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Project Summary

Estimating the Risk of Lung Cancer from Inhalation of Radon Daughters Indoors: Review and Evaluation

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A review of the dosimetric models and epidemiological studies with regard to the relation between indoor radon exposure and lung cancer indicates that the Working Level is an appropriate unit for indoor radon exposure; that the uncertainty in applying risk estimates derived from uranium miner data may be reduced by determining nose vs. mouth breathing ratios, residential aerosol characteristics, and lung cancer risk vs. age at exposure; that there is persuasive evidence of an association between radon exposure indoors and lung cancer; and that epidemiological studies in progress may provide a basis for revision or validation of current models but only if experimental designs are employed that will permit pooling of data to obtain greater statistical power.

This Project Summary was developed by EPA's Environmental Monitoring Systems Laboratory, Las Vegas, NV to announce key findings of the research project that is fully documented in a separate report of the same title (see Project Report ordering information at back).

Introduction

Inhalation of radon gas was the first situation in which radiation was implicated as a cause for cancer. The problem can be traced back for more than 400 years. In the sixteenth century, an unusual fatal disease was occurring among underground miners in Bohemia. About 100 years ago, this disease was diagnosed as lung cancer and at that time about 50 percent of the miners in the region died from lung cancer.

Around 1924, it was suggested that the high rate of lung cancer may be attributed to elevated concentrations of the radioactive noble gas, radon. In many ways it was difficult to reconcile the fact that an insoluble gas could be responsible for the disease. However, in 1950 it was recognized that the true cause of high absorbed doses to the lung was inhalation of the short lived radioactive descendants (daughters) of radon which are initially created by the decay of radon in air.

It has recently become evident that this same mechanism could be responsible for the induction of lung cancer in the general public. Measurements of radon in dwellings indicate that 20-60 percent of the dose commitment from natural background radiation is due to radon. It is generally more pronounced in regions where dwellings must be closed and insulated to protect the occupants from the weather.

The objective of this report is to summarize state-of-the-art methodologies for deriving risk estimates from this environmental pathway. It also includes an evaluation of the uncertainties of each method and suggestions for improving the risk estimation process. The report is divided into the following major sections:

- DOSIMETRY
- EPIDEMIOLOGY
- RISK MODELS

Sections on dosimetry and epidemiology are included since each discipline has contributed to the derivation of risk models employed to assess public health detriment due to indoor radon exposure.

Current risk models are based on epidemiologic data from underground

miners. The intent of this report is to summarize the epidemiologic data available from indoor radon studies and investigate its usefulness as a basis for estimating risk coefficients or validating those derived from miner data. In addition, studies in progress are summarized and their potential contribution to quantitative risk estimation discussed.

The section describing epidemiologic risk models is included simply to enhance the usefulness of this report. No attempt was made to evaluate the merits and deficiencies of each of the models.

Method

Public concern for the health effects of exposure to indoor radon has made it necessary to make risk estimates based on inadequate and incomplete data. The challenge to the professional community is to evaluate available information on occupational exposure and adapt it to non-occupational environments using basic concepts of radiation dosimetry. The results must then be validated on the basis of epidemiologic evidence and data on residential exposures.

To attempt to evaluate the problem, a literature search was conducted to locate suitable epidemiological studies and personal and professional contact were used to obtain information on unpublished work and on studies in progress. There were 21 published reports that were suitable and 6 data from unpublished studies were obtained. Finally, interim results were obtained from 14 epidemiological studies now in progress, 8 in the United States and 6 in other countries.

Discussion

There have been a number of attempts to model absorbed dose to the lung and portions of the respiratory tract from inhalation of radon daughters. These have been summarized by the National Council on Radiation Protection and Measurement (NCRP84) and James (Ja87). Three recent models were the focus of this report, by Harley (Ha86), Jacobi (Ja80, OECD83) and James (Ja87).

Dose conversion factors for inhalation of radon daughters have appeared in the literature since 1956. They range from 0.7-29 mGy WLM^{-1} (Ja87), but recently the range of values has been reduced considerably. The results from the three models indicate that the spread between models is greater than the conversion from occupational to environmental exposures within each model.

Each model was formulated by distinguished scientists who have selected input variables according to their interpretation of available and often identical data. At this time, there is no indisputable evidence that permits ranking or elimination of any of the computations.

The average of all three models gives a ratio of dose conversion factors for residential to occupational exposure of 1.3 $\pm$ 1.3. The only conclusion that can be made with confidence is that the ratio of dose conversion factors is greater than 1. The added uncertainty of deriving risk coefficients using data from underground miners may not be significant.

The concept of cumulative potential alpha energy is sufficient for describing the exposure of individuals and there is no justification for redefining or modifying the WLM or J h m^{-3}. However, there are several important factors which influence the conversion from exposure to dose. Improved data on the following could reduce the uncertainty in the risk estimates to the general public:

- Fraction of time nose breathing *vs.* mouth breathing
- Unattached fraction
- Aerodynamic median diameter and geometric standard deviation of attached aerosols
- Age dependence
- Location of radiosensitive targets

The common denominator for dose conversion factors is cumulative exposure to potential alpha energy (WLM). Most epidemiological studies of indoor environments measure radon gas only. It is important to understand the relationship between radon gas and radon daughter concentrations.

James (Ja87) has reported that the conversion to dose can be related directly to radon concentrations indoors. The reason is that for a constant level of radon the potential alpha energy, WL, increases as the concentration of room aerosols increases. However, the availability of condensation nuclei reduces the unattached fraction, f_p. These compensating factors tend to dampen variations in the dose conversion factor for a given concentration of radon gas. These concepts should be carefully evaluated in future studies.

Two general types of epidemiologic studies are represented in this body of literature: ecological and case-control. Ecological studies may have an inherent systematic bias towards showing no association between lung cancer and indoor radon due to the effect of population migration. A second source of bias in such studies may involve secondary characteristics of geographic regions studied which may either dilute or enhance an apparent association. Due to the inherent problems with interpretation of ecological studies, they can be weighted less heavily than case-control studies in the assessment of the strength of the evidence for a causative role of radon daughter exposure in lung cancer etiology.

The majority of the case-control studies relied on surrogate measures of radon daughter exposure. However, at least these measures were determined on an individual home basis. The studies are so diverse in design and execution that the data cannot be pooled or combined in order to increase the statistical significance. However, each of the published studies can be treated as an independent trial to test the hypothesis of an association between radon and lung cancer.

Six of the seven published case-control studies have indicated a relative risk or odds ratio greater than one. If there is no association between indoor radon and lung cancer and there is no systematic bias among the studies, it can be assumed that there would be a 50 percent chance of finding a positive association (relative risk or odds ratio greater than one) and a 50 percent chance of finding a negative association. Using the binomial probability distribution, the probability of six of seven such studies showing a positive association if, in fact, none exists, is approximately 0.06. This analysis depends on the assumption that the results of the published studies represent a random sample from a binomial population of results of all possible studies. The question of bias in publication of studies could invalidate this analysis.

The studies in progress are generally of case-control design and will use actual radon measurements. Several also have common design features. Collectively, they have the potential to show an association between indoor radon exposure and lung cancer which would withstand a more rigorous statistical analysis if such an association truly exists. It is much more difficult to provide definitive evidence that an association does not exist if, in fact, this is the case.

Even under the best circumstances, the exposure data from studies in progress may not be sufficiently refined to allow for development of risk models and risk coefficients independent of the information already obtained from studies of underground miners. It is likely that the studies in progress will provide a means for validating the adaptation of risk models derived from miner data to non-

occupational exposures among the general population.

Conclusions

Dosimetric analyses that take into account differences between underground miners and members of the general public, in terms of lung morphometry, breathing patterns, and environmental aerosol characteristics, indicate that the dose per unit exposure to radon daughters may be marginally higher for non-occupational exposures than for miners. Therefore, there is no apparent rationale for redefining the Working Level (WL) for indoor radon exposure simply on the basis of the reduced volume of air inhaled per unit time.

The uncertainty in applying risk estimates derived from studies of underground miners to the general public may be reduced by determining the fraction of the time persons inhale through the nose vs. the mouth, the physical characteristics of residential aerosols which would influence the unattached fraction, and the relationship between lung cancer risk and age at exposure.

The results of epidemiologic studies dealing with indoor radon provide persuasive evidence of an association between lung cancer and residential radon exposure. However, these data are not sufficient to allow derivation of quantitative risk estimates specific for indoor radon or validation of risk estimates derived from underground miner data.

Epidemiologic research in progress may provide a basis for revision or validation of current risk models and coefficients. This is feasible only if the individual investigations employ designs which allow for pooling of data to obtain greater statistical power than that possible for any single study.

References

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The complete report, entitled "Estimating the Risk of Lung Cancer from Inhalation of Radon Daughters Indoors: Review and Evaluation," (Order No. PB 88-218 979/AS; Cost $19.95, subject to change) will be available only from:

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