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Health Risks Due to Radon in Drinking Water

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Following more than a decade of scientific debate about the setting of a standard for ^{222}Rn in drinking water, Congress established a timetable for the promulgation of a standard in the 1996 Amendments to the Safe Drinking Water Act. As a result of those Amendments, the EPA contracted with the National Academy of Sciences to undertake a risk assessment for exposure to radon in drinking water. In addition, the resulting committee was asked to address several other scientific issues including the national average ambient ^{222}Rn concentration and the increment of ^{222}Rn to the indoor-air concentration arising from the use of drinking water in a home. A new dosimetric analysis of the cancer risk to the stomach from ingestion was performed. The recently reported risk estimates developed by the BEIR VI Committee for inhalation of radon decay products were adopted. Because the 1996 Amendments permit states to develop programs in which mitigation of air-producing health-rsik reductions equivalent to that which would be achieved by treating the drinking water, the scientific issues involved in such "multimedia mitigation programs" were explored.

Introduction

Of all the radioisotopes that contribute to natural background radiation, radon presents the largest risk to human health. There are three naturally occurring isotopes of radon, but the use of the term *radon* in this paper refers specifically to ^{222}Rn, which is a decay product of ^{238}U. A recent report by the National Research Council suggested that between 3000 and 33 000 lung cancer deaths in the United States (the most likely number of deaths in the United States is 19 000) are associated with exposure to ^{222}Rn in indoor air, largely because radon substantially increases the lung cancer risk for smokers (*1*).

Most ^{222}Rn enters homes via migration of soil gas. The mean annual ^{222}Rn concentration measured in the living areas of homes in the United States is 46 Bq m^{-3} (*2*). [Radon concentrations are cited in the SI unit of becquerels per cubic meter (1 Bq m^{-3} = 0.027 pCi L^{-1}.]

Radon has also been identified as a public health concern when present in drinking water (*3*). Surface water contains very small amounts of dissolved radon. Typically, concentrations in surface waters are less than 4000 Bq m^{-3}. Water from wells can have high radon concentrations. A summary of the groundwater concentrations and the characteristics of groundwater supply systems is presented in Table 1 (*4*).

Because radon is relatively insoluble in water, water use releases radon into the indoor air and contributes to the total indoor-airborne radon concentration. Ingestion of radon in water may also pose a direct health risk through irradiation of sensitive cells in the gastrointestinal tract and other organs once it is absorbed into the bloodstream (*5, 6*). Thus, radon in drinking water could potentially produce adverse health effects in addition to lung cancer.

Policy Background

Drinking water quality in the United States is regulated by the Environmental Protection Agency (EPA) under the Safe Drinking Water Act originally passed in 1974. In the 1986 amendments to the act, the EPA was specifically directed to promulgate a standard for ^{222}Rn as one of several radionuclides to be regulated. Because of delays in implementing the regulation, the EPA was sued by the Bull Run Coalition. In the resulting consent decree, the EPA agreed to publish final rules for radionuclides in drinking water, including ^{222}Rn, by April 1993.

The EPA proposed national primary drinking water regulations for radionuclides in 1991. Because radon is a known carcinogen, its maximum contaminant level goal (MCLG) was automatically set at zero (0 Bq m^{-3}). A maximum contaminant level (MCL) of 11 kBq m^{-3} was proposed because that concentration could be measured routinely with an uncertainty of no more than 30%. Public comments on the proposed regulations suggested that the MCL for radon be set somewhere from less than 1 to 740 kBq m^{-3}; a large majority favored setting the MCL at higher than 11 kBq m^{-3}.

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TABLE 1. Summary of Number and Size of Groundwater Systems and Average Radon Concentrations in Water in 1994

system size	no. of community groundwater supply systems	population served ($\times 10^3$)	av radon concn (Bq m^{-3})	credible range of mean concn (Bq m^{-3})
very, very small, 23−100 people	16 600	1 000	31 000	24 000−40 000
very small, >100−500 people	15 400	3 900	25 000	9 300−32 000
small, 50−3300 people	10 000	13 800	10 500	7 700−15 000
medium, 3300−10 000 people	2 300	14 000	7 500	5 400−10 000
large/very large, >10 000 people	1 300	48 700	7 600	5 000−11 000
total	45 600	81 000	9 100	7 600−11 000

In 1992, Congress directed the Office of Technology Assessment to analyze the EPA health-rsik assessment and outline actions that could address regulation of radon, considering both air and water. Also in 1992, the Chaffee−Lautenberg Amendment to the EPA FY 1993 appropriation bill directed the agency to seek an extension of the deadline for publishing a final rule until October 1993 and to submit a report, reviewed by the EPA's Science Advisory Board (SAB), to Congress by July 1993. That report was to address the risks posed by human exposure to radon and to consider both air and water sources, the costs of controlling or mitigating exposure to waterborne radon, and the risks posed by treating water to remove radon. The SAB review of the report questioned the EPA's estimates of the number of community water supplies affected, the extrapolation of the risk of lung cancer associated with the high radon exposures of uranium miners to the low levels of exposure in homes, and the magnitude of risk associated with ingestion. The SAB report also emphasized that the risk of cancer from radon in domestic settings was a multimedia issue and that the risk for radon in water must be considered within the context of the total risk from radon, which is dominated by radon in indoor air. The Office of Management and Budget also expressed concern about the EPA's analysis of the cost of mitigation. In the Agency's FY 1994 appropriation bill, Congress ordered the EPA to delay publishing a radon in drinking water rule.

The 1996 Amendments to the Safe Drinking Water Act required the EPA to contract with the National Academy of Sciences (NAS) to perform a risk assessment of radon in drinking water and an assessment of the health-risk reduction benefits associated with various measures to reduce radon in indoor air. The EPA was also required to publish an analysis of the health-risk reduction and the costs associated with compliance with any specific MCL before issuing a proposed regulation and to promulgate an alternative maximum contaminant level (AMCL) if the proposed MCL is less than the concentration of radon in water "necessary to reduce the contribution of radon in indoor air from drinking water to a concentration that is equivalent to the national average concentration of radon in outdoor air". Under the law, states may elect to allow utilities whose water has radon concentrations higher than the MCL but lower than the AMCL to use a multimedia approach to mitigation. The multimedia programs to mitigate radon in indoor air may include "public education; testing; training; technical assistance; remediation grant, loan or incentive programs; or other regulatory or nonregulatory measures". If a state does not have an EPA-approved multimedia mitigation program, a public water supply in that state may submit such a program to the EPA directly. Public water supplies exceeding the AMCL and choosing to institute a multimedia mitigation program to achieve equivalent health-risk reductions must, at a minimum, treat their water to reach the AMCL. The EPA is required to evaluate the effectiveness of proposed multimedia programs on the basis of an assessment of such programs prepared by National Research Council of the National Academy of Sciences (*3*). This paper is a summary of the review and assessments conducted by Committee on Risk Assessment of Exposure to Radon in Drinking Water that was established by the National Research Council. The full committee report is given in ref *3*.

Critical Scientific Issues

It has been difficult to set a standard for radon, as opposed to other radioactive or chemical contaminants, in drinking water. For most contaminants, the risk assessment can be based exclusively on exposure from drinking water. Under these circumstances, an appropriate MCL can be set for each waterborne contaminant. However, ^{222}Rn presents a regulatory problem in that its efficient transfer from water into indoor air produces a risk from the inhalation of its decay products, which represents most of the actual risk associated with the drinking water, and yet this risk is small as compared to the radon in most homes that is present because of soil gas.

Because of the limited total volume of water used in homes, the large volume of air into which the radon is emitted, and the exchange of indoor air with the ambient atmosphere, the radon in water typically adds only a small increment to the indoor-air concentration. Specifically, radon at a given concentration in water adds only about 1/10 000 as much to the indoor-air concentration; that is, typical use of water containing 10 000 Bq m^{-3} radon will increase the air concentration by only 1 Bq m^{-3}. There is always radon in indoor air from the penetration of soil gas into homes, so only a relatively high concentration of radon in water will make an important contribution to the airborne concentration.

Even though water generally makes only a small contribution to the indoor radon concentration, the risk posed by radon released from water, even at typical groundwater concentrations, is estimated to be larger than the risks posed by the other drinking water contaminants that have been subjected to regulation, such as disinfection byproducts. Thus, in most homes, the risk to the occupants posed by indoor radon is dominated by the radon from soil gas, which is not subject to regulation, and a change in the radon in drinking water would produce a minimal change in the risk posed by airborne radon. That problem led to the suggestion that mitigation of radon in indoor air be considered an alternative means of achieving risk reduction equal to or greater than that which would be achieved by reducing the concentration of radon in drinking water.

Radon in water also presents a direct ingestion risk. Questions were raised with respect to the ingestion risk assessment that the EPA used in the 1991 proposed regulations and in the revised multimedia risk assessment of 1994. The questions were related to the applicability of some of the data used as the basis of the risk model and to the resulting assumptions that were used to estimate risk. The substantial uncertainties in the risks other than those posed by inhalation add to the problems of setting an appropriate MCL to protect public health. Thus, a reevaluation of the ingestion risks was needed. Each of these major scientific issues is addressed in our analysis.

National Average Ambient Radon Concentration. The ambient concentration of radon varies with distance from

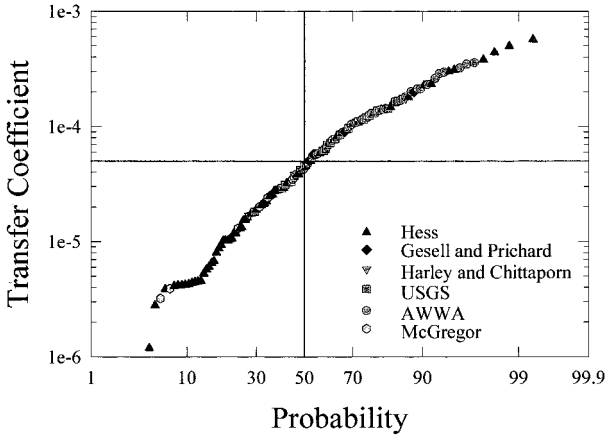

FIGURE 1. Probability distribution of the state-average measurements of the ambient ^{222}Rn based on the measurements of ref *7*.

and height over its principal source in the ground (rocks and soil) and other sources that can locally or regionally affect it, such as lakes, mine or mill tailings, vegetation, and fossil-fuel combustion. However, diurnal changes due to air stability and meteorologic events account for most of the variability. Average ambient radon concentrations were measured by the EPA over 9 seasons at 50 sites across the United States (*7*). The distribution of state-average ambient concentrations are shown in Figure 1. Most sites coincided with the capital city of the state, but these locations did not statistically represent the population across the United States, nor were the sites necessarily representative of ambient radon in each state. Thus, the data are not sufficiently representative to permit a population-weighted annual average ambient radon concentration to be calculated. However, this EPA data set is the only one with a fully national extent. An unweighted arithmetic mean of 15 Bq m^{-3}, with a standard error of 0.3 Bq m^{-3}, was calculated from these data, and the use of this value is recommended as the best available national ambient average concentration that can be currently estimated. After reviewing the other ambient radon concentration data that are available from specific sites [e.g., Ft. Collins, CO (*8*) and Soccoro, NM (*9*)], it was concluded that the average ambient radon concentration probably lies within the range of 14−16 Bq m^{-3} with high probability.

Transfer Coefficient. The transfer coefficient is the fraction of the initial average radon concentration in water that is contributed to the indoor-airborne radon concentration. It is defined as

$$\text{transfer coefficient} = \frac{\overline{\Delta C_a}}{\overline{C_w}} \qquad (1)$$

where $\overline{\Delta C_a}$ is the average increment in the indoor-air radon concentration and $\overline{C_w}$ is the average concentration of radon in the household water. Gesell and Prichard first estimated the transfer coefficient to be 10^{-4} (*10*). The transfer coefficient has been measured in a number of houses. The distribution of these values is shown in Figure 2. The sources of these data points are described in the Committee report (*3*). The median value is 4.5×10^{-5}, and the arithmetic mean value is 8.7×10^{-5} with a standard deviation of 1.2×10^{-4}. When graphed on a log-normal probability scale, the central portion of the distribution is fairly linear, and thus the geometric properties have also been calculated. The geometric mean is 3.8×10^{-5} with a geometric standard deviation of 3.3.

The average transfer coefficient can be estimated by a model used by Nazaroff et al. (*11*)

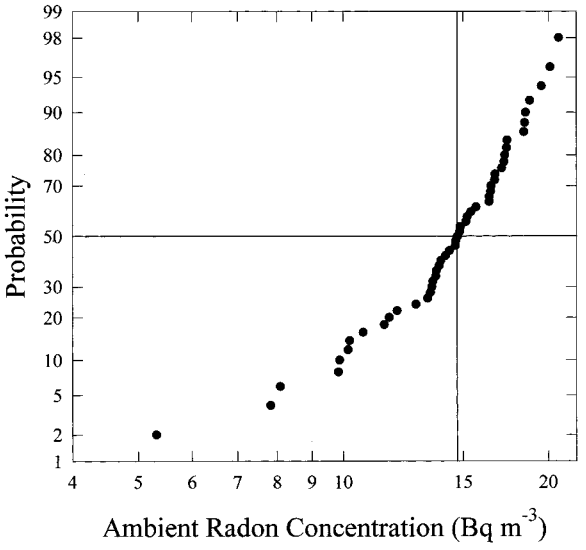

FIGURE 2. Probability distribution of the available measurements of the transfer coefficient. Data were obtained from refs *9* and *23*−*30*.

$$\frac{\overline{\Delta C_a}}{\overline{C_w}} = \frac{We}{\lambda V} \qquad (2)$$

where W is the time-averaged water use rate, e is the use-weighted transfer efficiency of ^{222}Rn from water to air, λ is the air exchange rate that is assumed to be uncorrelated with the water use rate, and V is the volume of the dwelling. New data were available for input to this model relative to those employed by Nazaroff et al. The detailed descriptions of these input values are presented in the Committee's report (*3*). There is a significant problem of estimating the distribution of ventilation rates in U.S. homes, and that leads to two different values. The model's average transfer coefficient was estimated to be either 9×10^{-5} or 12×10^{-5} depending on the choice of input parameter values. The average estimated from measurement data (8.7×10^{-5}) is in reasonable agreement with these values. Having considered the problems with both the measurements of the transfer coefficient and the measurements that are the input values into the model, it was recommended that the transfer coefficient be considered to lie in the range of 8×10^{-5}−12×10^{-5} and recommended that the EPA continue to use 1.0×10^{-4} as the best central estimate of the transfer coefficient that can now be derived from the best data that are currently available (*3*).

Risk Assessment

Biologic Basis of Risk Estimation. The biologic effects of radon exposure under the low exposure conditions found in domestic environments are postulated to be initiated by the passage of single alpha particles. The α-particle tracks produce multiple sites of DNA damage that result in deletions and rearrangements of chromosomal regions and lead to the genetic instabilities implicated in tumor progression. Because low exposure conditions involve cells exposed to single tracks, variations in exposure translate into variations in the number of exposed cells rather than in the amount of damage per cell. This mechanistic interpretation is consistent with a linear, no-threshold relationship between high-LET radiation exposure and cancer risk, as was adopted by the BEIR VI Committee (*1*). However, quantitative estimation of cancer risk requires assumptions about the probability of an exposed cell becoming transformed and the latent period before malignant transformations is complete. When these values are known for singly hit cells, the results might lead

to reconsideration of the linear no-threshold assumption used at present.

Ingestion Risk. The cancer risk arising from ingestion of radon dissolved in water has to be derived from the calculated dose absorbed by the tissues at risk because no direct studies have quantified the risk. Studies of the behavior of radon and other inert gases have established that they are absorbed from the gastrointestinal tract and readily eliminated from the body through the lungs. The stomach is of particular concern. The range of an α-particle emitted when radon decays is such that α-particles emitted within the stomach are unable to reach the cells at risk in the stomach wall. Thus, the dose to the wall depends heavily on the extent to which radon diffuses to and into the wall. Once radon has entered the blood, through either the stomach or the small intestine, it is distributed among the organs according to the blood flow to them and the relative solubility of radon in the organs and in blood. Radon dissolved in blood that enters the lung will equilibrate with air in gas-exchange region and be removed from the body.

New mathematical models of the diffusion of radon in the stomach and the behavior of radon dissolved in blood and other tissues were developed. A simplified, spherical model of the stomach and its contents was used to examine the sensitivity of the expected time-integrated concentration of radon at the depth of the cells at risk to the choice of diffusion coefficient. The result, based on a diffusion coefficient of 5×10^{-6} cm^2 s^{-1}, indicated that the integrated concentration in the wall was about 30% of that in the stomach content.

A physiologically based pharmacokinetic (PBPK) model of the behavior of radon in the body then was employed to examine the transfer of radon from the gastrointestinal tract into and through the body. Various investigators have assessed the retention of inhaled and ingested radon in the body, but their observations do not relate directly to the distribution of radon among the tissues. The PBPK model was formulated from information on blood flow to the tissues and on the relative solubility of radon in blood and tissue to determine the major tissue of deposition (which was adipose tissue) and retention within this tissue. The results calculated by the PBPK model are consistent with the observations regarding radon behavior in the body. Unlike previous estimates of the radiation dose, this analysis also considered that each radioactive decay product formed from radon decay in the body exhibited its own behavior with respect to tissues of deposition, retention, and routes of excretion.

The estimates of cancer risk are based on calculations with risk-projection models for specific cancer sites. The computational method was that described in the EPA's *Federal Guidance Report 13* (*12*). An age- and sex-averaged lifetime cancer death risk associated with ingestion of radon dissolved in drinking water of 1.9×10^{-9} at 1 Bq m^{-3} for ^{222}Rn was derived. Stomach cancer is the major contributor to the risk. The EPA (*13*) had previously estimated the risk to be about 6.3×10^{-9} at 1 Bq m^{-3} based on the work of Crawford–Brown (*4, 5*). Thus, the risk estimate recommended in this study is about one-third the value that was assumed by the EPA.

Inhalation Risk. Lung cancer arising from exposure to radon and its decay products is bronchogenic. The α-particle dose delivered to the target cells in the bronchial epithelium is necessarily modeled on the basis of physical and biologic factors. The dose depends particularly on the diameter of the inhaled ambient aerosol particles to which most of the decay products attach. These particles deposit on the airway surfaces and deliver the pertinent dose, and the dose can vary because of changes in particle size, by about a factor of 2 in normal home conditions.

TABLE 2. Committee Estimate of Lifetime Risk Posed by Exposure to Radon in Drinking Water at 1 Bq m^{-3}

	lifetime risk		
exposure pathway	male	female	U.S. population[a]
inhalation (smokers)	3.2×10^{-8}	1.29×10^{-8}	
inhalation (nonsmokers)	1.1×10^{-8}	0.41×10^{-8}	
inhalation (population)			1.3×10^{-8}
ingestion	0.15×10^{-8}	0.23×10^{-8}	0.2×10^{-8}
total risk (inhalation and ingestion			1.5×10^{-8}

[a] These rounded values combine the various subpopulations, with appropriate weighting factors taken from the 1997 U.S. Census.

The dose from ^{222}Rn gas itself is smaller than the dose from decay products on the airways, mainly because of the location of the gas in the airway relative to the target cells. The dose from ^{222}Rn gas that is soluble in body tissues is also smaller than the decay-product dose. Two of the underground-miner studies showed no statistically significant risk of cancer in organs other than the lung due to inhaled radon and radon decay products. The dosimetry supports that observation, although there is a need to continue the miner observations.

The risk of lung cancer associated with lifetime inhalation of ^{222}Rn in air at 1 Bq m^{-3} was estimated on the basis of studies of underground miners. The value was based on the risk projections in the BEIR VI report (*1*) and is the rounded average derived from the two BEIR VI models. The risk equals 1.3×10^{-4} at 1 Bq m^{-3}. The lung cancer risk to smokers is statistically significantly higher than the risk to nonsmokers. Given the adopted transfer coefficient of 1×10^{-4}, the risk of lung cancer posed by lifetime exposure to ^{222}Rn in water at 1 Bq m^{-3} was calculated to be 1.3×10^{-8}.

Summary of Risk Estimates. The risk estimates for ^{222}Rn in drinking water are summarized in Table 2. Although the Committee was asked to estimate the risks to susceptible populations such as infants, children, pregnant women, smokers, and elderly and seriously ill persons, there is insufficient scientific information to permit such estimation except for the lung cancer risk to smokers, which is presented in the table. The adopted lifetime risk of lung cancer for a mixed population of smokers and nonsmokers, men and women, resulting from the air exposure to radon from a waterborne radon concentration of 1 Bq m^{-3} is 1.3×10^{-8}. The adopted lifetime risk of stomach cancer for the same water concentration is 0.2×10^{-8}; the Committee could not make a distinction in risk for any specifically identified subpopulation

The Committee identified the issues of uncertainty and variability as likely to have important policy implications for the health impacts attributable to radon in drinking water. One overarching issue is how uncertainty and variability can affect the reliability of the estimated health impacts of a given standard or the health benefits of alternative standards and control strategies.

There is insufficient information to quantify the inter-individual variability in the cancer risk models that are available for this study. There is simply not enough information to characterize this type of variability. In contrast, radon exposure data, including concentrations in water and soil, transfer factors, and equilibrium factors, have been collected with sufficient resolution to explicitly represent population variability within the Unite States. However, uncertainties about the parameters, i.e., distributional moments describing this variability, can be quantified using methods used by the EPA (*14*) and Rai et al. (*15*). These methods use combined uncertainty/variability analysis methods to characterize the

relative importance of these two sources of variance in risk estimates. However, the Committee felt that the uncertainties in the quantification of the variability and that the uncertainties in the risk modeling were sufficiently large that a Monte Carlo parameter analysis to predict population variability in risk would provide a false sense of reliability in the risk estimates.

Multimedia Approach to Risk Reduction. The 1996 Safe Drinking Water Act Amendments permit states to develop a multimedia approach to health-risk reduction if the MCL is stringent enough as to make the contribution of the radon to waterborne indoor radon concentration less than the national average concentration in ambient air. Under those circumstances, an AMCL is defined such that the resulting contribution of waterborne radon to the indoor-air concentration is equal to the national average ambient radon concentration. The EPA administrator is required to publish guidelines including criteria for multimedia approaches to mitigate radon in indoor air that result in a reduction in risk to the population living in the area served by a public water supply that contains radon in concentrations greater than the MCL. There are implementation issues involved in a multimedia mitigation approach that have been explored through a sequence of scenarios that explore the possible options.

The ratio of the average ambient ^{222}Rn air concentration to the transfer coefficient defines the AMCL. However, the MCL will be determined by the Administrator of the EPA on the basis of a variety of considerations including their risk assessment, measurement technology, and economic factors; thus, a specific value has not yet been determined. On the basis of the Committee's recommended values for the average ambient radon concentration and the average transfer coefficient, the AMCL would be 150 000 Bq m^{-3} (about 4000 pCi L^{-1}). By statute, water in excess of the AMCL must be mitigated at least to the AMCL, and alternative means can then be used to provide a health-risk reduction equivalent to what would be obtained by mitigation of the water to the MCL. However, because of the relatively small cost difference between mitigating the water to the AMCL and to the MCL, the Committee believes that in most cases multimedia mitigation programs will probably not be considered for public water supplies with water concentrations in excess of the AMCL. For high radon concentration water, it will generally be most cost-effective to mitigate radon in water to the MCL.

For water supplies with radon concentrations between the MCL and the AMCL, the feasibility of implementing a multimedia mitigation program depends on the availability of homes in which the airborne radon concentration is high (greater than 150 Bq m^{-3}). The EPA has divided the country into three regions of different potentials for elevated indoor radon concentration. For water supplies in areas of low indoor-air radon potential, it will be difficult to identify and mitigate enough homes to achieve an equivalent or better health-risk reduction by treating the air. For such water supplies, it is unlikely that multimedia mitigation programs will be practical unless the water concentration of radon is only slightly above the MCL.

In areas of medium and high indoor-air radon potential, it is more feasible to mitigate a small number of high indoor concentration homes to provide an equivalent health-risk reduction at a cost less than the cost of mitigating the water. One way to achieve this is to install active (mechanical) systems to reduce radon entry into existing or new houses. Adequate testing (long-term measurements in the living space to reflect actual exposures) will be necessary to determine which existing houses should be mitigated. Routine followup measurements will be needed, both to determine the risk reduction achieved by the mitigation and to ensure continued successful operation of the mitigation systems. To ensure that equivalent or greater health-risk reductions have been achieved, there should be an excess of home indoor-air mitigation to the extent of probably 10−20% over the minimum calculated on the basis of current indoor radon concentration measurements.

There is a question of who would be responsible for the multimedia program costs. It is possible that the public water supply would have to recruit high indoor-air radon concentration homes actively and mitigate them. Alternatively, homeowners could be encouraged to mitigate their houses. Incentives could perhaps be used to get participation of homeowners in these multimedia programs. If the utility was responsible, then they would have to monitor and maintain the air mitigation systems routinely. This would require water utilities to become involved in air mitigation in individual homes, something with which they are likely to have little experience. If the state governments have the responsibility, it is not clear how they could ensure that the equivalent health-risk reductions had been achieved. Thus, there remains considerable uncertainties in how such a multimedia risk reduction program would operate.

Radon-resistant new construction methods could also be used. However, the Committee decided that the technical and practical bases of their implementation are still poorly developed. Evaluation of the baseline radon exposure would require use of radon-monitoring data from existing houses in the community of interest or estimates of average indoor concentrations based on calculated radon potentials for the region. Careful attention to the followup monitoring results would be important, both for determining how much radon reduction has resulted (on the basis of aggregate comparisons) and for determining whether radon persists at unacceptable concentrations.

Discussion

There are several significant policy issues associated with the regulation of radon in drinking water. However, the Committee did not explicitly address these issues since they were not part of its charge or its expertise. For example, there is the tradeoff of individual versus population risk. The mitigation of indoor-air radon concentrations in a small number of homes means risk reduction among only a few people who had high initial risk rather than uniform risk reduction for a whole population served by the water utility. This approach raises questions of equity among the various groups that are being exposed to differing levels of risk associated with radon. Equity issues would also result if the airborne-radon risks in one community were traded for the risks in another with a resulting identical or improved public health effect and a commensurate economic benefit to both communities. Thus, risk and benefit distribution considerations could play a large role in the evaluation of multimedia mitigation programs and might be the deciding factors in determining whether such programs are socially and politically feasible. In any planning process, a careful program of public education will be essential to give the public information as a basis of an adequate perspective and an opportunity for input on the tradeoffs in risks being proposed and of the health and economic costs and benefits that will be produced by the various alternatives [see, for example, Stern and Fineberg (*16*) or Renn (*17*)].

The EPA and the State agencies responsible for water quality will continue to be faced with the problem of the health risks associated with the presence of radon in drinking water. The increment in indoor radon that emanates from the water will generally be small as compared with the average concentration of radon already present in the dwellings from other sources. Thus, except in situations where concentrations of radon in water are very high, the reduction of radon

in water will generally not make a substantial reduction in the total radon-related health risks to occupants of dwellings served by the water supply. However, the risks associated with the waterborne radon are large in comparison with other regulated contaminants in drinking water. Thus, a focus limited within a single media regulation, the Safe Drinking Water Act, appears to dictate the need for action, while a multimedia consideration across programs and options for risk reduction suggests that such action may not be cost-effective for the overall benefit of public health. While a variety of regulatory, management, and philosophical approaches may be adopted to address this conflict (*18*−*22*), the availability in this case of an option that can both satisfy regulatory requirements and yield more cost-effective protection of public health suggests a basis for action. Using mitigation of airborne radon to achieve equivalent or greater health-risk reductions therefore makes good sense from both a regulatory and a public health perspective. As long as the equity issues associated with the multimedia approach and other related issues discussed above are considered as part of the efforts to obtain agreement by all of the stakeholders, implementation of multimedia mitigation programs in lieu of water treatment should receive further study and consideration by public water supplies and regulatory agencies seeking to reduce the risks of cancer from indoor radon. The results presented in this study can help provide a basis for such efforts.

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