

Shelter and Indoor Air in the Twenty-First Century—Radon, Smoking, and Lung Cancer Risks

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Recognition that radon and its daughter products may accumulate to high levels in homes and in the workplace has led to concern about the potential lung cancer risk resulting from indoor domestic exposure. While such risks can be estimated with current dosimetric and epidemiological models for excess relative risks, it must be recognized that these models are based on data from occupational exposure and from underground miners' mortality experience. Several assumptions are required to apply risk estimates from an occupational setting to the indoor domestic environment. Analyses of the relevant data do not lead to a conclusive description of the interaction between radon daughters and cigarette smoking for the induction of lung cancer. The evidence compels the conclusion that indoor radon daughter exposure in homes represents a potential life-threatening public health hazard, particularly in males, and in cigarette smokers. Resolution of complex societal interactions will require public policy decisions involving the governmental, scientific, financial, and industrial sectors. These decisions impact the home, the workplace, and the marketplace, and they extend beyond the constraints of science. Risk identification, assessment, and management require scientific and engineering approaches to guide policy decisions to protect the public health. Mitigation and control procedures are only beginning to receive attention. Full acceptance for protection against what could prove to be a significant public health hazard in the twenty-first century will certainly involve policy decisions, not by scientists, but rather by men and women of government and law.

Introduction

This paper discusses the quality of the indoor air we breathe and how its condition may affect the public health as we enter the twenty-first century. My comments are confined to three problem areas: those of indoor radon, cigarette smoking, and lung cancer and how they may be interrelated. The paper only addresses the risks of lung cancer in exposed populations, how these have been estimated, and the assumptions and uncertainties both in the estimation process and in the assessment of the potential health hazards to the public. An overview is not attempted of radon and its decay products in indoor air, nor does the paper discuss sources and transport processes, the characteristics and behavior of radon decay products, or controlling indoor exposures. These comments deal solely with the basis for health concerns.

Radon and Its Progeny

The terrestrial radionuclide of increasing importance to public health is radon-222, a noble gas and a decay product of radium-226 in the uranium-238 series. This gas emanates from the soil and from building materials

of terrestrial origin, e.g., stone, bricks, and concrete. It seeps into homes and office buildings. When ventilation is restricted this gas may accumulate in concentrations substantially higher than those prevailing outdoors. In response to the recent need to conserve energy in the heating of homes and office buildings, construction methods that sharply restrict ventilation have been introduced. As a result, the control of radon levels in indoor air is becoming increasingly important.

As radon-222 and its progeny undergo radioactive decay, they emit alpha radiations. Deep within the soil, radon-222 concentrations can exceed 1000 pCi/L (37,000 Bq/m³). Outdoor concentrations of radon-222 vary considerably, but average about 0.2 pCi/L (7.4 Bq/m³) with much higher concentrations at ground level. In terms of concentration of radon progeny, an average value of 0.001 WL* is representative for an outdoor radon concentration of 0.1 pCi/L (3.7 Bq/m³). The major pathway for exposure of members of the general public is through exposure indoors, where on the average of 70 to 80% of the time is spent. Because closed structures do not allow for extensive mixing of air, the concentrations of radon in buildings tend to be higher than outdoor concentrations. Indoor levels are only moderately higher,

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*WL = working level; i.e., a unit of air concentration of potential alpha energy released from radon and its daughters.

averaging about 1.5 pCi/L (55 Bq/m³) and up to 8 pCi/L (300 Bq/m³) or more, when ventilation is not greatly restricted. These indoor radon concentrations can vary widely from the ambient air outdoor value to values that are a few thousand times higher. On the average, the level of indoor radon progeny is reported by the National Council on Radiation Protection and Measurements (NCRP) to be about 0.004 WL (0.4 pCi/L or 15 Bq/m³). In contrast, radon concentrations of 100 pCi/L (3700 Bq/m³) or more have been measured in some older homes and in recently constructed homes designed to limit ventilation as far as possible. These can be far greater than levels measured in many uranium mines.

The tissues at risk from exposure to radon and its progeny include the surfaces of the bronchi, segmental bronchioles, and alveolar membranes. These tissues are exposed primarily to radon daughters, e.g., polonium-218, which attach themselves to dust particles. When inhaled these deposit themselves within the respiratory system at locations influenced by particle size. The epithelium of alveoli receives an estimated dose equivalent of approximately 0.5 rem/y (5 mSv/yr) when radon concentrations in air are 1 pCi/L (37 Bq/m³). The dose equivalent to the segmental bronchioles may be approximately five times higher.

Thus, the important tissue is the bronchial epithelium, which is the site of most lung cancers thought to be induced by radiation. The major contributors to the alpha-radiation exposure are the short-lived decay products of radon, measurements that show an apparent log-normal distribution of concentrations in indoor air. For smokers, the additional exposure to the lungs from naturally occurring radionuclides in tobacco products increases the dose equivalent to the bronchial epithelium considerably.

Human Populations at Risk

Current scientific reports concentrate on the health outcomes due to exposure to radon and its progeny, primarily because of a need for a comprehensive characterization of the lung cancer risk associated with exposure to radon and its short-lived daughters in indoor domestic environments. Estimation of lung cancer risk appears to be best derived from epidemiological surveys of underground miners throughout the world who breathe widely differing levels of radon-222 progeny. Calculations based on dosimetric models of the respiratory tract are complex, and values are based largely on the location of the target cells in the bronchial epithelium, the physiological processes involved in the variable dosimetry, and uncertainties introduced by numerous confounding risk factors such as smoking. All of the epidemiological surveys are presently in progress; the human data on lung cancer induction by radon progeny are limited. No data are completed, and the person-years of follow-up are still relatively small, so that until a sufficient number of the study populations have died—most in the next century—the lifetime carcinogenic

risks of alpha-radiation exposure remain uncertain.

Permissible concentrations of radon progeny in air can be derived mathematically by calculating the concentrations in the tissues. The mathematical procedures are quite straightforward; it is the fundamental and physiological assumptions that have proved difficult. For these reasons the need for guidance on protection from the potential health hazards of radon and its daughter products is of current and future concern. For a considerable period, such guidance has been directed primarily to those occupationally exposed in the workplace, for example, uranium miners. Now, other groups of persons are being considered, and their circumstances differ from those occupationally exposed. We now include the general population, pregnant women, children, and persons who suffer from health conditions that might render them more sensitive to radiation injury. Furthermore, the biological assumptions, mathematical models, and radiation dosimetry are uncertain. Following deposition of the radionuclide within the body, the radiation exposure usually has a complex time pattern with varying distribution, and the actual dose and dose rate in the tissue is often inadequately known.

Estimation of Radon-induced Lung Cancer Risk

Numerous studies of underground miners exposed to radon daughters in the air of mines have shown an increased risk of lung cancer in comparison with non-exposed populations (1). Laboratory animals exposed to radon daughters also develop lung cancer. Abundant epidemiological and experimental data establish the carcinogenicity of radon progeny (1-6). These observations are of considerable importance because uranium, from which radon and its progeny arise, is ubiquitous in the earth's crust, and radon in indoor environments can reach relatively high levels. Nevertheless, while the carcinogenicity of radon daughters is established and the hazards of high levels of exposure during mining are well recognized, the risks of exposure to lower levels of radon progeny have not yet been adequately characterized. However, risk estimates of the health outcomes of lower levels of exposure are needed to address both the potential health effects of radon and radon daughters in homes and to determine acceptable levels of exposure in occupational environments.

Two approaches are currently used to characterize the lung cancer risks of radon-daughter exposure. The first is the use of mathematical representations of the respiratory tract that model radiation doses to target cells. The second is the use of epidemiological investigations of exposed populations, mainly underground miners. The dosimetric approach provides estimates of lung cancer risks of radon-daughter exposure that are based specifically on modeling the radon-daughter dose to target cells. A number of different dosimetric models have thus far been developed; all require certain relevant assumptions. Some are not subject to direct verification con-

cerning the deposition of radon daughters in the respiratory tract and the type, nature, and location of the target cells for cancer induction. Because of these assumptions, the uncertainties, and the technical difficulties encountered in this approach, it appears prudent not to use dosimetric models solely for calculating the lung cancer risk estimates. However, the dosimetric approach is of considerable value, and the results of such dose-effect models are used to extrapolate lung cancer risk coefficients derived from the epidemiological studies of occupational exposure of the underground miners to the general population in indoor domestic environments.

The use of available epidemiological data has advantages because the studies of radon-daughter exposed miners provide a direct assessment of human health effects. While each investigation has limitations, the approach of a combined analysis of the major data sets permits a comprehensive assessment of the lung cancer risks of radon-daughter exposure and of factors influencing the risk of exposure. In analyzing original data sets, a descriptive analytical approach may be used rather than statistical methods based on conceptual models of carcinogenesis or radiation (dose-response) effects. In the current 1988 BEIR IV Report (1), the National Research Council Committee obtained primary data sets from four of the principal and most complete epidemiological studies of radon-exposed underground miners (the Ontario uranium miners, the Saskatchewan uranium miners at Beaverlodge, the Swedish iron miners at Malmberg, and the Colorado Plateau uranium miners) and developed risk models for lung cancer that were derived from its own formal statistical analyses.

The follow-up experience of the groups analyzed totals about 500,000 person-years at risk and includes 459 lung cancer deaths. There are important differences among the four studies including the duration of follow-up, the exposure rate, and the degree of uncertainty and potential biases in the estimated exposures. These factors were evaluated extensively and were examined to the possible extent in the epidemiological analysis.

Using statistical regression techniques appropriate for survival time data, the risk or probability of dying of lung cancer because of radon-daughter exposure in the combined cohorts and in the absence of smoking may be best described by a complex time-since-exposure statistical model. In this time-since-exposure relative risk model, although simple in its mathematical formulation, the excess relative risk varies with time since exposure rather than remaining constant and depends on age at risk. This expression, therefore, is a departure from most previous risk models that have assumed that the relative risk is constant over both age and time. Radon exposures more distant in time have a somewhat lesser impact on the age-specific excess relative risk than more recent exposures. Moreover, the age-specific excess relative risk is higher for young persons and declines with older ages. The relative risk form provides a simpler description of observed lung cancer risks in the

miner cohorts; it requires fewer variables than would an absolute risk form.

Recognition that radon and its daughter products may accumulate to high levels in homes has led to concern about the potential lung cancer risk resulting from exposure to radon progeny in indoor domestic environments. While such risks can be estimated with the derived mathematical expression for excess relative risks, it must be recognized that the epidemiological model is based on data from occupational exposure of underground miners. Several assumptions are required to transfer risk estimates from an occupational setting to the indoor domestic environment: *a*) that the epidemiological findings in the underground miners could be extended across the entire lifespan, *b*) that cigarette smoking and exposure to radon daughters interact multiplicatively, *c*) that exposure to radon progeny increases the risk of lung cancer proportionally to the sex-specific ambient risk of lung cancer due to other causes, and *d*) that a unit of radon-daughter exposure yields an equivalent radiation dose to the respiratory tract and to the bronchial epithelium in both occupational and domestic environmental settings. It was concluded that additional data on ventilation rates and aerosol characteristics in mines and homes are needed to address quantitatively the comparative dosimetry of radon daughters in the occupational and domestic environmental settings.

Based on the estimates of excess relative risks per WLM* (the unit of exposure to radon progeny that is derived from analysis of the four miner cohorts examined) and the assumptions, it is possible to project well into the twenty-first century, the lung cancer risks, lifetime risks, risk ratios, average lifespans, and average years of life lost for U.S. males and females for various exposure rates and durations of exposure, and estimated risks conditional on survival to a particular age and for smokers and nonsmokers of either sex.

These risk projections cover exposure situations of current public health concern. Lifetime exposure to 1 WLM/yr is estimated to increase the number of deaths due to lung cancer by a factor of about 1.5 over the current rate for both males and females in a population having the current prevalence of cigarette smoking. Occupational exposure to 4 WLM/yr from ages 20 to 40 is projected to increase lung cancer deaths in males by a factor of 1.6 over the current rate of this age cohort in the general population. In all of these cases, most of the increased risk occurs to smokers, for whom the risk is up to ten times greater than for nonsmokers.

Comparisons of estimates of the lifetime risk of lung cancer mortality due to a lifetime exposure to radon progeny in terms of WLM and alpha-particle dose to the target cells of the bronchial epithelium, made by this and other scientific committees over the past decade, yield similar lung cancer risk coefficients (Table 1). It must be

*WLM = working level month; i.e., a unit of exposure to air concentrations of potential alpha energy released from radon daughters.

Table 1. Estimates of lung cancer risk due to exposure to radon progeny.

Study	Excess lung cancer deaths/10 ⁶ person-WLM	Reference
1988 BEIR IV National Academy of Sciences	350	(1)
1987 International Commission on Radiological Protection	180-230	(2)
1984 National Council on Radiation Protection and Measurements	130	(3)
1981 International Commission on Radiological Protection	150-450	(4)
1980 BEIR III National Academy of Sciences	730	(5)
1977 United Nations Scientific Committee on the Effects of Atomic Radiation	200-450	(6)

remembered, however, that in each of the six studies, the epidemiological data available, the dosimetric and statistical models applied, and the assumptions introduced, were quite different and with differing and alternative methods of analysis. Nevertheless, the excess lung cancer deaths per million person year WLM range within a factor of about two at most.

The uncertainties that affect the estimates of the lung cancer risk include *a*) random and possibly systematic errors in the original data on exposure and lung cancer in the miner populations analyzed, *b*) inappropriate statistical models for analysis or incorrect specification of the components of the models, *c*) sampling variation, and *d*) incorrect description of the interaction between radon-daughter exposure and cigarette smoking. In addition, the actual computed lifetime lung cancer risks and expected years of life-shortening depend on the age-specific disease rates of the referent population, here, the 1980 to 1984 U.S. population mortality rates. Projections based on a different referent population would be expected to differ, although the ratio of lifetime risks and years of life lost to ambient values may be more stable across populations.

Review of the literature and analyses of the relevant data do not lead to a conclusive description of the interaction between exposure to radon daughters and cigarette smoking. Several data sets have been analyzed and suggest a multiplicative interaction for risk projections on a relative risk scale. A submultiplicative model is also consistent with the data analyzed. Neither additive nor subadditive models appear consistent with these data.

Ongoing research in the U.S. and other countries has provided data on concentrations of radon and radon progeny in homes. These studies have also described the

sources of radon and determinants of its concentration. There appears to be a log-normal distribution with very wide variation of levels of radon and radon progeny in U.S. homes(7), with an average of about 1.5 pCi/L (55 Bq/m³); about 2% of homes exceed levels of 8 pCi/L (300 Bq/m³), much greater than permissible levels of 4 pCi/L (150 Bq/m³) in mines recommended by the U.S. Environmental Protection Agency. A few epidemiological investigations of the lung cancer risk associated with radon-daughter exposure in homes have been carried out, but the study populations have been small and the results remain inconclusive. These studies are, at present, inadequate for the purposes of risk estimation. For this reason, the lung cancer risk projections for the general population can only be based on the epidemiological studies of miners. Estimates of lung cancer risks from studies on miners can be used to estimate the potential lung cancer risk from elevated levels of indoor radon. However, the estimates derived are uncertain, particularly since differences between mining and indoor domestic environments and the interaction between smoking and exposure to radon progeny remain incompletely resolved.

Comparisons of Lung Cancer Rates

To provide some perspective of the lung cancer risk due to radon exposure, comparisons might be made with the expected risk in the U.S. An estimated 130,000 lung cancer deaths occurred in 1986; 89,000 in males and 44,000 in females. About one death in twenty is due to lung cancer, a lifetime risk of 5%. It has been estimated that cigarette smoking is responsible for 85% of lung cancers among men and 75% among women, some 83% overall. The lifetime risk of lung cancer for nonsmokers is somewhat less than 1%. Even for the nonsmoker, passive smoking may contribute to this 1% or less; it has been estimated that passive smoking may be a contributor to this 1% in U.S. nonsmokers. On average, a smoker's risk is about 10 times that of a nonsmoker.

However, the role of smoking as a confounding factor is still not clear from analyses of the underground miner data, and the effect of smoking on radon risk depends strongly on the type of interaction, whether additive or multiplicative. Accordingly, it is very difficult to determine the precise risk of exposure to indoor radon progeny to the general public in the presence of the more proven causative agent, cigarette smoking. Based on NCRP modeling and risk estimates, the annual number of lung cancer deaths attributable to an average indoor air radon exposure of 0.004 WL in a continuously exposed population of 240 million is about 7000/year, but could be as high as 10,000/year. Based on the BEIR IV modeling and risk estimates, the lung cancer deaths attributable to radon progeny exposure are calculated to be higher. In both estimates, the excess deaths are in both smokers and nonsmokers and include exposure to passive smoke.

A satisfactory method of treating the confoundin

factor of smoking in lung cancer risk assessment and in establishing levels for protecting the health of the public has not as yet been developed. Thus, the precise radon-induced lung cancer risk in the nonsmoker is uncertain, and the overall effectiveness of mitigating and protective measures remain in doubt. Based on the available information, however, the radon risk to the nonsmoker appears to be much less than has been presently estimated. Protective measures are likely to be most effective in reducing radon risk to smokers who are already at very high risk.

Radon-related Problems Evolving in the Twenty-First Century

Radon-related research undertaken during the past decade has provided much of our current information on radon concentrations in homes and in the workplace, factors that influence indoor concentrations and the risk of lung cancer associated with indoor exposures. The results indicate the need for further health-related investigations. Discoveries of exceptionally high indoor radon concentrations in the eastern U.S. also raised the level of public concern. The increased scientific and public interest resulted in the initiation of more comprehensive research programs in order to respond to the public health concerns and provide a stronger basis for formulating national policy decisions.

Only a small number of homes and occupational environments have been surveyed to characterize the vector aerosols in indoor air to which radon progeny attach. Better knowledge of the types of aerosols in homes and in office buildings is required before predictive models of radon progeny behavior can be formulated. It is likely that the vector aerosols are influenced by seasonal variations, geographic location, presence of cigarette smokers, and home and office ventilation patterns.

New health-related research is also needed to compare the deposition patterns of inhaled radon progeny for adult men in mine atmospheres with those for male and female adults and children in homes. Such studies can then be applied to determine the influence of aerosol parameters, body size, and breathing patterns for the purpose of extrapolating exposure-dose-risk relationships obtained for underground miners to evaluate exposures to the general population.

While respiratory tract cancer risk from exposure to radon progeny in homes may be estimated from the epidemiological studies of underground miners, these estimates are uncertain for several reasons. There are important differences between miners and the general public: *a*) the uranium miners only include men with a limited range of ages; *b*) the miners were exposed in an occupational setting that included a variety of toxic airborne pollutants; *c*) miner populations have a higher percentage of cigarette smokers than the general population; *d*) nonmalignant respiratory disease is more prevalent among miners; and *e*) miners perform heaviest manual labor. Also, in dusty mine atmospheres, a larger

fraction of the radon progeny is attached to particles. Because the fraction of radon progeny attached to particles is probably lower in most indoor atmospheres in homes and office buildings compared to mines, this may result in higher doses to miners for any given exposure level.

Information on radiation doses to underground miners and potential confounding factors such as cigarette smoking is incomplete and its validity questionable. Because the miner population is not typical of the public, carefully planned epidemiological studies must be carried out to provide more information on the potential confounding factors, particularly smoking. Comprehensive mathematical models must be developed to relate levels of exposure, the radiation dose to bronchial target cells, and respiratory tract cancer in order to assess the carcinogenic risk associated with indoor radon. The health-related risk models will likely be based on the results of epidemiological studies of underground miners, but they must properly account for differences in exposure conditions, age at exposure, cumulative exposure levels, and cigarette smoking. This will influence evolving construction methods, environmental health strategies, and national policy decisions affecting air, shelter, and occupational standards for air quality in domestic and workplace settings.

Conclusions

The present need to apply lung cancer risk projections from surveys of underground miners to estimate risk to the general population from indoor radon introduces numerous uncertainties and technical difficulties. The domestic environment has not, as yet, been characterized adequately in terms of the variables affecting the dose and risk from radon progeny. Variations in indoor radon levels, alterations of aerosol characteristics, and the impact of active and passive smoking and non-smoking risk factors suggest that health consequences resulting from indoor radon exposures require much more study. There is some wisdom in recommending continuation of epidemiological studies of lung cancer resulting from indoor radon exposure and underground mining surveys, provided such studies have sufficient statistical power to quantify any significant differences between the risks in the domestic environmental and occupational settings. This will permit us to assess the magnitude of the potential lung cancer risk to the general public from exposure to radon progeny in indoor domestic environments, and thereby help place into perspective the potential ill-effects of this environmental hazard with those pernicious diseases afflicting our nation's health in the twenty-first century.

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REFERENCES

1. National Academy of Sciences. *The Health Effects of Radon and Other Internally-Deposited Alpha Emitters. BEIR IV*, National Academy Press, Washington, DC, 1988.
2. International Commission on Radiological Protection. *Lung Cancer Risk from Indoor Exposure to Radon Daughters*. ICRP Publication 50, Pergamon Press, New York, 1987.
3. National Council on Radiation Protection and Measurements. *Evaluation of Occupational and Environmental Exposures to Radon and Radon Daughters*. NCRP Report 78, National Council on Radiation Protection and Measurements, Bethesda, MD, 1984.
4. International Commission on Radiological Protection. *Limits for Intake of Radionuclides by Workers*. ICRP Publication 30, Pergamon Press, New York, 1979.
5. National Academy of Sciences. *The Effect on Populations of Exposure to Low Levels of Ionizing Radiation*. National Academy Press, Washington, DC, 1980.
6. United Nations Scientific Committee on the Effects of Atomic Radiation. *Sources and Effects of Ionizing Radiation*. United Nations, New York, 1977.
7. Nero, A. V., Schwehr, M. B., Nazaroff, W. W., and Revzan, K. L. Distribution of airborne radon-222 concentrations in U.S. homes. *Science* 234: 992-997 (1988).