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HUMAN DISEASE FROM RADON EXPOSURES: THE IMPACT OF ENERGY CONSERVATION IN BUILDINGS*

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Abstract

The level of radon and its daughters inside conventional buildings is often higher than the ambient background level. Interest in conserving energy is motivating home-owners and builders to reduce the rate of infiltration of fresh air into homes, and hence to increase the concentration of indoor air contaminants, including radon. It is unlikely, but possible, that the present radiation levels from radon daughters account for much of the lung cancer rate in non-smokers. In any event, it is likely that some increased lung cancer risk would result from increased radon exposures; hence, it is desirable not to allow radon concentrations to rise significantly. There are several ways to circumvent the increased risk without compromising energy conservation considerations.

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INTRODUCTION

Reduced air exchange rates in buildings, proposed as an important energy conservation measure, can lead to elevated levels of indoor generated air contaminants. One such contaminant is radon-222, for which several indoor sources have been identified.

Radon and its decay daughters have always been present as part of man's natural radiation burden. Since radon may emanate from indoor sources, reduced ventilation could lead to higher indoor concentrations of radon daughters and the attendant increased radiation exposure to lung tissue. The possible increased risk of disease, especially lung cancer, must be considered when adopting building energy conservation standards. The risk should be assessed in the context of the naturally occurring exposure to radon daughters and the possible health impact of this exposure to the general population. Measures are available that would limit increases in radon daughter concentrations indoors while still achieving energy conservation in buildings.

RADON AND ITS DAUGHTERS: CHARACTERISTICS & CONCENTRATIONS

Radon-222 is an inert, radioactive, naturally occurring gas which is part of the uranium-238 decay chain. Any substance that contains radium-226, the precursor of radon, is a potential emanation source. Since radium-226 is a trace element in most rock and soil, indoor radon sources include concrete, brick, and other building materials. Radium-226 has a half life of 1602 years, so its presence in building materials results in a continuous source of radon for the life of the building. Other potentially significant sources of radon in buildings include the soil beneath the foundation and tap water, especially if the water is taken from certain wells or underground springs.

The alpha decay of radium-226 produces a chemically inert, recoiling radon-222 atom which has a 3.8 day half-life. If the atom ends its recoil in an interstitial space of the solid source material, it may migrate to the surface and enter the air. Radon has four short-lived daughters, each with a half life of less than 30 minutes (see Fig. 1). The subsequent production of lead-210, with a 22-year half-life, effectively ends the sequence as far as biological effects are concerned.

The four radioactive daughters of radon are not inert. Most attach themselves by chemical or physical means to airborne particulates, generally less than a micron in size. These particulates, when inhaled, may be retained in the lung bronchii where the subsequent decays to lead-210 result in a radiation dose to the lung. The primary hazard is due to the alpha emissions of polonium-218 and polonium-214. Since alpha particles have a very short range (a few tens of microns), essentially all of the energy is deposited near the surface of the lung.

Although the inert radon is not the principal health hazard in the decay chain, its concentration is a good indicator of exposure to the biologically important daughters. Radon concentrations can be expressed in nCi/m^3 (this S.I. unit equals the more commonly used unit, pCi/liter). Ambient concentrations are typically 0.01 to 1 nCi/m^3: measurements in the San Francisco Bay Area showed radon concentrations ranging from 0.01 to 0.15 nCi/m^3 (1); average levels at four study sites in Colorado and Utah were in the range 0.34 to 0.8 nCi/m^3 (2); near Chicago, levels were found to vary diurnally from about 0.1 to 1.4 nCi/m^3 (3). Concentrations an order of magnitude greater have been found above tailings from uranium mining and milling activities(4). In terms of absolute concentrations, these numbers are minute; 1 nCi/m^3 of radon-222 is equivalent to 1.8×10^7 $atoms/m^3$, or less than one part in 10^{18}.

In the literature there are numerous examples of radon measurements showing higher indoor than outdoor concentrations. Recent measurements in the New York City area showed annual mean radon concentrations in 21 homes ranging from 0.2 to 3 nCi/m^3, with a geometric mean of 0.8 nCi/m^3 [5]. For the same locations, outdoor concentrations were 0.1 to 0.2 nCi/m^3. Average levels in Swedish homes of various construction were found to range from 1 to 12 nCi/m^3 [6]. However, Swedish homes, with air exchange rates of about 0.2 to 1.0 air changes per hour (ach),[6,7] are tighter than typical U.S. homes, where air exchange rates are on the order of 0.5 to 1.5 ach[8].

The concentration of radon in indoor air depends on the emanation rate from the parent material and on the mechanisms for removal, including ventilation. Most U.S. single-family homes are ventilated by infiltration through cracks in the building envelope: between walls and floors, around windows, doors, plumbing, vents, electrical wiring, and so on. Commercial (non-residential) buildings are usually mechanically ventilated, with fresh air supplied at about 10 cubic feet per minute (cfm) per occupant. A calculation based on the emanating surface per volume and the air exchange rate shows that the concentrations of outgassing pollutants could be comparable in typical commercial and residential buildings.[9] Due to the fact that the population spends most of its time indoors, the total exposure of the general public to radon daughters will be largely determined by the elevated indoor concentrations. Good data on indoor levels is, therefore, a prerequisite for accurate assessments of populations-at-risk to radon daughters.

DISEASE EFFECTS

Radon daughter concentrations may be expressed in terms of the Working Level (WL), a unit designed to indicate relative health hazard. One WL is defined as any combination of radon daughters in one liter of air such that the decay to lead-210 will result in the ultimate emission of 1.3×10^5 MeV of alpha energy. This unit is insensitive to the degree of radioactive equilibrium existing among the airborne daughters and radon. If radon and its first four daughters are in radioactive equilibrium, 100 nCi/m^3 of radon implies 1 WL. In well ventilated air, where the daughters have not become completely ingrown, somewhat more than 100 nCi/m^3 is necessary to generate 1 WL. An equilibrium ratio of about 0.5 has been measured in both Swedish and New York homes(5,6); for this discussion we will assume that 200 nCi/m^3 of radon yields 1 WL.

Radon daughter exposures are usually expressed in terms of working level months* (WLM), where 1 WLM is realized by exposure to 1 WL for a working month of 173 hours. Members of the general public are probably exposed to concentrations which average less than one percent of a WL (at <u>most</u>, a few nCi/m^3), so that annual exposures are fractions of a WLM. For example, exposure to 1 nCi/m^3 for a full 8760-hour year would result in an annual exposure of about 0.25 WLM/year, as follows:

$$\left(1\ \frac{\text{nCi}}{\text{m}^3}\right) \cdot \left(\frac{1\ \text{WL}}{200\ \text{nCi/m}^3}\right) \cdot \left(\frac{1\ \text{WLM}}{1\ \text{WL} \times 173\ \text{hrs}}\right) \cdot \left(8760\ \frac{\text{hrs}}{\text{year}}\right) \simeq 0.25\ \frac{\text{WLM}}{\text{year}}$$

Thus, subjects spending a 50-year lifetime in an environment with such a concentration would receive an exposure of about 10 to 15 WLM.

High levels of exposure to radon daughters clearly suggest an increased risk of lung cancer. The principal evidence arises

*The literature contains disagreements as to the actual radiation dose (in energy per mass of tissue) delivered to the bronchii, the area of concern, from exposure to radon daughters. We consistently use WLM as a measure of exposure or, equivalently, dose.

from epidemiological studies of uranium miners who worked underground in poorly ventilated areas before proper occupational health controls were imposed. For example, Figure 2 [10] shows the results of one study of excess lung cancer mortality as a function of dose. In this study increased incidence of lung cancer was observed at doses in the range of hundreds to thousands of WLM, much larger than doses to the general public.

Since epidemiological studies have not observed effects at doses much below 100 WLM, the limited high dose information must be used together with other information, such as animal experiments, to predict effects at lower doses. A commonly used method for rough estimates is based on the "linear hypothesis" that risk is directly proportional to dose. For example, 1% of a given dose would cause 1% as much risk as the risk at the full dose. The validity of this hypothesis is not known. Biological defense mechanisms may repair low dose damage, thereby providing a threshold for exposure below which no adverse effects are realized. It is also possible that the linear hypothesis may underestimate the risk.[11] Even within the linear hypothesis, there is disagreement among the experts in interpreting any dose response data, including the increased lung cancer incidence among miners. In "absolute risk" models, an additional dose to a given population causes additional risk strictly proportional to that dose, but independent of the normally occurring disease rate. Relative risk models assign additional risk proportional to the normally occurring disease rate for the population group being considered. For either school of the thought, the risk estimates in the literature vary, probably because of the differences in populations under study, the duration of the follow-up, the doses received, the dose rates, and perhaps other factors.

Considering both types of models, the data and their analyses provide risk estimates that range over an order of magnitude.

For continuous exposure to 1 nCi/m^3, corresponding to about 10 to 15 WLM, such estimates suggest an added annual risk of lung cancer within the range of about 20 to 200 cases per million.[6,12-14]

In the United States, the 45-64 year age group is at highest risk to lung cancer. Annual incidence rates during 1969-1971 for this age group were 1200 cases per million for white males and 300 cases per million for white females.[15] Although precise quantification is difficult, tobacco smoking is generally thought to be causally associated with 80% or more of the male cases.[16] Presumably, the same relationship holds for females. Based on the above estimates of risk due to exposure to 1 nCi/m^3, lifetime exposure to a few nCi/m^3 could yield increased lung cancer incidence equal to the observed rate for non-smokers.

These statements are not meant to imply that radon daughter exposures are the proper explanation for 20 to 200 annual cases of lung cancer per million, in part because it is unlikely that the average concentration of radon is 1 nCi/m^3. Additionally, the etiology of lung cancer is undoubtedly more complicated than such a simple model allows. As we do not yet know enough about the actual dose-response characteristics of low-level radiation exposure, we cannot say with certainty whether there is any added risk from a lifetime exposure of 10 to 15 WLM. However, use of the linear hypothesis is considered prudent for radiation protection purposes until we do have a better understanding of the dose-response characteristics of radiation exposure.

ENERGY CONSERVATION IN BUILDINGS

Because of increased energy prices, there is financial incentive to reduce air exchange rates and the resulting heat losses.* Measures presently considered that would halve ventilation rates could roughly double indoor concentrations of radon daughters and perhaps increase disease rates.

However, this is not the only impact that decreased ventilation rates may have on indoor air quality. Combustion products from gas stoves may require special kitchen ventilation.[17] Also, as air exchange rates decrease, other problems occur, including those with moisture and mold, odors, and chemicals outgassing from building materials and plastics. Such increases in non-radioactive pollutant levels must also be considered in formulating building standards.

Nevertheless, the possible increase in radon levels requires specific attention. Two regulatory approaches are possible for limiting exposure to radon and its daughters. One is to specify a maximum permissible concentration level and to accept the disease incidence, if any, that may be associated with increases of radon levels to this limit. There is a precedent for selecting such a level in the setting of occupational exposure standards+ and standards for the general public are sometimes selected by comparison with occupational standards. The other approach is to set standards based on an explicit comparison of the disease incidence that may be caused by increased radon concentrations with the cost of preventing these increases. Such a comparison would be made considering the benefit to be gained from reduced energy usage, balanced with the adverse effects of increased indoor pollutant levels. A decision on this matter must be preceded by substantial work on characterizing

*For example, heating 1 ach through a Chicago winter (6000 degree days) requires 30 to 40 million Btu, costing about $100 at current gas or oil prices (see Ref. 9 for more details).

+"Threshold limit values" (TLV) have been established for several chemicals and physical agents encountered in the occupational environment.[18]

both the sources of radon and the impact of various building designs on indoor concentrations.

Radon emanation rates vary widely from one substance to another, and indoor levels are strongly affected by the manner in which materials are incorporated into a building, as well as other aspects of the building design, particularly the infiltration or ventilation rate. There are several design features discussed in greater detail in a companion article[9] that might be adopted specifically to limit increases:

1) Mechanical ventilation could be coupled with an air-to-air heat exchanger to transfer heat from the exhaust air to the fresh air stream. Already in use in larger buildings, heat exchangers are now being marketed for homes in Europe and Japan. These could be used to maintain air exchange rates (and, therefore, radon and other pollutant concentrations) at the current level, while reducing heat losses from air exchange.
2) Indoor air could be circulated through electrostatic precipitators or particle filters, substantially reducing the concentration of radon daughters.[19]
3) Measures could be incorporated to seal or eliminate radon at the source. Radon from soil could be reduced by crawl space ventilation. Walls or floors could be sealed with polymers. Materials could be selected for low emanation rates.

The effectiveness and advisability of such measures depend on various circumstances, such as the type of building and the geographical location. Some of these measures may also be effective for controlling pollutants other than radon. At this time, however, we have insufficient information to provide a basis for a considered regulatory decision. The effects of elevated radon levels are highly uncertain, and the impact of building energy conservation measures is not known in detail. Moreover, the regulatory authorities will have to choose whether or not to make an explicit risk-benefit comparison.

A relatively simple interim approach to the radon question alone would be to avoid substantially altering radon levels. In most situations this may be done without compromising efforts to conserve energy in buildings. For example, in some cases the heat exchangers noted above may be used to save energy while maintaining air exchange rates around the current levels of 1 ach. Other measures may be more appropriate for some situations.

A long term solution requires a comprehensive approach which balances factors such as the impact of radon and other pollutants and the need for energy conservation. Such an approach demands substantial work to delineate more precisely the sources of radon, the effects of conservation measures on radon levels, and the disease effects of such changes.

ACKNOWLEDGEMENT

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REFERENCES

1. G. E. Jones and L. M. Kleppe, "A Simple and Inexpensive System for Measuring Concentrations of Atmospheric Radon-222", Lawrence Berkeley Laboratory Report #UCRL-16952 (1966).

2. S. D. Shearer and C. W. Sill, "Evaluation of Atmospheric Radon in the Vicinity of Uranium Mill Tailings", Health Physics 17, 77 (1969).

3. J. E. Pearson, "Natural Environmental Radioactivity from Radon-222", U.S. Public Health Service Publication No. 999-RH-26 (1967).

4. "Evaluation of Radon-222 Near Uranium Tailings Piles", U.S. Bureau of Radiological Health, Rockville, MD 20852 (1969).

5. A. C. George and A. J. Breslin, "The Distribution of Ambient Radon and Radon Daughters in Residential Buildings in the New Jersey and New York Area", Presented at the Symposium on the National Radiation Environment III, Houston, TX, April 23-28, 1978.

6. G. A. Swedjemark, "Radon in Dwellings in Sweden", Presented at the Symposium on the National Radiation Environment III, Houston, TX, April 23-28, 1978.

7. F. A. Olsson, "Ventilation and the Draught-Proofing of Windows in Old Blocks of Flats", Lund Institute of Technology, Department of Building Science Report (1977).

8. T. H. Handley and C. J. Barton, "Home Ventilation Rates: A Literature Survey", Oak Ridge National Laboratory Report No. ORNL-TM-4318 (1973).

9. See "Relations Between Reduced Ventilation/Infiltration & Indoor Air Quality" prepared by the authors of the present paper for Energy in Buildings (1978).

10. Federal Radiation Council, "Guidance for the Control of Radiation Hazards in Uranium Mining", Report FRC No. 8 (Revised), Washington, D.C. (1967). Available from Office of Radiation Programs, U.S. Environmental Protection Agency, Washington, D.C. 20460.

11. UNSCEAR, "Sources and Effects of Ionizing Radiation", United Nations, Official Records, 32nd Session, Supplement No. 40 (A/32/40), New York (1977).

12. W. E. Ellett, "Recent Advances in Understanding the Risk Due to the Inhalation of Radon Daughters", p. 102 in Radon Workshop, February 1977 Report No. HASL-325, Health and Safety Laboratory, U.S. Department of Energy, New York (1977).

13. W. Jacobi, "Relation Between Cumulative Exposure to Radon Daughters, Lung Dose, and Lung Cancer Risk", p. 492 in Noble Gases (R. E. Stanley and A. A. Moghissi, ed.), U.S. Environmental Protection Agency, Las Vegas (1975).

14. J. O. Snihs, "The Significance of Radon and Its Progeny as Natural Radiation Sources in Sweden", p. 115 in Noble Gases (R. E. Stanley and A. A. Moghissi, ed.), U.S. Environmental Protection Agency, Las Vegas (1975).

15. S. J. Cutler and J. L. Young, Jr. (Eds.) "3rd National Cancer Survey: Incidence Data", Monograph 41, DHEW Publication No. (NIH) 75-787. U.S. Department of Health, Education & Welfare, National Institutes of Health (1975).

16. Private Communication with Warren Winkelstein, Jr., School of Public Health, University of California, Berkeley (July, 1978).

17. C. D. Hollowell and G. W. Traynor, "Combustion-Generated Indoor Air Pollution" in Proceedings of the 13th International Atmospheric Pollution Conference, Paris, France, April 26-29, 1978.

18. "TLVs - Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1977", American Conference of Governmental Industrial Hygienists, P.O. Box 1937, Cincinnati, Ohio 45201 (1977).

19. USEPA, "A Preliminary Evaluation of the Control of Indoor Radon Daughter Levels in New Structures", EPA-520/4-76-018, Office of Radiation Programs, Washington, D.C. 20460 (1976).

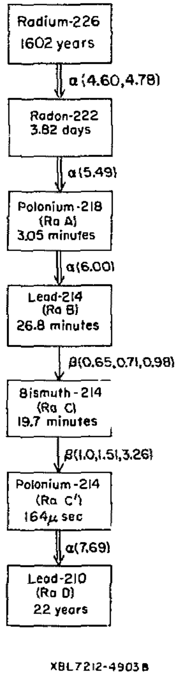

Figure 1 - Decay Chain, Radium-226 to Lead-206 (α, β energies in MeV)

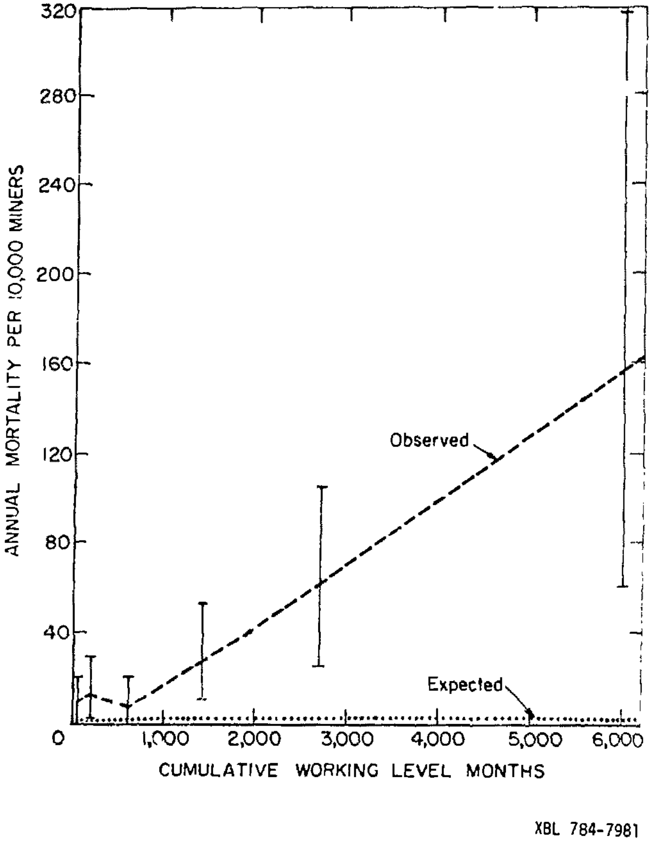

Figure 2 - Observed and expected annual lung cancer mortality per 10,000 miners and 95-percent confidence limits in relation to exposure. The expected line corresponds to an annual lung cancer mortality of 3 per 10,000. (from Ref. 10)