Effects of Radon Mitigation vs Smoking Cessation in Reducing Radon-Related Risk of Lung Cancer

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Introduction

The Environmental Protection Agency (EPA) considers radon a major cause of lung cancer that is responsible for 7000 to 30,000 deaths in the United States annually. The agency has urged Americans to test their homes and remediate those in which radon readings exceed 4 pCi/L. EPA distinguishes the risks of radon exposure for smokers and never smokers in its A Citizen’s Guide to Radon. The risks are dramatically higher for smokers, reflecting an interaction effect between radon and cigarette smoking. Although the guide recommends quitting smoking, along with remediating homes with high radon readings, no publication has compared the risk reduction attainable by radon mitigation, smoking cessation, or the two combined.

Methods

In our analysis, we used the standard radon lung cancer risk model used by EPA, BEIR IV, linked with 2 other models, one describing the distribution of radon in homes in the United States and one characterizing Americans’ patterns of moving to new homes (averaging 10 or 11 moves throughout their lives). Introducing realistic patterns of residential mobility greatly reduces estimates of the individual risk confronted by people currently residing in high-radon homes, simply because they will spend most of their lives in lower-radon homes. The models and the analytical process have been described elsewhere (a technical appendix is available from the authors).

In using the BEIR IV model, we assumed a multiplicative relationship between smoking and radon exposure to estimate the effects on risk reduction of radon mitigation alone, smoking cessation alone, or both together. We assumed that mitigation means reducing all elevated radon exposures to 2 pCi/L, the level that EPA believes can be attained on average. If individuals were to remediate one home and then move to another high-radon home, we assumed that they would remediate that home as well.

To compute the risk reduction attributable to quitting smoking, we assumed that background lung cancer risk declines linearly from levels for current smokers to the average for former smokers in 15 years. This was an extremely conservative approach that, for 2 reasons, considerably underestimates risk reduction: (1) it treats the average risk for former smokers as the risk attained 15 years after quitting, although the average former smoker has been abstinent only a few years, and (2) empirical evidence on lung cancer risk reduction after cessation of smoking indicates a rate of decline more rapid than linear.

As with EPA’s results, our findings represent averages for both sexes. In actuality, risks will be higher for men, and risk reductions greater, because men have higher background lung cancer rates.

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To illustrate model results, we consider here two representative 40-year-olds who have smoked since 20 years of age, one in a home with a radon reading of 4 pCi/L and the other in a home with a reading of 10 pCi/L, a very high level reached or exceeded in only 0.7% of residences. We assumed that these individuals would live in their current homes for 10 more years and thereafter move according to the residential mobility model.

### Results

Table 1 presents the 2 hypothetical 40-year-olds’ lifetime percentage risks of contracting radon-related lung cancer, with the risks of a never smoker of the same age presented for comparison.

For the smoker living in a home with a radon reading of 4 pCi/L (columns 1 and 2), doing nothing produces a 1.69% lifetime chance of radon-related lung cancer. Mitigating the radon problem but not quitting smoking would decrease risk by a fifth, to 1.35%. Quitting smoking but not mitigating radon would decrease risk by more than half, to 0.81%. Both mitigating radon and quitting smoking reduces risk by just over 60%, to 0.65%. The incremental gain of mitigation, once one has quit smoking, is small relative to the incremental gain of quitting smoking, given that one has mitigated. (The never smoker who does not mitigate has less than 0.1% chance of contracting radon-related lung cancer.)

For the smoker living in a home with a radon reading of 10 pCi/L (columns 3 and 4), doing nothing translates into a 3% lifetime chance of radon-related lung cancer. Mitigating the radon problem while continuing to smoke reduces risk by nearly 40%, to 1.83%. Quitting smoking but not mitigating decreases risk by more than half, to 1.45%. Combining mitigation and quitting smoking reduces risk by fully 70%, to 0.9%. As before, mitigating after having quit smoking creates a smaller incremental risk reduction than does quitting smoking after having mitigated the radon problem. (The never smoker confronts a lifetime risk of 0.16%, which mitigation would lower by nearly 40%.)

Not shown are results associated with varying the age of the individual or the minimum length of continued residence in the same home. These results would not qualitatively alter the findings. Varying the age at which one quits smoking would influence the findings in obvious ways. For example, mitigation undertaken at 40 years of age reduces risk to a greater extent than does smoking cessation at 70 years of age. (However, quitting smoking at age 60 affords risk reduction comparable to that achieved with mitigation at age 40.)

### Discussion

The striking finding of this analysis is that quitting smoking will reduce the risk of radon-related lung cancer more than will directly addressing the home’s radon problem itself. This is true even at unusually high levels of radon and despite modeling assumptions that clearly underestimate the risk reduction benefits of quitting smoking. Of course, to maximize risk reduction, smokers in high-radon homes should both quit smoking and remediate the home. In most situations, however, the latter will produce only a relatively small marginal gain once the former has been achieved.

Our specific findings depend critically on the nature of the interaction between smoking and radon in producing radon-related lung cancer. We used the same risk model used by EPA (and widely accepted elsewhere as well). This model posits a multiplicative relationship between smoking and radon exposure.\(^1\) Alternative models, consistent with the existing data, that specify a submultiplicative relationship\(^2\) might imply that quitting smoking has a lesser impact in reducing radon-induced lung cancer. However, they will not change our qualitative finding that smoking cessation is more effective than mitigation in reducing radon-related lung cancer risk.

The radon-related health benefits of quitting smoking pale in comparison with its other contributions to health.\(^3\) The principal health motivation for quitting smoking, therefore, is to not solve a radon problem. Still, we find it intriguing that mastering America’s premier cause of preventable premature mortality, cigarette smoking, dominates strategies to deal with the problem posed by indoor radon as well. □

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### References