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High-LET Alpha-Emitters: Radon, Lung Cancer and Smoking

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HIGH-LET ALPHA-EMITTERS: RADON, LUNG CANCER AND SMOKING ^{1,2}

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- ³ Professor of Radiology, University of California, Berkeley and San Francisco
- ⁴ Dr. Fabrikant was Chairman of the BEIR IV Committee, i.e., the Committee on the Biological Effects of Ionizing Radiation of the National Academy of Sciences-National Research Council. The Committee authored the BEIR IV Report, "Health Effects of Radon and Other Internally Deposited Alpha-Emitters: BEIR IV" (1).

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ABSTRACT

The National Academy of Sciences BEIR IV Report deals with the health effects in human populations exposed to internally-deposited alpha-emitting radionuclides and their decay products. Quantitative risk estimates for cancer induction are derived, mainly from analyses of epidemiological data. The Report addresses the health outcomes of exposure to radon and its daughters, primarily lung cancer risks of worker exposure to radon progeny in underground mines and in the general public in indoor domestic environments. An excess relative risk model of lung cancer mortality and exposure to radon progeny is developed; this models the excess risk per Working Level Month in terms of time intervals prior to an attained age, and is dependent on time-since-exposure and age at risk. Risk projections are presented and cover exposure situations₁ of current public health concern. For example, lifetime exposure to 1 WLM y^{-1} 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 having₁ the current prevalence of cigarette-smoking. Occupational exposure to 4 WLM y^{-1} from ages 20 y to 40 y is projected to increase lung cancer deaths 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 non-smokers.

INTRODUCTION

The BEIR-IV Report (1) addresses demonstrated and potential health effects in human populations exposed to internally deposited alpha-emitting radionuclides and their decay products. Emphasis has been placed primarily on the carcinogenic effects in humans, and where possible, quantitative risk estimates for cancer induction are presented. The largest part of our National Academy of Sciences report concentrates 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.

Alpha-emitting radionuclides and their daughter products may be absorbed into the tissues of the body following inhalation or ingestion, through wounds in the skin, or after injections and instillations for diagnostic or therapeutic medical applications, and irradiate adjacent cells, tissues and organs. Laboratory animal experiments and human studies demonstrate that

radiation effects depend not only on the physical properties of the emitted radiations, but also on the physical and chemical characteristics of the radionuclides, which control their deposition, transport, metabolism, excretion, and reutilization within the body. Radiation health effects in humans include cancer induction, genetic disease, teratogenesis (induction of developmental abnormalities), and degenerative changes.

The BEIR IV Report (1) attempts to respond to a broad range of scientific questions that impact current public health issues; not all of these questions can be addressed adequately with currently available information. There is considerable variation in the amount of data from epidemiological studies of human populations and from experimental animal investigations that are available. For certain of the alpha-emitting radionuclides, such as radon and its daughters, some epidemiological data are available. However, dependence must also be placed on extensive experiments with animals. As in all experimental studies with animals, the extent to which the information and conclusions can be extrapolated to humans and the confidence that can be placed on such an extrapolation remain uncertain. Where human data are available, wherever possible we relied on our own analyses using current epidemiological methods to address these problems rather than relying solely on published information. We also explored statistical methods for analysis of interspecies comparisons of the risks from different radionuclides where useful animal data are available and the human data are limited. The Committee recognized the uncertainties inherent in such animal-to-human extrapolation; nevertheless, we believe the methodology introduced helps provide for more detailed comparison as additional data from epidemiological and animal studies become available.

RADON IN THE ENVIRONMENT

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 and, when ventilation is restricted, 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.

Deep within the soil, radon-222 concentrations can exceed 1000 pCi L^{-1} ($37,000 \text{ Bq m}^{-3}$). Outdoor concentrations of radon-222 vary considerably, but average about 0.2 pCi L^{-1} (7.4 Bq m^{-3}) with much higher concentrations at ground level. In terms of concentrations of radon progeny, an average value of 0.001 WL is representative for an outdoor radon concentration of 0.1 pCi L^{-1} (3.7 Bq m^{-3}). The major pathway of exposure of members of the general public is through exposure indoors, where on the average 70-80 percent 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, averaging about 1.5 pCi L^{-1} (55 Bq m^{-3}) and up to 8 pCi L^{-1} 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 in the United States is reported by the NCRP to be about 0.004 WL (0.4 pCi L^{-1} or 15 Bq m^{-3}). In contrast,

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 and, when inhaled, deposit themselves within the respiratory system at locations influenced by particle size. The epithelium of alveoli received an estimated dose equivalent of approximately 0.5 rem y^{-1} (5 mSv y^{-1}) when radon concentrations in air are 1 pCi L^{-1} (37 Bq m^{-3}). The dose equivalent to the segmental bronchioles may be approximately 5 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 contributions to the alpha-radiation exposure are the short-lived decay products of radon, measurements of which show an apparent log-normal distribution of concentrations in indoor air, based on surveys in homes and office buildings. For smokers, the additional exposure to the lungs from dose equivalent to the bronchial epithelium considerably. This becomes particularly important with the introduction of improved techniques and ventilation control for conservation of heat in domestic and occupational environments.

HUMAN POPULATIONS AT RISK

Current scientific reports concentrate on health outcomes due to exposure to radon and its progeny, primarily because of a need for a comprehensive characterization of 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 cell in the bronchial epithelium, the physiological processes involved in the variable dosimetry, and uncertainties introduced by numerous confounding risk factors, such as smoking.

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 proven difficult. It is for these reasons that 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. 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 adequately known.

The evaluation of the lung cancer risk from radon and its progeny was the most

challenging task of the Committee. 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 nonexposed populations. Laboratory animals exposed to radon daughters also develop lung cancer. There is abundant epidemiological and experimental data to establish the carcinogenicity of radon progeny. 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 is well recognized, the risks of exposure to lower levels of radon progeny have not yet been precisely characterized. However, risk estimates of the health effects of lower levels of exposure are needed to address the potential health effects of radon and radon daughters in homes and to determine acceptable levels of exposure in occupational environments.

EPIDEMIOLOGICAL STUDIES OF UNDERGROUND MINERS

Two approaches are currently being used to characterize the lung cancer risks of radon daughter exposure: mathematical representations of the respiratory tract that model radiation doses to target cells and epidemiological investigation of exposed populations, mainly underground miners. The dosimetric approach used by other investigators and committees provides an estimate of lung cancer risk of radon daughter exposure that is based specifically on modelling the dose to target cells. A number of different dosimetric models have thus far been developed; all require certain relevant assumptions, some not subject to direct verification, concerning the deposition of radon daughters in the respiratory tract and the type, nature and location of the target cells for cancer induction. Accordingly, our committee chose not to use dosimetric models for calculating the lung cancer risk estimates in this Report. The results of such dose-effect models were 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 environments. However, the lung cancer risk estimates for radon daughter exposure derived by the BEIR IV Committee in its Report (1) are based solely on the epidemiological evidence.

We turned to the available epidemiological data because the studies of miners exposed to radon daughters provided a direct assessment of human health effects. While each of the investigations has limitations, the approach of a combined analysis of major data sets permitted a comprehensive assessment of the health risks of radon daughter exposure and of factors influencing the risk of exposure. In analyzing the data, the Committee used a descriptive analytical approach rather than using statistical methods based on conceptual models of carcinogenesis or radiation (dose-reponse) effects.

Data were obtained from four of the principal studies of radon-exposed miners (the Ontario uranium miners, the Saskatchewan uranium miners, the Swedish metal miners, and the Colorado Plateau uranium miners) and developed risk models for lung cancer from our own analyses. The follow-up experience of the groups analyzed total about 500,000 person-years and includes 459 lung cancer deaths. There are important differences among the four studies

including the duration and person-years of follow-up, the exposure rate, and average duration of exposures. These factors were evaluated extensively and were examined to the extent possible in the epidemiological analyses.

TIME-SINCE-EXPOSURE MODEL

The analyses indicated that the age at risk and the time since cessation of exposure were significant factors modifying the excess relative risk of lung cancer mortality. The Committee's epidemiologists and statisticians developed a time-since-exposure model, which modelled the excess risk per WLM in terms of time intervals prior to an attained age. This model was tested by analyzing the data for each of the underground miner cohorts separately. These analyses indicated that common values of the factors for age at risk and time since exposure could be applied to all four miner cohorts and that these factors operated largely independent of one another. As a final step, the combined data were re-analyzed with these values to obtain a maximum likelihood estimate of the excess lung cancer risk per WLM: $r(a) = r_0(a)(1 + 0.025)(a)[W_1 + 0.5 W_2]$ where $r(a)$ is total risk of lung cancer at age a , and $r_0(a)$ is the baseline lung cancer risk which varies with sex, smoking status, and calendar period. The age factor (a) is 1.2 for ages <55 y, 1.0 for ages 55-64 y, and 0.4 for ages 65 y or more. W_1 is the cumulative WLM incurred between 5 and 15 y prior to age a and W_2 is the WLM incurred 15 y or more before age a . The diminishing risk of lung cancer mortality after age 64 y and the discounting of exposures occurring more than 14 y before age a results in considerably smaller estimates of lifetime risk than a constant relative risk model fitted to the same data.

Thus, using statistical regression techniques appropriate for survival time data, the risk or probability of dying or lung cancer due to 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 relative risk model, although simple in its mathematical formulation, the excess relative risk after a 5 y lag period 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 which 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 is higher for younger persons and declines at older ages. Our analysis did not assume a priori that analysis based on the relative risk was necessarily more appropriate than alternatives, such as the absolute risk. However, an absolute risk model would have involved a complex power function of age. 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.

LUNG CANCER RISKS

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 indoor domestic exposure. While such risks can be estimated with the current BEIR IV model for excess relative risks, it must be recognized that our risk estimates from an occupational setting to the indoor domestic environment. Accordingly, we assumed (1) that the epidemiological findings in the underground miners could be extended across the entire lifespan, (2) that cigarette smoking and exposure to radon daughters interact multiplicatively,

(3) that exposure to radon progeny increases the risk of lung cancer proportionally to the sex-specific baseline risk of lung cancer, and (4) that exposure to a WLM yields an equivalent dose to the respiratory tract and to the bronchial epithelium in both occupational and environmental settings. This last assumption was a qualitative decision by the members of the Committee. We 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 environmental settings.

Based on the estimates of excess relative risks per WLM of exposure to radon progeny derived from analysis of the four miner cohorts, and the assumptions outlined, the Committee projected lung cancer risks for United States males and females. The BEIR IV risk projections estimate lifetime risks, ratios of lifetime risk, average lifespans, and average years of life lost for various exposure rates and durations of exposure. Tables are provided in the Report for estimating risks conditional on survival and exposure to a particular age and for smokers and nonsmokers of either sex. For projecting lifetime effects due to radon exposure, the Committee used the 1980-1984 United States mortality rates as referent rates, and a 5-y lag period.

These risk projections cover exposure situations of current public health concern. Lifetime exposure to 1 WLM y^{-1} 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 y^{-1} from ages 20 y to 40 y 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 non-smokers.

COMPARISONS OF LIFETIME RISK OF LUNG CANCER MORTALITY

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 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. Although our Report (1) uses much information not available for the earlier reports, the differences reflect mainly differences in assumptions made and the models used by the various --- our Committee developed risk models for lung cancer mortality from its own analyses of the epidemiologic studies. Nevertheless, the excess lung cancer deaths per million person year WLM range within a factor of about 2 most.

Table 1

LIFETIME RISK OF LUNG-CANCER DUE TO LIFETIME EXPOSURE TO RADON PROGENY

Study			Deaths/10 ⁶ Person-WLM
1988	BEIR IV	(1)	350
1987	ICRP	(2)	180-230
1984	NCRP	(3)	130
1983	ICRP	(4)	150-450
1980	BEIR III	(5)	730
1977	UNSCEAR	(6)	200-450

Our committee's modified relative risk model differs from the others in that it incorporates a dependence of the relative risk of lung cancer mortality on both time since exposure and age at risk. This model was derived from a formal statistical analysis of primary data from four of the most complete epidemiological studies of underground miners.

UNCERTAINTIES

The uncertainties that affect the estimates of the lung cancer risk due to exposure to radon progeny given in our Report (1) must be considered by users. These uncertainties include (1) random and possibly systematic errors in the cigarette-smoking. In addition, (5) the actual computed lifetime risk and expected life shortening depend on the age-specific disease rates of the referen population, in our examples the 1980-84 United States 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.

SMOKING

Review of the literature and the our own analyses of the relevant data did not lead to a conclusive description of the interaction between radon daughters and cigarette smoking for the induction of lung cancer. Several data sets were analyzed, and while we chose a multiplicative interaction on a relative risk scale for its risk projections, it recognizes that a submultiplicative model is also consistent with the data analyzed. However, neither additive nor subadditive models appear consistent with the available data.

INDOOR RADON STUDIES

A few exploratory 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 inconclusive. The Committee judged these exploratory studies to be inadequate for the purposes of risk estimation. For this reason, our risk projections for the general population are based on the studies of miners. We concluded that 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. We has provided estimates of the effects of indoor exposure to radon in homes while recognizing that differences between mining and domestic environments and the interaction between smoking and exposure to radon progeny remain incompletely resolved.

GAPS IN INFORMATION

In our review and analysis, we found gaps in information related to some aspects of radiation carcinogenesis by radon daughters. The cells of the respiratory tract that give rise to radon-daughter-associated lung cancer are still not known. A unique link between radon daughter exposure and small cell carcinoma of the lung was not found; in the studies of underground miners, this histological type occurred in greatest excess, but other cancer cell types were also increased.

OTHER HEALTH OUTCOMES

Health effects of exposure to radon daughters other than respiratory cancer are also of concern, but the data are sparse and associations are weak. Reductions in lung function in some uranium miners cannot be attributed directly to radon daughter exposure. The data on increased occurrence of chromosomal aberrations in lymphocytes and on adverse reproductive outcomes in uranium miners are inconclusive.

RADON AND RADON PROGENY IN HOMES

Ongoing research in the United States and other countries has provided some 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, with an average of about 1.5 pCi L^{-1} (55 Bq m^{-3}) (7); about 2 percent of homes exceed levels of 8 pCi L^{-1} (300 Bq m^{-3}), much greater than permissible levels of 4 pCi L^{-1} (150 Bq m^{-3}) in mines recommended by the United States 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 estimations 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

To provide some perspective of the lung cancer risk due to radon exposure

comparisons might be made with the expected risk in the United States. An estimated 130,000 lung cancer deaths occurred in 1986; 89,000 in males and 44,000 in females. About one death in 20 is due to lung cancer, a lifetime risk of 5 per cent. It has been estimated that cigarette smoking is responsible for 85 per cent of lung cancer among men and 75 per cent among women, some 83 per cent overall. The lifetime risk of lung cancer for non-smokers is somewhat less than 1 per cent. Even for the non-smoker, passive smoking may contribute to this one per cent or less; it has been estimated that passive smoking may be a contributor to this one per cent in U.S. non-smokers. On average, a smoker's risk is about ten 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 modelling 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 y^{-1} , but could be as high as $10,000 \text{ y}^{-1}$. Based on the BEIR IV (1) modelling 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 non-smokers, and include exposure to passive smokers.

HOME RADON EXPOSURES AND LUNG CANCER RISK

Nero et al (7) have presented a systematic appraisal of data from several surveys of home radon exposure in the United States. Their work suggests that the distribution of radon exposure rates in homes follows approximately a log-normal distribution with geometric mean (GM) of 0.96 pCi/L (35.5 Bq m^{-3}) and geometric standard deviation (GSD) 2.84 pCi L^{-1} (105.1 Bq m^{-3}). This estimate was based on 22 sets of data from surveys which were generally carried out to ascertain potential increases in radon exposure resulting from energy conservation procedures. The shape of the distribution and the estimates of its parameters are at best only an approximation of the true levels of radon found in American homes. In addition, homes that were selected for measurement were provided by volunteers, so that the overall sample may be weighted towards housing of the middle and higher socioeconomic groups where energy conservation awareness is relatively higher estimated exposures.

The risk of radon-induced lung cancer among residents of single family homes in the United States (approximately 70 per cent of the housing stock) has now been estimated by Lubin and Boice (8) using the time-since-exposure models recently developed by the BEIR IV (1) Committee. These models, based on extrapolation to lower radon exposure levels from exposure-response relationships observed at higher doses among radon-exposed miners, predicted that approximately 14 per cent of lung cancer deaths among such home-residents (about 13,300 deaths per year, or 10 per cent of all U.S. lung cancer deaths) may be due to indoor radon exposure. The 95 per cent confidence interval is 7-25 per cent or approximately 6,600 to 24,000 lung cancer deaths. The attributable risks, defined as the excess lung cancer rate in the United States in a population due to exposure to radon progeny as a fraction of the lung cancer rate, are similar for males and females and for smokers and nonsmokers. However, higher baseline risks of lung cancer result in much larger numbers of

radon attributable cancer deaths among males (approximately 9,000) and among smokers (approximately 11,000). Since the radon concentration in single family dwellings appears to be log-normally distributed, because of the apparent skewness of the exposure distribution, most of the contribution to the attributable risks arises from exposure rates which exceed 4 pCi L^{-1} (150 Bq m^{-3}) (approximately 8 per cent of homes) were estimated, the models predict that the total annual lung cancer burden in the U.S. would decrease by 4-5 per cent, or by about 3,800 lung cancer deaths, in contrast to a maximum reduction of 14 per cent if all indoor radon exposure were eliminated.

A satisfactory method of treating the confounding 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 the protective measures remains 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.

CONCLUSIONS

As in the case of the previous BEIR Committee (5), the BEIR V Committee cautioned that the risk estimates derived from the epidemiological and the experimental animal data should not be considered as precise numerical values. All were derived from analyses of incomplete data and involved numerous uncertainties. It is expected that these risk estimates will change as new information and methods for analysis become available. And lastly, the Committee assumed no responsibility to recommend regulatory limits, or to address cost-benefit issues involving the radionuclides of concern. Such issues were beyond the scope of the task and the expertise of the Committee.

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 impacts of active and passive smoking and nonsmoking risk factors suggest that health consequences resulting from indoor radon exposure and underground mining surveys provided such studies has sufficient statistical power to qualify 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 radon exposure as an environmental hazard with those pernicious diseases afflicting the nation's health now and into the twenty-first century.

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