UC-95d
LBL-7809 Rev.

Lawrence Berkeley Laboratory
UNIVERSITY OF CALIFORNIA

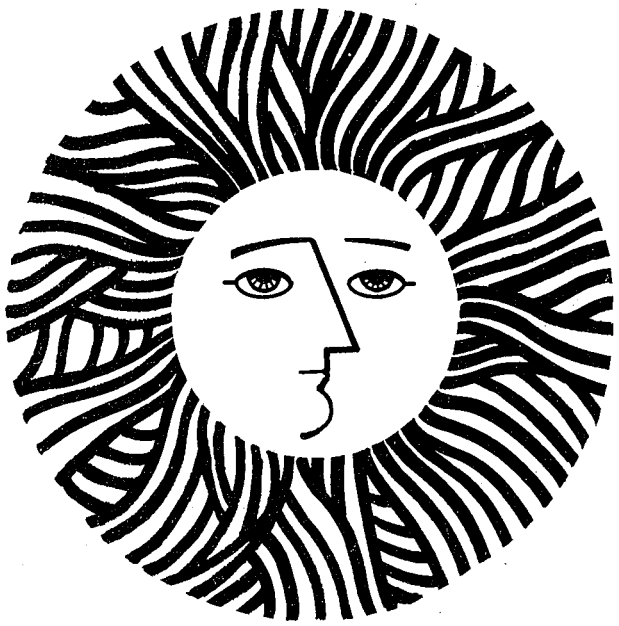
ENERGY & ENVIRONMENT DIVISION

Submitted to Energy and Buildings

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

R. J. Budnitz, J. V. Berk, C. D. Hollowell, W. W. Nazaroff,
A. V. Nero and A. H. Rosenfeld

July 1979



Prepared for the U. S. Department of Energy
under Contract W-7405-ENG-48

Submitted to Energy and Buildings

LBL-7809 Rev.
EEB-Vent 78-6

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

R. J. Budnitz[1], J. V. Berk, C. D. Hollowell,
W. W. Nazaroff, A. V. Nero and A. H. Rosenfeld

Energy Efficient Buildings Program
Energy and Environment Division
Lawrence Berkeley Laboratory
University of California
Berkeley, CA 94720
August 15, 1979

The work described in this report was funded by the Office of Buildings and Community Systems, Assistant Secretary for Conservation and Solar Applications of the U.S. Department of Energy, under contract No. W-7405-ENG-48.

---

1. On leave from Lawrence Berkeley Laboratory; Present Address: U.S. Nuclear Regulatory Commission, Washington, D.C. 20555

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 ventilation and hence to increase the concentration of indoor generated air contaminants, including radon. It is unlikely that the current radiation levels in conventional homes and buildings from radon daughters could account for a significant portion of the lung cancer rate in non-smokers. However, it is likely that some increased lung cancer risk would result from increased radon exposures; hence, it is prudent not to allow radon concentrations to rise significantly. There are several ways to implement energy conservation measures without increasing risks.

Keywords: air pollution, energy conservation, houses, indoor air quality, infiltration, radon, ventilation

INTRODUCTION

Reduced ventilation in buildings, a major 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 products have always been present as part of man's natural radiation burden. Radon is present in soil, concrete, and various building materials. Since radon may emanate from indoor sources or be transported indoors in high concentrations, reduced ventilation could lead to higher indoor concentrations of radon daughters and the attendant increased radiation exposure of building occupants. 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 of radon daughter concentrations indoors while still achieving energy conservation in buildings.

SOURCES AND CONCENTRATIONS

Radium-226, which is part of the uranium-238 decay chain, has a half-life of 1602 years. Its alpha decay produces a chemically inert, recoiling radon-222 atom having a half-life of 3.8 days. Radon has four short-lived daughters, each with a half-life of less than 30 minutes. The subsequent production of lead-210, with a 22 year half-life effectively ends the sequence as far as disease risks are concerned. Figure 1 shows the decay chain of radium-226.

The four radioactive daughters of radon are not inert. Most attach themselves by chemical or physical means to airborne particulates. When inhaled, these particulates may be retained in the tracheobronchial and pulmonary regions. Subsequent decays to lead-210 result in a radiation dose to those areas. 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 in tissue), essentially all of the energy is deposited near the surface of the lung tissue.

Because radon itself is inert, it is not the principal health hazard in the decay chain; however, its concentration* is a good indicator of exposure to the biologically important radon daughters.

---

*Radon concentrations can be expressed in nanocuries per cubic meter (nCi/m^3), which is equivalent to the more commonly used unit, picocuries per liter (pCi/l), or in Becquerels per cubic meter (Bq/m^3); 1 nCi equals 37 Bq.

Any substance containing radium-226, the precursor of radon, is a potential emanation source. Since radium-226 is a trace element in most rock and soil, sources of indoor radon include building materials, such as concrete or brick, and the soil under building foundations. The 1602 year half-life of radium-226 insures a continuous source of radon for the life of a building.

There are at least three distinct physical mechanisms by which radon from soil or building materials may be transported indoors. Radon atoms which end their recoil in an interstitial (or pore) space of the solid source material may diffuse to the surface and enter the air. Diffusion through material is a result of the radon thermal motion of gas molecules and occurs whenever a concentration gradient exists. Air infiltration through the envelope of the house provides a second important transport mechanism. Infiltration results primarily from wind blowing against and over the house and from temperature differences between indoor and outdoor air and can carry radon in high concentrations from pore spaces of building materials and soil into the house. In addition, radon gas from soil and building materials can be forced into the house by a drop in barometric pressure. The third potential transport mechanism is via tap water taken from wells or underground springs. This water may enter the house with a high concentration of radon, which is then transferred to the indoor air.

Figure 2 illustrates the primary pathways by which radon in building materials and soil gas enters a building. The relative importance of these pathways depends on the specific location, design and construction materials and techniques used in a given building.

Scattered observations have shown that indoor concentrations of radon are generally higher than local ambient concentrations (see Figure 3). The Environmental Measurements Laboratory measured radon concentrations in 21 homes in the New York/New Jersey area [1]. The geometric mean of the annual average radon concentration on the first floor of these homes, 0.83 nCi/m^3, was five times the comparable ambient level of 0.18 nCi/m^3. A study in Salzburg, Austria, measured radon concentrations at several hundred sites [2]. The results are similar to the New York study: geometric mean radon concentrations were found to be 0.42 nCi/m^3 indoors and 0.16 nCi/m^3 outdoors.

In Florida, homes built on land reclaimed from phosphate strip mining show radon concentrations much higher than in other homes in the state [3]. These elevated radon levels are associated with the high radium concentration in Florida phosphate deposits.

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 reached secular equilibrium, somewhat more than 100 nCi/m^3 are necessary to generate 1 WL. An equilibrium fraction* of about 0.5 has been measured in both New York and Swedish homes [1,4]. 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 a few percent of a WL, so that annual exposures are fractions of a WLM.**

Experience with high levels of exposure to radon daughters clearly suggest an increased risk of lung cancer. The principal evidence arises from epidemiological studies of uranium miners who worked underground in poorly ventilated areas before proper occupational health controls were imposed. For example, Figure 4 shows the results of one study of excess lung cancer mortality as a function of dose [5]. 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 [6]. 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

---

*The equilibrium fraction, F, is defined as

$$100 \times \frac{\text{WL}}{\text{radon concentration}}$$

where the radon concentration is in nCi/m^3.

**For example, exposure of the general public to 1 nCi/m^3 for a full 8760-hour year would result in an annual exposure of about 0.25 WLM/year, derived as follows:

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

Assuming a 20 year latency period for lung cancer induction, a person living a 70 year lifetime in an environment with such a concentration would be exposed to about 12.5 WLM.

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 considered. For either school of thought, 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 12.5 WLM, such estimates suggest an added annual risk of lung cancer in the vicinity of 100 cases per million.*

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 [9]. Although precise quantification is difficult, tobacco smoking is generally thought to be causally associated with 80% or more of the male lung cancer cases [10]. 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 male non-smokers.

These statements are not meant to imply that radon daughter exposures are the proper explanation for approximately 100 annual cases of lung cancer per million, because in part it is unlikely that the average person is exposed to radon at 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.

CONTROL STRATEGIES

Rising energy prices have generated a financial incentive to reduce ventilation rates and thereby reduce heating and cooling loss. This may have the effect of increasing concentrations of indoor generated contaminants. It is well known that moisture accumulates on walls and windows of poorly ventilated buildings. Recent studies have shown that special kitchen ventilation may be required to prevent the buildup of combustion products from gas stoves [11]. Organic chemicals outgassing from

---

*For example, the recent UNSCEAR review of the uranium miner data arrives at a risk of $200\text{-}450 \times 10^{-6}$ excess cancers per WLM [7], which we convert to 50-110 cases per million at our nominal concentration of 1 nCi/m^3. However, using this or other estimates [8] for a particular environmental concentration entails other uncertainties of a factor of two or more, arising from possible differences in exposure range, manner of deposition, age group, etc.

building materials and plastics, as well as odors from cooking and from occupants, may reach unacceptable levels if ventilation rates are reduced. Increased concentrations of indoor contaminants must be considered in formulating building standards. However, the increase in radon levels and the rise in the attendant risk of lung cancer that could occur as a result demand 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 balance the benefits of reduced energy usage with the adverse effects of increased indoor pollutant levels.

Although there is currently no standard specifically limiting radon daughter concentrations in the general housing stock, the U.S. Environmental Protection Agency has recommended a guideline** to the state of Florida for houses on phosphate reclaimed land [13]. A similar standard has been promulgated in Canada*** to limit radon daughter concentrations in houses in four communities associated with uranium mining and processing [14]. At an equilibrium fraction of 0.5, the EPA guideline of 0.02 WL is equivalent to 4 nCi/m^3 radon. In the Nordic countries (Denmark, Finland, Norway and Sweden), concern about high radon levels has led to a recommended minimum ventilation rate standard of 0.5 air changes per hour (ach) in residential buildings [15].

A simple interim approach to the radon question would be to avoid increasing indoor concentrations. There are many ways to do this without compromising efforts to conserve energy in buildings.

We may broadly classify radon control strategies as passive or active. In passive systems, the control mechanism usually blocks or eliminates radon at the source, while active systems operate typically by removing radon and its daughters from the indoor air.

---

*"Threshold Limit Values" (TLV) have been established for several chemicals and physical agents encountered in the occupational environment [12].

**U.S. EPA Guideline: 0.01-0.02 WL remedial action should be taken to reduce such concentrations to as low as reasonably achievable.
above 0.02 WL remedial action should be undertaken.

***Canadian Standard: Prompt interim action - greater than 0.15 WL.
Primary criterion - greater than 0.02 WL.
Investigation level - greater than 0.01 WL.

The passive approach to radon control requires little or no maintenance. Unfortunately, this approach is not effective in reducing levels of other pollutants which may be more important than radon. Active systems, conversely, require some attention by the occupants but can act on other pollutants in addition to radon.

The best passive controls are those that eliminate the radon pathways into buildings. These pathways include the floor wall joints, the basement floor drain, loose fitting pipes, and cracks in the concrete. Eliminating these pathways requires some extra expense in new construction.

An example of an active radon control system is the recirculation of indoor air through an electrostatic precipitator or other type of or particle filter. Such devices could substantially reduce the concentrations of radon daughters as well as reducing other particulate contaminants but would not be effective in reducing concentrations of gaseous contaminants (including radon gas). Units which can maintain a recirculation rate of about 5 house volumes per hour in a 150 m^2 home are commercially available for $200 [16].

A promising active system is a mechanical ventilation system coupled to an air-to-air heat exchanger. Currently, most single family homes in the U.S. are ventilated by infiltration through cracks in the building envelope. One could construct the building much tighter and use a mechanical system to maintain ventilation rates (and, therefore, radon and other pollutant concentrations) at current levels. A savings would result from the reduced heat loss; however, more work is required to determine the circumstances in which this is a cost-effective strategy. Heat exchangers are already in use in larger buildings and are being marketed for homes in Europe and Japan [17].

The effectiveness and advisability of control measures as described above depend on various circumstances such as the type of building, the geographical location, and the cost of the control strategy. At this time, 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 long term solution requires a comprehensive approach which balances factors such as the impact on human health of radon and other contaminants and the need for energy conservation. For radon, such an approach demands substantial work to delineate more precisely its sources, the effects of conservation measures on radon levels, and the disease effects of such changes.

REFERENCES

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

2. F. Steinhausler, W. Hofmann, E. Pohl and J. Pohl-Ruling, "Local and Temporal Distribution Pattern of Radon and Daughters in an Urban Environment and Determination of Organ Dose Frequency Distributions with Demoscopical Methods," Presented at the Symposium on the Natural Radiation Environment III, Houston, TX, April 23-28, 1978.

3. J.E. Fitzgerald, Jr., R.J. Guimond and R.A. Shaw, "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. 20640, 1976.

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

5. Federal Radiation Council, "Guidance for the Control of Radiation Hazards in Uranium Mining," Report FRC No. 8 (Revised), Washington, D.C., 1967.

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

7. ibid., p. 398.

8. R.J. Guimond, Jr., W.H. Ellett, J.E. Fitzgerald, Jr., S.J. Windham and P.A. Cuny, "Indoor Radiation Exposure Due to Radium-226 in Florida Phosphate Lands," EPA 520/4-78-013, February 1979.

9. 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.

10. Private communication with Warren Winkelstein, Jr., School of Public Health, University of California, Berkeley, CA 94720, July 1978.

11. C.D. Hollowell and G.W. Traynor, "Combustion-Generated Indoor Air Pollution," Lawrence Berkeley Laboratory Report LBL-7832, published in Proceedings of the 13th International Atmospheric Pollution Conference, Paris, France, April 26-29, 1978.

12. "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, OH 45201, 1977.

13. Private communication with J. Cook, Environmental Protection Agency, Washington, D.C., June 1979.

14. Federal-Provincial Task Force on Radioactivity (Canada), Working Party on "Radiation Criteria; Recommendations," December 1976.

15. Private communication with J. Sundell, Nordiska Kommitten for Byggradsbestammelser, April 1979.

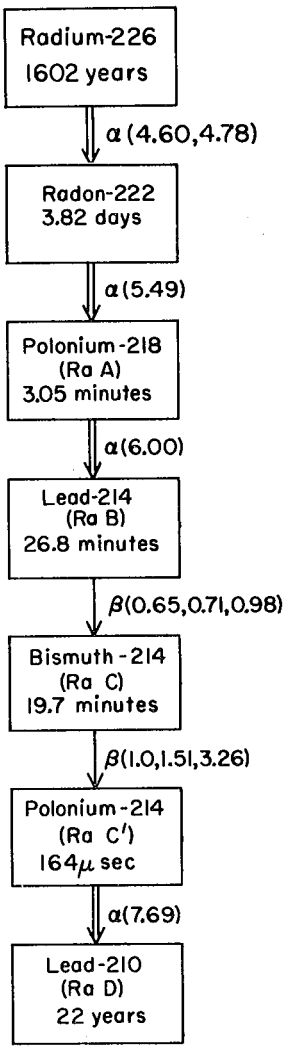
16. Private communication with R. Langenborg, Lawrence Berkeley Laboratory, Berkeley, CA, June 1979.

17. G.D. Roseme, C.D. Hollowell, A. Meier, A.H. Rosenfeld and I. Turiel, "Air-to-Air Heat Exchangers: Saving Energy and Improving Indoor Air Quality," Lawrence Berkeley Laboratory Report LBL-9381, EEB-Vent 79-11; presented at the 2nd International Conference on Energy Use Management, Los Angeles, CA, October 22-26, 1979.

18. Private communication with D.J. Bressan, Naval Research Laboratory, Washington, D.C., February 1978.

19. Federal Register, Vol. 33, No. 252, December 28, 1968.

20. "Vital Statistics of the United States," Vol. 2, Mortality, Part B, U.S. Department of Health, Education & Welfare, 1977.



XBL7212-4903B

Figure 1. Decay Chain, Radium-226 to Lead-210 (α,β energies in MeV).

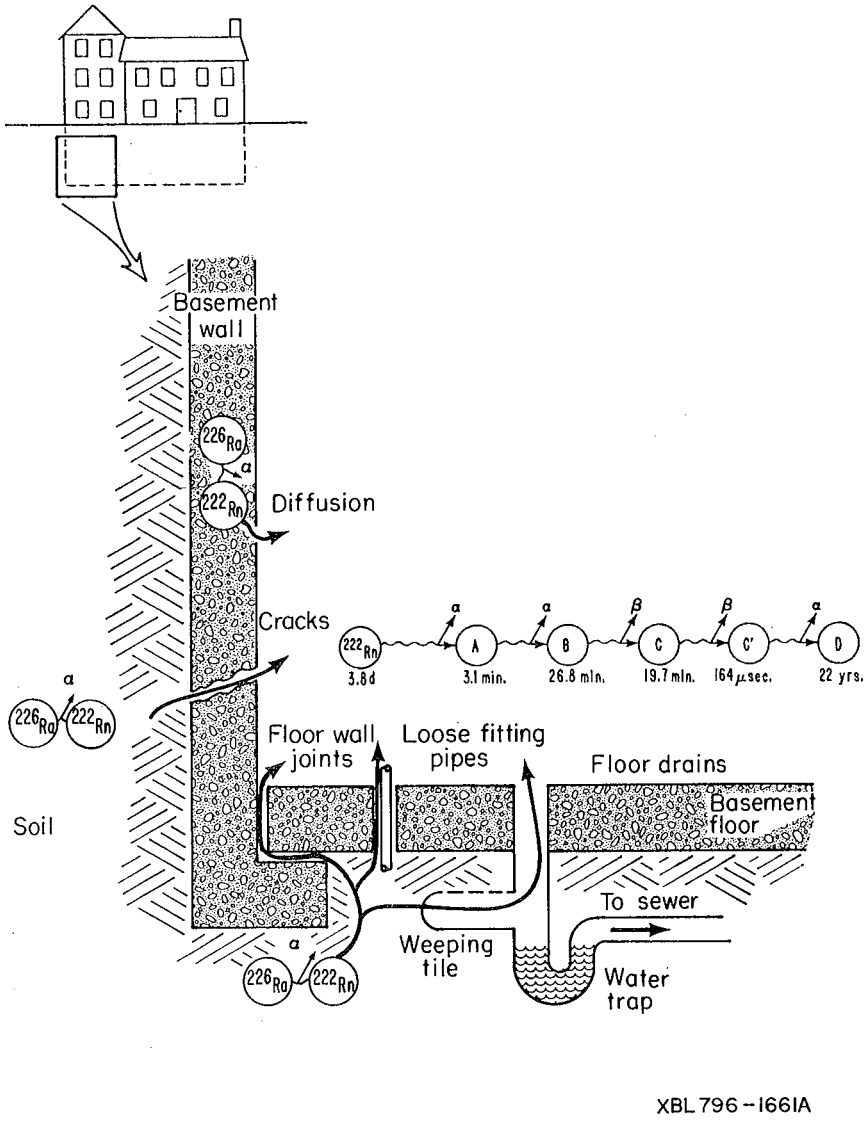


Figure 2. Some primary pathways for radon entry in residences.

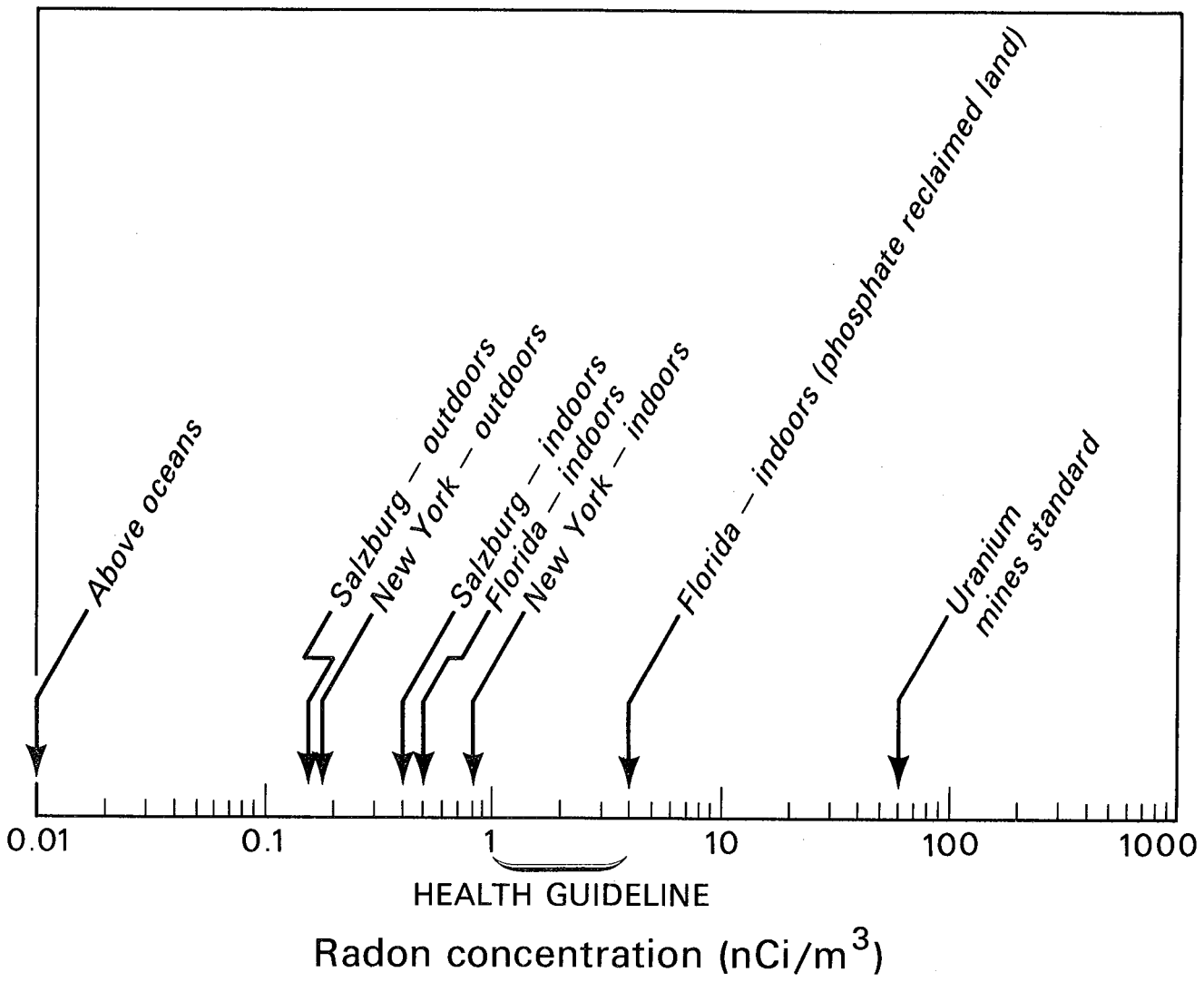
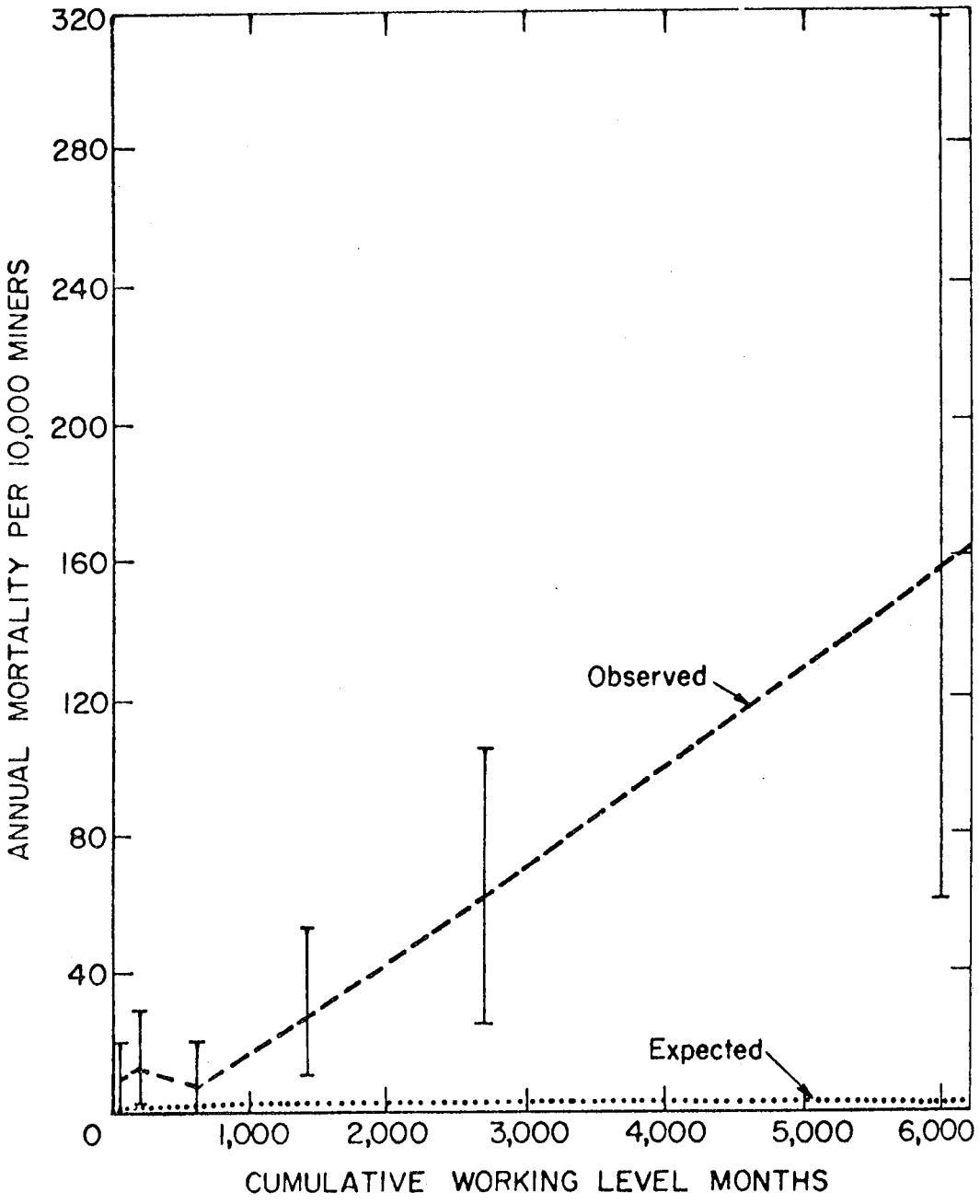


Figure 3. Radon Concentrations in Air.

The numbers for New York [1], Salzburg [2] and Florida [3] are geometric means of the average for each site sampled. The value above oceans is a typical number reported in one study [18]. The value given as the uranium mines standard is calculated (assuming an equilibrium fraction of 0.5) from the annual dose limit for occupational exposures of 4 WLM [19]. The health guidelines apply to houses built on land reclaimed from phosphate strip mining in Florida [13], and houses in four communities associated with uranium mining and processing in Canada [14].



XBL 784-7981

Figure 4. Observed and expected annual lung cancer mortality per 10,000 miners and 95% confidence limits in relation to exposure [5]. The expected line corresponds to an annual lung cancer mortality of 3 per 10,000 [20].

This report was done with support from the Department of Energy. Any conclusions or opinions expressed in this report represent solely those of the author(s) and not necessarily those of The Regents of the University of California, the Lawrence Berkeley Laboratory or the Department of Energy.

Reference to a company or product name does not imply approval or recommendation of the product by the University of California or the U.S. Department of Energy to the exclusion of others that may be suitable.

TECHNICAL INFORMATION DEPARTMENT
LAWRENCE BERKELEY LABORATORY
UNIVERSITY OF CALIFORNIA
BERKELEY, CALIFORNIA 94720