Toward a More Realistic Appraisal of the Lung Cancer Risk from Radon: The Effects of Residential Mobility

ABSTRACT

Objectives. A consideration of the effects of residential mobility produces much more realistic estimates of typical individuals' radon exposures and mortality risks than those of the Environmental Protection Agency (EPA).

Methods. A model linking residential mobility, the distribution of radon in US homes, and lung cancer risk is used to simulate lifetime radon exposure, with and without mitigation of high-radon homes, for typical mobile individuals. Radon-related lung cancer mortality risks are then estimated for smokers and never-smokers.

Results. Most individuals residing in high-radon homes have equivalent lifelong radon exposures well below those they are currently experiencing. Consequently, actual lung cancer risks are generally well below those implied in the EPA's radon risk charts. For most people who mitigate high-radon homes, risk reduction is modest.

Conclusions. Radon may indeed be responsible for as large a population risk of lung cancer as the EPA estimates. However, caution must be used in interpreting the EPA's risk assessment for individuals; in many cases, mitigation will have little effect on residents' health risks. (*Am J Public Health.* 1996;86:1222–1227)

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Introduction

The Environmental Protection Agency (EPA) has labeled radon "probably the biggest public health problem we have," the source of an estimated 7000 to 30 000 lung cancer deaths annually in the United States.[1] The agency has mounted a campaign calling for universal voluntary testing of all homes for radon and mitigation when readings exceed 4 pCi/L (the agency's "action level"). The campaign has engendered considerable controversy. This is partly because the mode of risk assessment is itself controversial[2,3]: data on lung cancer in miners exposed to high levels of radon have been linearly extrapolated down to the low levels found in homes. The campaign is also controversial because of the "scare tactics" used by the EPA to encourage testing and mitigation. These have included televised public service announcements in which exposure to radon is likened to "hundreds of chest x-rays yearly" and skeletons are superimposed on images of young children.[3]

In its principal publicly disseminated document on radon, *A Citizen's Guide to Radon: The Guide to Protecting Yourself and Your Family from Radon,*[4] the EPA poses the risk associated with radon in two tables, one for never-smokers and one for smokers (reflecting the hypothesized interaction of radon with smoking, the leading cause of lung cancer[2,5]). The tables estimate the number of people who would get lung cancer out of 1000 people exposed to a given level of radon over a lifetime, and they present a "comparison risk." For example, smokers are informed that at the agency's action level of 4 pCi/L, "about 29 people could get lung cancer." This risk is characterized as "100 times the risk of dying in an airplane crash." The other comparison risks in the table are all dramatic, frightening events: being killed in a violent crime, drowning, dying in a home fire, being killed in a car crash.

Although the tables clearly state that the exposure would have to be over a lifetime, lay readers might be expected to interpret the table as indicating their personal risk, associated with the radon level in their own homes. The table headings encourage this perception: "Radon Risk if *You* Smoke" and "Radon Risk if *You*'ve Never Smoked" (emphasis added). However, because people move frequently throughout their lives (an average of 10 to 11 times[6]), their *current* radon exposure is not generally a good guide to their *cumulative* lifetime exposure; it is the latter that determines lung cancer risk. In particular, for people living in the high-radon homes the EPA targets for action, normal patterns of residential mobility mean that the vast majority will experience cumulative lifetime exposures equivalent to residing in homes having, on average, much lower radon levels.[7] The reason for this is that homes targeted for mitigation constitute only 7% of the nation's housing stock.[8] The average house has a radon reading of 1.25 pCi/L.[1] Thus, the small minority of people currently

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This paper was accepted February 1, 1996.

Note. The views expressed here are the authors' and do not necessarily represent those of the National Cancer Institute.

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living in high-radon homes are very likely to have lived in the past—and to live in the future—in homes with much lower levels of radon.

To illustrate, consider a 20-year-old smoker currently living in a home with 10 pCi/L of radon (found in fewer than 1% of all homes) but experiencing a typical pattern of residential mobility over his or her lifetime. Using the model described in this paper, we estimate that this individual will have a lifetime exposure equivalent to living permanently at 2.6 pCi/L, *without* mitigating any of the homes in which he or she ever resides. According to the EPA's *Citizen's Guide* risk charts, someone living at 10 pCi/L has a risk of radon-related lung cancer death of 71 per 1000 (see Table 1). We employ the same model used by the EPA to associate radon exposure with lung cancer death,[1,9] but we recognize the normal pattern of mobility. Thus, we find that this typical 20-year-old smoker's risk of a radon-related death is actually only 20 per 1000, or 28% of that estimated by the EPA.

Because the EPA's tables may tend to mislead, we present here more realistic estimates of the lung cancer risk associated with radon for typical individuals currently living in high-radon homes (≥ 4 pCi/L), based on the individuals' age, smoking status, and anticipated length of residence in their current homes. We also examine the benefit to these individuals of mitigating these high levels of radon in terms of reduced risk of lung cancer mortality, and we compare it with the benefit that would accrue to individuals who lived all their lives in a single home, as implied in the EPA risk charts. (The EPA's *Citizen's Guide* does not specifically estimate the benefit of mitigation. Rather, the reader is left with the possible inference that mitigation will largely "solve the problem," which it will not do, as we discuss further on.)

As we explain later in this paper and elsewhere,[7] our differences with the EPA with regard to individual risk do not translate into differences in our estimates of population risk.

Methods

To demonstrate the effects of normal residential mobility on cumulative radon exposure and hence on lung cancer risk, a previous paper presented and described simulation results from a model that integrated residential mobility into a radon exposure and lung cancer risk model.[7] The present paper uses that model to produce realistic estimates of lifetime radon exposures for people based on age, smoking status, and expected length of residence in their current homes. Employing the same model used by the EPA (BEIR IV) to link cumulative exposure to lung cancer risk,[1,9] this paper then estimates these individuals' risks of radon-related mortality and compares those estimates with estimates found in the EPA's *Citizen's Guide* tables.[4] The reduction in lung cancer mortality risk associated with individuals is then examined, assuming that all current and future residences at or above 4 pCi/L are mitigated down to 2 pCi/L, the level that the EPA believes mitigation of high-radon homes can achieve on average.[1] These estimates are compared with estimates of reduced mortality for the EPA's hypothetical lifetime residents of high-radon homes.

To be consistent with the EPA's approach, this analysis has not differentiated between men and women. Thus, all figures should be construed as representing an average of results for both sexes. In fact, risks for men will be higher than those found in the tables and risks for women lower, reflecting gender differences in the relative risk of smoking-related lung cancer and in background lung cancer rates.

Model

As described in our previous paper,[7] to estimate lifetime residential radon exposure and hence lung cancer risk, a model was developed that links three component models: (1) a residential mobility model that describes Americans' typical patterns of movement over a lifetime; (2) a residential radon exposure model that describes the distribution of radon throughout homes in the United States; and (3) BEIR IV, the aforementioned model relating radon exposure to lung cancer risk,[9] as modified by the EPA.[1]

The residential mobility model considers the effects of distance, age, and population size in determining the propensity of an individual to migrate to a specific state or move within a state or county. Parameters for this model were estimated from 1980 and 1990 Census data. A variety of evidence suggests that the model effectively captures the actual dynamics of residential mobility.[7]

For the radon exposure model, the distribution of radon levels in homes across the United States was built up from distributions of radon in large counties and in clusters of small counties. The distribution in each of these regions was assumed to be lognormal, consistent with previous research.[10] This procedure allows us to account for the fact that radon concentrations are less variable within regions than across them, and that most moves occur within regions or to nearby regions. Our exposure data were derived by combining the information contained in the EPA's State Residential Radon Surveys[11] and National Residential Radon Survey.[8]

TABLE 1—Lifetime Lung Cancer Risk for Current and Never-Smokers According to the Environmental Protection Agency (EPA)

Radon Level, pCi/L	Mortality in 1000 Smokers	Mortality in 1000 Never-Smokers
4	29	2
6	44	2
10	71	4
20	135	8

Source. All the figures in this table, except for the ones corresponding to the exposure level of 6 pCi/L, were taken from the EPA's *Citizen's Guide to Radon.*[4] The risk figures associated with 6 pCi/L were calculated according to the procedure described in the EPA's *Technical Support Document for the 1992 Citizen's Guide to Radon.*[1]

Analysis

The analysis requires two tasks: (1) determination of lifetime radon exposure, with and without mitigation of high-radon homes, for typical mobile individuals differentiated by current age, radon exposure in current homes, and length of expected residence in current homes; and (2) conversion of these age and exposure patterns into lung cancer mortality risks, differentiated by smoking status (smoker and never-smoker). Subjects for examination were individuals currently 20, 40, and 60 years old who resided in homes having radon readings of 4, 6, 10, or 20 pCi/L and who expected to continue living in those homes for 5, 10, or 25 years or permanently, the last being the assumption implicit in the EPA's risk tables.

Three of the four exposure levels are included in the EPA's *Citizen's Guide* tables; the analysis presented here has substituted 6 pCi/L, which is approximately the 50th percentile of high-radon

TABLE 2—Estimated Average Lifetime Radon Exposure and Associated Mortality for US Residents Currently 20 Years of Age, Accounting for Residential Mobility

Current Radon Exposure, pCi/L	Anticipated Length of Residence in Current Home: Permanent WOM	Permanent WM	5 Years WOM	5 Years WM	10 Years WOM	10 Years WM	25 Years WOM	25 Years WM
Lifetime exposure, expressed as equivalent constant exposure level								
4	4	2.6	1.9	1.5	2.1	1.5	2.7	1.8
6	6	3.2	2.3	1.6	2.6	1.7	3.7	1.9
10	10	4.3	2.9	1.9	3.4	2.0	5.5	2.2
20	20	7.2	4.2	2.5	5.7	2.6	9.9	2.8
Mortality in 1000 smokers								
4	30	20	15	11	16	12	20	14
6	45	24	17	13	20	13	28	15
10	73	33	22	15	26	15	42	17
20	139	54	32	19	43	20	73	21
Mortality in 1000 never-smokers								
4	2	1	1	1	1	1	1	1
6	2	1	1	1	1	1	1	1
10	4	2	1	1	1	1	2	1
20	8	3	2	1	2	1	4	1

Note. WOM = without mitigation; WM = with mitigation.

homes, for 8 pCi/L, which is included in the EPA's charts. (According to the EPA's surveys, 56% of all homes with radon readings of 4 pCi/L or more have readings between 4 and 6 pCi/L.[8]) Thus, inclusion of 4, 6, 10, and 20 pCi/L permits consideration of the consequences of radon for the low end of the "actionable" levels (4 pCi/L), the median level (6 pCi/L), and two very extreme levels (10 and 20 pCi/L, with fewer than an eighth of all high-radon homes having readings at or above 10 pCi/L).

Because the distribution of radon levels among residences is continuous (lognormal), the chance of finding a person in this model who is subject precisely to any predetermined radon level is minuscule. Therefore, the specific radon levels of interest were construed as an interval that covers ±5% of the designated reading. For instance, reference to a radon level of 4 pCi/L includes any radon level between 3.8 and 4.2 pCi/L.

To illustrate the simulation procedure, suppose the goal is to compute the annual average radon exposure over a lifetime for individuals living in homes with 4 (3.8 to 4.2) pCi/L of radon at age 20 who will remain in those homes for the next 10 years. First, a person's county of birth is selected with probability proportional to the 1990 population census. A mobility and radon history for that person is simulated until the individual is 20 years of age. If the individual's radon level at age 20 is not between 3.8 and 4.2 pCi/L, that person is discarded from the sample and another is simulated from birth. However, if the radon level at age 20 *is* between 3.8 and 4.2 pCi/L, the same radon level (and residence) is held constant for the next 10 years. After age 30, the person resumes normal patterns of mobility, dictated by the mobility model, until age 110 (the maximum age in most life tables).

To calculate the annual radon averages over a lifetime for the population of 20-year-olds living in homes with between 3.8 and 4.2 pCi/L of radon for the next 10 years, the experience of a large number of such individuals was simulated and their annual radon exposures were averaged. The same was done for each of the other age/exposure/length-of-residence cases. The number of individuals simulated in each case (3000 to 5000) was selected so that the width of a 95% confidence interval around the average exposure at age 70 was less than 10% of the estimated average. (Note that age 70 was chosen arbitrarily. Because there is no reason to believe that the variance of exposures at this age is larger (or smaller) than at any other age, any other age could have been used to determine the number of simulation replications with similar results. Note also that by following all the simulated individuals to age 110, this analysis proceeds as if mortality rates were uncorrelated with radon exposure. Obviously, this is not precisely correct, because people exposed to higher radon doses die at a higher rate; consequently, the estimated average exposures are upwardly biased at old ages. However, the contribution of radon to overall mortality is so low that the bias in the estimates is of no practical consequence.)

This procedure yielded estimates of the average annual radon exposures of the designated individuals over a lifetime, assuming no mitigation of high-radon homes. For the mitigation case, the identical simulated mobility histories were used, and all current and future radon exposures at or above 4 pCi/L were simply adjusted down to 2 pCi/L, the level that the EPA believes can be attained on average.[1]

To determine the lung cancer mortality risk for each case, the exposure data and smoking status were entered into the BEIR IV Time Since Exposure model,[9] as revised and used by the EPA in its analysis.[1] Thus, the resultant risk estimates, like those of the EPA, are subject to the uncertainty inherent in the BEIR IV model. Details on how the model was applied are presented in a technical appendix available from the authors on request.

Results

Table 1 presents mortality rates from the EPA's *Citizen's Guide* risk charts[4] by smoking status and radon exposure levels at or above the "action level" of 4 pCi/L. The table shows the radon-induced mortality in a cohort of 1000 individuals permanently exposed to various levels of radon from birth to a maximum of 110 years.

Tables 2 through 4 present the results of our simulation for individuals currently aged 20, 40, or 60 years. The rows in each table indicate the current radon exposure level at the given age. Columns 3 through 8 indicate the length of time the individual expects to continue living in the current home; columns 1 and 2 are equivalent to the EPA case of an individual who spends an entire lifetime at the indicated radon exposure level. In all the results presented below, when we

say that someone lives (or plans to live) in a house for *y* years, we are assuming that he or she will stay in that residence for that period of time unless the individual dies before *y* years elapse. Our numbers in the first column differ from those of the EPA (Table 1) because we have taken into account that the 1000 individuals of the cohort have survived to the current age of 20, 40, or 60 years. In the EPA's charts, mortality is computed from birth and so the "current age" of the EPA cohort is 0. (Implicitly in the EPA analysis, smokers begin smoking at birth.)

In the upper third of Tables 2 through 4 are our estimates of average lifetime radon exposures, expressed in terms of the equivalent permanent exposure, for the case of no mitigation (the left-hand figure in each pair) and for mitigation of all current and future exposures above 4 pCi/L down to 2 pCi/L (the right-hand figure). (We define "equivalent permanent exposure" as the constant radon exposure that produces the same number of radon deaths from current age, when the 1000 individuals in the cohort are known to be alive, up to a maximum age of 110.) The middle third of each table gives the expected number of radon-related lung cancer deaths for smokers, without and with mitigation. The bottom third gives the mortality estimates for never-smokers. These mortality estimates are intended for comparison directly with the EPA's *Citizen's Guide* chart figures (Table 1). The estimates in Tables 2 through 4 may be used by individuals with those age, smoking status, exposure, and expected-length-of-residence characteristics to obtain a realistic assessment of their mortality risks from radon.

Our findings can be summarized as follows:

Typical mobile individuals residing in high-radon homes have equivalent lifelong radon exposures well below those they are currently experiencing, whether or not they mitigate. For example, all 20- and 40-year-olds currently residing in homes with 4, 6, or 10 pCi/L of radon and never mitigating will experience lifetime exposures equivalent to living permanently below the EPA's action level of 4 pCi/L; this is true except for those expecting to live for an additional 25 years in homes with 10 pCi/L (Tables 2 and 3). To take one specific case, a 20-year-old residing in a home with 6 pCi/L, close to the median value of high-radon homes, will experience a lifetime exposure equivalent to living permanently at 2.3 pCi/L if the individual does not mitigate and moves after 5 years, a common length of residence for individuals of that age (Table 2).

TABLE 3—Estimated Average Lifetime Radon Exposure and Associated Mortality for US Residents Currently 40 Years of Age, Accounting for Residential Mobility

Current Radon Exposure, pCi/L	Anticipated Length of Residence in Current Home							
	Permanent		5 Years		10 Years		25 Years	
	WOM	WM	WOM	WM	WOM	WM	WOM	WM
Lifetime exposure, expressed as equivalent constant exposure level								
4	4	3.2	1.9	1.6	2.1	1.7	2.6	1.8
6	6	4.4	2.3	1.8	2.7	1.9	3.5	2.0
10	10	6.7	3.2	2.3	3.8	2.3	5.3	2.4
20	20	12.6	4.9	3.1	6.4	3.2	9.6	3.3
Mortality in 1000 smokers								
4	31	25	15	13	17	13	20	15
6	46	34	18	14	21	15	27	16
10	75	52	25	18	30	18	41	19
20	142	94	38	24	49	25	73	26
Mortality in 1000 never-smokers								
4	2	1	1	1	1	1	1	1
6	2	2	1	1	1	1	1	1
10	4	3	1	1	2	1	2	1
20	8	5	2	1	3	1	4	1

Note. WOM = without mitigation; WM = with mitigation.

TABLE 4—Estimated Average Lifetime Radon Exposure and Associated Mortality for US Residents Currently 60 Years of Age, Accounting for Residential Mobility

Current Radon Exposure, pCi/L	Anticipated Length of Residence in Current Home							
	Permanent		5 Years		10 Years		25 Years	
	WOM	WM	WOM	WM	WOM	WM	WOM	WM
Lifetime exposure, expressed as equivalent constant exposure level								
4	4	3.7	2.2	2.0	2.3	2.0	2.4	2.1
6	6	5.4	2.7	2.5	2.9	2.5	3.1	2.5
10	10	8.8	3.9	3.4	4.3	3.4	4.6	3.4
20	20	17.3	6.3	5.1	7.1	5.2	7.9	5.2
Mortality in 1000 smokers								
4	25	23	13	12	14	13	15	13
6	37	33	17	15	18	15	19	15
10	60	53	24	21	26	21	28	21
20	113	99	38	31	43	32	48	32
Mortality in 1000 never-smokers								
4	1	1	1	1	1	1	1	1
6	2	2	1	1	1	1	1	1
10	3	3	1	1	1	1	2	1
20	6	6	2	2	2	2	3	2

Note. WOM = without mitigation; WM = with mitigation.

For the typical mobile individual, the current radon reading by itself is not a reliable indicator of lung cancer risk. To illustrate, we focus on the mortality

implications for smokers, who have dramatically higher radon-related mortality risks than do never-smokers, as seen by comparing the middle and bottom thirds of each of Tables 2 through 4.

Consider, for instance, 20-year-old smokers living currently at a radon exposure level of 10 pCi/L (Table 2). Such individuals experience very different radon mortality risks depending on their expected tenure in their current residences. If they live permanently in their current residences, they will face a radon risk of 73 deaths per 1000. However, if they have reached their current residences through normal patterns of mobility and remain there for 5, 10, or 25 more years, they will experience a radon mortality risk per 1000 of 22, 26, or 42, respectively. Thus, living in the same very high radon home for an additional 5 or 10 years translates into a radon mortality risk that is roughly a third the risk of the rare individual who never moves from his or her home. Even living in the same high-radon home for an additional quarter of a century implies a radon mortality risk less than 60% that of someone who never moves.

Current age is also an important determinant of radon risk, although age, like expected length of residence, is ignored in the EPA risk charts (Table 1). For example, 60-year-old smokers who live at 10 pCi/L and plan to stay in their current residences for 25 more years (if they survive that long) will experience a radon risk of 28 deaths per 1000 (Table 4), two thirds the risk experienced by an equivalent group of 20-year-old smokers.

Mitigation reduces but does not eliminate the risk of radon-induced lung cancer. This point is true independent of mobility. Individuals reading the EPA's *Citizen's Guide* may derive the false impression that mitigating their homes will eliminate radon-related risk. According to the EPA, however, mitigation of houses above 4 pCi/L will reduce radon levels to only around 2 pCi/L, with further reductions very difficult to achieve.[1] This implies that individuals who mitigate will still be at risk, exposed to a radon level higher than the indoor US average of 1.25 pCi/L.

To illustrate, consider smokers currently living at 6 pCi/L. For 40-year-olds (Table 3) under the EPA's implicit assumption of no mobility, mitigation will reduce radon risk from 46 deaths per 1000 to 34, a 26% reduction. However, the risk is far from being totally eliminated. In fact, these individuals, after mitigation, are still left at more risk than those living permanently at 4 pCi/L. Mitigation is proportionately less effective for people exposed to lower radon levels. For example, mitigation reduces mortality risk by only 19% for 40-year-old smokers living permanently at 4 pCi/L.

Mitigation effectiveness also depends on the age of the individuals who mitigate. The older the individuals, the less the risk reduction they obtain by mitigating their homes. Looking at different age groups of smokers living at 6 pCi/L and again assuming no mobility, we observe that for 20-year-olds, mitigation reduces radon risk from 45 deaths per 1000 to 24, a 47% reduction, and for 60-year-olds, from 37 to 33, an 11% reduction.

The qualitative nature of these conclusions still applies if we drop the assumption of no mobility. However, there are important quantitative differences introduced by the fact that typical individuals do not spend all their lives in the same residence, as discussed immediately below.

Compared with individuals who spend their whole lives in the same residence (the EPA's assumption), typical mobile individuals obtain a smaller reduction in risk by mitigating high-radon homes. As seen in Tables 2 through 4, mitigation reduces absolute lifetime exposure for people residing permanently in high-radon homes more than it does for more typical mobile individuals. To illustrate, consider the case of 40-year-old smokers currently exposed to 20 pCi/L of radon (Table 3). Under the EPA's assumption of no mobility, mitigation will yield a reduction in risk of 48 deaths per 1000 (from 142 to 94). However, for typical mobile individuals, a reduction in risk close to this magnitude can be achieved only if such individuals remain in their current residences 25 more years. A shorter tenure will produce smaller mitigation benefits: by mitigating this and all future homes above the EPA's action level, individuals who remain in their current homes an extra 10 years will obtain a reduction in radon risk of 24 deaths per 1000; those who remain 5 more years will obtain a risk reduction of 14. These figures are, respectively, 50% and 30% of the mitigation benefit implied by the EPA's assumption of no mobility. (Apparent exceptions to the general rule are artifactual, attributable to rounding.)

Discussion

Ignoring normal patterns of residential mobility leads to considerable overestimation of the lung cancer risk associated with radon for the vast majority of people living in the high-radon homes that the EPA targets for remedial action. For individuals currently living in the highest-radon homes and expecting to continue living in them for a relatively long period of time, mitigation can significantly reduce lifetime exposure to radon and, for smokers, the associated relatively high risk of lung cancer. However, that risk remains a fraction—typically less than half—of that which uncritical interpretation of the EPA's risk charts might suggest.

Our findings are particularly salient for the majority of residents living in homes with radon levels that are close to the EPA's action level (between 4 and 6 pCi/L). According to our model, typical mobile individuals residing in homes with 4 pCi/L face risks that are roughly half those implied by the EPA's risk charts; mitigation of such homes—and of all future homes exceeding 4 pCi/L—can reduce those risks by no more than 30% and usually by much less.

As we explain elsewhere,[7] given the EPA's assumptions, we concur with the EPA concerning the total *population* risk associated with radon. While mobility greatly reduces the variance of individuals' lifetime cumulative radon exposures, it does not affect the mean. Thus, if lifetime cumulative exposures are concentrated closer to the mean, the effect of mobility will be to concentrate radon-related mortality toward the average of the distribution as well. This means that people who currently live in high-radon homes are generally at much lower risk of lung cancer than is suggested by the EPA's risk charts, whereas people who currently live in low-radon homes are at greater risk than the EPA's charts indicate.

That the EPA has long understood that mobility will affect risk seems clear. In the current version of the *Citizen's Guide,*[4] published in 1992, the tables clearly label the risk as requiring exposure to the various radon levels "over a lifetime," even if the presentation of the charts tends to deemphasize this critically important variable. In a note toward the end of the first edition of the *Citizen's Guide,* published in 1986, the EPA states, "The risk estimates in this booklet are based on the assumption that you will be exposed to the radon level found in your home for roughly 70 years. As you evaluate your potential risk, therefore, you might consider the total amount of

time you expect to live in your home. But remember: other houses you have lived in—or will live in—may have the same or higher radon levels."[12(p12)] This note was dropped from the second edition. In any case, the caveat at its end might be deemed misleading, given that occupants of high-radon homes have relatively low probabilities of living in comparably high-radon homes in their past or future.[7]

It is not our purpose to question the EPA's motivation for presenting the risk charts as they have. Rather, our intent is to use the same risk model the EPA uses but to include ordinary patterns of residential mobility so as to determine a more realistic estimate of the risks posed by radon, and of the health benefits of mitigation, for typical citizens. As we have demonstrated, the radon-related risk of lung cancer for typical individuals living in high-radon homes is significantly lower than that suggested by the EPA's risk charts. Similarly, the benefits of mitigation, in terms of risk reduction, are considerably smaller than those that would be experienced by someone who mitigated a high-radon home in which he or she lived permanently.

These conclusions do not mean that we recommend ignoring high radon exposures. In ongoing research,[13] we demonstrate that universal compliance with the EPA's radon recommendations would be socially cost-effective. We conclude, however, that a voluntary approach to dealing with radon, as advocated by the EPA, is extremely unlikely to succeed. As a result of residential mobility, when homeowners remediate their high-radon homes, they subsidize the health of the future occupants of those homes more than improving their own.[7] As we discuss in our ongoing work, attainment of the public health goal of radon control will likely require explicit regulatory policy. □

Acknowledgment

This article was supported by grant number R01 CA57691 from the National Cancer Institute.

References

1. *Technical Support Document for the 1992 Citizen's Guide to Radon.* Washington, DC: US Environmental Protection Agency; 1992. EPA 400-R-92-011.
2. Lubin JH, Boice JD Jr, Edling C, et al. *Radon and Lung Cancer Risk: A Joint Analysis of 11 Underground Miners Studies.* Bethesda, Md: National Cancer Institute; 1994. NIH publication 94-3644.
3. Cole LA. *Element of Risk: The Politics of Radon.* Washington, DC: AAAS Press; 1993.
4. *A Citizen's Guide to Radon: The Guide to Protecting Yourself and Your Family from Radon.* 2nd ed. Washington DC: US Environmental Protection Agency; 1992. EPA ANR-464.
5. *Reducing the Health Consequences of Smoking: 25 Years of Progress. A Report of the Surgeon General.* Washington, DC: US Dept of Health and Human Services, Centers for Disease Control; 1989. DHHS publication CDC 89-8411.
6. Long LE. Changing residence: comparative perspectives on its relationship to age, sex, and marital status. *Popul Stud.* 1992;46: 141–158.
7. Warner KE, Courant PN, Mendez D. Effects of residential mobility on individual vs population risk of radon-related lung cancer. *Environ Health Perspect.* 1995;103: 1144–1149.
8. *The National Residential Radon Survey.* Washington, DC: US Environmental Protection Agency; 1993.
9. Committee on the Biological Effects of Ionizing Radiation, National Research Council. *Health Risks of Radon and Other Internally Deposited Alpha-Emitters. BEIR IV.* Washington DC: National Academy Press; 1988.
10. Nero AV, Schwehr MB, Nazaroff WW, Revzan KL. Distribution of airborne radon-222 concentrations in US homes. *Science.* 1986;234:992–997.
11. *The EPA/State Residential Radon Surveys.* Washington, DC: US Environmental Protection Agency; 1993.
12. *A Citizen's Guide to Radon: What It Is and What to Do about It.* Washington, DC: US Environmental Protection Agency; 1986. EPA OPA-86-004.
13. Courant PN, Warner KE, Mendez D. The cost-effectiveness of alternative approaches to residential radon control. Ann Arbor, Mich: University of Michigan; 1996. Working paper.