Presented at Workshop on Exposure Modeling and Monitoring, September 30 - October 2, 1985, Cambridge, Mass. Proceedings to be published.

LBL--21642

DE87 000052

Estimated Risk from Exposure to Radon Decay Products in U.S. Homes

Anthony V. Nero, Jr.
Indoor Environment Program
Applied Science Division
Lawrence Berkeley Laboratory
University of California
Berkeley, CA 94720

May 1986

This work was supported by the Director, Office of Energy Research, Office of Health and Environmental Research, Human Health and Assessments Division and Pollutant Characterization and Safety Division, and by the Assistant Secretary for Conservation and Renewable Energy, Office of Building Energy Research and Development, Building Systems Division, of the U.S. Department of Energy under Contract No. DE-AC03-76SF00098.

Abstract

Recent analyses now permit direct estimation of the risks of lung cancer from radon decay products in U.S. homes. Analysis of data from indoor monitoring in single-family homes yields a tentative frequency distribution of annual-average ^{222}Rn concentrations averaging 55 Bq m^{-3} and having 2% of homes exceeding 300 Bq m^{-3}. Application of the results of occupational epidemiological studies, either directly or using recent advances in lung dosimetry, to indoor exposures suggests that the average indoor concentration entails a lifetime risk of lung cancer of 0.3% or about 10% of the total risk of lung cancer. The risk to individuals occupying the homes with 300 Bq m^{-3} or more for their lifetimes is estimated to exceed 2%, with risks from the homes with thousands of Bq m^{-3} correspondingly higher, even exceeding the total risk of premature death due to cigarette smoking. The potential for such average and high-level risks in ordinary homes forces development of a new perspective on environmental exposures.

Keywords: radon, lung cancer, indoor air quality, epidemiology, risk assessment

INTRODUCTION

During the last decade, increasing attention has been given to airborne pollutants in the indoor environment. The occasion for this interest has been, on the one hand, growing evidence that human exposures to several classes of pollutants can be dominated by exposures incurred indoors and, on the other hand, the possibility that measures to decrease energy use in buildings by reducing ventilation rates might raise levels of pollutants whose origin is predominantly indoors.

A significant difficulty in assessing the importance of current exposures (or of potential changes due to alterations in the manner in which buildings are constructed or operated) has been limited knowledge of the exposure distribution or, indeed, of the health effects of exposures to major pollutant classes. Thus, we now know that exposures to combustion emissions can be substantially increased by utilization of unvented combustion appliances. However our knowledge of the actual exposure distribution - in terms of average exposures (or concentrations) and the fraction of houses with high levels - is tentative at best, and the dose-response relationship required to estimate the actual risk is virtually unquantifiable. Similarly, it has become clear that indoor concentrations of organic chemicals are often one or two orders of magnitude above outdoor levels and even above levels of regulatory concern in the general environment. But - except for formaldehyde - there is little information on the exposure distribution and, for almost all

chemicals observed indoors, risk factors must be derived from animal studies at exposures orders of magnitude higher than encountered indoors, involving order-of-magnitude uncertainties in the extrapolation therefore required to estimate the risks from indoor exposures.

The potential for estimating the risk from exposures to the decay products of radon 222 in the indoor environment has been somewhat better: it has been known for some time that typical annual-average radon concentrations in U.S. single-family houses are in the vicinity of 40 Bq m^{-3} and that a significant number of homes have concentrations more than an order of magnitude higher. Furthermore, a substantial body of epidemiological data is available from studies of uranium and other miners, yielding direct evidence that large exposures to radon decay products significantly increase the risk of lung cancer. These data provide a quantitative dose-response factor that, if applied to typical indoor radon decay-product concentrations, implies an average lifetime risk of lung cancer, associated with these levels, exceeding 0.1%. The risks associated with houses with higher levels would, similarly, exceed 1%.

However, despite these indications of the degree of risk from indoor radon exposures, quantitative estimation of the risk has not been possible. Although a variety of monitoring efforts have been undertaken in various areas of the United States, none has included monitoring in a statistical sample of the entire housing stock. Nor have epidemiological studies been performed to elucidate any relationship between radon decay-product exposures and lung cancer incidence among the general population. Thus quantitative risk assessment has been deferred until

more complete information on indoor exposures and health effect relationships became available.

Two recent efforts have provided results that substantially improve capabilities for estimating the risk from indoor exposures to radon decay products. The first is a systematic examination of the entire body of data available from monitoring of radon in U.S. homes, yielding a tentative frequency distribution of indoor radon concentrations. The second is detailed study of the manner in which the radiation dose from radon decay products is delivered to the lung, thereby providing quantitative guidance for the application of the miner epidemiological data to exposures of the general population indoors. Together, these permit more reliable estimation of the risk, from both average and high exposures, and provide a partial basis for developing a perspective on risk in the indoor environment, as well as objectives and strategies for control of indoor radon.

FREQUENCY DISTRIBUTION OF INDOOR RADON CONCENTRATIONS

A recent analytical effort has systematically examined the U.S. radon data, aggregating them in a consistent fashion to estimate the frequency distribution of concentrations in U.S. homes (Nero *et al.*, 1986). Data were accumulated from 38 U.S. areas (typically urban centers or states), including results available in the literature or obtained through direct communication with researchers. The total number of houses monitored in these sets was 1377, virtually all single-family, with numbers in individual data sets ranging from a few to approximately 100. A key ele-

ment in the analysis was explicit consideration of the differing incentives for the studies - resulting from different scientific or regulatory objectives - and differing monitoring protocols, including various measurement techniques and periods.

The basic method of aggregation was to consider the U.S. distribution to be represented as a weighted sum of the distributions representing the individual data sets. Aggregate distribution parameters, either arithmetic mean and fraction above 300 Bq m^{-3} (8 pCi l^{-1}) ^{222}Rn, or geometric mean (GM) and geometric standard deviation (GSD), were calculated from the parameters for the individual data sets. Aggregations differed from one another, depending on which data sets were included, on whether or not data taken during the heating season were normalized to obtain annual-average concentrations, and on what weighting factors were applied to different data sets.

The differing origins for the U.S. studies led to separation of the data sets into two groupings, based on the fact that for 16 of the data sets there had been some prior indication of the possibility of high-than-average concentrations. In contrast, for 22 of the data sets (totaling 817 homes), there was no explicit indication of such expectation. Aggregations including only these 22 sets would be expected to yield lower concentrations, and to be more representative of the U.S. housing stock, than aggregations utilizing all 38 data sets. Figure 1 displays an unsophisticated aggregation of the 19 of the 22 "unbiased" data sets for which individual data were available. Note the good correspondence to a lognormal function with the indicated parameters.

Many of the U.S. results are from monitoring performed only during the heating season, while the parameter of interest from a health standpoint is the annual-average concentration or exposure. A total of four data sets included monitoring in both winter and summer, thereby providing a basis for relating annual-average distributional parameters to heating-season parameters. The ratios of annual-average to heating-season AM, GM, and GSD varied somewhat among the four sets, but we applied the average of the four results - 0.72 for the AM, 0.81 for the GM, and 0.89 for the GSD - to heating-season parameters to obtain a reasonable approximation for annual-average parameters.

Finally, three weighting schemes were utilized: number weighting, where each distribution was given a weight in proportion to the number of houses sampled; equal weighting, with each distributional form weighted equally; and - for the 22-set aggregations - population weighting, assigning to each state the state's population or to each city the population within 50 miles.

The annual-average 22-set grouping, the aggregation of primary interest, yielded an AM of 53-57 Bq m^{-3} (1.42-1.54 pCi l^{-1}), depending on weighting, a GM of 31-33 Bq m^{-3} (0.85-0.89 pCi l^{-1}), and a GSD of 2.6-2.9. The percentage of homes found to exceed 300 Bq m^{-3} (8 pCi l^{-1}), obtained in several different fashions, ranged from 1% to 3%. The other aggregations, either including all 38 data sets or using data that had not been normalized to annual averages, tended to have higher concentrations than those just cited, as expected.

Thus the aggregate distribution resulting from this analysis has an AM

of about 55 Bq m^{-3} (1.5 pCi l^{-1}) and a GM and GSD of about 32 Bq m^{-3} (0.9 pCi l^{-1}) and 2.8, respectively. Associated with these parameters are uncertainties that cannot be estimated in a simple fashion, since the set of housing sampled was not chosen in a systematic way. Examination of the internal structure of the data, and indeed the mere fact that 22 sets were used - having GMs that were themselves distributed with a GSD of 2.0 - suggests that the uncertainty in the means is about 15%. The total uncertainty is somewhat larger, but probably not substantially larger, judging from the substantial number of states and cities (including major metropolitan areas) represented. It must be emphasized that this tentative distribution represents only single-family housing, and probably has some underrepresentation of low-income housing and of the South (although both of these components of the housing stock are represented to a significant degree). However, the fact that single-family housing is the bulk of the housing stock, serving roughly three quarters of the population (Energy, 1984), implies that this distribution is a reasonable basis for considering exposures of the population.

Based on extremely scanty monitoring data, and also on our presently very substantial knowledge of the factors affecting radon entry, it would be expected that concentrations in multifamily housing would be substantially lower, e.g., by a factor of three or so on the average. In any case, the fact that multifamily housing serves a modest portion of the population implies that estimates of population exposures are relatively independent of uncertainties about the smaller concentrations found in apartment structures.

Recent unpublished and incomplete studies tend to confirm the general

distribution indicated on the basis of this analysis. In particular, year-long monitoring of radon concentrations in approximately 500 homes of physics faculty from about 100 institutions around the country yield an arithmetic mean of 54 Bq m^{-3} (1.47 pCi l^{-1}). The data fit a lognormal distribution admirably, with a GM and GSD of 38 Bq m^{-3} and 2.36, respectively, implying that 0.8% of homes have concentrations exceeding 300 Bq m^{-3}, at the low end of the range discused above (Cohen, 1986). Additionally, substantial numbers of homes having extremely high concentrations have been found in the Reading Prong of Pennsylvania and its extension into New Jersey, raising the possibility in fact that the lognormal distribution resulting from the LBL analysis may significantly underestimate the number of homes at very high levels. However, on the whole, these incoming data substantially confirm results of this analysis, at least as a basis for risk estimation and strategy development.

HEALTH EFFECTS OF EXPOSURE TO RADON DECAY PRODUCTS

The general potential for radiation exposures to cause increased incidence of cancer, mutations, and other effects has led to development of a substantial framework of radiation protection, including guidance for limiting exposures both of the general population and of those who receive higher-than-average doses in the course of their work (NCRP, 1971). In addition, the specific experience of uranium miners, found decades ago to to have much increased risks of lung cancer as compared with the general population, has led to substantial epidemiological work

among various mining populations, which - in turn - has linked these elevated risks to exposures to ^{222}Rn decay products and has served as the basis for standards limiting such occupational exposures. This linkage is also supported by results from animal studies and by detailed investigation - experimental and theoretical - of the nature of the radiation dose from ^{222}Rn decay products that are inspired and collected in the lung.

The discovery of significant, and sometimes large, exposures among the general population has led to the need for utilizing epidemiological and dosimetric results as a basis for estimating lung cancer risks from "environmental" exposures. In such application, the essential issue is whether or how the results from miner studies can be used to estimate risks at somewhat lower exposure levels, in a different setting with different conditions, and among a more general population mix than healthy adult males who were performing physical labor and most of whom were cigarette smokers. Modest epidemiological studies among the general population have yielded inconclusive results, and even the larger studies now being designed cannot be expected to yield quantitative dose-response factors in the next several years, if at all.

However, two major research directions are providing an improved basis for estimating risks among the general population. One is the completion of more and diverse studies among miners, including some that directly provide tentative risk factors at relatively low exposures, well into the range experienced in homes, although not as low as average population exposures. The second is the development of more complete dosimetric models, permitting detailed examination of potential differences in the doses to the lungs of miners and of members of the public.

The data from epidemiological and animal studies are reviewed in a recent report of the National Council on Radiation Protection and Measurements (NCRP, 1984). More recent important papers include reports of a study of Swedish iron miners (Radford and Renard, 1984) and of a reanalysis (Thomas *et al.*, 1985) of data from five primary miner populations: United States, Czechoslovakian, and Ontario uranium miners, Newfoundland fluorospar miners, and Swedish metal miners. While these data are not unequivocal in their interpretation, they give risk factors that lie within a single order of magnitude, with the U.S. results yielding risk coefficients at the low end of the range. The main uncertainties in the risk estimates are not statistical limitations arising from the number of cancers observed, but rather difficulties in estimating doses retrospectively, in matching the mining populations with comparable nonmining populations, and - to some extent - in considering the potential effect of cofactors in the mining environment (such as metal dusts and diesel exhaust fumes).

Nonetheless, taken as a whole, the data provide a risk factor whose uncertainty is only a factor of 2 or 3 in either direction. Specifically, they indicate that an individual would suffer approximately 1% added risk of lung cancer from each 40 WLM exposure during one's lifetime. By way of comparison, an average indoor concentration of 40 Bq m^{-3} (about 1 pCi l^{-1}), two thirds occupancy, and a normal amount of decay products relative to the radon, implies an exposure rate of approximately 0.2 working level month (WLM) per year, or about 15 WLM in a lifetime. (One WLM exposure occurs from presence for a working month of 173 hours in an atmosphere with 1 working level [WL] of decay products, which at 50% equi-

librium would be caused by 200 pCi l^{-1} of radon.) Furthermore, for houses at higher levels, such as 300 Bq m^{-3}, the exposures and risks are larger and lie within the range where the epidemiological data are directly applicable, requiring no extrapolation to lower exposures.

These risks may also be expressed relative to lung cancer rates as a whole, but it is first useful to consider how to apply this information to the general public. Considerable study of the dosimetry of radon decay products has taken place during the last several years, with two illuminating, and quite different, papers on the subject having been presented at an international conference on indoor radon in 1983 (Harley, 1984 and James, 1984). These explore the manner in which the dose along the bronchial epithelium depends on the characteristics of the radon decay products (in terms of fraction not attached to particles, size distribution of those attached), details of the lung (branching pattern, clearance rates, target cells, mucus thickness), and personal characteristics (age, breathing rate and pattern). Such studies can be thought of as having two related objectives: 1) to examine the dosimetry of miners versus various element of the general population as a basis for normalizing the miner epidemiological results for use in estimating environmental risks and 2) to provide basic dosimetric information that links alpha doses from radon decay products to the much larger body of information on other kinds of radiation exposures and health effects (and that even permits estimates of the risks from radon decay products without the use of the miner data).

Such detailed examinations, which have progressed even further in the last two years, lead to some important conclusions. One is that the risk

factors derived from the mining populations can be applied almost directly to the general population, due to the presence of offsetting factors. For example, although the average person does not breathe as rapidly as a miner performing physical labor, lower flow rates increase the probability of diffusion of decay products to the tracheobronchial lining. A further conclusion is that the risk factors from the miner data are consistent with, although somewhat higher than, those from estimates based on other radiobiological information. These results have provided a more complete knowledge of how to utilize the miner data for purposes of risk estimation, permitting quantitative risk assessment in the environmental setting.

The overall result of epidemiological studies, complemented by recent dosimetric investigations, is to confirm the estimate that approximately 40 WLM lifetime exposure induces, on the average, an added lifetime risk of lung cancer of approximately 1%. This may be put in relative risk terms by stating that the risk of lung cancer (averaging about 4% in the United States, including both smokers and nonsmokers) would be doubled by decay product exposures of 100 WLM or so. It must be emphasized that these are only approximate risk factors: different investigators, analyzing essentially the same data, find values both higher and lower by factors of 2 or 3.

INDOOR RISKS AND IMPLICATIONS

These recent estimates of the concentration distribution U.S. homes and

of lung-cancer risk factors now permit effective assessment of the risk from radon decay products in indoor atmospheres. It is still necessary to make an estimate of exposures, as distinguished from concentrations, but - for the case of radon - this is relatively straightforward. In particular, for those who live in single-family homes, with an average concentration of 55 Bq m^{-3} (1.5 pCi l^{-1}), the exposure is dominated by the time spent in this environment. Indeed, this major portion of the population receives a total exposure of approximately 0.3 WLM per year. Because a quarter of the population receives significantly less than this, the average population exposure rate is probably not far from 0.25 WLM/y. This implies 15-20 WLM in a lifetime as the average decay-product exposure, to which we can attribute a risk of lung cancer of about 0.4%, a very large figure compared with most risks from environmental pollutants. However, it must be remembered that, failing elucidation of the potential interaction between decay-product exposures and cigarette smoking, it must be assumed that any incidence implied by this estimate would appear among both smokers and nonsmokers. In fact if, as some believe, there is a synergism between these two insults, the "radon" risk would appear disproportionately among smokers and therefore - ironically - be virtually unobservable at low exposure levels.

The risk for the portion of the population living in houses with high levels depends, of course, both on the exposure rate and on the period of occupancy. For example, the million or so houses estimated to have radon concentrations exceeding 300 Bq m^{-3} (8 pCi l^{-1}) would be the occasion of exposures of about 1.5 WLM/y, so that 30-year occupancy (considerably longer than average) would imply a 1% added risk and lifetime occupancy would incur risks of the order of 3%. Correspondingly higher risks are

associated with higher levels, a sobering thought considering that houses exceeding 2000 Bq m^{-3} are now being found with startling frequency.

These risks exceed by orders of magnitude those usually considered in the environmental setting, and even in occupational contexts. However, as discussed more thoroughly elsewhere (Nero, 1986), the indoor environment must be considered in its own right. The exposures and risks encountered there, while higher than often considered, are not higher than risks accepted by individuals in other contexts, in particular in using automobiles or cigarettes. Indeed, the average risk from radon is entirely comparable to the risk of premature death from fires and accidents in the home.

This is not to suggest that indoor radon exposures are unimportant. Indeed, it is utterly clear that risks range to such high levels that some strategy for control must be adopted, both for existing homes and for future structures. However, it is equally important that strategic objectives be framed only after formulation of a reasonable perspective on risks in the indoor environment.

This work was supported by the Director, Office of Energy Research, Office of Health and Environmental Research, Human Health and Assessments Division and Pollutant Characterization and Safety Division, and by the Assistant Secretary for Conservation and Renewable Energy, Office of Building Energy Research and Development, Building Systems Division, of the U.S. Department of Energy under Contract No. DE-AC03-76SF00098.

REFERENCES

Energy Information Administration (1984) Housing Characteristics 1982, U.S. Government Printing Office, report DOE/EIA-0314.

Cohen B. L. (1986) A national survey of radon concentrations in U.S. homes. To be published in *Health Physics*.

Harley N. H. (1984) Comparing radon daughter dose: environmental versus underground exposures. *Radiation Protection Dosimetry* 7, 371.

James A. C. (1984) Dosimetric approaches to risk assessment for indoor exposure to radon daughters. *Radiation Protection Dosimetry* 7, 353.

NCRP (1971) *Basic Radiation Protection Criteria*, Report No. 39. National Council on Radiation Protection and Measurements, Bethesda, Md., U.S.A.

NCRP (1984) *Evaluation of Occupational and Environmental Exposures to Radon and Radon Daughters in the United States*, Report No. 78. National Council on Radiation Protection and Measurements, Bethesda, Md., U.S.A.

Nero A. V., Schwehr M. B., Nazaroff W. W., and Revzan K. L. (1986) Distribution of airborne radon 222 concentrations in U.S. homes. Submitted to *Science*.

Radford E. P. and Renard K. G. (1984) Lung cancer in Swedish iron miners

exposed to low doses of radon daughters. *New Engl. J. Med.* **23**, 1485.

Thomas D. C., McNeill K. G., and Dougherty C. (1985) Estimates of lifetime lung cancer risks resulting from Rn progeny exposure. *Health Phys.* **49**, 825.

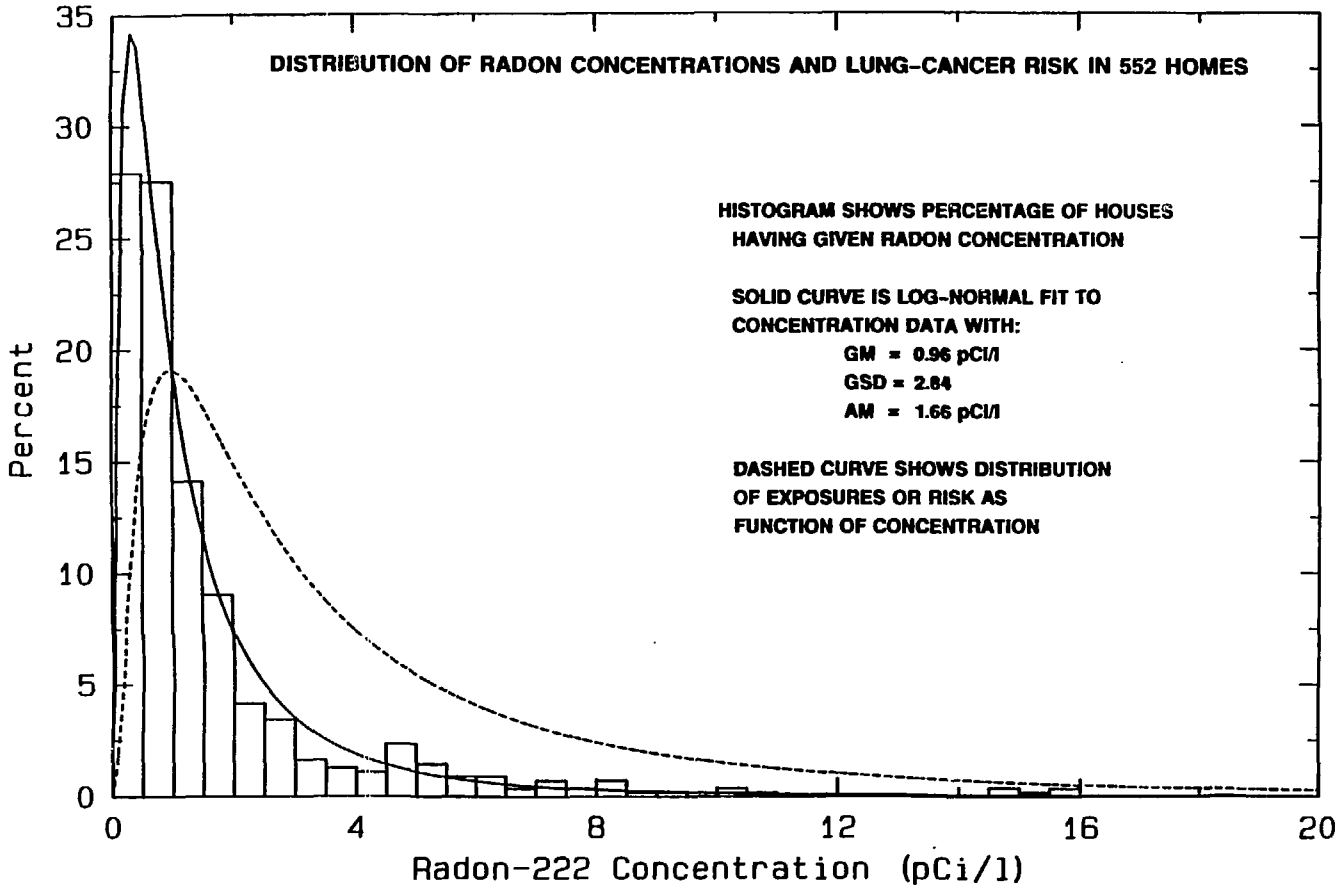

Figure 1. Histogram of directly aggregated data from 19 local surveys totaling 552 houses. The solid curve is the lognormal function with geometric mean (GM) and geometric standard deviation (GSD) calculated directly from the data. The dashed curve is the distribution of exposure (effectively probability x concentration) as a function of indoor concentration, which - for a risk that is linear with exposure - is also the distribution of risk.