

Effects of Residential Mobility on Individual Versus Population Risk of Radon-related Lung Cancer

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The U.S. Environmental Protection Agency (EPA) does not consider the effects of normal patterns of residential mobility in estimating individual radon-related lung cancer risks. As a consequence, the EPA's population risk estimates may have little bearing on individual risks, and remediation of high-radon homes may have only small health benefits for the individuals who remediate their homes. Through a simulation analysis, we examine the effects of residential mobility on radon exposure and lung cancer risk. Given normal mobility, only 7% of eventual radon-related mortality among current 30 year olds will occur in the 5% currently living in homes above 4 pCi/l (the EPA's action level for remediation), in contrast with an estimate of 31% of deaths when mobility is ignored. Above 10 pCi/l, the no-mobility assumption implies 10.3% of deaths, compared to only 0.4% when mobility is taken into account. We conclude that knowledge of one's current radon exposure is not necessarily a useful guide to one's risk, especially for residents of the high-radon homes targeted for remediation by the EPA. The risk of such individuals is likely to be substantially lower than that implied in the EPA's risk charts. If people currently living in high-radon homes remediate their houses, the majority of the resulting health benefits will accrue to future occupants of their homes. *Key words:* computer simulation, lung cancer, radon, residential mobility, risk analysis. *Environ Health Perspect* 103:1144-1149 (1995)

Models that extrapolate mortality from the high-dose radon exposures of miners to the low-dose exposures typically experienced in homes imply that radon-222 and its decay products cause from 7,000 to 30,000 lung cancer deaths annually in the United States (1,2). Although this extrapolation is controversial (3-6), the U.S. Environmental Protection Agency (EPA) has labeled radon "probably the biggest public health problem we have" and has called for the testing of every home for radon, with remediation of all homes found to exceed the EPA action level of 4 pCi/l. The EPA believes that full compliance would avoid thousands of radon-related deaths annually (1).

The EPA has mounted an aggressive and controversial risk communication program intended to achieve this objective on a voluntary basis (3,7). The "Citizen's Guide to Radon" (8) includes a table that estimates the risk of lung cancer associated with living in homes with each of several levels of radon exposure. Risks are posed both in terms of lifetime risk of lung cancer per 1,000 people and in comparison with other familiar and more dramatic risks. For example, living in a home with 4 pCi/l is equated to "100 times the risk of dying in an airplane crash" for smokers and "the risk of drowning" for never-smokers.

The EPA's estimates for individual risk depend on the premise that individuals always have lived and always will live in their current residences, or at least that all of their residences will expose them (on average) to the same level of radon (8,9).

This assumption deviates significantly from actual experience, however. This is particularly important for the group the EPA targets as being at highest risk and who, therefore, according to the EPA, should remediate their homes: the 5% of individuals living in residences estimated to have radon exposures of 4 pCi/l or greater. [The EPA estimates that 7% of homes have radon concentrations of 4 pCi/l or more (1). These homes house only 5% of the population, however, reflecting a small negative correlation between population density and radon levels.] Precisely because these homes fall in the upper tail of the distribution, other homes these individuals have occupied previously and will occupy in the future are not likely to expose them to comparably high levels of radon. Rather, on average their past and future homes will be closer to the mean of the distribution, 1.25 pCi/l. Since the average American moves 10-11 times over a lifetime (10), exposure to the current high levels of radon will occur during only a small fraction of that lifetime. Thus, typical persons currently exposed to high levels of radon will experience cumulative lifetime exposure reflecting a much lower average rate of exposure. As a consequence, the risk of radon-induced lung cancer for such persons will fall well below that estimated in the EPA's risk charts. Similarly, typical persons currently exposed to low levels of radon will experience cumulative lifetime exposure reflecting a higher average rate of exposure, meaning that their

risk will exceed that found in the EPA's charts, although still falling below average.

Mobility, and its consequences for assessment of individual risk, does not alter the EPA's conclusion about the aggregate mortality burden associated with radon (assuming the validity of the model used by the EPA to estimate the relationship between cumulative radon exposure and lung cancer risk, as we do throughout this analysis). Rather, mobility implies that the distribution of individuals' cumulative radon exposure is clustered much more around the mean than is the distribution of residential radon itself. In the model employed in this study, the variance of people's actual average exposures to radon is less than 30% of the variance of radon exposures among residences. As a consequence, the number of Americans at very high risk of radon-related lung cancer is dramatically smaller than would be inferred from a model that does not allow for mobility.

To assess the differences between typical individual risks and those presented in the EPA's risk charts, we used a simulation model that incorporates realistic patterns of residential mobility into the standard radon risk model. Although we produce specific quantitative estimates, our purpose is to develop a sound qualitative understanding of the relative importance of mobility in defining radon-related risk. Other analysts have recognized that mobility complicates determination of the relationship between cumulative exposure to radon and the incidence of lung cancer (11,12), but no one has explicitly studied the implications of mobility for individual versus collective risk.

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Methods

Model

To estimate lifetime residential radon exposure and hence lung cancer risk, we developed a model 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) a model relating radon exposure to lung cancer risk.

Residential mobility model. For a person of a given age, the residential mobility model uses age-specific mobility rates from Long (13), derived from 1980 U.S. Census data, to compute the likelihood of moving from one's current residence to another residence in a given year. There are three possible destinations for a move, in decreasing order of probability: 1) the same county, 2) the same state, but a different county, 3) another state.

Conditional on moving to another state, we use a gravity model (14), estimated with 1990 Census data, to evaluate the likelihood of an individual's moving to any particular state within the continental United States. The gravity model yielded the following equations, which estimate the number of people who migrate each year from state i to state j (M_{ij}) (adjusted $R^2 = 0.80$):

$$\log(M_{ij}) = -15.50 + 0.92\log(\text{Pop}_i) + 0.87\log(\text{Pop}_j) - 0.52\log(\text{Dist}_{ij})$$

if i and j are not contiguous states, and

$$\log(M_{ij}) = -3.09 + 0.47\log(\text{Pop}_i) + 0.50\log(\text{Pop}_j) - 0.31\log(\text{Dist}_{ij})$$

if i and j are contiguous states. Pop_i represents the population of state i and Dist_{ij} the distance between the most populous cities of states i and j .

Conditional on making an interstate move, we then compute the probability of moving from state i to state j , as:

$$P_{ij} = \frac{M_{ij}}{\sum_k M_{ik}}$$

The probability of moving to a specific county, given a target state, was taken to be the proportion of the population of the state living in that county.

Intrastate moves fall into two categories: intracounty and intercounty. Intracounty moves are assigned the age-

specific probability of such moves from Long (13). Within counties, movers are assumed to move at random with respect to the distribution of residential radon in the county. For intercounty moves, estimated destination probabilities are proportionate to each county's share of the state's 1990 population.

Note that mobility-induced changes in radon levels for an individual may be less random than is implied by our model. For example, apartment dwellers may tend to move to other apartments. The highest radon exposures are typically found in basements and first floors.

Radon distribution model. The distribution of radon levels in homes across the United States was assumed to follow a log-normal distribution, consistent with previous research (15). To estimate the geometric mean and standard deviation of the distribution, we used data from the EPA State Residential Radon Surveys (SRRS) (16) and the EPA National Residential Radon Survey (NRRS) (17).

The SRRS were conducted in 42 states and 6 Indian lands to characterize the distribution of radon in the lowest livable area of owner-occupied homes and to identify areas within the states with elevated levels of radon. The SRRS contain short-term radon screening measurements in over 63,000 randomly selected houses during the winter heating season. The EPA grouped counties within states into areas based on the geology of the states to identify zones of homogeneous radon levels. The EPA claims that the SRRS results provide an accurate representation of the distribution of radon at the state and substate (area) levels, but the estimates from the survey cannot be used to assess health risks directly because winter screening measurements can be up to 3 times higher than annual average measurements (18).

The NRRS was designed to provide an estimate of the frequency distribution of annual average radon concentrations in all lived-in levels of residences for each of the 10 EPA national regions. The NRRS contains information collected in 5,694 housing units used by the EPA to assess potential health risks associated with radon (18).

Combining the information contained in the two surveys, we estimated parameters to describe the annual average radon concentrations over all lived-in housing levels, at the state and area level. We transformed the EPA data as follows. First, we eliminated 9,169 observations from the SRRS representing negative radon readings (which we considered to be errors), observations above the second floor, and readings from Indian reservations and the states

of Alaska and Hawaii. Then we normalized all basement readings in the SRRS to an equivalent first-floor reading, using the average ratio of radon readings between basement and first floor for each EPA national region. Finally, we normalized the resulting radon readings of the SRRS to the average radon levels by EPA national region obtained from the NRRS.

With the transformed data, we estimated the parameters of the lognormal radon distribution for each intrastate area in each of the states included in the survey. To the states that did not participate in the SRRS, we assigned the radon geometric mean of the EPA national region to which they belong and the national geometric standard deviation, both estimated from the NRRS.

Lung cancer risk model. To evaluate the risk at each age a due to radon exposure, $r(a)$, we used the model developed by the Committee on Biological Effects of Ionizing Radiation (BEIR IV) of the National Research Council (19), which is the model used by the EPA. A recent study concluded that, as time since exposure increases, the influence of radon likely diminishes somewhat more than is reflected in the BEIR IV model (2). The use of an alternative model incorporating this phenomenon and others discussed in the study would change the quantitative details of our results. For the purposes of this research, however, differences in the results produced by BEIR IV and such alternative models are qualitatively indistinguishable, as confirmed by the senior author of the new study (J. Lubin, personal communication). We used BEIR IV to make our work directly comparable to that of the EPA. Thus, we accept all of the EPA's other assumptions, including implicit assumptions about time spent at home and exposure outside the home. The BEIR IV model expresses risk as a linear function of cumulative exposure to radon, subject to adjustments for current age and time since exposure, as given in the following equation.

$$r(a) = r_o(a)[1 + 0.025 \gamma(a)(W_1 + 0.5W_2)]$$

where $r_o(a)$ is the age-specific background lung cancer mortality rate from all causative agents; $\gamma(a)$ is 1.2 when age (a) is less than 55 years old, 1.0 when 55–64 years old, and 0.4 when 65 years or older; W_1 is the radon exposure expressed in working level months (WLM) incurred between 5 and 15 years before this age; and W_2 is WLM incurred 15 years or more before this age. A WLM is defined as the total exposure derived from a radon concentration of 1 working level (WL) for 1

working month (170 hr). A WL is the concentration of radon daughters in 1.0 l of air that results in the ultimate release of 1.3×10^5 MeV of α energy during complete decay. Under typical indoor conditions, a concentration of 200 pCi/l of radon-222 produces the α emission of 1 WL (20). Thus, for example, living in a house at 4 pCi/l for 1 year produces exposure over the year of about a quarter of a WLM. Because presentations to the U.S. public about the dangers of radon are usually expressed in picocuries per liter, throughout our paper we have converted WLM into the equivalent constant picocuries per liter.

The impact on radon-related risk of cigarette smoking, the principal cause of lung cancer, is estimated in the equation through differences in the background lung cancer mortality risks, $r_p(a)$, for smokers, former smokers, and never smokers.

Analysis

Using the small-area distributions of radon generated by our model, for each of 100,000 individuals, and for every year of life to age 80, we simulated radon exposure and geographic area of residence. The number of people originally assigned to each geographic area was proportional to the 1990 population of the area. Then, for each location and every year of life, the distribution of cumulative radon exposure was estimated. Finally, combining the estimated cumulative radon distribution, the BEIR IV model, and the distribution of the U.S. population by location and age, we computed radon-induced lung cancer rates by age and region.

This "mobility model" generates distributions of cumulative radon exposures and lung cancer deaths, by age and location. We compare these to distributions of the same variables generated using exactly the same initial conditions, but applying the effective assumption in the EPA's risk tables that there is no mobility: exposures and deaths are the same as if people never move from the residences in which they are born. Hence we refer to this as the "no-mobility model."

Results

The means of the distributions of exposure (and mortality) generated by the two models are approximately the same, the equivalent of residing permanently in a home with 1.18 pCi/l in the case of the no-mobility model and 1.22 for the mobility model. The small difference reflects the fact that people show a slight tendency to move toward higher-radon areas in the mobility model. This is consistent with cross-sectional data from the 1990 Census,

which show a slight positive correlation between age and average radon by area of residence. (These estimates differ slightly from the mean for houses, 1.25 pCi/l, due to population-weighting of the housing stock in the models.)

Although the means of the exposure distributions are nearly identical, the variance of the no-mobility model distribution is dramatically larger than that of the mobility model distribution, 3.5 and 1.0 pCi/l, respectively. Given the lognormal distribution, the substantial reduction in variance due to mobility means that a much larger proportion of lifetime exposures clusters within any given interval around the mean value. In the no-mobility model, 5% of lifetime exposures equal or exceed a lifetime of being exposed to the EPA's action level of 4 pCi/l, compared to 2% in the mobility model. Similarly, the very high-risk population with an average exposure equivalent to living permanently at or above 10 pCi/l is 0.7% of all people in the no-mobility model, but only 0.1% in the mobility model.

To illustrate how this occurs, Table 1 presents the lifetime mobility and radon exposure experience of a single individual from our simulation who has the following three traits: a residence with radon expo-

sure of 10 pCi/l at age 30, close to the average number of lifetime moves (10.4), and lifetime radon exposure close to that of the average person in the model exposed to 10 pCi/l at age 30 (the equivalent of living permanently in a home with 2.5 pCi/l). This typical high-exposure individual (at age 30) thus has a lifetime exposure equaling a quarter of that of someone who always lived at 10 pCi/l. The individual's effective lifetime exposure rate falls well below that which would be experienced by living permanently in a house with a radon concentration equal to the EPA's action level. Furthermore, if the individual had followed the EPA's recommendations and successfully mitigated the 10 pCi/l exposure down to 2 pCi/l when he or she first occupied the house, the person's cumulative lifetime exposure would have been the equivalent of living permanently at 2.18 pCi/l, a very modest reduction from the rate of 2.56 pCi/l without mitigation.

Tables 2 and 3 translate the exposure differences under the two models into differences in the distribution of radon-associated lung cancer deaths, employing a cohort longitudinal perspective (Table 2) and a national cross-sectional perspective (Table 3).

Table 2 shows the relationship between radon readings at age 30 for a cohort of 100,000 individuals, half male and half female, and expected lifetime lung cancer deaths due to radon. The no-mobility model finds that nearly one-third (30.8%) of all radon-related deaths in this cohort will occur in people currently (and in that model, permanently) residing in homes with radon readings at or exceeding 4 pCi/l. This proportion of radon-related deaths is six times the percentage of 30 year olds living in such high-radon homes. In contrast, the mobility model concludes that only 6.8% of deaths will be experienced by persons who are residents of these homes at age 30, representing less than 1.4 times their percentage of the cohort.

Table 1. Residential mobility and radon exposure history of a simulated individual in a home with 10 pCi/l at age 30

Age	Location	Radon exposure (pCi/l)
1	Erie, Pennsylvania	0.64
10	Newark, New Jersey	0.03
15	Somerset, Kentucky	0.70
18	Paducah, Kentucky	2.28
21	Bowling Green, Kentucky	0.40
28	Fargo, North Dakota	10.02
32	Fargo, North Dakota	1.52
36	Des Moines, Iowa	5.44
38	Cedar Rapids, Iowa	5.75
57	Cedar Rapids, Iowa	1.15
Effective average lifetime exposure		2.56

Table 2. Lifetime lung cancer mortality attributable to radon in a cohort of 50,000 males and 50,000 females, by exposure at age 30

	Radon, pCi/l				Total
	≤0.5	0.5–4	>4	>10	
No-mobility model	40 (8.7%)	277 (60.5%)	141 (30.8%)	47 (10.3%)	458 (100%)
Mobility model	158 (34.9%)	264 (58.3%)	31 (6.8%)	2 (0.4%)	453 (100%)

Table 3. Annual U.S. lung cancer mortality attributable to radon in a typical year, by exposure at time of death

	Radon, pCi/l				Total
	≤0.5	0.5–4	>4	>10	
No-mobility model	1,294 (9.8%)	7,990 (60.3%)	3,970 (29.9%)	1,232 (9.3%)	13,254 (100%)
Mobility model	4,379 (32.3%)	7,683 (56.7%)	1,495 (11.0%)	356 (2.6%)	13,557 (100%)

Even more dramatic, the no-mobility model predicts more than one-tenth of all radon-associated deaths (10.3%) in persons who live, at age 30, in residences with radon concentrations greater than or equal to 10 pCi/l. The mobility model predicts one-twenty-fifth as many radon-related deaths for this group (0.4%). The reason in both instances is that, followed to age 80, more than 90% of 30 year olds living at or above the EPA's action level will have experienced lifetime radon exposure less than they would have if they never moved from their residences at age 30. Table 1 illustrates this phenomenon. This analysis illustrates why it is difficult to find correlations between lung cancer deaths and the radon levels of decedents' homes. Even if cumulative radon exposure is an important cause of lung cancer, the correlation between radon level in decedents' final residences and their lifetime exposures may be too small to observe the underlying relationship.

Because the aggregate number of radon-related deaths is essentially the same in both the mobility and no-mobility models, the differences in deaths for people who experience high exposures at age 30 imply that a much larger proportion of total radon-related deaths is accounted for by people whose residences at age 30 have relatively low radon concentrations. According to the mobility model, more than one-third (34.9%) of eventual radon-related deaths will occur in people in residences below 0.5 pCi/l at age 30, compared to less than one-tenth (8.7%) in the static no-mobility model. In both models, close to 60% of deaths will occur in people currently residing above their minimal level of radon but below the EPA's action level.

We selected age 30 for this cohort analysis to illustrate how mobility affects radon-related risk in young adults, who have the greatest opportunity to reduce cumulative lifetime exposure to radon. The qualitative findings of this analysis hold for all age groups, although with less dramatic quantitative differences for the middle-aged and the elderly. For example, a cohort analysis of 45 year olds finds that 13.6% of eventual radon-related lung cancer deaths would occur among people currently living in homes registering above 4 pCi/l, compared with 6.8% for the 30 year olds (30.8% in the no-mobility model). For individuals living at or above 10 pCi/l, the share of radon-related deaths is 1.8% for the 45 year olds, compared to 0.4% for the 30 year olds (10.3% in the no-mobility model).

Table 3 shows the distributions of lung cancer deaths attributable to radon in a sin-

gle year for the entire nation, by current level of exposure. Both models predict essentially the same total mortality [slightly in excess of 13,000 deaths, consistent with previous estimates by the EPA (1)]. Compared to the mobility model, however, the no-mobility model estimates nearly three times as many deaths occurring in people currently residing in homes above the EPA's action level (29.9% in the no-mobility model, 11% in the mobility model). Above 10 pCi/l, the no-mobility model finds 9.3% of deaths, while the mobility model indicates only 2.6%. In contrast, at exposures below 0.5 pCi/l, the mobility model implies three times more mortality than does the no-mobility model (32.3% and 9.8%, respectively).

The proportional differences in deaths between the no-mobility and mobility models in the cross-sectional analysis in Table 3, though still large, are relatively smaller than in the cohort analysis in Table 2. This results because Table 3 presents a cross-sectional view of the current radon exposures of all people who die in a given year as a result of their cumulative radon exposures over their lifetimes. Current radon exposure at the end of life is much more highly correlated with cumulative exposure than is exposure at age 30 (the subject of the cohort analysis presented in Table 2), because as people age, and hence become more vulnerable to lung cancer, their residential mobility declines.

Mobility affects estimates of mortality by geographic region in a similar manner, causing state and area death rates to cluster more closely around the national mean than does the geographic distribution of radon per se. In analyses not shown here, we found that the no-mobility model overestimated the number of deaths in the five highest-radon states by 25–50%, while underestimating mortality in the lowest five by 11–42%. The differences are proportionately larger for areas within states, reflecting the much larger number of areas, their greater range and variance of radon readings, and the greater mobility among areas than among states.

Figure 1 further illustrates how mobility influences cumulative radon exposure. Each line shows the expected cumulative exposure of an individual of the indicated age, given the individual's location in a state and an area within that state, and given one of four assumptions about how cumulative exposure is generated. Each of the three straight lines shows what would happen to an individual subject to constant (lifetime) exposure at the national, state, or area average. The violet line shows what happens, on average, to residents of an area

who have been subject to normal patterns of lifetime mobility. Consistent with the smaller variance of the mobility model, mobility-affected cumulative exposures tend more toward the state and national averages (i.e., away from the area-specific average). The state average is more important than the national in influencing any individual's exposure because intrastate mobility is much more common than interstate mobility.

The two graphs in Figure 1 illustrate phenomena that occur consistently. On average, residents of an area that has a radon reading exceeding the state average in a higher-than-average radon state will experience less cumulative radon exposure with mobility than without it, as seen in Figure 1, which depicts a radon "hot spot" in Pennsylvania, a modestly above-average radon state. For the typical resident of this area, cumulative exposure at any age is approximately two-thirds that of a permanent resident of the area. Because exposure in this hot spot is so high (an average of 4.0 pCi/l) and, specifically, so much above that of the state (1.8 pCi/l) and nation (1.18 pCi/l), normal patterns of mobility decrease cumulative exposure from the hot spot average toward the state and national averages.

Figure 1 shows the implications of living in a low-radon area in a lower-than-average radon state, in this case Louisiana (state average exposure of 0.5 pCi/l). With normal mobility, cumulative exposure will average 1.5 times that of the never-moving resident of the area. In this instance, interstate mobility is sufficiently influential to increase average exposure well above the state's average.

Discussion

This analysis has important implications for both individual and collective societal responses to the hazard posed by radon. To deal with radon, the EPA and many state environmental agencies have emphasized voluntary remedial action based on individual interest in one's own health. Given the effects of normal residential mobility on cumulative radon exposure, however, we find that the EPA's assessment of risk, as conveyed to the public in its risk charts, greatly exaggerates the actual risk faced by most residents of high-radon homes. This conclusion does not depend on the strengths or weaknesses of our specific mobility model. Any reasonable model that incorporates residential mobility will demonstrate a similarly dramatic reduction in the variance of lifetime cumulative exposures and hence the variance of radon-related mortality.

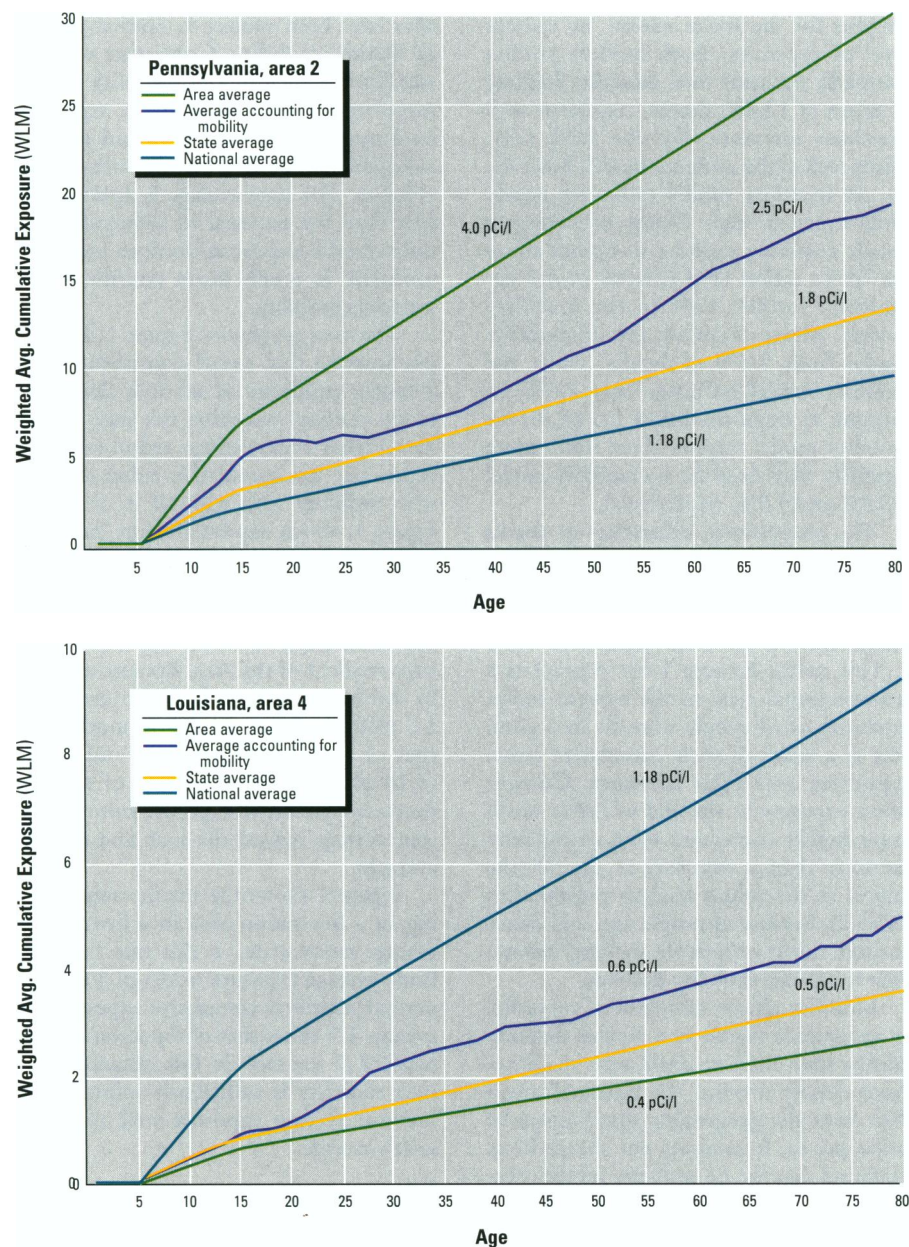


Figure 1. Influence of mobility on cumulative radon exposure, by age, for residents of a high-radon area in Pennsylvania (a high-radon state) and for residents of a low-radon area in Louisiana (a low-radon state). Each of the three straight lines shows what would happen to an individual subject to constant (lifetime) exposure at the national, state, or area average. (The turn in these lines at age 15 reflects the fact that the BEIR IV model applies a weight of 50% to the cumulative exposure incurred 15 years or more before the age at risk, effective after age 15.) The violet line shows what happens, on average, to residents of an area who have been subject to normal patterns of lifetime mobility.

Assuming the validity of the basic risk model (19), the results from our mobility model concur with those of the EPA concerning the aggregate mortality burden of radon. Thus, if Americans voluntarily followed the EPA's guidelines, remediating all homes above the action level, thousands of lives could be saved annually, just as the EPA states. However, most of the lives saved would not be those of the people who undertook the effort and expense of remedi-

ation. Using our model, for example, we find that if all homes with exposures in excess of 4 pCi/l were remediated today to 2 pCi/l, the level the EPA believes attainable on average (1), only between a quarter and a third of the reduction in mortality would occur among those who occupied the homes at the time of the remediation.

Thus, a policy directed at voluntary individual behavior effectively asks current homeowners to subsidize improved health

for others. If people truly understand their actual risk and respond in a self-interested manner, only a small fraction of the lives that could be saved through complete compliance with the EPA's preferred approach will be saved. Private, voluntary action is unlikely to have much success dealing with radon.

One might expect that real estate markets would generate widespread testing and mitigation of radon levels, without public intervention. Just as pollution is reflected in the market value of a home (21), radon might be expected to affect market value, with low-radon homes selling or renting at a premium. Thus, quite independent of the effects on their own health, homeowners would remediate to the point that the cost equaled the increase in house value.

Generally, however, profit-seeking behavior in real estate markets will have only small effects on testing and mitigation. Increases in house value arising from mitigation will be determined by the discounted valuations that future residents of the house place on the radon level, and those valuations are highly uncertain. They depend on the size of each future household, the age of the residents, the length of time that each future resident will stay in the house, and the residents' preferences for risk reduction. Equally important are the future supply and demand for housing with different radon levels in each local housing market. Where there is an abundant supply of low-radon housing relative to demand, as will be typical, remediation of a given high-radon house will not be profitable, because the equilibrium premium for low radon will be small. Moreover, the equilibrium premium can never exceed the cost of testing and mitigation, because any future owner can choose to test and remediate. Thus, all the financial risk is on the downside. The best possible outcome for current owners who test and remediate is that they will recover much of their radon investment in higher property values. Given the many sources of uncertainty in determining future valuations, however, as well as the abundant supply of low-radon homes, the vast majority of remediators are likely to recover only a small fraction of their investment, if anything at all.

The policy implication is strong, if likely difficult for the EPA and perhaps the public to accept: assuming, as the evidence suggests, that radon constitutes a genuine threat to health, the effects of mobility make it primarily a public health problem, rather than an individual risk problem. Despite formidable political obstacles (3,22,23), collective action may well be the only appropriate and effective approach to

dealing with radon. Short of legal requirement of universal near-term testing and mitigation, collective action could (and in a few jurisdictions does) take such forms as revision of building codes to ensure minimal radon exposure and requirement of radon testing and, where appropriate, mitigation at the time a residence is sold. These more moderate policies, although more politically feasible, would take more time to achieve a given reduction in mortality than would policies with broader coverage.

Ultimately, the decision of whether or not to adopt radon testing and mitigation policies, at either the individual or societal level, must rest on consideration of the costs of testing and mitigation, as well as the health benefits. To date there have been only a handful of cost-benefit evaluations of radon intervention strategies (1,24,25); we are examining the issue in ongoing research. It is virtually certain, however, that there are health, economic, and technological circumstances under which formal regulatory policies would pass a cost-benefit test, while reliance on individual voluntary action would consistently fail.

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