Indoor Radon: A Case Study in Decisionmaking

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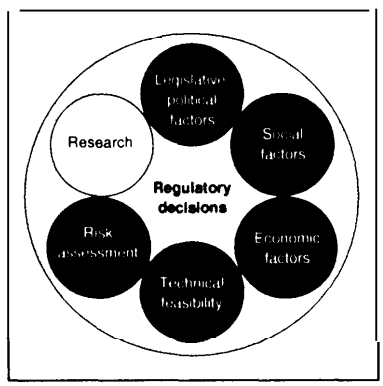
When radon gas, which originates in the Earth's crust, is emitted into the open air, it is rapidly diluted to the low 'background' or 'outside' levels that are found everywhere and are inevitable. When it is emitted into a home, school, or other building, dilution is slower, and the concentrations of radon inside structures are usually higher than the concentrations outside. These higher levels raise health concerns because studies have shown higher levels of radon are associated with higher rates of lung cancer in uranium miners and other workers exposed.

Responding to those concerns, Congress and the Environmental Protection Agency (EPA) have considered methods to reduce the risks posed by indoor radon. Most indoor radon enters buildings directly from the soil, and efforts to reduce those exposures include EPA programs to inform homeowners about the radon risks and how to reduce radon inflow into buildings. The private sector has also acted to reduce radon in homes by imposing requirements for measuring and, if deemed necessary, reducing indoor radon as a condition in real estate contracts.

EPA cannot, of course, regulate radon from soil because radon from that source enters homes directly without passage through any entity that can be regulated. Some radon, however, enters buildings through the water supply, and the agency can regulate radon in water just as it regulates other contaminants under the Safe Drinking Water Act (SDWA) (P.L. 93-523 and 99-339).



Some Members of Congress, including the Chairman of the House Committee on Science, Space, and Technology, asked the 0ffice of Technology Assessment (OTA) to examine an ' 'inconsistency' in EPA's approach to radon. The request, which

arrived after this study of health risk assessment research had begun, resulted in an analysis of issues related to radon, which is included in this report.

This chapter reviews and comments on the bases for assessing the risks posed by indoor radon and radon in water and discusses ongoing and possible future research projects. It also discusses the policy issues surrounding the congressionally identified "inconsistency' that arises because of differences between the goal of the Indoor Radon Abatement Act (IRAA) (P.L. 100-551), EPA's proposed level for the regulation of radon in drinking water under the SDWA, and the level of indoor radon at which EPA urges homeowners to take voluntary action to reduce radon infiltration (box 6-A). The inconsistency is quantitative: The IRAA sets the target for indoor air concentrations of radon as equal to concentrations in outdoor air. The proposed regulation under the SDWA sets a stricter level, imposing regulations on water suppliers so that emissions of radon from water to air would be reduced to about one-tenth of the level of radon in outdoor air. The voluntary action level EPA urges for homeowners-about eight-times higher than the level of radon in out-door air-is higher than either the IRAA goal or the SDWA regulatory limit. In response to the congressional request, this chapter also includes a rationale for "policy options for developing a consistent approach to reducing the risk from radon. '

HOW LARGE IS THE WATERBORNE RADON PROBLEM?

EPA (U.S. EPA, 1993b) estimates that about 19 million people are served by water systems that exceed its proposed regulatory limit for radon in water of 300 picoCuries per liter (pCi/L).[1] To reduce current concentrations that exceed that hit, the agency has selected aeration as the Best Available Technology (BAT). A number of aeration methods are available, which, according to EPA, will eliminate up to 99.9 percent of the radon as well as some fraction of other volatile, toxic contaminants from water (U.S. EPA, 1991). The volatility of radon, which makes aeration EPA's treatment of choice, results in waterborne radon being a problem primarily in water supplies that depend on groundwater because radon in surface water volatilizes into the outdoor air before it enters buildings.

Some background information is necessary to put the proposed regulation of radon in water into perspective. The proposed regulation depends upon the interpretation of epidemiologic studies, congressional actions in response to the projected regulation, some risk assessment models developed by EPA, and a series of reviews by EPA's Science Advisory Board (SAB).

RADON, MINING, AND INDOOR EXPOSURES

Radon is a decay product of radium, which itself is a decay product of the uranium found ubiquitously in the Earth's crust. Radon also undergoes radioactive decay, and it is the products of radon decay (called radioactive "progeny' or "daughters") that are associated with lung cancer. Radium and radon, of course, are especially abundant in radioactive deposits, and the Federal Government's demand for uranium to make atomic bombs during and after World War II resulted in a rush to mine such deposits. As a consequence, miners were exposed to high levels of radon. Beginning in the 1950s, results from

[1] A pico (p) Curie (Ci) is a measure of radioactivity. "Pico" means one-trillionth, so a pico Curie (pCi) is one-trillionth of a curie. One Curie is equal to 3.7×10^{10} radioactive disintegrations per second and a pCi is then 3.7×10^{-2} per second or 2.2 disintegrations per minute. The measure 4 pCi/L means that the radioactivity in one liter (L) of air (or water) produces 4 X 2.2 disintegrations per minute = 8.8 disintegrations per minute. Although pCi/L is the unit of measure most often used in the United States to express concentrations of radioactivity, in other countries, "Bq/rn^3" is more commonly used. A becquerel (Bq) is equal to 37 pCi, and one cubic meter (m^3) is equal to one L. Therefore, 1 pCi/L is equal to 37 Bq/m^3. (Usually, when conversions between the two units of measure are made and no calculator is available, the conversion factor is 40; that is, 1 pCi/L is about equal to 40 Bq/m^3.) Various detectors are available to measure radioactive disintegrations.

Box 6-A--Reducing Exposures to Radon: A Goal, an Action Level, and a Regulatory Standard

Nazaroff and Teichman (1990)[1] calculate that **current** exposures to radon are associated with about 15,700 lung cancer deaths annually. They estimate that 97 percent of those deaths will occur in smokers, and 3 percent will occur in nonsmokers. Indoor concentrations of radon are higher than those outdoors, and the Federal Government is directing several efforts at reducing indoor exposures. At present, there is a goal for reducing indoor radon concentrations, an action level to guide voluntary reductions, and a proposed regulation to reduce concentrations of radon in water.

A **Goal:** The Indoor Radon Abatement Act sets the goal of reducing indoor radon concentrations to the concentrations found outdoors-0.4 pCi/L. Currently, the average indoor concentration is about 1.5 pCi/L, with about 6 percent of all houses having concentrations greater than 4 pCi/L. The Environmental Protection Agency states that it is difficult to reduce indoor levels below 2 pCi/L (apparently for houses that have levels greater than 4 pCi/L).

An Action Level: EPA recommends that indoor radon concentrations be reduced to 4 pCi/L or below, a level considered technologically feasible for all houses. Reducing all indoor radon concentrations that are now greater than 4 to 2.7 pCi/L is expected to eliminate about 3,500 deaths (a reduction of about 17 percent). (The level of 2.7 pCi/L is the mean between the national average of about 1.5 pCi/L and the action level of 4 pCi/L.)

A Regulatory Standard: Under provisions of the Safe Drinking Water Act, EPA proposes regulating radon in drinking water so t hat t he concentration of radon in air that results from the volatilization of radon from drinking water is no more than 0.03 pCi/L. According to EPA, reducing all higher concentrations of radon in water to this level would eliminate 80 radon-associated lung cancer deaths annually (a reduction of about one-hatf of 1 percent).

[1] W.W. Nazaroff and K. Teichman, "Indoor Radon," *Environmental Science and Technology* 24(1990):774-782.

SOURCE: Office of Technology Assessment, 1993.

studies by the Atomic Energy Commission showed that lung cancer was more common in U.S. uranium miners than in other men, and studies of miners elsewhere—in Czechoslovakia, Sweden, and Canada—reported similar results (Brill, 1990).

Results from studies of miners identify radon as a hazard to human health, and assessing the risk that radon poses to human health is free from the problem of animal-to-human extrapolation that besets most health risk assessments. Those results identify radon as a hazard, but they leave risk assessors and decisionmakers with the problem of extrapolating from the effects seen at ''high exposure levels' in the miners to estimates of expected effects at the, generally, ''low exposure levels" found in houses, Such high-to-low extrapolations are a common issue with substances that are identified as a hazard to human health as a result of studying human populations. Usually, researchers accumulate the human data from studies of people exposed to high concentrations of chemicals or radiation in the workplace or in medical practice. Then those data must be used to extrapolate to the risks at lower ''environmental' exposures (U.S. Congress, OTA, 198 1; U.S. DHHS, National Toxicology Program, 1991).

In the late 1970s, Congress recognized some risks posed by nonoccupational exposures to radon and passed the Uranium Mill Tailings Radiation Control Act of 1978 (P.L. 95-604). That act directed EPA to set limits on radon emissions from inactive uranium processing sites and to establish acceptable levels for indoor radon in buildings associated with those sites.

That narrow focus on occupational or residual exposures that remained from closed-down mining and refining operations disappeared in 1984.

As the story is commonly told, it ended when Stanley Watras, an engineer at the Limerick Nuclear Power Plant in eastern Pennsylvania, passed through a radiation detector at the plant. He triggered the detector's alarm every day for almost 2 weeks in a row, which was surprising given that his co-workers seldom triggered the alarm. Mr. Watras guessed that his radioactive contamination might be coming from a source other than his work, and as an experiment one morning, he went directly to the detector before he went to his job. The alarm sounded. A subsequent inspection showed that Mr. Watras was bringing in radon from his house on his clothing and his person. Measurements in his house showed radon levels that resulted in a radiation dose well above those permitted in industrial settings (Taylor, 1990). What is less commonly reported is that Mr. Watras' house was directly over the tunnel of a uranium mine and that the house next door had only background levels of radon, about one-thousandth of those detected in his house (Moeller, 1989).

Although some scientists had identified indoor radon as a hazard by the late 1970s (Nero, 1990), Mr. Watras's saga began the process that widely publicized radon in homes as a health risk. Within 2 years, EPA (1986) published *A Citizen's Guide to Radon,* which" attributed between 5,000 and 20,000 lung cancer deaths annually to exposure to radon. A year later, when the agency (U.S. EPA, 1987) cataloged sources of environmental cancer risks, the numerical estimate for cancer mortality from indoor radon (between 5,000 and 20,000 annual deaths) was about the same as the estimate for mortality from skin cancer (10,000) caused by exposure to sunlight. Both of those estimates were much higher than risks of cancer associated with other sources (table 6-l). In the 1992 revision of *A Citizen's Guide to Radon,* EPA and the Department of Health and Human Services (DHHS) estimated that radon causes about 14,000 cancer deaths annually (U.S. EPA and DHHS, 1992). Although EPA's estimates have varied across the years, they have consistently associated several thousand cancer deaths with exposures to radon.

Table 6-1—Major Environmental Cancer Risks and Cancer Risks From Water

Source of risk	Estimated annual cancer mortality
Indoor radon	*5,000-20,000'*
Sunlight	10,000
All airborne cancer risks (excluding radon and environmental tobacco smoke)	2,267-3,294
Pesticides	3,075-6,150
Radiation in drinking water	37-730[b]
All chemicals in drinking water	215-430

a Other EPA estimates vary upwards from this range.
b Other EPA estimates vary within this range.

SOURCE: M. Gough. 1989. "Estimating Cancer Mortality." *Environmental Science and Technology* 23:925-930, *based* on U.S. Environmental Protection Agency. 1987. *Unfinished Business.*

Smokers are much more likely than nonsmokers to develop lung cancer as a result of radon exposure. Nazaroff and Teichman (1990) estimate that only 3 percent of the projected mortality from radon-associated lung cancer will occur in nonsmokers; EPA (1992a) estimates that 70 percent of deaths from radon-related lung cancer will occur in smokers, 24 percent in former smokers, and 6 percent in nonsmokers. In the 1992 *Citizen's Guide (U.S.* EPA and DHHS, 1992), EPA and DHHS point out that, for smokers, the most important step to reduce risks from radon is to quit smoking.

Currently, EPA recommends that homeowners take action to reduce indoor radon concentrations to 4 pCi/L, a level that can be reached in almost every home. The agency also states that levels in many homes can be reduced even more, to about 2 pCi/L (U.S. EPA and DHHS, 1992). This goal of 2 pCi/L is a little higher than the average indoor concentration in the United States (1.25 pCi/L), and it is from about 3 to 6 times higher than the outdoor average concentration of 0.3 to 0.5 pCi/L. Remediation methods recommended by EPA include increasing ventilation below slabs and sealing basements and foundations to reduce entry of radon.

Interest in the health effects of indoor radon has prompted more than a dozen epidemiologic studies comparing the rates of lung cancer in people who live in homes with higher levels of radon with those of people who live in homes with lower levels. Interpreting results from the various studies and attempting to reconcile conflicting results require that some attention be given to how the studies were designed and executed.

EPIDEMIOLOGIC STUDIES OF RADON-RELATED LUNG CANCER

Epidemiology is the study of the distributions of diseases in populations and the conditions that contribute to the appearance or progression of diseases. The most basic epidemiologic information is provided in a case report, which describes the occurrence of a disease (usually rare and therefore attracting attention) or a cluster of cases of a disease. Such reports identify populations for further investigation or study, but they provide no analysis of the putative links between exposure and disease, For example, a report of a rare form of muscle disease in a worker in a chemical plant would alert health professionals to a possible link between that disease and exposures to some toxic agent in the plant. But the disease could have occurred completely by chance, and further investigation would be necessary to examine the worker's exposures to specific chemicals and other studies would be needed to see whether other exposed workers suffered from the same disease or some precursor to it.

Beyond case reports, most epidemiologic research can be classified as one of three kinds: ecological studies, case-control studies, and cohort studies. Researchers have used all three types to investigate relationships between radon exposure and lung cancer. In general, ecological and case-control studies have been used to examine questions about indoor radon and case-control, and cohort studies have been employed for investigations in occupational populations.

In ecological studies, scientists compare rates of lung cancer in populations in geographical areas that have different average levels of exposure to radon. Case-control studies involve locating cases (persons who have lung cancer or the records of people who have died from lung cancer) and comparing the exposures of the cases to the exposures of controls (people who do not have lung cancer). In a cohort[2] study, scientists compare the rates of lung cancer in a group of people, such as miners, who share types, times, and intensities of radon exposure that differ from those of other groups. In ecologic and cohort studies of a disease as common as lung cancer, many cases of the disease will be expected in all the studied populations. Finding that the rate of the disease is higher in a population exposed to higher levels of radon is taken as evidence of a connection between exposure and disease after ruling out other factors that might account for the difference. For instance, if smoking was more common in the group with higher rates of lung cancer, a careful analysis would be necessary to ascertain the separate and combined effects of smoking and radon.

Ecological studies provide no direct comparisons between the exposures of individuals and their diseases. Such studies are relatively easy to do in areas in which records of disease incidence or deaths are available and in which there have been enough measurements of indoor radon that scientists can estimate average levels of exposure for the area. When completed, however, ecological studies provide no information about whether the persons who developed the disease had exposures near the average level or well above or below it.

Case-control studies are useful for studies of both indoor radon and exposures in the workplace, and they can provide information

[2] As an epidemiologic term, a' 'cohort' is a group of people who share certain characteristics (the word cohort originally identified one-tenth of a Roman legion).

about individual exposures and frequency of disease. One stumbling block for such studies is the difficulty of determining all past exposures to radon. For instance, many people move several times during their lifetimes, and a complete inventory of their exposures to indoor radon would require measurements in each of their homes. A further complication in interpreting such studies is how to make allowances for ''competing risks' that contribute to the risks of developing lung cancer. The most important is smoking; the second most important is probably environmental tobacco smoke (Brownson et al., 1992).

Most of the available quantitative information about the risks posed by radon comes from cohort studies of uranium miners that compare the rates of lung cancer among the miners with rates among other workers. Because of the latent period of 20 or more years between exposure to radon and the appearance of lung cancer, scientists who study rates of lung cancer among miners are most interested in their levels of exposure over two decades ago and more. That need for a long-term view complicates interpretation of the studies because accurate measurements are seldom available for past exposures.

Ecological Studies

The absence of direct ties between the exposure of an individual and his or her health status complicates interpreting the results of ecological studies. Using average (or group) information to estimate exposures results in the "ecological fallacy,' which links together specific health consequences among individuals and estimates or measures of average exposures. Only careful (and perhaps impossible) analysis would clarify whether the group measure was appropriate to describe the exposure of a person with a disease.

Although it may seem reasonable to conclude that people with an illness in a group exposed to a higher average level of radon were exposed to more radon than people exposed (on average) to lower levels of radon, there is no way to be certain of that. No single ecological study nor the complete set of such studies taken together will resolve the question of whether nonoccupational exposure to radon increases the rate of lung cancer.

Samet (1989) reviewed 11 ecological studies:

> In spite of crude exposure measures, most of these studies showed associations between exposure to radon decay products and the incidence of or mortality from lung cancer. Two studies of counties in the Reading Prong [the area of Pennsylvania in which Mr. Watras lived] are of particular interest because of the high number of homes in this region with high radon concentrations.

Of particular interest in discussions of radon in water and health risks are two studies reviewed by Samet (1989) that analyzed rates of lung cancer in relation to levels of radioactivity in water supplies. One of the two studies found an increase in rates of lung cancer in both men and women as a function of estimated greater exposures to radon; the other found an increase in men but not in women. The latter result would be an unexpected one if radon in household water made a significant contribution to the risk of lung cancer.

Overall, Samet (1989) concluded that 5 of the 11 studies were consistent with a correlation between exposure of higher levels of radon exposure and lung cancer, and one (one of the "water' studies) found an increase in men but not in women. Four of the 11 found no statistically significant increase in rates of lung cancer; one study reported an inverse correlation between exposures to radon and rates of lung cancer. That ''negative study and others done by the same scientist and his colleagues have received a great deal of attention, perhaps because the associations run counter to conventional ideas about radon and risk (Hanson, 1989).

Cohen (Cohen, 1990, 1992) investigated associations between rates of lung cancer and levels of radon using tens of thousands of measurements in

living areas and basements in houses in the 48 contiguous States of the United States. On the one hand, Cohen accepts that the ecological fallacy means that such studies cannot shed light on the question whether radon causes lung cancer because there is no way of knowing the levels of exposure of the people who develop lung cancer. On the *other* hand, he argues that the ecological fallacy does not prevent such studies from answering the question of whether a linear, no-threshold relationship exists between exposure to radon and lung cancer (see the discussion in ch. 2). According to Cohen, if that relationship is correct, cancer rates should vary directly with average countywide exposures. Cohen found that rates went down as exposures increased.

Immediately, objections were raised to Cohen's finding. For instance, how does smoking vary from county to county? Cohen's response was to compare cigarette sales in different States, factor that information into his analysis, and demonstrate that the negative correlation between levels of radon and lung cancer persists. What kind of correlation would be expected between current rates of lung cancer and current household exposures? Given the latent period between exposure and manifestation of disease, the exposures of interest occurred many years ago. Cohen has adjusted his analysis to consider that fact and has accumulated measurements of radon levels in far more houses than any other investigator. Nevertheless, other investigators have reported no replications of Cohen's results.

Taken together, the ecological studies present a confusing picture. Each additional study, whether it shows a positive association, no association, or a negative association, can be added to the tally, but no one study by itself nor all the ecological studies taken altogether will convince everyone about whether low-level radon is associated with lung cancer. Moreover, calculating reliable, quantitative estimates of risk from such studies is impossible.

Case-Control Studies

Case-control studies provide more definitive information about exposure than do ecological studies. In case-control studies of indoor radon, scientists (Blot et al., 1990; Schoenberg et al., 1990; Svensson, Pershagen and Klominek, 1989) often focus on women because fewer women smoke compared with men, and women typically spend more time at home.

Like the ecological studies, the case-control studies have yielded contradictory results. For instance, both Schoenberg et al. (1990) and Svensson et al. (1989) reported elevated levels of lung cancer among women who lived in houses with higher levels of radon. As the authors of those papers pointed out, the numbers of women included in the studies, especially the numbers of women exposed to higher levels of radon, were quite small. Only 24 of 433 women with lung cancer in the Schoenberg et al. (1990) study had lived in homes with concentrations of radon greater than 2 pCi/L. The small number of cases makes it difficult to interpret those studies, and many results showing excesses of cancer in the more highly exposed women were not statistically significant; that is, the excesses that were detected might have arisen by chance. Furthermore, no consistent relationship was found between smoking habits and lung cancer in the Schoenberg et al. (1990) and Svensson et al. (1989) studies, which introduces some uncertainty in interpretation because smoking and rates of lung cancer rates usually vary directly with each other.

Blot et al. (1990) studied women in a province of China and found that 'No association between radon and lung cancer was observed regardless of cigarette-smoking status, except for a nonsignificant trend among heavy smokers. ' Those authors go on to interpret their results as indicating that "projections (of cancer risk) from surveys of miners exposed to high radon levels may have overestimated the overall risks of lung cancer associated with levels typically seen in this

Chinese City. ” Letourneau et al. (1993) reported comparable results from a case-control study of 750 people with lung cancer in Winnipeg, Canada. They found that ‘‘no increase in the relative risk of any of the histologic types of lung cancer observed among cases was detected in relation to cumulative exposure to radon. ’

Lubin et al. (1993) prepared an analysis and comparison of the Blot, Schoenberg, and Svensson studies and concluded that any link between exposure to radon and risk of lung cancer is only weakly demonstrated in the studies, if it is present at all. Nevertheless, the fact that no increase was detected does not necessarily mean that none was there. It might have been present but undetectable because of the (small) size of the study.

Similarly, Ruosteenoja (1991) found “no significant correlation between the average radon exposure and incidence of male lung cancer. Yet, as the author pointed out, her study had little chance to detect the level of risk predicted from the miner studies. As in other studies, the small number of cases made it possible that any effect of radon that was present went undetected against the number of lung cancer cases expected regardless of the presence or absence of radon.

So far, case-control studies leave open the two possibilities that either the risk of developing lung cancer from exposures to indoor radon are zero (or at least below the limit of detection) or that it is compatible with the level of risk estimated from the miner studies. Additional case-control studies of sufficient size and ‘‘power’ might provide the information needed to determine whether risks projected from the miner studies are realized in people exposed to lower levels.

One alternative to a single large study is to carry out a meta-analysis of the already completed and soon to be completed studies and to combine those results to produce a more definitive answer. Meta-analysis is not a panacea, but it is a developing subdiscipline with applications to epidemiology (Dickersin and Berlin, 1992) and risk analysis (Society for Risk Analysis, 1993) as well as in health and behavioral sciences in general (Olkin, 1992). The Department of Energy has begun preparations for conducting a meta-analysis of case-control studies to begin in about 2 years when some ongoing studies will have been completed.

Researchers expect important findings from two ongoing studies in the United States that involve Midwesterners who tend to live in one house for long periods and who live in either Missouri or Iowa, States with relatively high radon concentrations. Of 524 homes examined in Missouri, 33(8 percent) had radon concentrations of 4 pCi/L or greater, and 8 (2 percent) had concentrations more than 8 pCi/L. Results already reported from that study verified predictions by Lubin et al. (1990) that people who move frequently have lower exposures than people who remain in a single home. Given the relative rarity of ‘hot homes, ’ a person who moves from such a home is more likely than not to move to a house with lower levels. Alavanja et al. (1992) report that 11 percent of Missouri women who lived in a single house for 30 years had been exposed to concentrations greater than 4 pCi/L for that time; 6 percent of women who lived in two houses had exposures that high; and none of the women who lived in three or more houses had such exposures. Investigators expect to complete their analysis of the relationships between levels of radon and cancer incidence in the 600 nonsmoking women with lung cancer in the study by mid-1993 and to publish them by the end of the year.

A research team at the University of Iowa is conducting the second Midwestern study. The investigators are studying women who smoke, to shed light on interactions between smoking and radon in cancer causation; studies of women who do not smoke are expected to identify any direct relationships between radon and lung cancer (Lynch, 1993). Equal in size to the Missouri study, the Iowa study includes a total of 600 cases of lung cancer and 1,400 controls. From the results of the EPA survey of radon in homes, it appears that about 70 percent of Iowa homes have radon concentrations greater than 4 pCi/L, and

those higher radon 1evels favor detecting associations between exposures and lung cancer-if they exist. The principal investigator of the study expects results to be published in late 1997.

Cohort Studies

Since the 1950s, scientists have studied the health of miners (in particular, uranium miners, who were frost seen to be at risk) and determined that exposure to radon increases the incidence of lung cancers. Samet (Samet, 1989) reviewed 20 studies of underground uranium miners and concluded that the data show consistent relationships between exposure to radon and elevated rates of lung cancer.

Exposure levels of the miners are expressed in working-level months (WLM), which are an approximation of the radiation exposure experienced by a uranium miner in 1 month's work. Miners in the various epidemiologic studies of radon had histories consistent with cumulative occupational exposures that ranged from 1 to 10,000 WLM. The current occupational limit for exposure to radon is 4 WLM annually (NRC, 1988), and a miner exposed at the current limit for 40 years would accumulate 160 WLM from his workplace. In comparison, the 70 years lifetime cumulative exposure of residents of homes with average concentrations of indoor radon is about 20 WLM (Samet, 1989).

As might be expected, cancer is far more frequent in the miners exposed to hundreds or thousands of WLM than in those exposed to lower levels. However, Bodansky (1990), in a review of those data, stated " . . . miner studies do seem to suggest a statistically significant positive effect, for cumulative exposures as low as 20 to 50 WLM. When the cumulative exposure is low, however, either due to low radon levels or short duration of employment, the data is vulnerable to confounding factors. ' Despite the suggested effect at exposures below 100 WLM, correlations between rates of lung cancer and higher levels of exposure dominate the risk assessment.

A question is raised whether the relationships seen between cancer and radon at hundreds of WLM, which are experienced in a few years, accurately predict cancer risks at levels of 10 or 20 WLM accumulated over a lifetime. Some scientists in EPA's Office of Science, Planning and Regulatory Support (Ulsamer, 1993) describe the problem this way: "The potential effects of differences in dose rates between miners (who are exposed for an average of 7 years to 20+ WLM/yr) and home residents (who are exposed for an average of 72 years to 0.22 WLM/yr from soil radon and 0.01 WLM/yr from water radon) . . . needs to be discussed." In this respect, indoor radon is a prime example of a problem in high-to-low-dose extrapolation.

Other Cancers

Henshaw and his colleagues (Henshaw et al., 1990; 1992) reported associations between levels of radon and the incidence of some cancers other than lung cancer in several countries. Those investigators relied upon ecological studies, making their results subject to the ecological fallacy, which reduces their value for decisionmaking. Although some scientists have treated the associations as possibly indicating a role for radon in other cancers (Pete, 1990), Doll (1992) points out that rates for none of the cancers that Henshaw et al. (1990) associate with radon exposure were elevated among miners, and a recently published study of 4,000 Czech miners found no association between radon exposure and leukemia (Anon, 1993a). Moreover, Miller et al. (1993) directly examined the possibility of an association between residential exposure to radon and the occurrence of a form of leukemia that had been suggested by Henshaw et al. (1990). They found no evidence for the association. Currently, there is little support for an association between exposures to radon and other cancers.

POLICY

EPA divides its regulatory programs along media lines-air, water, industrial wastes, and so forth. It has approached the issue of indoor radon as a media problem; thus, it has different policies toward radon entering buildings in air and water. The agency has not proposed regulating radon that is emitted directly from the soil, but it has proposed regulation of water suppliers as a method to reduce exposures to radon. Some scientists, Members of Congress, and other policymakers have recognized that indoor radon is only a single part of the larger issue of indoor air pollution, which presents assessment, remediation, and regulatory difficulties different from those associated with pollutants in outside air.

Air

In 1986, EPA estimated that 7 million U.S. homes had concentrations of radon above 4 pCi/L, the level at which the agency would recommend remedial action. Subsequently, as a result of its National Residential Radon Survey (EPA, 1992b) which involved measurements of radon in houses around the country, the agency reduced that estimate. Based on that survey, EPA now estimates that between 60,000 and 100,000 homes have concentrations of radon of 4 pCi/L or more and that the average home has a concentration of around 1.25 pCi/L.

The fact that EPA does not regulate airborne radon does not mean that those exposures have gone unaddressed. EPA distributed *A **Citizen's Guide to Radon in 1988*** and a revised document in 1992 as part of an information program to alert citizens about the risks from indoor radon. Some experts have questioned whether the guides provide the appropriate information. In particular, Nero (1992) and others have criticized EPA for urging that all houses be tested because they see that policy as distracting attention from homes in the areas with the highest concentrations of radon. The critics have also faulted EPA for not focusing on persuading residents to mitigate concentrations of radon in houses with levels of 20 pCi/L or higher. Those levels are higher than the exposures currently allowed for miners and other workers exposed to radon. According to those critics, EPA has dissipated the force of its message by calling for remediation in any house with levels greater than 4 pCi/L. Some experts have also objected to EPA's telling citizens to act on the basis of short tests of 2 to 7 days rather than testing for a year to obtain more accurate results.

Table 6-2-Comparison of Short-Term In-Basement Measurements of Radon in Air and Estimated Annual Exposures in Living Areas

If short-term result is:	Then estimated annual radon level is:
1 pCi/L	0.3 pCi/L
2	0.7
3	1.0
4	1.3
5	1.7
6	2.0
7	2.3
8	2.7
9	3.0
10	3.3
11	3.7
12	4.0

NOTE: pCi/L = pico Curies per liter.

SOURCE: Office of Technology Assessment, based on U.S. Environmental Protection Agency, *Reporting on Radon* (1989), personal communication from M. Reimer, U.S. Geological Survey.

The data in table 6-2 demonstrate the importance of appropriate testing techniques. Several years ago, EPA recommended that testing for radon be done with radiation detectors placed in the basements of homes and that the homes be kept closed up during the measurement period. As a result, the measurements were taken in the area of the house with the highest level of radon, regardless of whether anyone spent any time in that area, and under conditions that reduced dilution of indoor radon by outside air entering through open doors and windows. As shown in the table, measurements under those conditions were three times higher than the year-round

Table 6-3-Current Estimates of Radon-Associated Lung Cancer Deaths and Reductions Expected From Reducing Indoor Exposures

Source	Smokers	Former smokers	Non-smokers	Total
Estimated number of annual radon-associated cancer deaths				
Nazaroff and Teichman	15,200'		**500**	**15,700**
Environmental Protection Agency	9,600	3,200	800	13,700

Estimated number of annual averted deaths at reduced exposures					
	Exposures reduced to	Smokers	**Former** smokers	Non-smokers	Total
Nazaroff and Teichman	2.7 pCi/L	2,300		**200**	**2,500**
Environmental Protection Agency	4.0	1,500	**500**	**100**	**2,200**
Environmental Protection Agency	3.0	1,800	600	**200**	**2,600**
Environmental Protection Agency	2.0	2,300	**700**	**200**	**3,100**
Office of Technology Assessment	1.3[b]				**5,000'**

NOTE: pCi/L = pico Curies per liter.
a Nazaroff and Teichman combine current and former smokers in their calculations.
b Average indoor concentration of radon in the United States.
c Interpolated from figure 6-1.

SOURCES: W.W. Nazaroff and K. Teichman, "indoor Radon," *Environmental Science and Technology* 24(1990):774-782; U.S. Environmental Protection Agency, *Technical Support Document for the 1992 Citizen's Guide to Radon,* EPA 400-R-92-011 (Office of Technology Assessment, 1993).

average measurement of radiation in the living quarters of the house with ordinary ventilation and household traffic. EPA now recommends that measurements be made in the lowest living quarters of the house rather than in the basement.

Despite disagreements about the content of EPA's information materials, some people have clearly heard the message that indoor radon is a risk that can be addressed. Indeed, in some States and counties, radon inspections, like inspections for termites, are now part of real estate transactions. As a rule, inspecting for radon is not required by law or regulation but is part of the agreement between buyer and seller. For example, in Montgomery County, Maryland, most sales contracts require a 2-day sampling for radon. If the concentration is 4 pCi/L or higher, the buyer may require the seller to take remedial action to reduce the level.

Both Nazaroff and Teichman (1990) and EPA (1992a) have calculated the number of deaths from lung cancer that might be avoided by reducing exposures to indoor radon. Nazaroff and Teichman (table 6-3) estimate that reducing concentrations of radon to 2.7 pCi/L in all homes that currently have concentrations above 4 pCi/L would prevent 200 deaths per year from lung cancer among nonsmokers and 2,300 deaths per year among smokers (leaving about 12,000 radon-associated lung cancer deaths).

EPA's (1992a) estimates are quite similar. EPA currently recommends that all homeowners take action to reduce any exposure in excess of 4 pCi/L, and that level *is* often called the ''action level.' Reductions of all current exposures above the action level to 4 pCi/L are calculated to reduce the lung cancer death rate by 2,200, with 100 deaths being prevented in nonsmokers (table 6-3). The expected reductions in death rates increase with further reductions in exposures to radon: reducing all indoor exposures now above 3 pCi/L to 3 pCi/L would prevent about 2,400 deaths annually in smokers and former smokers and 200 deaths among nonsmokers; reducing exposures to 2 pCi/L, which EPA (1992a) considers near the practical limit for mitigation efforts, is calculated

to lower the annual death rate from lung cancer by about 3,100, leaving about 10,500 such deaths associated with radon (figure 6-l).

As is apparent from figure 6-1, the bulk of cancers associated with radon exposure occurs in the population exposed to low levels, below 2 pCi/L. The primary reason for that is that many more people are exposed to those levels than to higher levels. Given EPA's conclusion that it is impossible to reduce levels below 2 pCi/L in some houses, the practical lower limit on the number of deaths associated with radon maybe as high as 10,500. This estimate is based, of course, on extrapolations from the miners studies, and refinement of those extrapolations might reduce or increase the estimate of the number of cancers associated with radon.

Interpolating from the data on figure 6-1, OTA estimates that reducing all indoor exposures now above 1.25 pCi/L to that level, which is the U.S. average, would avert about 5,800 radon-associated lung cancer deaths annually (table 6-3). That would leave 7,900, a little over half, of radon-associated lung cancer deaths unabated.

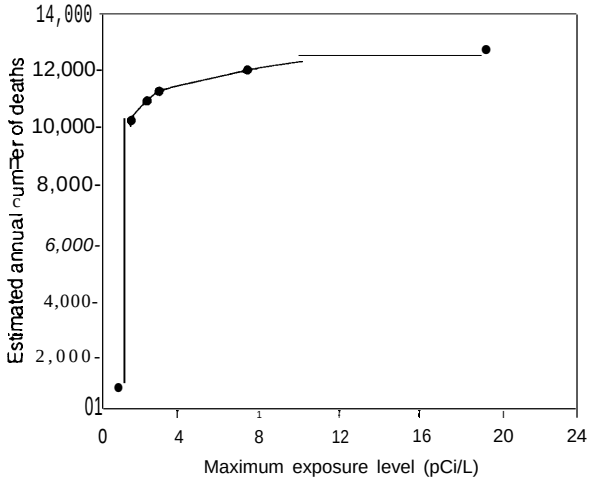
Because radon is present in all air, both inside and outside, it is impossible to have zero radon exposures. Thus, some risk of death from radon-associated lung cancer is always present if it is assumed that there is no threshold for radon-associated lung cancer deaths, and as is shown on figure 6-1, exposures to radon in outside air are associated with about 500 lung cancer deaths annually.

The National Research Council's (NRC, 1983) distinction between risk assessment and risk management calls for deliberations at two levels:

1. Is there a risk?
2. If there is one, what methods are most suited for its control?

For radon in homes, EPA's ***Technical Support Document for the 1992 Citizen's Guide to Radon*** (EPA, 1992a) provides the agency's reasoning behind choosing 4 pCi/L as the level at which homeowners should obtain more information about exposure and remediate to bring levels below that concentration. But, because EPA does not regulate radon in air, the Federal Government did not have to provide an administrative forum to debate whether the projected benefits of reaching 4 pCi/L of radon justified the associated costs. Figure 6-2 summarizes EPA's cost-effectiveness analysis for reducing concentrations of indoor radon to various levels. Reducing exposures to 8 pCi/L is expected to save lives at a cost of less than $0.5 million per life; the cost per life saved just about doubles (to a little less than $1.0 million) at 4 pCi/L and increases further at lower action levels.

Figure 6-1—Estimates of Deaths From Lung Cancer at Different Levels of Radon Exposure



SOURCE: Office of Technology Assessment, 1993.

Water

The Safe Drinking Water Act Amendments of 1986 require EPA to develop regulations for toxic chemicals in water. The agency has decided to regulate radon like any other waterborne carcinogen; it also considers radon to pose, quantitatively, the greatest risk of cancer from water (table 6-l). That regulatory process can be considered in two time periods. Before the summer of 1992, EPA was developing the regulation under its

usual procedures, but at that time Congress intervened in the process. Congress mandated EPA to make a reassessment of its estimates of risks and costs in relation to radon in water. In its action, Congress reflected some opinions expressed by EPA's Science Advisory Board.

THE SCIENCE ADVISORY BOARD'S COMMENTS

EPA's Science Advisory Board weighed into the radon in water issue in 1992. It w-rote a letter to the EPA Administrator:

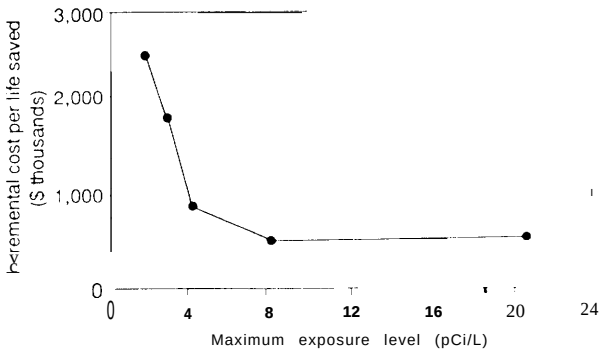
> ... to convey its concern about the inconsistent approach within the Agency regarding reducing risks from radon exposures in homes. . . .
>
> The purpose of this letter is two-fold: (a) to address the fragmented and inconsistent approach regarding reduction of radon risk, and (b) to provide our closing comments on the revised drinking water criteria documents that support the proposed regulations (Loehr et al., 1992).

The letter points out the proposed regulation would reduce the concentration of radon in water so that the amount that volatilizes from water to air would be more than 100 times smaller than EPA's action level (a voluntary guideline) of 4 pCi/L for indoor air. It also notes that that concentration is well within normal variations in levels of radon in homes, and about 10 times smaller than the average concentration of radon in outdoor air.

The SAB concluded that "Frankly, radon in drinking water is a very small contributor to radon risk except in rare cases and the Committee suggests that the Agency focus its efforts on primary rather than secondary sources of risk" (Loehr et al., 1992). The board also acknowledged that it understood that the SDWA required the regulation of radon in water. But it returned to a theme developed in its 1990 report *Reducing Risk* (U.S. EPA, SAB, 1990) and urged EPA to base its plans on "ongoing assessments of remaining environmental risks, the explicit comparison of those risks, and the analysis of opportunities available for reducing risks, rather than on past efforts at risk reduction or existing programmatic considerations. It went on to urge EPA to conduct a multimedia risk assessment of the options for regulating radon in drinking water and to include risks engendered by the treatment process and the disposal of any wastes produced from it. The board recommended that EPA develop and present better treatments of uncertainty in the water criteria documents.

Several of the SAB's recommendations, including the multimedia risk assessment, became part of the 1992 congressional mandate.

Figure 6-2—Cost-Effectiveness of Different Action **Levels for Reducing Indoor Radon Exposures**



SOURCE: Office of Technology Assessment, 1993.

PUBLIC LAW 102-389 AND THE MULTIMEDIA RADON RISK ASSESSMENT

Section 591 of the Housing and Urban Development, Veterans Administration, and Independent Agencies Appropriations Bill of 1992 put a hold on EPA's proposed regulation of radon in water. That section, commonly called the Chaffee-Lautenberg Amendment after its senatorial sponsors, directed EPA to complete a study July 6, 1993, that considers "the risks from various pathways of radon exposure-air and water, inhalation and ingestion. " The study was also to examine the costs of controlling various pathways, detailing the costs to households and communities (including small communities), and any risks posed by disposing of materials used to

remove radon from water. The study was to be reviewed by EPA's Science Advisory Board, and the board was to submit its recommendations to the EPA Administrator, who would then report to Congress. After completing the analyses and reviews, EPA was to issue regulations for radon in water by October 1, 1993.

Congress adopted the Chaffee-Lautenberg Amendment after the Senate narrowly defeated the broader Domenici Amendment that would have placed an outright moratorium on EPA's capacity to promulgate drinking water standards. According to Senator Chaffee's discussion of the amendment:

> The dispute here is about the relative risk of radon in drinking water. And since the Federal Government does not require that any steps be taken to correct the principal source of the risk, namely the gas that comes from the soil, the drinking water suppliers, quite rightfully, wonder why they should be required to clean up drinking water at a great expense. In other words, yes, some radon comes up with the drinking water, but more of it comes from infiltration through basement walls, et cetera.
>
> So there is much to be said for the line of reasoning for those who object to the testing of it in water. Thus our amendment delays promulgation of the radon standard until the end of 1993. During the interim, the EPA is asked to provide better data on the relative risk of radon from various sources, from water, from cellars, and so forth. So we can revisit that next year in 1993, because this postponement goes to the end of 1993 (Chaffee, 1992).

Given the time the amendment allotted for the risk assessment, EPA could do Little more than review the existing literature about radon risks and address specifically some uncertainties in its risk assessment. Although the conclusions from the reassessment were very close to those in EPA's (1991) water criteria document for radon in water, the multimedia risk assessment (or reassessment) did not answer all the questions raised by the SAB and by the amendment.

PROPOSED REGULATION

The SDWA imposes a goal of zero for concentrations of carcinogens in water. That goal is unattainable for radon (extensive aeration of radon-bearing water would discharge the radon into the air but there would always be radon at least at the concentration found in outside air). EPA bases its proposed regulation on its determination that the lowest ''practical quantification level" for radon in water is 150 pCi/L, and it set the regulatory maximum-contaminant level at that value in its proposed rule in 1991 (U.S. EPA, 1991). The half-life of radon is 4 days; that is, half of the radon decays in 4 days. Because EPA allows up to 4 days for transporting the water to the testing lab, the agency decided that a measurement of 300 pCi/L was the lowest feasible level for its regulation. Differences in procedures for measuring radon in air and water account for the fact that airborne measurements of 2 pCi/L of air are routinely obtained while EPA contends that measurements below 150 pCi/L in water are not practical.

There is general agreement that 10,000 pCi/L of radon in groundwater results in 1 pCi/L of radon in air from volatilization (U.S. EPA, 1991). Therefore, the 300-pCi/L limit on radon in water, if imposed, would mean that no more than 0.03 pCi/L of radon in indoor air would result from the waterborne radon. That concentration is 10 percent or less of the radon in outdoor air, and it would contribute about 5 percent to total indoor exposures. Supplying a house with water that contains 1,000 pCi/L of radon does not increase the airborne radon content by 1 pCi/L because when no water is running, there is little transfer of radon from water to air. EPA has carefully examined such things as how much radon is released into the air from water during showering, laundering, and flushing the toilet in order to estimate the contribution of radon from water to indoor air.

The Natural Resources Defense Council (NRDC) and the Friends of the Earth (FOE) dispute EPA's claim that 150 pCi/L in water is the lowest

practical quantification level (Olson et al., 1991). They point to published studies that show that changes in sampling and methods of analysis for radioactivity can lower the detection level to 25 pCi/L, which makes it possible to set a standard 12 times more stringent than the one EPA proposed. EPA's own analysis of amounts of radon in water casts doubt on 150 pCi/L being the minimal detection level because it reports on the number of water systems that exceed 100 pCi/L and presents some information about those between 50 and 100 pCi/L (U.S. EPA, 1991).

NRDC and FOE also point out that EPA calculates that the cancer risk at the proposed regulatory limit of 300 pCi/L is 2×10^{-4}. Not only is that risk level twice as high as the 10^{-4} level that is EPA's usual upper limit on acceptable risk; it is also much higher than the risks from other waterborne carcinogens, which are often in the range of 10^{-6}.

Arguing from the viewpoint that concentrations of radon in water below 300 pCi/L can be measured, NRDC and FOE also claim that imposing regulations on water supplies with concentrations below 300 pCi/L would greatly reduce exposures and risks at little additional cost. In particular, they calculate that such a regulation could avert twice as many cancers for an increase in cost of between 28 and 40 percent.

Water suppliers also disagree with EPA's proposed standard. They question whether the risk assessment is accurate and whether the proposed standard will save 80 deaths annually as EPA calculates; they also draw attention to the estimate that about 90 percent of the risk of lung cancer risk is confined to smokers. Like NRDC and FOE, but for very different reasons and by reaching very different conclusions, water suppliers draw attention to the fact that the proposed standard is based on a measurement level. Improvements in the capacity to measure radon in water (which NRDC and FOE contend are already here) could be translated into a constantly shifting, and constantly decreasing, standard. That kind of situation would leave the water suppliers facing an uncertain future of tighter standards and higher costs.

RISK

In 1991, EPA (1991) estimated that current concentrations of radon in water were associated with about 200 deaths annually from cancer, and the agency estimated that lowering all water supplies that were then higher than 300 pCi/L to 300 pCi/L would avert about 80 cancer deaths. At that time, EPA associated 80 percent of the risk from waterborne radon with radon that volatilized from water and was inhaled. Because of a National Research Council (1991) study that said certain adjustments were necessary to allow for differences between radon exposures in mines and in homes, EPA, in its reassessment, reduced its estimate of the number of cancer deaths associated with volatilized radon. At the same time, it increased its estimate of the number of deaths from cancer expected to result from ingested radon. When EPA (1993) added together the number of deaths from cancer that it associates with inhalation and ingestion of radon from water, the total came to about 160 annually, a number not different from the approximately 200 deaths from cancer that it previously associated with inhaled radon from water.[3]

EPA predicts ingested radon will cause cancer of the stomach and other digestive system organs. However, unlike other estimates of radon risk, the risk from ingested radon is not based on direct evidence of adverse effects in miners or other

[3] EPA calculates precise point estimates for risks along with a range of possible risks, For example, its estimate for annual deaths from ingested radon is 46, with a range of 11 to 212. Such precision is unwarranted because of uncertainties in measurement and models, and OTA prefers to present EPA results in less precise terms, such as 'about 50.' More importantly, the calculated range is not the same as the uncertainty that surrounds the estimate. As the Science Advisory Board noted (Loehr and McClellan, 1993), the uncertainty of the risk from ingested radon is so great that there may be zero risk.

populations. Indeed, there is no evidence for increases of those cancers in miners. Moreover, according to the Agency for Toxic Substances and Disease Registry (1992), there is no evidence for an association between groundwater radon and gastrointestinal cancers or leukemias. EPA bases its estimates of risk for ingested radon on modeling of the distribution of xenon gas in the human body and on the observed increase of stomach and other digestive system cancers in survivors of atomic bomb blasts. The modeling, which is the basis for estimating doses of internal radiation from ingested radon, is taken from a paper that has not been peer-reviewed. And, although both radon and atomic bombs release radiation, they release different kinds of radiation—alpha particles are released from radon, and gamma rays and neutrons from atomic bombs. Moreover, the two sources deliver radiation quite differently: ingested radon is a long-term internal exposure, and atomic bombs produced an external, one-time exposure. Those differences point to the problems involved in estimating the risk from ingested radon.

The upward revision of the number of deaths expected from ingested radon elicited several negative comments. In particular, Harley and Robbins (1993) estimated that the exposure of the stomach to radiation from ingested radon is about 100 times less than did EPA. EPA scientists defended their process (Chiu, Puskin, and Barry, 1993), but Crawford-Brown (1993), the author of the paper on which EPA depends for its estimate of radiation exposure to the stomach, agrees with Harley and Robbins that the estimate of exposure may be too high.

More fundamentally, Crawford-Brown (1993) objects to EPA's assuming that the mathematical equation it used to extrapolate risk is correct: "I believe the USEPA is both philosophically and scientifically far from the mark in suggesting that uncertainties in extrapolation equations are to be characterized . . . (as if) . . . there is no uncertainty in these equations . . . ." This comment questions the risk assessment based on miner data that EPA has used to estimate the cancer risks from radon in water. Some scientists within EPA have made parallel comments about the uncertainties in the dose-response equation that is used in EPA's risk assessment (Ulsamer, 1993).

Scientists at Brookhaven National Laboratory (BNL) responded to a request from EPA's Office of Research and Development that the Department of Energy (DOE) review EPA's risk assessment of radon in water. The resulting review was sent from DOE to EPA accompanied by a letter (Pelletier, 1993) that summarized the DOE position: "BNL concludes that the draft report contains significant flaws which seriously detract from its usefulness." The review itself is quite critical of EPA's risk analysis (Morris, Rowe, and Baxter, 1993). The BNL scientists agree with Crawford-Brown (1993) that EPA may have overestimated exposures from ingested radon by a factor of 100, and they point to a number of computational errors that they found in the EPA report. The EPA scientists who developed the risk assessment profoundly disagreed with the DOE review (Chiu, Puskin, and Barry, 1993) and responded to its summary comments.

In its review of the reassessment, EPA's Science Advisory Board was quite critical of the methods used to estimate cancer rates from ingested radon. It characterized the methods as more indirect than those used to estimate risks from airborne radon and concluded: "In the absence of direct evidence, it is not possible to exclude the possibility of zero risk from ingested radon" (Loehr and McClellan, 1993). As it did in 1992, the SAB drew attention to the small risk associated with radon in water as compared to the overall risks from radon. The SAB also, as in 1992, made comparisons between the number of deaths that might be associated with waterborne radon (about 160) and the 2,500 deaths that are expected to occur annually from radioactive potassium that occurs in the human body and the 500 or so expected from outdoor radon.

COSTS

The three parties-EPA, NRDC, and FOE, and the water suppliers-also disagree about expected costs. In 1991, EPA estimated that the costs to reduce radon to 300 pCi/L in all 25,907 water supplies that exceeded that level would be $1.6 billion in capital costs and $0.18 billion in annualized costs (U.S. EPA, 1991). The cost of averting a case of radon-associated cancer was estimated at $2.3 million. In February 1993, EPA increased those estimates. Currently, its best estimate is that 41,000 water supplies exceed 300 pCi/L and that the best estimates for capital and annualized costs are $1.8 billion and $0.26 billion, respectively (U.S. EPA, 1993a).

NRDC and FOE accept EPA's cost estimates for reducing concentrations to 300 pCi/L and use the agency's estimates to project the additional cost of reducing radon in water to lower levels (Olson et al., 1991). EPA's proposed regulation would reduce current levels by 80 percent. NRDC and FOE contend that reducing levels to 1 percent of current levels, which might double the expected health benefits, would cost only an additional 28 to 40 percent, and in fact, the greater reductions might be achieved at even smaller cost increments. (Experts who work for water suppliers have said that they would expect costs to fall as more efficient aeration systems are developed to remove radon from water.)

The SAB (U.S. EPA, SAB 1993) did not endorse EPA's proposal for wholesale adoption of aeration to reduce radon concentrations in water. EPA had considered and rejected granulated activated charcoal (GAC) as a control measure, in part because of the problems raised by disposing of the radioactive charcoal after its use. The SAB urged the agency to look again at GAC because of its potential to hold down costs as compared to the costs of aeration in some applications. The Board made no projections of the costs. The board also urged EPA to revisit its estimates of costs for water supplies of different sizes and to consult with the water suppliers to obtain more information. Whether aeration or GAC is used to remove radon, either technique allows the introduction of microbes into water supplies, and SAB (U.S. EPA, SAB 1993) cautions that "costs of disinfection, especially in small systems, needs to be reviewed thoroughly.

The Association of California Water Agencies commissioned an engineering study of the costs of bringing public water suppliers in California into compliance with the 300-pCi/L standard (Fensterheim, 1992). According to the association, the capital costs to bring 9,420 California wells into compliance would be $3.73 billion; the annualized costs would be $0.7 billion. According to one projection from the associations results, total national capital costs are expected of between $12 and $20 billion; those expenditures are expected to reduce total radon exposures by about 1 percent (Abelson, 1993).

The SAB (U.S. EPA, SAB 1993) compares EPA's estimate of $3.2 million to avert a death from lung cancer from waterborne radon to the EPA estimate that remediation to reduce airborne radon to 4 pCi/L will avert a death for about $700,000. The Association of California Water Agencies calculates much higher costs; it estimates that the cost to avert a death from radon-associated lung cancer would range between $65 and $87 million in California. The cost to avert a death from lung cancer in a nonsmoker in that State would be between $433 and $592 million. (The much higher cost for averting the deaths of nonsmokers results from the fact that lung cancer is so much rarer among them.)

Part of the reason for the California Association's much higher cost estimate is its survey that shows many more water supplies exceed the proposed 300-pCi/L regulatory limit than is estimated by EPA. The SAB (Loehr and McClellan, 1993) also points to uncertainty in the estimates of how many water supplies would be subject to regulation (''may seriously underestimate the number of community water systems impacted . . . ") and states that "this uncertainty

in exposure estimates ultimately impacts the costs of mitigation.

EPA has estimated that the average annual cost for radon reduction for houses served by the smallest water utilities would be $120; most houses are served by larger systems and would pay about $50 per year (Wilcher, 1991). Based on Association of California Water Agencies estimates, former Senator William E. Dannemeyer wrote to EPA that every household that has to pay for radon reduction would pay an extra $340 per year (Dannemeyer, 1991). The town of Hastings, Nebraska, has a population of 23,000 and water that exceeds EPA's proposed limit for radon in water. According to an analysis that the town conducted, a water treatment plant to remove the radon would cost $65 million and be the single largest drain on the town's treasury (Schneider, 1993).

NRDC and FOE do not accept that small water suppliers will bear sizable new costs as a result of setting a standard of 300 pCi/L or lower for radon in water (Olson et al., 1991). They argue that smaller suppliers could tie into larger suppliers to gain economies of scale or look for water that contains less radioactivity. Moreover, Olson et al. (1991) cite experts who state that technologies are available that are much less costly than those EPA considered in its 1991 cost estimates. In EPA's 1993 recalculation of estimated costs, total capital costs increased by 20 percent and annual costs increased by 44 percent, whereas the number of water suppliers increased by 60 percent, indicating that EPA had found some savings in costs per supplier.

The costs of the proposed regulation on radon in water regulation can also be compared with the costs of a public health measure that has become more expensive in recent years and that has produced public outcries for reducing the profits of pharmaceutical companies. The cost of childhood immunizations has increased from between $7 and $23 in 1982 to between $129 and $244 in 1992 (Orenstein, 1993). Even so, the annual cost of the radon-in-water regulation-estimated by EPA to be about $50 per family served by averaged-sized systems and $120 per family served by small systems-ranges from between a fifth to a little less than half the one-time cost of immunization. The estimate of the Association of California's Water Agencies of $340 per family per year for the radon-in-water regulation exceeds the one-time cost of immunization.

The continuing, annual estimated family cost of the regulation, which will affect about 1 percent of all exposures to radon, of between $50 and $340 can also be compared with EPA's estimate of the one-time cost of bringing indoor radon concentrations down to 4 pCi/L or lower. EPA (1992a) estimates for the one-time cost for remediation of a house ranges from $500 to $2,500 with an average of $1,200 and average operating expenses of $68.

SCIENCE ADVISORY BOARD COMMENTS, JULY 1993

The Science Advisory Board review gave EPA high marks for its general approach to the multimedia risk assessment, but it focused on areas such as estimates of the population exposed to concentrations of radon greater than 300 pCi/L in water, calculated risks from ingested radon, and capital cost estimates in which it thought the agency could make efforts to refine its approaches and calculations. A letter from the chair of the SAB Executive Committee and the chair of the SAB Chafee-Lautenberg Study Review Committee (Loehr and McClellan, 1993) returned to the Board's 1992 position that EPA should apply relative risk approaches in its consideration of risks from radon. The relative risk approach ''caLls for giving the highest priority to mitigating the largest sources of risks first, especially when the cost-effectiveness of risk reduction of such sources is high. '

As part of that approach, Loehr and McClellan (1993) encourage EPA to continue its efforts "to encourage voluntary actions to reduce indoor air radon in view of the cost effectiveness of this approach for reducing risks. " About radon in

water, they conclude that the proposed regulatory limit of 300 pCi/L is "the most costly in terms of costs per cancer death avoided. " They suggest that EPA also consider setting limits for radon in water at either 1,000 or 3,000 pCi/L. Even the higher numbers would result in water contributing no more radon to indoor air than is present in outdoor air.

Nero (1993) has also suggested alternatives to the 300-pCi/L limit on radon in water. Like the SAB, he suggests setting the water limit so that radon from water would make no more of a contribution to indoor air than does the radon in outside air, which would be in the range of 1,000 to 3,000 pCi/L in water. An EPA official also reported to a newspaper that agency staff were forwarding at least three options for a radon-in-water rule to the EPA Administrator: 300, 1,000, and 2,000 pCi/L (Anon, 1993 b).

Should a limit of 1,000 to 3,000 pCi/L in water be set, EPA could continue to accumulate information about the levels of radon in water, the number of water supplies with various concentrations, and the risks from ingestion of radon. The additional information would reduce the uncertainties in the estimates of risks, costs, and cost per life saved, and pave the way for alterations in the regulation if needed.

"Inconsistency" in EPA's Approach to Radon

The letter that requested this OTA examination of indoor radon cited the SAB 1992 concerns about inconsistencies in EPA's approach to reducing risks from radon. It contrasted the goals of the IRAA both with EPA's action level for indoor radon and with its proposed level for regulating radon in water under the SDWA: The IRAA goal is to bring indoor radon level down to those commonly found outdoors (0.1 to 0.5 pCi/L), whereas EPA urges that remediation be undertaken to reduce concentrations of radon in homes to 4 pCi/L or lower. In contrast, EPA's proposed regulation would set 300 pCi/L of radon in drinking water as the highest permitted level, limiting radon in indoor air to 0.03 pCi/L from this source (given the assumption that 10,000 pCi/L of radon in water produces 1 pCi/L of radon in air because of volitalization). Clearly, the goal, the action level, and the proposed regulation set different exposures as acceptable (box 6-A).

These inconsistencies are no surprise given the way that the goal, the action level, and the regulation were derived. Congress, in the IRAA, acknowledged that the outdoor level of radon in air is unavoidable and that concentrations cannot be reduced below that level. At the same time, reducing concentrations to that level would be as health protective as possible.

EPA, in setting the 4-pCi/L action level, accepts a risk of cancer from radon that is far higher than the 1 X 10^{-6} (one excess cancer per million people) exposed for a lifetime that the agency routinely uses as a goal in regulating exposures to toxic chemicals. *The Citizen's Guide to Radon* (U.S. EPA and U.S. DHHS, 1992) provides some examples of comparative risk; for instance, the risk that a nonsmoker bears from constant exposure to radon at 4 pCi/L is roughly the same as that person's risk of drowning.

The proposed radon-in-water standard under the SDWA is risk- and measurement-based. The level of 300 pCi/L of radon in water, set at what EPA had determined is the practical limit on quantification, was projected to reduce risks to about 2 X IN. In its preamble to the proposed rule, EPA raised the question of the significance of waterborne radon to the total radon issue: ''In evaluating the various alternatives for proposing a radon MCL [maximum contaminant level; the regulatory standard], EPA considered the critical policy questions of whether radon in water should be regulated like other drinking water contaminants, or whether it should be regulated more in accord with its importance compared to overall radon exposure. EPA decided to regulate radon as other waterborne contaminants, and the SAB (Loehr et al., 1992) criticized that action because

of the small contribution that waterborne radon makes to overall exposure to radon.

Congress's mandating of the multimedia risk assessment produced some refinements in EPA's risk and cost assessment, but whether it will make a difference in regulation remains to be seen. The risk estimate hardly changed at all, and, according to EPA% assessment, radon in water remains associated with a risk greater than 10^{-4}, which is the usual upper limit on the risk that EPA finds tolerable.

Indoor Air

Risks to health from contaminants in indoor air-lead paint, asbestos in buildings, environmental tobacco smoke (ETS), and other substances—have spotlighted the indoor environment as a source of hazards. In the 102d Congress, Representative Joseph Kennedy and Senator George Mitchell introduced the Indoor Air Quality Act of 1991 in the House and Senate, respectively (H.R. 1066 and S. 455). Had either bill passed, it would have authorized research, development, and demonstration projects concerned with improving air quality; the House bill would have imposed some regulations. Both Kennedy and Mitchell have introduced bills in the 103d Congress that focus on research and development.

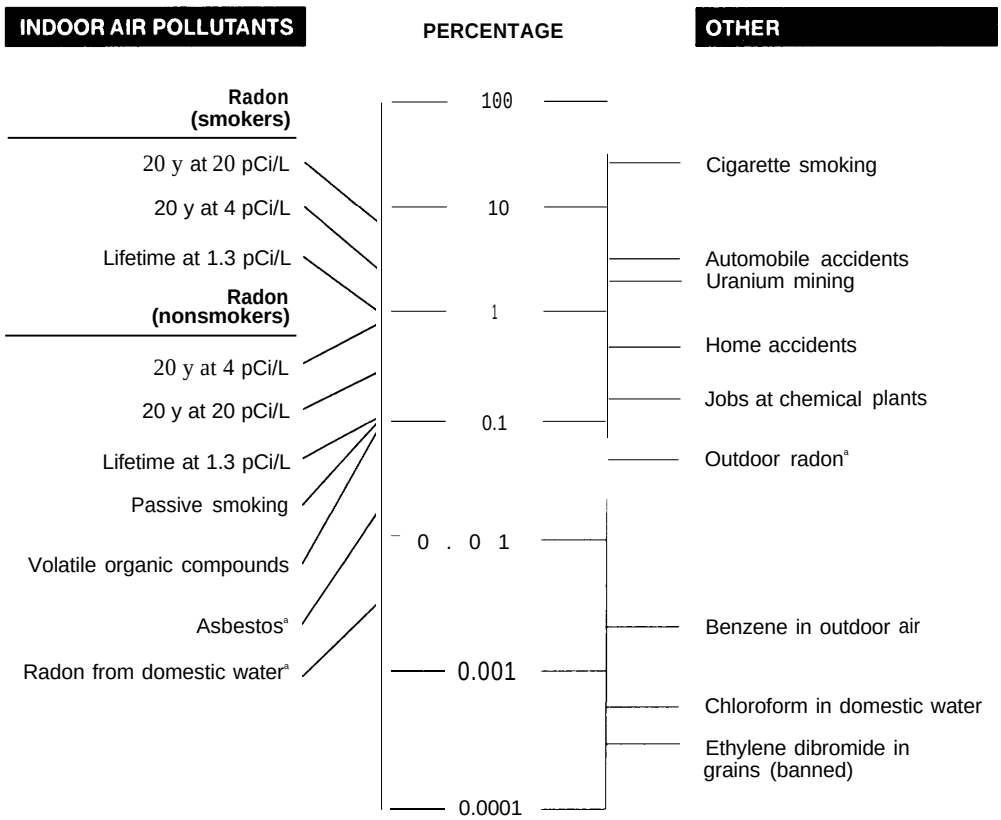
The contents of the bills demonstrate the complexity of the issues arising in legislation regarding the quality of indoor air. In addition to the agents mentioned above, indoor air can contain hazardous chemicals that are carried home in clothes and on the skin from the workplace; any number of volatile organic compounds from common household chemicals such as paints and soaps; and allergens that arise from pets, insects, molds, and mildews. The bills addressed indoor air quality in homes, which are probably the setting that is most often considered when thinking about indoor air, but they also treated air in educational facilities and commercial and Federal buildings. Clearly, legislation dealing with indoor air would apply to many substances and various kinds of buildings.

A decade ago, Spengler and Sexton (1983) discussed the special problems of indoor air pollution; more recently, Nero (1992) has argued that indoor air problems merit approaches different from other environmental issues. As shown on figure 6-3, risks from substances in indoor air fall over a wide range, with the radon-related risk of death for smokers as high as 8 percent and the risk from waterborne radon about 0.006 percent (more than 1,000 times lower). [Because of computational differences, figure 6-3 shows that the risk of death from waterborne radon is less that 0.01 percent, which is less than the approximately 0.02 percent (2 X l@) risk that EPA associates with waterborne radon.] The highest risk shown on the figure is for smoking, which increases the risk of premature death by about 25 percent. Accidents and certain occupations are associated with levels of risk around the level associated with radon.

EPA has concluded that the risks associated with indoor radon are greater than any other that it contends with (with the exception of risks of cancer posed by sunlight). The agency thus argues that indoor radon and waterborne radon require immediate attention. Nero (1993) counters that the risks from indoor radon are not put in the proper context when compared with outdoor risks. Instead, he maintains that indoor radon should be considered in the context of indoor risks that are typically higher than outdoor risks.

Considering the risks posed by indoor radon in the context of indoor risks would create monumental obstacles to setting the SDWA's limit of 10^{-4}(0.01 percent) on cancer risks as a consistent goal for risk reduction. As shown in figure 6-3, many indoor risks are far above the proposed regulatory limit for radon in water. It is very unlikely that the risks from radon in indoor air can be lowered from the nearly 1 percent associated with the average concentration in U.S. homes of 1.3 pCi/L or that the risk of fatal home accidents can be lowered from about 0.8 to 0.01 percent.

Figure 6-3-Estimated Lifetime Risk of Premature Death From Various Sources



SOURCE: Office of Technology Assessment, 1993.

Treating the risks presented by indoor air in a concerted fashion, which might result from legislation on indoor air quality, would probably lead to greater reductions in overall exposures than would be achieved under current laws. For instance, improved ventilation could be designed to reduce the concentrations of environmental tobacco smoke, volatile organic compounds, radon and other substances in the air, with an expected decrease in risks. In general, the solutions to indoor air problems are likely to follow similar paths-that is improving ventilation and filtration, considering the volatility of substances introduced into the indoor environment, and so forth. A single piece of legislation might facilitate considering the risks together rather than piecemeal.

The SAB (Loehr and McClellan, 1993) recognized 'that the large number of laws under which EPA operates makes it difficult to implement a relative risk reduction strategy across the Agency. . . . The SAB strongly encourages the Agency and the Congress to work together to consider changes in existing statues that would permit implementation of relative risk reduction strategies in a more efficient and effective manner.

THE FUTURE

"Enforcement' of the 4 pCi/L level for radon in indoor air is being accomplished through nonregulatory means, and given the possible liability concerns that might result if a house were sold with a higher level, realtors, attorneys,

buyers, and sellers probably will not alter their practices even if research findings show that risks at that level are smaller than is now believed. On the other hand, an increase in the estimate of risk would probably be quickly reflected in real estate transactions because of reasons of liability.

EPA's proposed regulation of radon has been delayed beyond October 1, 1993. The Agency is, reportedly, still deciding on its response to the Science Advisory Board's comments on its multimedia risk assessment. It is expected that work on the proposed regulation will follow that response. The regulation may be delayed for 1 year by Congress, if an amendment passed by the Senate is also approved by the House. Whenever EPA writes the regulation, the Science Advisory Board has offered alternatives to its proposed limit of 300 pCi/L radon in water. It is possible that the agency could set a higher limit that would, in effect, apply only to water systems that contribute a significant fraction of overall radon exposures. Setting a limit higher than 300 pCi/L would be expected to engender lawsuits from citizens and organizations concerned about risks from waterborne radon.

In contrast, if NRDC's and FOE's petition to set a stricter standard were successful, it would require that EPA reduce concentrations of waterborne radon, tightening the standard. Alternatively, Congress could relieve EPA of the responsibility for regulating radon in water, or shift it from the SDWA to the IRAA, or enact a new law on indoor air. Any shift might result in a standard different from 300 pCi/L.

No regulatory agenda requires a new study about the level of risk presented by indoor radon. The current risk assessment, based on the miner studies, is sufficient for regulatory action and indeed, being based on studies of humans, is more certain than animal-based risk assessments that form the basis for many regulations. Nevertheless, a convincing study of the risks associated with indoor radon would provide a great deal of information as well as a technical foundation for future policy decisions. A study that answered the question of whether the risks predicted from the miners studies were accurate would do more than inform the radon debate. It would also provide the first test of the accuracy of any extrapolated estimate of an environmental risk. Moreover, because it would provide more certain information about risks from radon in air at low levels, it would reduce the uncertainty of the risk assessment for radon in water because the major part of that risk is associated with inhalation.

Such a study would make a real contribution to scientific understanding and, depending on what it reveals, could have different effects on debates about regulatory levels. If the study revealed that the current levels were about right, it would confirm the appropriateness of the methods used to generate current risk estimates. If the study showed that current risk estimates were too low, EPA could tighten up the regulations. If, however, the results of the study indicated that the risks were lower than are now estimated, EPA might be confronted with the problem of backing off on some of its regulations and guidelines.

Doll (1992) is confident that studies now being performed will produce valid data about relationships between indoor radon and cancer within the next few years. In anticipation of the completion of those studies, both EPA and DOE are planning to carry out meta-analyses of the findings from those studies. Yet, despite Doll's optimism, there is no guarantee that the ongoing studies will produce a clear-cut answer about cancer risks from indoor radon. In that case, the government could assemble a group of experts to decide whether it is possible to design such a study, and design it, if it is feasible. A study of that kind would probably have to be larger than any done to date, and it would have to be carried out in areas (such as Missouri and Iowa) in which radon exposures are higher than average. To have scientific and political credibility, the study would have to be planned in an open process with explicit discussions of what results would be expected under different planning assumptions.

Furthermore, the planning would have to determine the study's chances of resolving the issue. If the chances were low, policymakers could decide not to go ahead with the study. Still another nonconvincing, nonconclusive study would not justify the expenditure of resources necessary for its completion.

At quite a different level of research, studies of molecular mechanisms of radon-caused carcinogenesis and of movements of radon in buildings (for examples, see DOE, 1993) and of carcinogenesis in general (see chs. 3 and 4) may provide more information about risks from radon. Scientists can design epidemiologic studies and decide, in advance of doing them, whether the studies have sufficient power to answer questions important for policymaking and how long the studies will take---certainly they will take years. But, advances in molecular studies, which may provide better estimates of exposure, pre-disease conditions, or mechanisms of action, cannot be put on a timetable. They may come in months, or they may take years.

The specific questions raised by radon maybe answered by congressional or EPA decisions that impose new regulations or that leave the current approaches intact. New epidemiologic results may inform those decisions by revealing more certain evidence of the level of risk posed by radon at environmental levels. And it is possible that research into mechanisms of carcinogenesis may shed some light on such risks. More generally, radon is a case that illustrates the difficulties posed by an environmental risk of uncertain size that reaches human beings through different media.

As of mid-October 1993, Senator Baucus had introduced a bill that would direct EPA to regulate radon in water by a method different from that now being considered. In addition, Senator Chaffee was considering introduction of legislation as was Representative Slattery. This legislative action indicates that policy on radon in water may well be set by legislative modifications to the SDWA.

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