Free Executive Summary

Health Effects of Exposure to Radon: BEIR VI

Committee on Health Risks of Exposure to Radon (BEIR VI), National Research Council

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Radon progeny--the decay products of radon gas--are a well-recognized cause of lung cancer in miners working underground. When radon was found to be a ubiquitous indoor air pollutant, however, it raised a more widespread alarm for public health. To develop appropriate public policy for indoor radon, decisionmakers need a characterization of the risk of radon exposure across the range of exposures people actually receive. In response, the BEIR VI committee has developed a mathematical model for the lung cancer risk associated with radon, incorporating the latest information from epidemiology and scientific studies.In this book the committee provides a fresh assessment of exposure-dose relationships. The volume discusses key issues--such as the weight of biological evidence and extrapolation from radon-exposed miners to the larger population--in estimating the risk posed by indoor radon. It also addresses such uncertainties as the combined effects of smoking and radon and the impact of the rate of exposure.The committee considered the entire body of evidence on radon and lung cancer, integrating findings from epidemiological studies with evidence from animal experiments and other lines of laboratory investigation. The conclusions will be important to policymakers and environmental advocates, while the technical findings will be of interest to environmental scientists and engineers.

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Public Summary:
The Health Effects of
Exposure to Indoor Radon

Radon is a naturally occurring gas that seeps out of rocks and soil. Radon comes from uranium that has been in the ground since the time the earth was formed, and the rate of radon seepage is variable, partly because the amounts of uranium in the soil vary considerably. Radon flows from the soil into outdoor air and also into the air in homes from the movement of gases in the soil beneath homes. Outside air typically contains very low levels of radon, but it builds up to higher concentrations indoors when it is unable to disperse. Some underground mines, especially uranium mines, contain much higher levels of radon.

Although radon is chemically inert and electrically uncharged, it is radioactive, which means that radon atoms in the air can spontaneously decay, or change to other atoms. When the resulting atoms, called radon progeny, are formed, they are electrically charged and can attach themselves to tiny dust particles in indoor air. These dust particles can easily be inhaled into the lung and can adhere to the lining of the lung. The deposited atoms decay, or change, by emitting a type of radiation called alpha radiation, which has the potential to damage cells in the lung. Alpha radiations can disrupt DNA of these lung cells. This DNA damage has the potential to be one step in a chain of events that can lead to cancer. Alpha radiations travel only extremely short distances in the body. Thus, alpha radiations from decay of radon progeny in the lungs cannot reach cells in any other organs, so it is likely that lung cancer is the only potentially important cancer hazard posed by radon in indoor air.

For a century, it has been known that some underground miners suffered from higher rates of lung cancer than the general population. In recent decades, a growing body of evidence has causally linked their lung cancers to exposure to

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high levels of radon and also to cigarette-smoking. The connection between radon and lung cancer in miners has raised concern that radon in homes might be causing lung cancer in the general population, although the radon levels in most homes are much lower than in most mines. The National Research Council study, which has been carried out by the sixth Committee on Biological Effects of Ionizing Radiation (BEIR VI), has used the most recent information available to estimate the risks posed by exposure to radon in homes.

The most direct way to assess the risks posed by radon in homes is to measure radon exposures among people who have lung cancer and compare them with exposures among people who have not developed lung cancer. Several such studies have been completed, and several are under way. The studies have not produced a definitive answer, primarily because the risk is likely to be very small at the low exposure encountered from most homes and because it is difficult to estimate radon exposures that people have received over their lifetimes. In addition, it is clear that far more lung cancers are caused by smoking than are caused by radon.

Since a valid risk estimate could not be derived only from the results of studies in homes, the BEIR VI committee chose to use the lung-cancer information from studies of miners, who are more heavily exposed to radon, to estimate the risks posed by radon exposures in homes. In particular, the committee has drawn on 11 major studies of underground miners, which together involved about 68,000 men, of whom 2,700 have died from lung cancer. The committee statistically analyzed the data to describe how risk of death from lung cancer depended on exposure. In this way, the committee derived two models for lung-cancer risk from radon exposure.

In converting radon risks from mines to homes, the committee was faced with several problems. First, most miners received radon exposures that were, on the average, many times larger than those of people in most homes; people in a few homes actually receive radon exposures similar to those of some miners. It was necessary for the committee to estimate the risks posed by exposures to radon in homes on the basis of observed lung-cancer deaths caused by higher exposures in mines. The committee agreed with several earlier groups of experts that the risk of developing lung cancer increases linearly as the exposure increases; for example, doubling the exposure doubles the risk, and halving the exposure halves the risk. Furthermore, the existing biologic evidence suggests that any exposure, even very low, to radon might pose some risk. However, from the evidence now available, a threshold exposure, that is, a level of exposure below which there is no effect of radon, cannot be excluded.

The second problem is that the majority of miners in the studies are smokers and all inhale dust and other pollutants in mines. Because radon and cigarette smoke both cause lung cancer, it is complicated to disentangle the effects of the 2 kinds of exposure. That makes it especially difficult to estimate radon risks for nonsmokers in homes using the evidence from miners. A final problem is that the

miners were almost all men, whereas the population exposed to radon in homes includes men, women, and children.

The committee used the information from miners and supplemented it with information from laboratory studies of how radon causes lung cancer. Then, with facts about the U.S. population, including measurements of radon levels in homes, it estimated the number of lung-cancer deaths due to radon in homes. In 1995, about 157,400 people died of lung cancer (from all causes including smoking and radon exposure) in the United States. Of the 95,400 men who died of lung cancer, about 95% were probably ever-smokers; of the 62,000 women, about 90% were probably ever-smokers. Approximately 11,000 lung-cancer deaths are estimated to have occurred in never-smokers in 1995.

The BEIR VI committee's preferred central estimates, depending on which one of the two models are used, are that about 1 in 10 or 1 in 7 of all lung-cancer deaths—amounting to central estimates of about 15,400 or 21,800 per year in the United States—can be attributed to radon among ever-smokers and never-smokers together. Although 15,400 or 21,800 total radon-related lung-cancer deaths per year are the committee's central estimates, uncertainties are involved in these estimates. The committee's preferred estimate of the uncertainties was obtained by using a simplified analysis of a constant relative risk model based on observations closest to residential exposure levels. The number of radon-related lung-cancer deaths resulting from that analysis could be as low as 3,000 or as high as 33,000 each year. Most of the radon-related lung cancers occur among ever-smokers, and because of synergism between smoking and radon, many of the cancers in ever-smokers could be prevented by either tobacco control or reduction of radon exposure. The committee's best estimate is that among the 11,000 lung-cancer deaths each year in never-smokers, 2,100 or 2,900, depending on the model used, are radon-related lung cancers.

Radon, being naturally occurring, cannot be entirely eliminated from our homes. Of the deaths that the committee attributes to radon (both independently and through joint action with smoking), perhaps one-third could be avoided by reducing radon in homes where it is above the "action guideline level" of 148 Bqm^{-3} (4 $pCiL^{-1}$) to below the action levels recommended by the Environmental Protection Agency.

The risk of lung cancer caused by smoking is much higher than the risk of lung cancer caused by indoor radon. Most of the radon-related deaths among smokers would not have occurred if the victims had not smoked. Furthermore, there is evidence for a synergistic interaction between smoking and radon. In other words, the number of cancers induced in ever-smokers by radon is greater than one would expect from the additive effects of smoking alone and radon alone. Nevertheless, the estimated 15,400 or 21,800 deaths attributed to radon in combination with cigarette-smoking and radon alone in never-smokers constitute a public-health problem.

Executive Summary

INTRODUCTION

This National Research Council's report of the sixth Committee on Biological Effects of Ionizing Radiations (BEIR VI) addresses the risk of lung cancer associated with exposure to radon and its radioactive progeny. Radon, a naturally occurring gas formed from the decay of uranium in the earth, has been conclusively shown in epidemiologic studies of underground miners to cause lung cancer. There is supporting evidence from experimental studies of animals that confirm radon as a cause of lung cancer and from molecular and cellular studies that provide an understanding of the mechanisms by which radon causes lung cancer.

In addition to being present at high concentrations in many types of underground mines, radon is found in homes and is also present outdoors. Extensive measurements of radon concentrations in homes show that although concentrations vary widely, radon is universally present, raising concerns that radon in homes increases lung-cancer risk for the general population, especially those who spend a majority of their time indoors at home. For the purpose of developing public policy to manage the risk associated with indoor radon, there is a need to characterize the possible risks across the range of exposures received by the population. The higher end of that range of exposures is comparable to those exposures that caused lung cancer in underground miners. The lower end of that range includes exposures received from an average indoor lifetime exposure which is at least one order of magnitude lower.

Risk models, which mathematically represent the relationship between exposure and risk, have been developed and used to assess the lung-cancer risks

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associated with indoor radon. For example, the precursor to this committee, the BEIR IV committee, developed one such model on the basis of statistical analysis of data from 4 epidemiologic studies of underground miners. The BEIR IV model has been widely used to estimate the risk posed by indoor radon. Since the 1988 publication of the BEIR IV report, substantial new evidence on radon has become available: new epidemiologic studies of miners have been completed, existing studies have been extended, and analysis of the pooled data from 11 principal epidemiologic studies of underground miners has been conducted involving a total of 68,000 miners and to date, 2,700 deaths from lung cancer. Other lines of scientific evidence relevant to assessing radon risks have also advanced, including findings on the molecular and cellular basis of carcinogenesis by alpha particles. Radon itself does not directly cause lung cancer but alpha particles from radon progeny directly damage target lung cells to cause cancer. There is additional information for calculating the dose of alpha particles received by the lung from inhaled radon progeny, the topic of a 1991 follow-up report to the BEIR IV report, the report of the National Research Council's Panel on Dosimetric Assumptions. Finally, during the last decade, a number of epidemiologic case-control studies that estimated the risk associated with indoor radon directly have also been implemented.

The BEIR VI committee faced the task of estimating the risks associated with indoor radon across the full range of exposures and providing an indication of the uncertainty to be attached to risk estimates across this range. In preparing this report, the BEIR VI committee, in response to its charge, reviewed the entire body of data on radon and lung cancer, integrating findings from epidemiologic studies with evidence from animal experiments and other lines of laboratory investigation. The committee also considered the substantial evidence on smoking and cancer and the more limited evidence on the combined effect of smoking and radon. The report's elements include comprehensive reviews of the cellular and molecular basis of radon carcinogenesis and of the dosimetry of radon in the respiratory tract, of the epidemiologic studies of miners and the general population, and of the combined effects of radon and other occupational carcinogens with tobacco-smoking. The committee describes its preferred risk models, applies the models to estimate the risk posed by indoor radon, and characterizes uncertainties associated with the risk estimates.

THE MECHANISTIC BASIS OF RADON-INDUCED LUNG CANCER

Information on radon carcinogenesis comes from molecular, cellular, animal, and human (or epidemiologic) studies. Radiation carcinogenesis, in common with any other form of cancer induction, is likely to be a complex multistep process that can be influenced by other agents and genetic factors at each step. Since our current state of knowledge precludes a systematic quantitative description of all steps from early subcellular lesions to observed malignancy, the com-

mittee used epidemiologic data to develop and quantify an empirical model of the exposure-risk relationship for lung cancer. The committee did draw extensively, however, on findings from molecular, cellular, and animal studies in developing its risk assessment for the general population.

The committee's review of the cellular and molecular evidence was central to the specification of the risk model. This review led to the selection of a linear nonthreshold relation between lung-cancer risk and radon exposure. However, the committee acknowledged that other relationships, including threshold and curvilinear relationships, cannot be excluded with complete confidence, particularly at the lowest levels of exposure. At low radon exposures, typical of those in homes, a lung epithelial cell would rarely be traversed by more than one alpha particle per human lifespan. As exposure decreases, the insult to cell nuclei that are traversed by alpha particles remains the same as at higher exposures, but the number of traversed nuclei decreases proportionally. There is good evidence that a single alpha particle can cause major genomic changes in a cell, including mutation and transformation. Even allowing for a substantial degree of repair, the passage of a single alpha particle has the potential to cause irreparable damage in cells that are not killed. In addition, there is convincing evidence that most cancers are of monoclonal origin, that is, they originate from damage to a single cell. These observations provide a mechanistic basis for a linear relationship between alpha-particle dose and cancer risk at exposure levels at which the probability of the traversal of a cell by more than one alpha particle is very small, that is, at exposure levels at which most cells are never traversed by even one alpha particle. On the basis of these mechanistic considerations, and in the absence of credible evidence to the contrary, the committee adopted a linear-nonthreshold model for the relationship between radon exposure and lung-cancer risk. However, the committee recognized that it could not exclude the possibility of a threshold relationship between exposure and lung cancer risk at very low levels of radon exposure.

Extrapolation from higher to lower radon exposures is also influenced by the inverse dose-rate effect, an increasing effect of a given total exposure as the rate of exposure is decreased, as demonstrated by experiments in vivo and in vitro for high-LET radiation, including alpha particles, and in miner data. This dose-rate effect, whatever its underlying mechanism, is likely to occur at exposure levels at which multiple particle traversals per cell nucleus occur. Mechanistic, experimental, and epidemiologic considerations support the disappearance of the effect at low exposure corresponding to an average of much less than one traversal per cell location, as in most indoor exposures. Extrapolating radon risk from the full range of miner exposures to low indoor exposures involves extrapolating from a situation in which multiple alpha-particle traversals of target nuclei occur to one in which they are rare; such an extrapolation would be from circumstances in which the inverse dose-rate effect might be important to one in which it is likely to be nonexistent. These considerations indicated a need to assess risks of radon

in homes on the basis of miner data corresponding to as low an exposure as possible, or to use a risk model that accounts for the diminution of an inverse exposure-rate effect with decreasing exposure.

The committee also reviewed other evidence relevant to the biologic basis of its risk assessment approach. For the combined effect of smoking and radon, animal studies provided conflicting evidence on synergism, and there is uncertainty as to the relevance of the animal experiments to the patterns of smoking by people. Early attempts to identify a molecular "signature" of prior alpha-particle damage through the identification of unusual point mutations in specific genes have not yet proven useful, although approaches based on specific chromosomal aberrations show some promise, and all the principal histologic types of lung cancer can be associated with radon exposure. Available evidence, albeit limited, supports the likelihood that a typical human population would have a broad spectrum of susceptibility to alpha-particle-induced carcinogenesis.

THE BEIR VI RISK MODELS

For estimating the risk imposed by exposure to indoor radon, the committee chose an empirical approach based on analysis of data from radon-exposed miners. Other approaches that the committee considered but did not use included a "dosimetric" approach, and use of "biologically-motivated" risk models. A dosimetric approach, in which radon risks are estimated by applying risk estimates from A-bomb survivor studies to estimates of radiation doses delivered to the lung, was not pursued because of the major differences in the type of radiation and exposure patterns compared with radon-progeny exposure. A biologic-based approach to modeling with a description of the various processes leading to radon-induced cancer was not followed primarily because of the present incomplete state of knowledge of many of these processes.

The committee turned to the empirical analysis of epidemiologic data as the basis for developing its risk model. Two sources of information were available: data from the epidemiologic studies of underground miners and data from the case-control studies of indoor radon and lung cancer in the general population. Both groups include ever-smokers and never-smokers. Although the case-control studies provide direct estimates of indoor radon risk, the estimates obtained from these studies are very imprecise, particularly if estimated for never-smokers or ever-smokers separately, because the excess lung-cancer risk is likely to be small. Other weaknesses of the case-control studies are errors in estimating exposure and the limited potential for studying modifying factors, particularly cigarette smoking. Nonetheless, the committee considered the findings of a meta-analysis of the 8 completed studies.

In developing its risk models, the committee started with the recently reported analyses by Lubin and colleagues of data from 11 studies of underground miners—uranium miners in Colorado, New Mexico, France, Australia, the Czech

Republic, and in Port Radium, Beaverlodge, and Ontario in Canada; metal miners in Sweden; tin miners in China; and fluorspar miners in Canada. The data for 4 studies were updated with new information. These 11 studies offered a substantially greater data resource than had been available to the BEIR IV committee. The 11 epidemiologic studies covered a range of mining environments, times, and countries, and their methods of data collection differed in some respects.

The committee analyzed the data with a relative-risk model in which radon exposure has a multiplicative effect on the background rate of lung cancer. In particular, the committee modeled the excess relative risk (ERR), which represents the multiplicative increment to the excess disease risk beyond background resulting from exposure. The model represents the ERR as a linear function of past exposure to radon. This model allows the effect of exposure to vary flexibly with the length of time that has passed since the exposure, with the exposure rate, and with the attained age. The mathematical form of the model for ERR is:

$$\text{ERR} = \beta(w_{5\text{-}14} + \theta_{15\text{-}24}\, w_{15\text{-}24} + \theta_{25+}\, w_{25+})\phi_{\text{age}}\gamma_z$$

The parameter β represents the slope of the exposure-risk relationship for the assumed reference categories of the modifying factors. Exposure at any particular age has 4 components: exposure in the last 5 years—excluded as not biologically relevant to cancer risk—and exposures in 3 windows of past time, namely 5-14, 15-24, and 25 or more years previously. Those exposures are labeled $w_{5\text{-}14}$, $w_{15\text{-}24}$, and w_{25+}, respectively, and each is allowed to have its own relative level of effect, $\theta_{5\text{-}14}$ (set equal to unity), $\theta_{15\text{-}24}$, and θ_{25+}, respectively. With this weighting system, total exposure can be calculated as $w^* = w_{5\text{-}14} + \theta_{15\text{-}24}\, w_{15\text{-}24} + \theta_{25+}\, w_{25+}$. The rate of exposure also affects risk through the parameter γ_z; thus, the effect of a particular level of exposure increases with decreasing exposure rate, as indexed either by the duration of exposure or the average concentration at which exposure was received. The ERR also declines with increasing age, as described by the parameter ϕ_{age}.

Based on this analysis, the committee developed two preferred risk models referred to as the exposure-age-concentration model and the exposure-age-duration model. These two models differ only with respect to the parameter γ_z, which represents either duration of exposure or the average concentration over the time of the exposure. The models were equally preferred by the committee. The new models are similar in form to the BEIR IV model, but have an additional term for exposure rate and more-detailed categories for the time-since-exposure windows and for attained age.

RISK ASSESSMENT

The committee's risk models can be used to project the lung-cancer risk associated with radon exposure, both for individuals and for the entire US population. To extend the models that were developed from miner data to the general

population, the committee needed to make a set of assumptions on the following key issues.

Lung Dosimetry of Radon Progeny

Physical and biologic differences between the circumstances of exposures of male miners working underground and of men, women, and children in their homes could lead to differing doses at the same exposures. The committee estimated the value of a dimensionless parameter, termed the "K factor" in prior reports, that characterizes the comparative doses to lung cells in homes and mines for the same exposure. Using a model to estimate the dose to the cells in the lung, and incorporating new information on the input parameters of the model, the committee found that the doses per unit exposure in mines and homes were essentially the same. Thus, K is calculated to be about 1 for men, women and children (age 10 years), and slightly above $K = 1$ for infants (age 1). Consequently, a value of 1 was used in making the risk projections.

Extrapolation of Risks at Higher Exposures to Lower Exposures

Average exposures received by the miners in the epidemiologic studies are about one order of magnitude higher than average indoor exposures, although the lowest exposures of some miners overlap with some of the highest indoor exposures. To estimate risks of indoor radon exposures, it is thus necessary to make an assumption about the shape of the exposure-risk relationship across the lower range of the distribution of radon exposures.

The committee selected a linear-nonthreshold relationship relating exposure to risk for the relatively low exposures at issue for indoor radon. This assumption has significant implications for risk projections. Support for this assumption came primarily from the committee's review of the mechanistic information on alpha-particle-induced carcinogenesis. Corroborating information included evidence for linearity in the miner studies at the lower range of exposures, and the linearity and magnitude of risk observed in the meta-analysis of the case-control studies, which was fully consistent with extrapolation of the miner data. Although a linear-nonthreshold model was selected, the committee recognized that a threshold—that is, a level of exposure with no added risk—could exist and not be identifiable from the available epidemiologic data.

Exposure Rate

At higher exposures, the committee found evidence in the miner data of an inverse exposure-rate effect. Theoretical considerations suggested that the inverse exposure-rate effect found in the miner data should not modify risks for typical indoor exposures. Consequently, the exposure-rate effect in the lowest

range of miner exposure rates was applied for relevant indoor exposures without further adjustment.

Combined Effect of Smoking and Radon

Apart from the results of very limited in vitro and animal experiments, the only source of evidence on the combined effect of the 2 carcinogens (cigarette smoke and radon) was the data from 6 of the miner studies. Analysis of those data indicated a synergistic effect of the two exposures acting together, which was characterized as submultiplicative, i.e., less than the anticipated effect if the joint effect were the product of the risks from the two agents individually, but more than if the joint effect were the sum of the individual risks. The committee applied a full multiplicative relation of the joint effect of smoking and exposure to radon, as done by the BEIR IV committee, and also a submultiplicative relationship. Although the committee could not precisely characterize the joint effect of smoking and radon exposure, the submultiplicative relation was preferred by the committee because it was found to be more consistent with the available data.

Risks for Women

The risk model is based on epidemiologic studies of male miners. The effect of radon exposure on lung-cancer risk in women might be different from that in men because of differing lung dosimetry or other factors related to gender. The K factor was calculated separately for women and men, but did not differ by gender. The committee also could not identify strong evidence indicative of differing susceptibility to lung carcinogens by sex. Consequently, the model was extended directly to women, with the assumption that the excess risk imposed by radon progeny estimated from the male miners multiplies the background lung cancer rates for women, which are presently substantially lower than for men.

Risks Associated with Exposures in Childhood

Evidence was available from only one study of miners on whether risk was different for exposures received during childhood, during adolescence, and during adulthood. There was not a clear indication of the effect of age at exposure. The committee made no specific adjustment for exposures received at earlier ages. The K factor for children aged 10 was calculated as 1 and the value for infants was only slightly higher (about 1.08).

Characterization of Radon Risks

In making its calculations, the committee used the latest data on lung cancer mortality for 1985-1989 and for smoking prevalence for the U.S. in 1993. To

characterize the lung-cancer risk posed to the population by indoor radon, the two models for the exposure-risk relationship were applied to the distribution of exposures received by the population to estimate the burden of lung cancer sustained by the population as a result of indoor-radon exposure. To characterize risks to the population, we have used the population attributable risk (AR), which indicates how much of the lung-cancer burden could, in theory, be prevented if all exposures to radon were reduced to the background level of radon in outdoor air. The AR estimates include cases in ever-smokers and never-smokers. To characterize the risk to specific individuals, the committee calculated the lifetime relative risk (LRR), which describes the relative increment in lung-cancer risk resulting from exposure to indoor radon beyond that from exposure to outdoor-background concentrations of radon.

Radon-Attributable Risks

LRRs were computed using the committee's risk models. Estimates were computed for exposure scenarios which reflect concentrations of indoor radon of interest. Table ES-1 shows the estimated LRRs for lifetime exposures at various constant radon concentrations. The LRR values are quite similar for the preferred 2 models: exposure-age-concentration and exposure-age-duration. The LRR values estimated by the BEIR VI models and the BEIR IV model are also similar, in spite of the addition of exposure rate to the new models. As anticipated, LRR values increase with exposure. Women have a somewhat steeper increment in LRR with increasing exposure because of differing mortality patterns.

Attributable risks for lung cancer from indoor radon in the US population were computed with the committee's 2 preferred models and compared with the BEIR IV results. Based on the National Residential Radon Survey, the committee assumed a log-normal distribution for residential radon concentration, with a median of 24.3 Bqm^{-3} (0.67 $pCiL^{-1}$) and a geometric standard deviation of 3.1 (Marcinowski 1994). The AR was calculated for the entire US population and for males and females and ever-smokers and never-smokers under the preferred submultiplicative model (Table ES-2). For the entire population, the ARs calculated with the new models ranged from about 10% to 14% and were higher than estimates based on the BEIR IV model. Under the submultiplicative assumption which was described on page ES-9, the attributable risk estimates for ever-smokers tended to be lower than estimates for never-smokers, although the numbers of cases are far greater in ever-smokers than in never-smokers.

These AR estimates for the general population are further broken down with respect to the distribution of indoor concentrations in Table ES-3. This analysis provides a picture of the potential consequences of alternative mitigation strategies that might be used for risk-management purposes. The findings were the same for the committee's 2 models. The radon concentration distribution is highly skewed, with homes with higher radon concentrations contributing dispro-

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TABLE ES-1 Estimated lifetime relative risk (LRR) of lung cancer for lifetime indoor exposure to radon[a]

Exposure[b]					Exposure-age-concentration model				Exposure-age-duration model			
					Male		Female		Male		Female	
WLM/y	Jhm^{-3}/y	Bqm^{-3}	pCiL^{-1}	WL	Ever-smoker	Never-smoker	Ever-smoker	Never-smoker	Ever-smoker	Never-smoker	Ever-smoker	Never-smoker
0.10	0.00035	25	0.7	0.003	1.081	1.194	1.089	1.206	1.054	1.130	1.059	1.137
0.19	0.00067	50	1.4	0.005	1.161	1.388	1.177	1.411	1.108	1.259	1.118	1.274
0.39	0.00137	100	2.7	0.011	1.318	1.775	1.352	1.821	1.214	1.518	1.235	1.547
0.58	0.00203	150	4.1	0.016	1.471	2.159	1.525	2.229	1.318	1.776	1.352	1.819
0.78	0.00273	200	5.4	0.022	1.619	2.542	1.694	2.637	1.420	2.033	1.466	2.091
1.56	0.00546	400	10.8	0.043	2.174	4.057	2.349	4.255	1.809	3.053	1.915	3.174
3.12	0.01092	800	21.6	0.086	3.120	7.008	3.549	7.440	2.507	5.058	2.760	5.317

[a]Based on a submultiplicative relationship between tobacco-smoking and radon.
[b]Exposures are represented by concentrations in bequerels per cubic meter (Bqm^{-3}), picocuries per liter (pCiL^{-1}), or Working Levels (WL), assumed to be constant for home occupancy at the 70% level and 40% equilibrium between radon and its progeny, and also by joules-hours per cubic meter per year (Jhm^{-3}/y) and Working Level Months per year (WLM/y). For definitions of these terms, see the Glossary at the end of this report.

TABLE ES-2   Estimated attributable risk (AR[a]) for lung-cancer death from domestic exposure to radon using 1985-89 U.S. population mortality rates based on selected risk models

Model	Population	Ever-smokers[b]	Never-smokers[b]
Males			
Committee's preferred models			
Exposure-age-concentration	0.141	0.125	0.258
Exposure-age-duration	0.099	0.087	0.189
Other Models			
CRR[c] (<0.175 Jhm^{-3}; <50 WLM)	0.109	0.096	0.209
BEIR IV	0.082	0.071	0.158
Females			
Committee's preferred models			
Exposure-age-concentration	0.153	0.137	0.269
Exposure-age-duration	0.108	0.096	0.197
Other Models			
CRR[c] (<0.175 Jhm^{-3}; <50 WLM)	0.114	0.101	0.209
BEIR IV	0.087	0.077	0.163

[a]AR = the risk of lung cancer death attributed to radon in populations exposed to radon divided by the total risk of lung cancer death in a population.
[b]Based on a submultiplicative relationship between tobacco-smoking and radon.
[c]CRR = constant relative risk.

portionately to AR. Only 13% of the calculated AR is estimated to be contributed by the 50% of homes below the median concentration of about 25 Bqm^{-3} (0.7 pCiL^{-1}) and about 30% by homes below the mean of about 46 Bqm^{-3} (1.25 pCiL^{-1}). Homes above 148 Bqm^{-3} (4 pCiL^{-1}), the current action level established by the Environmental Protection Agency, contribute about 30% percent of the AR. This contribution to the total AR is indicative of the potential magnitude of avoidable deaths with a risk management program based on the current action guideline. While 10-15 percent of all lung cancers are estimated to be attributable to indoor radon, eliminating exposures in excess of 148 Bqm^{-3} (4 pCiL^{-1}) would prevent about 3 to 4 percent of all lung cancers, or, about one-third of the radon-attributable lung cancers.

The ARs were reestimated with assumption of thresholds, levels below which cancer risk is not increased, at 37, 74, or 148 Bqm^{-3} (1, 2, or 4 pCiL^{-1}). Even though the committee assumed that risk was most likely linear with exposure at lower levels, this analysis was conducted to illustrate the impact of assuming a threshold on risk-management decisions. Assuming an action level of 148 Bqm^{-3} (4 pCiL^{-1}) for mitigation, postulating a threshold reduces the total number of lung-cancer deaths that are attributable to indoor radon and also the number of lung-cancer deaths that can be prevented by reducing levels in homes to zero. For assumed thresholds below 148 Bqm^{-3} (4 pCiL^{-1}), there is little impact on the

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TABLE ES-3 Distribution of attributable risks for U.S. males from indoor residential radon exposure under BEIR VI models

Exposure Range (Bqm^{-3})	% of Homes in Range	Exposure-age-concentration model Contribution to AR			Exposure-age-duration model Contribution to AR		
		Actual	%	Cumulative %	Actual	%	Cumulative %
0- 25	49.9	0.018	12.8	12.8	0.013	12.8	12.8
26- 50	23.4	0.026	18.5	31.3	0.018	18.4	31.2
51- 75	10.4	0.020	14.2	45.5	0.014	14.2	45.4
76-100	5.4	0.015	10.5	56.0	0.010	10.5	55.9
101-150	5.2	0.020	13.9	69.9	0.004	13.9	69.8
151-200	2.4	0.013	9.2	79.1	0.009	9.2	79.0
201-300	1.8	0.014	9.6	88.7	0.010	9.7	88.7
301-400	0.7	0.007	5.2	93.9	0.005	5.3	94.0
401-600	0.4	0.006	4.5	98.4	0.005	4.6	98.6
601+	0.4	0.002	1.5	99.9	0.001	1.6	100.2
Total	100.0	0.141	100.0		0.099	100.0	

estimated numbers of preventable lung cancers by mitigation of homes with radon concentrations above 148 Bqm^{-3} (4 pCiL^{-1}).

These AR estimates can be translated into numbers of lung-cancer deaths (Table ES-4). In 1995, there were approximately 157,400 lung-cancer deaths—95,400 in men and 62,000 in women—in the United States. Most occurred in smokers and it is estimated that 95% of cases occurred in men and 90% in women. Table ES-4 shows the estimated lung-cancer deaths in the United States attributable to indoor radon progeny exposure under the BEIR VI models. A review of the data presented in Table ES-4 reveals some differences in the calculated radon-attributable lung-cancer deaths using the exposure-age-concentration model and the exposure-age-duration model. Further variability is evident for both models depending on the approach used to estimate the influence of cigarette-smoking on lung-cancer risk. The use of the two models with two approaches to dealing with smoking yields an array of estimates of lung-cancer risk attributable to radon exposure, and provides an indication of the influence of the model and of incorporating the effects of tobacco-smoking on the projections of population risk. The range of calculated values, however, is not a complete

TABLE ES-4 Estimated number of lung cancer deaths for the U.S. for 1995 attributable to indoor residential radon progeny exposure

Population	Number of lung-cancer deaths	Lung-cancer deaths attributable to Rn progeny exposure (No.)	
		Exposure-age-concentration model	Exposure-age-duration model
Males[a]			
Total	95,400	12,500[b]	8,800[b]
Ever-smokers	90,600	11,300	7,900
Never-smokers	4,800	1,200	900
Females[a]			
Total	62,000	9,300	6,600
Ever-smokers	55,800	7,600	5,400
Never-smokers	6,200	1,700	1,200
Males and Females			
Total	157,400	21,800	15,400
Ever-smokers	146,400	18,900	13,300
Never-smokers	11,000	2,900	2,100

[a]Assuming 95% of all lung cancers among males occurs among ever-smokers; 90% of lung cancers among females occurs among ever-smokers.

[b]Estimates based on applying a smoking adjustment to the risk models, multiplying the baseline estimated attributable risk per exposure by 0.9 for ever-smokers and by 2.0 for never-smokers, implying a submultiplicative relationship between radon-progeny exposure and smoking.

reflection of the uncertainty in estimating the lung-cancer risks of radon expo-
sures and especially for never-smokers at low levels of radon exposure.

Uncertainty Considerations

Quantitative estimates of the lung cancer risk imposed by radon are subject
to uncertainties—uncertainties that need to be understood in using the risk pro-
jections as a basis for making risk-management decisions (see Table ES-5). Broad
categories of uncertainties can be identified, including uncertainties arising from
the miner data used to derive the lung-cancer risk models and the models them-
selves, from the representation of the relationship between exposure and dose,
from the exposure-distribution data, from the demographic and lung-cancer mor-
tality data, and from the assumptions made in extending the committee's models
from the exposures received by the miners to those received by the general
population. The committee addressed those sources of uncertainty qualitatively
and, to a certain extent, quantitatively.

TABLE ES-5   Sources of uncertainty in estimates of lifetime risk of lung-
cancer mortality resulting from exposure to radon in homes

I   Sources of uncertainty arising from the model relating lung-cancer risk to exposure
   A   Uncertainties in parameter estimates derived from miner data
      1   Sampling variation in the underground miner data;
      2   Errors and limitations in the underground miner data;
        a)   Errors in health-effects data including vital status and information on cause of
           death;
        b)   Errors in data on exposure to radon and radon progeny including estimated
           cumulative exposures, exposure rates and durations;
        c)   Limitations in data on other exposures including data on smoking and on other
           exposures such as arsenic.
   B   Uncertainties in application of the lung-cancer exposure-response model and in its
      application to residential exposure to the general U.S. population
      1   Shape of the exposure/exposure rate response function for estimates at varying
        exposures and exposure rates;
      2   Temporal expression of risks;
      3   Dependence of risks on sex;
      4   Dependence of risks on age at exposure;
      5   Dependence risks on smoking status.
II   Sources of uncertainty arising from differences in radon progeny dosimetry in mines and
   in homes
III   Sources of uncertainty arising from estimating the exposure distribution for the U.S.
   population exposure distribution model
      1   Estimate of the average radon concentration;
      2   Estimate of the average equilibrium fraction;
      3   Estimate of the average occupancy factor.
IV   Sources of uncertainty in the demographic data used to calculate lifetime risk

The committee's models of lung-cancer risk were based on analyses of data from epidemiologic studies of miners. There are undoubtedly errors in the estimates of exposures to radon progeny for the miners, and information was limited on other key exposures including cigarette smoking and arsenic. The committee could not identify any overall systematic bias in the exposure estimates for radon progeny, but random errors might have led to an underestimation of the slope of the exposure-risk relationship. Although 6 of 11 study cohorts had some smoking information, sparse information on smoking limited the committee's characterization of the combined effects of smoking and radon-progeny exposure and precluded precise estimation of the risk of radon-progeny exposure in never-smokers.

The committee's models may not correctly specify the true relationship between radon exposure and lung-cancer risk. The models assume a linear-multiplicative relationship without threshold between radon exposure and risk. While the miner data provide evidence of linearity across the range of exposures received in the mines, the assumption of linearity down to the lowest exposures was based on mechanistic considerations that could not be validated against observational data. Alternative exposure-risk relations, including relations with a threshold, may be operative at the lowest exposures. However, the committee's analysis showed that assumption of a threshold up to exposures at 148 Bqm^{-3} (4 pCiL^{-1}) had little impact on the numbers of lung-cancer deaths theoretically preventable by mitigation of exposures above that level.

Additional sources of uncertainty in the risk projections reflect the approach used to evaluate possibly differing lung dosimetry for miners and for the general population, the limited information on cigarette-smoking, and the lack of data on risks of exposures of children and women.

The committee applied new quantitative methods for uncertainty analysis to evaluate the impact of variability and uncertainty in the model parameters on the attributable risk. Since not all sources of uncertainty could be characterized, this analysis was intended to be illustrative and not to replace the committee's more comprehensive qualitative analysis.

The quantitative analysis conducted by the committee provided limits within which the AR was considered to lie with 95% certainty. For the exposure-age-concentration model, the uncertainty interval around the central estimate of AR (14%) for the entire population ranged from about 9 to 25%. This range reflects a substantial degree of uncertainty in the AR estimate, although the shape of the uncertainty distributions indicated that values near the central estimates were much more likely than values near the upper and lower limits. For the exposure-age-duration model, the uncertainty interval ranged from 7 to 17% and was centered at about 10%. The committee's preferred uncertainty limits were obtained using a simple constant-relative-risk model fitted to the miner data below 0.175 Jhm^{-3} (50 WLM), which is based on observations at exposures closest to residential exposure levels. The latter analysis, which minimizes the degree of

extrapolation outside the range of the miner data, led to uncertainty limits of 2 to 21%, with a central estimate of about 11%.

Effects of Radon Exposure Other Than Lung Cancer

Health effects of exposure to radon progeny other than lung cancer have been of concern, including other malignancies and non-malignant respiratory diseases in miners. The findings of several ecologic studies in the general population have indicated a possible effect of radon exposure in increasing risk for several types of non-lung cancers and leukemias. A pooled analysis of 11 miner studies, differing in one study from the data used by the committee, showed no evidence of excess risk for cancers other than the lung. The committee concluded that the findings in the miners could be reasonably extended to the general population and that there is no basis for considering that effects would be observed in the range of typical exposures of the general population that would not be observed in the underground miners exposed at generally much higher levels.

The committee reviewed new studies of non-malignant respiratory disease in uranium miners. A case series of uranium miners with pulmonary fibrosis supported the possibility that exposures to radon progeny may cause fibrosis of the pulmonary interstitium, but the case series is insufficient to establish a causal link to radon progeny specifically.

CONCLUSIONS

Radon is one of the most extensively investigated human carcinogens. The carcinogenicity of radon is convincingly documented through epidemiologic studies of underground miners, all showing a markedly increased risk of lung cancer. The exposure-response relationship has been well characterized by analyses of the epidemiologic data from the miner studies, and a number of modifiers of the exposure-response relationship have been identified, including exposure rate, age, and smoking. For residences in the United States, a large national survey provides information on typical exposures and on the range of exposures.

On the basis of the epidemiologic evidence from miners and understanding of the genomic damage caused by alpha particles, the committee concluded that exposure to radon in homes is expected to be a cause of lung cancer in the general population. According to the committee's two preferred risk models, the number of lung-cancer cases due to residential radon exposure in the United States was projected to be 15,400 (exposure-age-duration model) or 21,800 (exposure-age-concentration model). Although these represent the best estimates that can be made at this time, the committee's uncertainty analyses using the constant relative risk model suggested that the number of cases could range from about 3,000

to 33,000[1]. Nonetheless, this indicates a public-health problem and makes indoor radon the second leading cause of lung cancer after cigarette-smoking.

The full number of attributed deaths can be prevented through radon mitigation only by eliminating radon in homes, a theoretical scenario that cannot be reasonably achieved. Nonetheless, the burden of lung-cancer deaths attributed to the upper end of the exposure distribution is expected to be reduced by lowering radon concentrations. Perhaps one-third of the radon-attributed cases (about 4% of the total lung-cancer deaths) would be avoided if all homes had concentrations below the Environmental Protection Agency's action guideline of 148 Bqm^{-3} (4 pCiL^{-1}); of these, about 87% would be in ever-smokers. It can be noted that the deaths from radon-attributable lung cancer in smokers could most efficiently be reduced through tobacco-control measures, in that most of the radon-related deaths among smokers would not have occurred if the victims had not smoked.

The committee's model and general approach to assessing lung-cancer risks posed by indoor radon and cigarette-smoking are subject to considerable uncertainty because of gaps in our scientific knowledge of effects at low levels of exposure. This uncertainty should be reduced as an improved understanding develops of molecular and cellular events in the induction of lung cancer at low levels of exposure to radon and other toxicants and of the role of various factors influencing susceptibility to lung cancer. The long-term follow-up of miner populations is strongly encouraged, as is completion of the case-control studies of residential exposures now in progress. The committee encourages further meta-analysis and pooling of case-control data. However, the committee recommends that new case-control studies not be initiated until those in progress are completed, data are analyzed and synthesized, and judgments rendered as to the likely value of further residential studies.

Despite the limitations of existing data, the committee found key observational and experimental data that, along with theoretical considerations in radiobiology and carcinogenesis, provided a basis for the models developed and used to estimate radon-attributable lung-cancer risks. The major shortcomings in the existing data relate to estimating lung-cancer risks near 148 Bqm^{-3} (4 pCiL^{-1}) and down to the average indoor level of 46 Bqm^{-3} (1.24 pCiL^{-1}), especially the risks to never-smokers. The qualitative and quantitative uncertainty analyses indicated the actual number of radon-attributable lung-cancer deaths could be either greater or lower than the committee's central estimates. This uncertainty did not change the committee's view that indoor radon should be considered as a cause of lung cancer in the general population that is amenable to reduction. However, the attributable risk for smoking, the leading cause of lung cancer, is far greater than for radon, the second leading cause. Lung cancer in the general population and in miners is related to both risk factors and is amenable to prevention.

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[1]The 95% upper confidence limit for the exposure-age-concentration model was approximately 38,600, but such an upper limit was highly unlikely based on the committee's review and analyses.

HEALTH EFFECTS OF

EXPOSURE TO

RADON

BEIR VI

Committee on Health Risks of Exposure to Radon (BEIR VI)

Board on Radiation Effects Research

Commission on Life Sciences

National Research Council

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Preface

Nearly a decade has passed since the fourth in a series of studies called Biological Effects of Ionizing Radiation (BEIR) assessed the risks posed by exposure to radon and other alpha emitters in 1988. Radon, a gas emitted into homes from the soil, from water and from building materials, becomes trapped in homes. Its radioactive daughters, the progeny of radioactive decay, are inhaled into human lungs, where further decay results in the exposure of lung cells to densely ionizing alpha particles. On the basis of considerable experience gained by studying health effects in uranium and other miners who worked in radon-rich environments, the radioactive radon progeny were identified as a cause of lung cancer. It has not been clear whether radon poses a similar risk of causing lung cancer in men, women, and children exposed at generally lower levels found in homes, but homeowners are concerned about this potential risk, and the Environmental Protection Agency (EPA) suggests "action levels," concentrations to which citizens are encouraged to reduce their levels of exposure.

Motivated by the ubiquitous exposure of the general population to radon and the continued concern about the risks of exposure to this natural radioactive carcinogen, EPA sought the advice of the National Academy of Sciences and the National Research Council in re-examining the approaches to assessing the lung-cancer risk associated with radon concentrations in the domestic environment. As requested by EPA, the National Research Council first conducted a scoping study; in its 1994 report *The Health Effects of Exposure to Radon: Time for Reassessment?,* the BEIR VI Phase I committee concluded that sufficient and appropriate new evidence had become available since the 1989 BEIR IV report to justify a new National Research Council BEIR VI study. Consequently, the

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BEIR VI committee, consisting of 13 scientists with the expertise required to explore EPA's charge, was formed in 1994 to reexamine the risk of health effects posed by exposure to radon in homes.

The report that follows includes 4 chapters and 7 appendixes. The chapters provide the committee's principal findings; supporting analyses and other related evidence are presented in the appendixes. After an introduction to the radon problem in chapter 1, the biologic evidence on mechanisms of radon-related lung cancer is reviewed in chapter 2, which summarizes information on issues identified by the committee as critical to assessing lung-cancer risk from radon exposure. Chapter 3 presents the committee's risk models and 3 major risk projections, provides the rationale for the committee's modeling decisions, describes the committee's preferred risk models and the projections of lung-cancer risk resulting from their use, and addresses the issues related to uncertainty in the risk projections. Chapter 4 reviews the evidence on health effects other than lung cancer that result from exposure to radon progeny.

The appendixes support the committee's findings and should be consulted by readers who want in-depth coverage of specific issues. Appendix A is devoted to previously used risk models, the committee's methods and approach to risk modeling, and the details of the uncertainty considerations and analyses. Radon dosimetry is discussed in appendix B, which updates a 1991 National Research Council report on this subject. The dominant cause of lung cancer in the United States and many other countries is tobacco-smoking; appendix C provides additional information on the risks of lung cancer in relation to smoking and reviews the available data on the combined effects of smoking and exposure to radon and its progeny.

Appendixes D, E, and F address the information available from epidemiologic studies on underground miners. Appendix D summarizes the characteristics and designs of the miner studies. Appendix E and its annexes review the details of the exposures in individual miner studies and provide the proceedings of a workshop on exposure estimation. Appendix F addresses exposures to agents other than radon—such as diesel exhaust, silica, and arsenic—which might be relevant to estimating lung-cancer risk posed by radon. The findings of studies—principally ecologic or case-control epidemiologic studies conducted to estimate directly the risk posed by radon exposure in homes—are described in appendix G. The findings of several individual studies and combined analyses of the case-control studies are summarized. Finally, a compilation of the literature cited in the report and a glossary of technical terms used in the report follows appendix G.

ACKNOWLEDGMENTS

In the course of doing its work, the committee held several meetings, including a meeting in which the scientific community and other interested groups were

invited to present suggestions regarding the problem. Other information-gathering meetings were held in which scientists with specific expertise were invited to discuss the results of their work. Several workshops were held in which the committee focused on particular aspects of the radon problem. The committee is grateful to participants in a January 1996 workshop, in which over 30 scientists discussed findings of work relevant to the shape of the dose-response curve at low doses of radiation with densely deposited energy applicable to the alpha particles emitted by radon and its progeny. The results of that workshop, which was sponsored by the Department of Energy's Office of Health and Environmental Research, were useful to the committee in selecting a risk-assessment model.

The committee is appreciative to the many scientists who contributed directly or indirectly to the work of the BEIR VI committee. Seven persons and groups deserve special recognition and thanks. First, the committee is indebted to the principal investigators (PIs) and other scientists who directed and analyzed the various cohort studies of miners and the case-control studies of lung cancer and radiation in the general population. Those scientists willingly and graciously provided their data to the committee and enabled the committee to apply analytical techniques to original data in order to derive its models and its risk estimates. For the miner studies, the scientists included Shu-Xiang Yao, Xiang-Zhen Xuan, and Jay H. Lubin (Chinese tin miners), Emil Kunz and Ladislav Tomášek (Czechoslovakian, now the Czech Republic, Uranium miners), Geoffrey Howe (Beaverlodge, Canada and Port Radium, Canada uranium miners), Howard I. Morrison (Newfoundland, Canada fluorspar miners), Robert A. Kusiak and Jan Muller (Ontario, Canada uranium miners), Margot Tirmarche (French uranium miners), Alistair Woodward (South Australian uranium miners), Edward P. Radford and Christer Edling (Swedish iron miners), Richard W. Hornung (Colorado, U.S.A. uranium miners), and Jonathan M. Samet (New Mexico, U.S.A. uranium miners).

Second, the committee sponsored a workshop on exposures of miners to radon progeny. The following persons, knowledgeable about the history of the mining industry with experience in geology or mine and ventilation engineering, were invited to the workshop and made contributions that were much appreciated: William Chenoweth, James Cleveland, Andreas George, and Douglas Chambers. During the workshop, Daniel Stram and Duncan Thomas provided advice to the committee regarding quantification of the measurement errors associated with the mine exposures.

Third, the committee expresses its thanks to Anthony James, who provided computational analyses of doses to lung cells resulting from radon and its progeny.

Fourth, the committee thanks Susan Rose of the Department of Energy for inviting the BEIR VI chair and study director to participate in meetings of the PIs of the case-control studies in Europe and North America in which plans for pooling of data and joint analyses were discussed.

Fifth, three scientists from Health Canada provided valuable computational analyses and assisted committee members in the development and application of

the risk assessment: Shesh N. Rai, Yong Wang, and Jan M. Zielinski. Their contributions were important in the development of chapter 3 and appendix A.

Sixth, the committee is especially grateful to Rosalyn Yalow, an orginal committee member whose health prohibited her from serving as a final author of the study. Dr. Yalow was especially effective in insuring that the committee not overlook the influence of cigarette-smoking on the lung-cancer problem.

Seventh, this report has been reviewed by individuals chosen for their diverse perspectives and technical expertise, in accordance with procedures approved by the National Research Council's Report Review Committee. The purpose of this independent review is to provide candid and critical comments that will assist the authors and the National Research Council in making the published report as sound as possible and to ensure that the report meets institutional standards for objectivity, evidence, and responsiveness to the study charge. The content of the review comments and draft manuscript remain confidential to protect the integrity of the deliberative process. We wish to thank the following individuals for their participation in the review of this report: Gregg Claycamp (University of Pittsburgh School of Public Health), Merril Eisenbud (deceased), Robert Forster (University of Pennsylvania School of Medicine), Naomi Harley (New York University Medical Center), Werner Hofmann (University of Salzburg), Maureen Henderson (University of Washington), Richard Hornung (University of Cincinnati), Donald Mattison (University of Pittsburgh School of Public Health), Suresh Moolgavkar (University of Washington), Lincoln Moses (Stanford University), Louise Ryan (Harvard University School of Public Health), and Duncan Thomas (University of Southern California). Although the individuals listed above have provided many constructive comments and suggestions, responsibility for the final content of this report rests solely with the authoring committee and the National Research Council.

The committee appreciates the assistance of the Board on Radiation Effects Research (BRER) staff who supported the committee's work, especially Doris Taylor for her assistance with meeting and travel arrangements and Amy Noel O'Hara and Dennis Gleeson, Jr., for their attention to the details of manuscript preparation.

The BEIR VI committee has faced many challenges in preparing this report. Committee members are hopeful that the report will meet the needs of the EPA as it considers risk management strategies for indoor radon and also will be informative to the public as homeowners make decisions about testing for radon and lowering radon concentrations in their homes.

Jonathan Samet, M.D.
Chair, Committee on Health Effects of
Exposure to Radon, (BEIR VI)

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