

Radon

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BACKGROUND

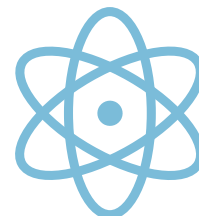
Radon-222 is a colorless, odorless, radioactive gas that forms from the decay of naturally occurring uranium-238. Since U-238 occurs in soil and rock throughout the world, radon exposure is universal; radon is present not only indoors but outdoors. Radon exposure in homes is largely a result of radon-contaminated gas rising from the soil. This makes it an unusual indoor air pollutant in that it has a natural source. Exposure to radon is also a known cause of lung cancer in underground miners of uranium and other ores. Thus, its presence in indoor air has raised concern that it may also be a cause of lung cancer for the entire population.

Radon decays with a half-life of 3.8 days into a series of solid elements called radon progeny. Among these elements—polonium-218, polonium-214, lead-214, and others—are several that emit alpha particles. An alpha particle consists of two protons and two neutrons, equivalent to the nucleus of a helium atom, and carries a positive charge. While alpha particles do not penetrate deeply into tissue, as gamma radiation can, they do carry enough energy to cause permanent changes in DNA if they reach the nucleus of a cell. As a result, when inhaled radon progeny reach the lungs, the alpha particles they emit can damage cells within the airways and thereby increase lung cancer risk.

RADON EXPOSURE

Suspicion that working in underground mines—later shown to be linked to radon exposure—is associated with cancer arose even before radon was identified as an element. In 1556, German scholar Georgius Agricola wrote in *De re metallica* of the high mortality of miners in the Carpathian Mountains of Eastern Europe. More than 300 years later, autopsy studies of miners in that region demonstrated that chest tumors were a common cause of death, and the type of tumor was later demonstrated to be primary lung cancer.

In the early 20th century, mines in Germany and Czechoslovakia (now the Czech Republic) were found to have high levels of radon, and researchers suspected that this exposure was the cause of the miners' lung cancer. In the 1950s, radiation scientists recognized that particulate radon progeny and not radon gas delivered the radiation dose ultimately responsible for causing cancer. Epidemiologic studies of radon-exposed miners during the 1950s and 1960s confirmed the association between radon exposure and lung cancer.¹ Less formal clinical and epidemiological studies of these miners also showed a clear excess of lung cancer in radon-exposed underground workers.

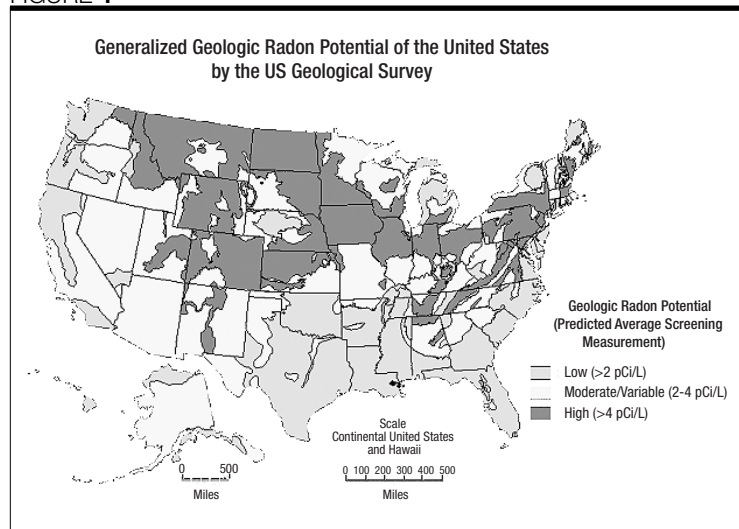


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This article is also available at www.cancer.org.

FIGURE 1



While occupational exposure to radon was established as a carcinogen decades ago, concern for the possibility that exposure of the general population to radon might also cause lung cancer is more recent in the general population. Adults and children are exposed to radon in homes, commercial buildings, schools, and other places. This exposure can occur when radon gas emitted by soil or rock enters buildings through cracks in floors or walls, construction joints, or gaps in foundations around pipes, wires, or pumps. Without ventilation or any other way of dissipating it, radon can accumulate and reach relatively high levels. On the other hand, radon emitted into outdoor spaces generally disperses and does not reach high levels. Indoor exposure is usually far lower than the occupational exposure reported for miners. However, the range of levels in the most contaminated homes approaches levels found in mines and may exceed standards of permissible exposures for underground miners.

Radon levels in the air are measured by units of radioactivity per volume of air. The most common concentration measure used is picocuries per liter, written pCi/L. Background outdoor levels of radon range from near zero to over 2 pCi/L.

The US Environmental Protection Agency (EPA) has set an action level of 4 pCi/L as an annual average for homes and schools, and the National Council of Radiation Protection recommends a limit of no more than 8 pCi/L indoors. Radon concentration may also be expressed in SI units of becquerels per cubic meter (Bq/m^3), which properly should be used, or as a Working Level (WL), a unit applied historically to underground mines. Exposure to radon or radon progeny incorporates the time spent at different concentrations; one commonly used unit for exposure is the Working Level Month, referring to spending one working month of time at a concentration of one WL.

The level of personal exposure to radon is quite variable, depending on the concentration at home and whether there is any occupational exposure. Some jobs do carry the potential for higher levels of radon exposure: Working in some underground mines, including not only uranium but also some other types; in caves; or in uranium processing factories. Contact with phosphate fertilizers, which have high levels of radium, the immediate precursor of radon, may also produce higher exposures. People who live near uranium mines are likely to be exposed to higher levels of radon than the rest of the population, but few facilities are now operating.²

With regard to indoor exposures, the range across the country is substantial, even within small geographic regions. The potential of the soil to release radon and contaminate homes varies with the concentration of radium and the characteristics of the earth. As a result, radon levels vary in the soil of different parts of the US. As shown by this United States Geological Survey map (Figure 1), an area in the mid-Atlantic states stretching from New York through Pennsylvania to Maryland and Virginia, as well as a broad stretch of the upper Midwest, has geological formations that yield higher radon levels. In contrast, radon levels are low in the Southeast as far west as Texas, and

along much of the West coast. However, elevated radon levels have been found in parts of almost every state.

The EPA estimates that as many as eight million homes in the US have elevated radon levels.² The EPA posts detailed geographic information on radon levels, including state maps, on its Web site at: www.epa.gov/iaq/radon/zonemap.html, and the United States Geological Survey publishes a series of "geologic radon potential books" that provide detailed geographic information also available at: energy.cr.usgs.gov/radon/radonhome.html.

Within buildings, radon levels are usually highest in the basement due to its proximity to the ground from which the radon-containing soil gas diffuses.³ Accordingly, people who spend much of their time in rooms in basements (at home or at work) would face a greater potential for exposure.

Radon exposure may also occur through drinking water and from exposure to building materials. Well water that comes from deep wells in rock with high radium concentrations may have high levels of radon, while surface water generally has very low radon levels. Exposure from ingestion of water is minimal; rather, it occurs when the radon moves from water to air and is inhaled. Overall, water provides a minor contribution to the overall exposure to radon.⁴ Similarly, building materials may contain radon if they are made from radon-containing substances. There have been specific instances of higher levels associated with particular building materials. In Sweden, for example, wall board made with phosphogypsum having a high concentration of radium led to elevated concentrations of radon. Such materials can make a major contribution to radon exposure.⁵

THE ASSOCIATION OF RADON AND CANCER

Radon is among the best-studied of environmental carcinogens. Present evidence

from multiple lines of investigation includes epidemiological studies of miners and of the general population, animal studies, and laboratory-based studies of responses of cellular systems to exposure to alpha particles. The evidence is complementary and comprehensive, providing a broad understanding of the mechanism by which alpha particles injure cells, of the patterns of radiation dose delivered to the lungs by inhaling radon progeny, and of the quantitative risk of lung cancer associated with exposure. This large body of evidence is summarized authoritatively in *Health Effects of Exposure to Radon*, the 1998 report of the National Research Council's Committee on the Biological Effects of Ionizing Radiation VI, often referred to as the BEIR VI Report.¹

THE EPIDEMIOLOGIC EVIDENCE

The three types of epidemiologic evidence regarding radon and lung cancer risk come from cohort studies of underground miners with relatively high levels of radon exposure, case-control studies of persons with lung cancer and appropriate controls from the general population, and ecologic studies comparing lung cancer mortality or incidence across areas with differing levels of radon exposure within their populations. Historically, the cohort studies have provided the first conclusive evidence that radon is a cause of lung cancer; they have also been an important source of data for quantitatively estimating the risk of radon exposure. The case-control studies were first initiated about 20 years ago, when recognition of the problem of indoor radon became widespread. The ecological studies were initiated for the same purpose.

Many groups of underground miners exposed to radon have been studied, initially to learn if radon causes lung cancer and subsequently to describe how risk varies with exposure. The main cohorts include uranium miners in Czechoslovakia, France, Canada,

Australia, and the US, fluorspar miners in Canada, iron miners in Sweden, and tin miners in China.^{1,6-8} For the purpose of quantitative risk estimation, data have been assembled from 11 cohort studies, including 68,000 miners with 2,700 deaths from lung cancer. These data have then been jointly analyzed to develop a statistical description of how risk varies with exposure to radon and other factors, including cigarette smoking. At all but the highest levels, risk increases in a linear fashion with exposure. At the lower end, the exposure-response relationship is linear and there is no indication that there is a threshold (a value that must be exceeded for there to be increased risk). In those miners known to be nonsmokers, a linear relationship between lung cancer risk and exposure also exists.

The designs of the studies are relatively similar: Cohort studies of mortality incorporating estimates of exposure to radon progeny and, in some instances, smoking. For example, a Public Health Service study followed 3,347 exposed miners on the Colorado Plateau (in Arizona, Colorado, New Mexico, and Utah). These individuals were exposed for an average of 3.9 years beginning in the late 1940s and 1950s; follow-up continues to the present. Investigators ascertained their vital status and cause of death from a combination of company, state, and federal sources, and studied the association of mortality with exposure levels. The results indicated an increasing risk of lung cancer with increasing exposure to radon.

In this study, as in most of the cohort studies of lung cancer in miners, methodologic issues complicate interpretation of the results. Complete data on levels of past exposures were unavailable, and the researchers needed to estimate exposures to fill the resulting gaps. Error resulting from their estimates would generally tend to lessen the apparent effect of radon on lung cancer risk, so that the actual risk of radon might be greater than actually observed. Statistical models representing the

relationship between risk and exposure were used to analyze the data to show how risk varies with level of exposure and other factors. The results of such models, as for any analysis based on estimates, are subject to uncertainty.

Second, although some information on smoking was available, this information was not complete, nor was smoking tracked regularly over time. Similarly, other relevant exposures, such as previous employment in other mines, exposure to other potential carcinogens in mines such as arsenic and diesel exhaust, and past medical treatment utilizing radiation, could not be fully accounted for, although the effect of radon on lung cancer risk was so strong as to be not readily explained by other factors.

When the problem of indoor radon was first recognized, numerous epidemiological studies of the case-control design were implemented. In case-control studies of residential radon exposure, lung cancer patients are compared with controls who are free of lung cancer with respect to their histories of radon exposure. Levels of radon are measured in their current homes and, if possible, in homes previously occupied. One major limitation of these studies, as for the studies of miners, is accurately estimating past exposures. Current levels of radon may not adequately represent those in the past because researchers have had difficulty in obtaining access to homes for the purpose of measuring radon levels. These studies have been reviewed by several authors.^{1,9-10} Since those reviews, several additional case-control studies have been published.¹¹⁻¹⁵

Two examples of case-control studies are illustrative. Pisa et al.¹² identified 138 deaths from lung cancer in an Italian alpine valley with high radon levels, and 291 controls matched by gender and year of birth. They evaluated exposure by measuring radon levels in the most recent residence and collected information on smoking and occupational history through interviews. Using a radon

exposure level of less than 40 becquerels (Bq/m³) as the reference category, the odds ratios for 40 to 76 Bq/m³, 77 to 139 Bq/m³, 140 to 199 Bq/m³, and greater than or equal to 200 Bq/m³ were 2.1, 2.0, 2.7, and 1.4, respectively. The association between radon and lung cancer seemed to be confined to male smokers.

Because of the possibility that smoking modifies the risk of radon exposure, as in the study of Pisa et al.,¹² Lagarde et al.¹³ studied nonsmokers, as have other researchers. They identified 436 lung cancer cases among nonsmokers in Sweden and 1,649 nonsmoking controls between 1980 and 1995. Exposure was evaluated by calculating the time-weighted residential radon concentration over three decades. Using a radon exposure level of less than 50 Bq/m³ as the reference category, the investigators calculated the odds ratios for 50 to 79 Bq/m³, 80 to 139 Bq/m³, and greater than or equal to 140 Bq/m³, which were 1.08, 1.18, and 1.44, respectively. The risk seemed to be higher among those exposed to environmental tobacco smoke.

Limitations of this study design have clouded interpretation of the findings. A major challenge in these studies, as in case-control studies generally, is the retrospective assessment of exposure. In addition, many of the case-control studies are relatively small, with limited power to detect and quantify increased risk, especially in subgroups. Finally, the case-control studies face the challenge of controlling for confounders and evaluating possible modifying factors. Smoking is a special challenge, since the effect of smoking on lung cancer risk appears to be an order of magnitude greater than the effect of radon. For these reasons, while many case-control studies demonstrate an association between radon and lung cancer, this finding has not been entirely consistent. Additionally, it has been difficult to characterize the joint effect of smoking and radon exposure with regard to potential synergism.

These limitations can be addressed to some

extent by combining the information from different studies. The technique of meta-analysis, which combines the summary results from studies, has been used for this purpose. The BEIR VI report¹ includes a meta-analysis of the then-available studies of residential radon. This analysis shows that risk of lung cancer increases with estimated exposure and that the exposure-response relationship is quite close to the predictions from the miner data. A pooling of the case-control studies from around the world has been implemented.

The third kind of epidemiologic evidence is ecologic studies, which compare population lung cancer rates in high-radon and low-radon areas. Without individual data on exposure to either radon or confounders such as smoking, these studies are less informative than case-control and cohort studies.¹⁶ Indeed, the results of ecologic studies of radon have been inconsistent, and numerous limitations of this body of evidence have been identified.^{1,17}

ANIMAL AND LABORATORY STUDIES

Like the studies of miners, experimental studies conducted on animals have clearly demonstrated a risk of lung cancer with exposure to radon. In studies using rats, hamsters, and dogs, inhalation of radon and its progeny resulted in a significant increase in the incidence of respiratory tract tumors.¹⁸ Nonspecific effects on the lungs have also been reported. In studies using human cells, radon and its decay products induced chromosomal abnormalities and other indications of permanent cellular change.¹⁸

WHAT EXPERT AGENCIES SAY

The National Toxicology Program (NTP) evaluates exposures that may be carcinogenic. Exposures that are thought to be carcinogenic are included in the *Reports on Carcinogens*, published every two years. Each exposure is assigned to one of two categories: "Known

to be human carcinogens” and “reasonably anticipated to be human carcinogens.” The first category includes substances for which human studies (epidemiologic studies and/or experimental studies) provide “sufficient evidence” of carcinogenicity in humans. The second category includes substances for which there is limited evidence of carcinogenicity in humans and/or sufficient evidence of carcinogenicity in experimental animals. Using this scheme, the NTP classifies radon as a “known human carcinogen.”¹⁹

The International Agency for Research on Cancer (IARC) also evaluates exposures that may be carcinogenic. The IARC classifies exposures into one of four categories: Group 1 exposures are those “known to be carcinogenic to humans,” usually based on “sufficient” human evidence, but sometimes based on “sufficient” evidence in experimental animals and “strong” human evidence. Group 2 exposures are divided into two categories; Group 2A (“probably carcinogenic to humans”) has stronger evidence, and Group 2B (“possibly carcinogenic to humans”) has weaker evidence. Group 3 exposures are not considered classifiable, because available evidence is limited or inadequate. Finally, Group 4 exposures are “probably not carcinogenic to humans,” based on evidence suggesting lack of carcinogenicity in humans and in experimental animals. The IARC rates radon as “carcinogenic to humans” (Group 1).^{18,20}

The Environmental Protection Agency (EPA), through its Integrated Risk Information System, uses a classification scheme very similar to that of the IARC. It classifies exposures into one of five categories: (A) human carcinogen; (B) probable human carcinogen; (C) possible human carcinogen; (D) not classifiable as to human carcinogenicity; and (E) evidence of noncarcinogenicity for humans. The EPA has not classified radon as to its carcinogenicity.²

The Agency for Toxic Substances and Disease Registry (ATSDR) has concluded that radon is carcinogenic.²¹

ASSOCIATION WITH OTHER HEALTH PROBLEMS

There is convincing evidence that radon causes lung cancer. Some studies of miners further suggest an association between radon exposure and nonmalignant respiratory disease, especially pulmonary fibrosis.²² These effects appear primarily in miners with high levels of exposure. The radiographic patterns are not typical of silicosis, also a problem in uranium miners.

Some evidence has also linked radon exposure with malignancies other than lung cancer. An analysis of the 11 cohort studies of miners²³ found an increase in leukemia and in cancers of the stomach and liver. Risk for these malignancies in the miners does not increase with the level of exposure, and these associations do not appear to be causal. Ecologic studies have linked radon with elevations of cancers other than lung cancer, but without any consistent pattern.¹ A recent case-control study of acute lymphocytic leukemia revealed no evidence of increased risk associated with radon exposure.²⁴ Since inhalation is the main route of absorption of radon, and since alpha particles penetrate tissues only superficially, effects on tissues other than lung tissue would be unexpected.

ADVISING PATIENTS AND THE GENERAL PUBLIC

Concerned patients may ask about medical tests for radon exposure. At present there is no test sufficiently sensitive to determine past levels of exposure. Some methods for estimating past exposure—e.g., special counting of levels of radiation in the skull—are under evaluation for research purposes, but these lack the needed sensitivity for most exposures in the general population.

On the other hand, radon exposure in homes can be assessed readily. Homeowners can purchase radon detection kits in hardware or home supply stores or hire a private radon contractor. Some common types of detectors

include charcoal canisters, alpha track devices, electret ion chambers, continuous monitors, and charcoal liquid scintillation detectors. Do-it-yourself kits are placed in the home for several days to as long as three months, then mailed to a laboratory for analysis. Alternatively, private contractors can be hired to perform this testing. Qualified contractors can be located through state radon offices, which are listed on the EPA Web site at www.epa.gov/iaq/contacts.html.

If test results show radon levels above 4 pCi/L, the EPA recommends remediation. This value refers to the annual average, and the short-term tests often used at the time of sale of a home tend to give measurements that are biased upwards from the actual value if the EPA protocol is followed. Values above the guideline should be followed by a repeat measurement, generally using one of the longer-term devices if circumstances permit. Like radon testing, remediation can be performed by the homeowner, depending on the approach, or by a commercial contractor. A variety of methods is used, ranging from sealing cracks in floors and walls to increased ventilation to "sub-slab depressurization" using pipes and fans. Again, qualified contractors can be located through state radon offices, which are listed on the EPA Web site at www.epa.gov/iaq/contacts.html.

Certain building materials may be more "radon-tight" and may help reduce exposure in areas where radon levels are high.²⁵ Further information can be obtained from state radon offices or from qualified contractors.

Since there is evidence of synergy between cigarette smoking and radon in the causation of lung cancer, it is especially important that people exposed to higher levels of radon quit smoking. For the miners, synergism between smoking and radon exposure has resulted in extremely high health risks.

Finally, for those miners whose health was compromised by exposure to radon, the US has established a national compensation approach

through the Radiation Exposure Compensation Act, passed in 1990²⁶ and amended in 2000. The act began with an apology to the uranium miners and offered compensation to miners for lung cancer and selected nonmalignant lung diseases, if selected criteria were met. Health care providers should be aware of the availability of this compensation for eligible persons.

THE BOTTOM LINE

Radon is a well-established, naturally occurring environmental carcinogen. The strongest and best-quantified human evidence comes from occupational studies of miners. Additional evidence comes from human studies of residential radon exposure and from animal and laboratory studies. The mechanism of cellular injury by alpha particles emitted by radon is well characterized. Based on extrapolation from occupational studies, it is estimated that radon exposure accounts for between 3,000 and 33,000 lung cancer deaths in the US each year, with central estimates of 15,400 or 21,800 (depending upon the model used).¹ This makes radon the second leading cause of lung cancer after cigarette smoking, although cigarettes account for far more cases than does radon. Most radon-induced cases of lung cancer occur in smokers, reflecting synergy between smoking and radon exposure, but the estimates of radon-caused lung cancer are also substantial.

Prevention of radon-induced lung cancer can be accomplished by reducing radon levels in homes and other buildings. It is estimated that approximately one third of radon-induced lung cancer could be avoided if homes with radon concentration exceeding 4 pCi/L, the EPA action level, could see a reduction in radon concentrations to below that level.¹ However, it is not technically feasible to eliminate all radon exposure. In addition, because of the importance of smoking in radon-induced lung cancer, smoking cessation is an essential part of the preventive approach.

FOR FURTHER INFORMATION:

One of the best sources of information on radon is the EPA's radon Web site, at www.epa.gov/iaq/radon. It includes links to several useful EPA publications, including *Radon: A Physician's Guide* (www.epa.gov/iaq/radon/pubs/physic.html) and *A Citizen's Guide to Radon: The Guide to Protecting Yourself and Your Family from Radon* (Third Edition) at www.epa.gov/iaq/radon/pubs/citguide.html.

The United States Geological Survey also has useful information about radon on its Web site. General background on radon entitled "Radon in Earth, Air, and Water" can be found at energy.cr.usgs.gov/radon/radonhome.html, and maps of radon exposure potential can be found at energy.cr.usgs.gov/radon/rnus.html.

Information on the Federal Radiation Exposure Compensation Program can be found at www.usdoj.gov/civil/torts/const/reca. CA

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