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Radon As an Indoor Air Pollutant

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Abstract. An overview of the environmental radon problem is presented, with special emphasis on risk estimation and its attendant uncertainties. Although remediation of radon in an individual house is usually fairly inexpensive, aggregate costs can vary greatly, depending on how many houses are deemed hazardous to health. Picking a danger level in the presence of large uncertainties (approximately an order of magnitude separates the high and low value) is a difficult regulatory decision.

Key words and phrases: Radon, lung cancer, smoking, modeling, environmental, risk assessment.

Radon gas is an indoor air pollutant which may pose serious health risks to a sizable fraction of the general population. Inhaling radon, or more accurately its decay products or "progeny," can expose lung tissue to significant doses of ionizing radiation, which may in turn cause lung cancer (National Council on Radiation Protection and Measurements (NCRP), 1984a,b), a usually fatal disease. Thus, the risk end point is indeed serious. Radon is also a colorless, odorless, inert gas, which cannot be detected without special instrumentation (Environmental Protection Agency (EPA), 1986a). As a gas, it can diffuse through cracks in foundations, can be transported in water and can move into basements through sewer lines or drain pipes (EPA, 1986a). In this, it is unlike all other members of the uranium decay chain (the succession of elements produced by a series of radioactive decays, which eventually transform uranium to lead), which are all fairly reactive metals (NCRP, 1984a,b). Because they are fairly reactive metals, they are bound as mineral compounds in the soil, and tend to stay put. Thus the exact magnitude of radon exposure in the general population is difficult to define. The central problem of this discussion is defining the probable magnitudes of the exposure-response coefficient and the exposures which prevail in the general population. Put another way, how likely is it that a given exposure will cause cancer, and what is the likelihood that such an exposure will actually be received?

The second question is fairly easy to answer. With public awareness of the problem has come a variety of radon measurement services (EPA, 1986a), and if one selects a vendor carefully (fraudulent and/or incompetent vendors are not unknown), it is fairly easy to obtain an accurate assessment of the radon levels present in one's home. (Note however that radon levels may change dramatically over time in the same house. Thus, especially if there is a priori reason to expect a radon problem, several measurements, spaced over time, are prudent (EPA, 1986a).)

The question of what a given level of radon means in terms of lung cancer risk is less straightforward. The source of our risk estimates for lung cancer induction by radon progeny is occupational health studies of miner (usually uranium) populations. Most recent studies have fairly good estimates of radon progeny exposure and careful followup of populations who have had substantial exposures. The risk estimates from these studies suggest that radon progeny exposure raises one's baseline lung cancer rate (risk) between 0.5 and 2.5% per "working level month" (WLM, a unit of radon progeny exposure) (Thomas, McNeill and Dougherty, 1985). This kind of model is termed a relative risk model because excess risk is a multiplicative function of exposure and one's baseline risk. In the model, radon progeny exposure is assumed to act multiplicatively with the risk from cigarette smoking. Thus, cigarette smokers have very much higher risks than nonsmokers for a given level of radon progeny exposure (Ginevan and Mills, 1986). Still it is unclear exactly where in the range of risk coefficients a general population risk coefficient might lie, or even if the model suggested here is the correct one.

First there is the problem of dose. All miner studies use exposure as a surrogate measure of dose but, for two reasons, there is not a necessary association between the two. First, the decay products are all metal ions, and as such, they tend to "stick" to dust particles. A mine is a rather dusty place, so most radon progeny are "stuck" or attached to dust particles. Small particles are much easier to inhale than large particles. Thus, for a fixed level of radon progeny in air, dose

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may be small or large depending on the size distribution of the dust particles (NCRP, 1984a,b). Second, only two of the progeny, polonium-218 and polonium-214, are really important in delivering dose to the lung. The relative abundance of isotopes in the radon-radon progeny decay scheme can change with factors like ventilation rate and rate of production of new radon by the decay of radium (NCRP, 1984a,b). Most measurements of radiation in uranium mines are in the form of counts per minute or total activity per liter of air, and thus do not entirely reflect probable radiation dose to the lung. Problem: How much is the air in your house like the air in a uranium mine?

Second, there is a problem of biology. Miners frequently have a life style which is optimal with regard to inducing cancer (poor nutrition, high alcohol consumption, heavy cigarette usage), and are often exposed to other occupational carcinogens such as diesel exhaust and asbestos. Miners are also performing heavy manual labor and thus have high respiratory rates. (If you breath more you can inhale more radiation.) These factors might increase lung cancer risk for a given level of exposure to radon progeny. On the other hand, miners have high levels of mortality from occupational diseases such as silicosis (Lundin, Wagoner and Archer, 1971), which may tend to reduce their apparent lung cancer mortality, and many have very high radon progeny exposures which may include wasted exposure (received after lung cancer has already developed), or which may actually kill cancerous lung cells before they can develop into a tumor. These phenomena will tend to reduce the magnitude of dose response coefficients. Problem: How much are you like a uranium miner?

Finally, there is the problem of whether the risk estimate has been derived from a model which reflects reality. One could, for example, fit a model which predicted excess risk as an absolute function of exposure (here excess risk per working level month might be around 0.00001 per WLM exposure per year at risk). Such models predict very much more lung cancer in nonsmokers and very much less lung cancer in smokers as compared to relative risk models. Likewise, one can suggest that radon progeny exposure damage may be repaired over time (NCRP, 1984b; Hornung and Meinhardt, 1987). If so, exposure at relatively low dose rates may be less hazardous because damage is repaired before it can cause an actual lung cancer. Happily the data are often good enough to reject a particular risk model (absolute risk doesn't seem to fit the available data well), but sometimes this is not so. Problem: How much do you trust your model?

This is not an exhaustive review of the sources of uncertainty, but should provide a useful perspective on the problem. If the direction of bias were clear, we could simply use the miner data plus a safety factor to determine safe levels for radon progeny. Risk assessments do tend to adopt coefficients which are toward the higher end of the range of coefficients implied by miner studies (EPA, 1986a). This may overstate risk because of the bad life style factors of miner populations and the exposure to other carcinogens inherent in the mine environment. However, factors like the relatively clean atmosphere in your home may increase your dose per unit of exposure and thus your risk for a given level of radon progeny (NCRP, 1984b) and, risk estimates from miners could be too low because of the strong competing mortality from diseases like silicosis.

Given this much uncertainty, why not be safe and keep radon progeny levels in homes at such a low level that they are safe no matter what? Zero risk is not attainable, but radon levels can usually be reduced to safe levels at reasonable cost. For example, if the radon source in a home is traced to a sump or floor drain, the remedy may be as simple as putting an air tight cover on the sump or a trap in the floor drain. In homes with cracks in basement walls, the strategy could consist of sealing the cracks and applying a paint vapor barrier to the wall. Some houses may require more elaborate solutions such as subfloor ventilation systems, but the cost of ameliorating the problem is modest in most homes (a few hundred dollars) and is usually less than $3000 (EPA, 1986b). Still, any definition of an allowable exposure involves risk estimates and varying the definition can involve millions of dollars in aggregate cost. Which brings us back to how much is too much?

At present the "action level" recommended (this is *not* a regulation) by the United States Environmental Protection Agency is 0.02 working levels (a unit of radon progeny exposure rate). Essentially, if your home is at or above this level, EPA suggests that you should take action to reduce it. My own calculations, which are similar to those of other authors, suggest that if one spent 24 hr/day in such a house, their lifetime excess risk of lung cancer would be between 0.4 and 5% (Ginevan and Mills, 1986). This range primarily reflects smoking habits. (Nonsmokers have much lower risks than smokers.) If smoking status is fixed, uncertainty in the dose-response coefficient and questions of dose versus exposure might still yield a factor of 3 between the risk estimate's low and high error bounds. Thus, a smoker could have a risk as great as 15%, and a nonsmoker could have a risk as low as 0.13%, at the EPA action level. So, is this level too high, just right or too low? I don't know, but answering the question in light of the available data, and its uncertainties, is the essence of regulatory decision making.

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