys* 1993;64:485–490.
- ICRP 23. *Reference man: anatomical, physiological and metabolic characteristics*. Oxford: Pergamon Press; 1975.
- Laurer GR, Gant QT, Lubin JH, et al. Skeletal ^{210}Pb levels and lung cancer among radon-exposed tin miners in southern China. *Health Phys* 1993;64: 253–259.
- Gunderson LCS. Role of geology in predicting radon potential. *Health Phys* 1992;62(suppl):S13.
- Nevessi AE, Bodansky D. Radon sources and levels in the outside environment. In: Bodansky D, Robkin MA, Stadler DR, eds. *Indoor radon and its hazards*. Seattle: Univ. of Washington Press; 1987:A2–A50.
- O'Conner P. Correlating geology with radon pockets. *Radon research notes, issue 11*. Oak Ridge TN: Oak Ridge National Laboratory; 1993.
- Pritchard HM, Gesell TF. An estimate of population exposure due to radon in public water supplies in the area of Houston, Texas. *Health Phys* 1981; 41:599–606.
- Tanner AB. The source of radon in houses. *Proceedings no. 10 of 24th annual meeting, March 30–31, 1988*. Washington DC: NCRP;1988.
- Stone R. EPA analysis of radon in water is hard to swallow. *Science* 1993;261.
- Bruno RC. Sources of indoor radon in houses: a review. *J Air Pollu Contr Assoc* 1983;133, 105.
- Nero AV, Schwehr MB, Nazaroff WW, Revzan KL. Distribution of airborne radon-222 concentrations in U.S. homes. *Science* 1986;234:992–997.
- Marcinowski F. Nationwide survey of residential radon levels in the U.S. *Health Phys* 1992;62(suppl):S13.
- Maher EF, Rudnick SN, Moeller DW. Effective removal of airborne Rn-222 decay products inside buildings. *Health Phys* 1987;53:351–356.
- Furth J, Lorenz E. Carcinogenesis by ionizing radiations. In: Hollaender A, ed. *Radiation biology*. New York: McGraw-Hill, 1954;1145–1201.
- Xiang-Zhen X, Lubin JH, Jun-Yao L, et al. A cohort study in southern China of tin miners exposed to radon and radon decay products. *Health Phys* 1993;64:120–131.
- Chamber DB, Reilly PM, Lowe LM, Stager RH, Dupont P. Effects of exposure uncertainty on estimation of radon risks. In: Cross FT, ed. *Indoor radon and lung cancer: reality or myth*. Columbus: Batelle Press; 1992;987–1012.
- Letourneau E. *Lung cancer mortality and indoor radon concentrations in*

- 18 Canadian cities. 16th Midyear Topical Meeting, Epidemiology Applied to Health Physics, Albuquerque, New Mexico, 1983.
43. Neuberger JS. Residential radon exposure and lung cancer: an overview of ongoing studies. *Health Phys* 1992;63:503-509.
 44. Neuberger JS. Residential radon exposure and lung cancer: an overview of published studies. *Cancer Detect Prev* 1991;15:435-443.
 45. Pershagen G, Zhong-Hua L, Hrubec Z, Svensson C, Boice JD. Residential radon exposure and lung cancer in Swedish women. *Health Phys* 1992;63:179-186.
 46. DOE. *Report on the second international workshop on residential radon*. CONF-9107220. Washington D.C.: Department of Energy, 1991.
 47. Pershagen G, Axelson, O, Clavensjo B, et al. Radon in dwellings and cancer: a country-wide epidemiological investigation (in Swedish). *IMM Report* 1993;1-26.
 48. Luxin W, Yongru Z, Zufan T, Weihui H, Deqing C, Yongling Y. Epidemiologic investigation of radiological effects in high background areas of Yand-Jiang. *China Journal of Radiation Research (Japan)* 1990;31:119-136.
 49. Shoenberg JB, Klotz HB, Wilcox HB, Szmeciasz SF. A case control study of radon and lung cancer among New Jersey women. In: Cross FT, ed. *Indoor radon and lung cancer: reality or myth*. Columbus: Batelle Press; 1992;905-922.
 50. Ennomoser O, Ambach W, Brunner P, Schneider P, Oberaigner W. High domestic and occupational radon exposures: a comparison. *Lancet* 1993; 47:342.
 51. Cohen BL. Multi-stratified multiple regression tests of the linear/no-threshold theory of radon-induced lung cancer. In: Cross FT, ed. *Indoor radon and lung cancer: reality or myth*. Columbus: Batelle Press. 1992;959-975.
 52. Cohen BL. Relationship between exposure to radon and various types of cancer. *Health Phys* 1993;65:234-251.
 53. Stidley CA, Samet JM. A review of ecologic studies of lung cancer and indoor radon. *Health Phys* 1993;65:529-531.
 54. Lubin JH, Boice JD. Estimating radon-induced lung cancer in the United States. *Health Phys* 1989;7:417-427.
 55. Reagan R. Title III. Indoor Radon Abatement. *Amendment to the Toxic Substances Control Act*. Signed into law by Ronald Reagan, October 1988.
 56. Schmidt A, Puskin JS, Nelson N, Nelson CB. EPA's approach to assessment of radon risk. In: Cross FT, ed. *Indoor radon and lung cancer 1 reality or myth*. Columbus: Batelle Press. 1992;923-933.
 57. ICRP65. Protection against radon-222 at home and at work. *Annals of the ICRP, Volume 23, no. 2*. New York: Pergamon Press; 1993.
 58. Cohen BL. Reducing hazards of nuclear power insanity in action. *Physics and Society*. 1987;16:2-4.