*Environmental Health Perspectives*
*Vol. 62, pp. 281-287, 1985*

# Potential Health Effects of Indoor Radon Exposure

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**Radon-222 is a ubiquitous noble gas arising from decay of radium-226 normally present in the earth's crust. Alpha radiation from inhaled short-lived daughters of radon readily irradiates human bronchial epithelium, and there is now good evidence of excess risk of lung cancer in underground miners exposed to higher concentrations. In homes, radon levels are highly variable, showing approximately log-normal distributions and often a small fraction of homes with high concentrations of radon and radon daughters. Factors affecting indoor concentrations include type of bedrock under dwellings, house foundation characteristics, radon dissolved in artesian water, and ventilation and degree of air movement in living spaces. Despite much recent work, exposures to radon daughters by the general public are not well defined. From application of risk assessments in miners to home conditions, it appears that about 25% or more of lung cancers among nonsmokers over the age of 60, and about 5% in smokers, may be attributable to exposure to radon daughters at home. It may be necessary to take remedial action to reduce this hazard in those dwellings with elevated levels of radon, and new construction should take account of this problem.**

One of the best documented hazards from ionizing radiation is the risk of lung cancer from exposure to alpha radiation arising from short-lived daughters of radon-222. Radon is a noble gas resulting from decay of radium-226, which is naturally present in the earth's crust and is also found in certain building materials. This gas is everywhere present in outdoor air but becomes more concentrated indoors. Radon can also reach human populations when it is dissolved in artesian well waters, which if used as a water supply in homes can lead to elevated concentrations indoors. It is also present in some natural gas sources, although this does not appear to lead to important exposures in homes.

Table 1 shows the decay scheme of radium through lead-210, whose long half-life provides time for this element to be transferred out of pulmonary tissues. Radon itself, because of its chemical inertness and low solubility in body fluids, does not deliver a significant dose to the lungs or other tissues. The immediate daughters of $^{222}Rn$, with their physical half-lives in minutes, decay approximately where they are deposited in the respiratory tract. Since these elements are solid materials and when formed are positively charged ions, they attach readily to dust particles in the air, and will follow the deposition of these particles in the respiratory tract. The small fraction present as free ions (depending on dust concentration and air movement) are attached readily to the surfaces of the upper respiratory structures. For daughters deposited on the bronchial walls, the alpha particles emitted, especially the energetic alpha from $^{214}Po$, can readily reach the basal stem cells of the epithelial layer. It is transformation of these stem cells that is believed to result eventually in cancer at these sites. The radiation dose delivered by radon daughters to the bronchial cells is the highest to any body tissues from natural background radiation.

## Radon Daughters and Lung Cancer

Evidence for the effectiveness of alpha radiation from radon daughters in inducing bronchial cancers is available from studies of several groups of miners exposed occupationally. Lung cancer from this type of radiation exposure was the first internal cancer ascribed to radiation (*1*); in the miners working in the metal and uranium mines of Bohemia and southern Germany, as many as 50–75% of all deaths were from lung cancer (*2*). Since that time, a number of epidemiologic follow-up studies have been carried out on these and other mining populations, with the result that we now have good information on the relationship of the cumulative dose from inhalation of radon daughters and excess risk of lung cancer in these workers. Table 2 shows the lung cancer risk coefficients derived from five cohort studies of underground miners currently under investigation. Absolute risk estimates are derived by the difference in observed and expected cases (excess cases) divided by the person-years at risk in millions, and by the average cumulative dose in Working Level Months. The Work-

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**Table 1. Main decay scheme of naturally radioactive radium-226 and its progeny.**

| Element[a] | Radiation emitted: Type | Radiation emitted: Energy, MeV | Physical half-life | Comments |
|---|---|---|---|---|
| $^{226}$Ra | α | 4.78 | 1602 yr | Common isotope of radium in uranium series |
| ↓ | | | | |
| $^{222}$Rn | α | 5.49 | 3.83 days | Noble gas (unreactive) Most stable radon isotope |
| ↓ | | | | |
| $^{218}$Po | α[b] | 6.00 | 3.05 min | Forms ions, attaches to surfaces |
| ↓ | | | | |
| $^{214}$Pb | β | ~0.7 (max) | 26.8 min | Beta radiation not important. Pb-214 and Bi-214 may translocate slightly in respiratory tract |
| ↓ | | | | |
| $^{214}$Bi | β | ~1.5 (max) | 19.7 min | |
| ↓ | | | | |
| $^{214}$Po | α[b] | 7.69 | 164 μ/sec | Most energetic α particle in uranium series |
| ↓ | | | | |
| $^{210}$Pb | β | 0.02 (max) | 22 yr | Long half-life means lead-210 leaves pulmonary area |

[a] Ra = radium, RN = radon, Po = polonium, Pb = lead, Bi = bismuth.
[b] These alpha decays are the radon daughters that account for the radiation dose to bronchial epithelial cells.

ing Level (WL) is defined as a concentration of the short-lived daughters giving $1.3 \times 10^5$ MeV of alpha energy from complete decay (a concentration approximately equal to daughters in equilibrium with 100 pCi/L of $^{222}$Rn) and a Working Level Month (WLM) is exposure in the mines to 1 WL for 170 hr, taken as the average working exposure per month. The WLM is a measure of cumulative exposure to radon daughters, and strictly speaking is defined only for miners. The absolute risk is thus excess lung cancer cases per million person-years at risk per WLM of exposure.

The relative risk coefficient is defined as the excess lung cancer cases divided by the expected cases and by the average cumulative dose. It represents the fractional increase in the normal or expected lung cancer rate per WLM of exposure. A value of 0.025/WLM means that there is a 2.5% increase in lung cancer per WLM of exposure, or at 40 WLM the lung cancer risk would be doubled. Both of these coefficients are based on the assumption that at low doses the excess risk is proportional to cumulative dose, an assumption that is supported by dose-response evidence in these studies. Moreover the relative risk appears from the present evidence to be reasonably constant over time once the full lung cancer effect is present (i.e., after the latent period has run), and it follows, therefore, that the absolute risk coefficient will be dependent on attained age, because the cases expected are strongly age-dependent.

The Ontario uranium miners being studied by Muller et al. (*8*) have a range of uncertainty in their cumulative dose, which is the reason for the range of coefficients in Table 2, but because they are still relatively young in comparison to the other mining groups their absolute risk coefficient is, as expected, lower than most of the others. The U.S. uranium miners (*4*) have lower coefficients because it is believed that the cumulative doses applied to this group were overestimated. The relative risk coefficient for the Swedish miners Dr. Renard and I have studied (*3*) is higher than the others because both these miners and the Swedish population used as a control smoke less than the other populations studied. Thus, when these epidemiologic or other factors are taken into account, the concordance of results among these five study populations is remarkable. Also remarkable are the recent results obtained by Lafuma and his colleagues in rats exposed to low doses of radon daughters but without cigarette smoking. They have found a doubling of lung cancers after exposure to 20 WLM (Lafuma, personal communication), roughly in agreement with our results in nonsmoking Swedish iron miners.

In our study we were able to determine smoking-specific expected lung cancer rates, and thus in Table 2 coefficients are given for smokers and nonsmokers separately. The absolute risk for smokers (current and recent ex-smokers) is only moderately higher than for nonsmokers (never-smokers and long-term ex-smokers), thus the risk of lung cancer from smoking is only slightly more than additive to the risk for radon daughter exposure. It follows from this observation that the relative risk for nonsmokers is much higher than for smokers, since the cases expected in smokers are higher than for nonsmokers. These observations of the effects of smoking in Swedish iron miners have the benefit of longer follow-up than for the U.S. uranium miners, in whom smoking is said to have a multiplicative effect on lung cancer from radon daughter exposure (*9*). For this reason the Swedish results are more indicative of lifetime lung cancer risk assessment.

## Outdoor Radon

Outdoor radon levels vary depending on whether prevailing winds are off oceans or have passed over continental masses (*10*). For example, in Hawaii the

concentration is only about 10% of values in continental U.S. Outdoor values are also low over Alaska, partly because of the persistent snow and ice cover that blocks radon diffusion from the soil. Outdoor values are also elevated in areas where uranium mine tailings are present, as in Canonsburg, PA, but the concentration decreases rapidly with distance away from this source. In well-ventilated houses, the indoor concentration may be close to the outdoor value, which in most of the continental U.S. is about 0.2–0.3 pCi/L of radon. There is a seasonal variation with higher values in the summer or late fall, and a small diurnal variation as well.

## Factors Modifying Radon in Homes

Most surveys of radon or radon daughters in homes have involved measurements in a small number of single family houses, and for this reason the general applicability of current survey information to whole populations is limited. Attention has especially focused on factors that may influence the concentrations present, and a number of these have been identified (*11*). First are sources of radon. These include radium content in soil around foundations or in home materials, radon in water supplies and, potentially at least, in natural gas sources. Radium in geologic formations on which houses are built have proved to be an important cause of elevated levels in Sweden (*12*) and certain parts of Canada (*13*) and the U.S. The use of uranium or phosphate mine tailings for fill or other purposes has led to high values in homes in Grand Junction, CO (*11*) and in Polk County, FL (*14*). In Butte, MT, abandoned mine shafts under the city have permitted radon to diffuse readily upward into homes and other buildings (*11*).

Building materials sometimes contribute importantly. In Sweden certain kinds of phosphate gypsum used as wallboard were found to be high in radium, and the Swedish government has issued limits of radium concentration above which these materials cannot be used in construction. Stones and brick used for house walls can contain variable amounts of radium, and usually stone or brick houses have somewhat higher radon indoors than frame houses. Rocks used for heat storage in basements of homes with solar energy systems or other energy conservation measures have been found to contribute increased radon unless sealed off from living spaces.

In Maine (*15*), artesian well water supplies in parts of the state have been found to have high concentrations of dissolved radon, and in some instances use of showers or dishwashers can increase indoor concentrations significantly. Hess et al. (*15*) have found that there is a correlation of radon in water supplies and radon in indoor air. This problem undoubtedly exists in other areas where deep wells are used for domestic water. Natural gas from southwestern fields contains elevated levels of radon, but because the volume used is so low in relation to the house volume, this source is not important.

The contribution of outside source factors to indoor radon can be variable depending on conditions in the house foundations. Unpaved basement areas permit radon to diffuse readily into living spaces, and if concrete

**Table 2. Summary of risk estimates of lung cancer from exposure to radon daughters by underground miners; cohort studies only.[a]**

| Study group of miners and period of observation | No. of men studied | Mean follow-up time after start of mining, yr | Abs. risk coefficient per $10^6$ PY per WLM | Relative risk coefficient per WLM | Reference |
|---|---|---|---|---|---|
| Swedish iron, 1951-76 | 1294 | 44 | 19 | 0.036 | Radford and Renard (*3*) |
| Smokers | | 44 | 22 | 0.024 | |
| Nonsmokers | | 44 | 16 | 0.107 | |
| U.S. uranium (white), 1950–74 | 3356 | 22 | 6–9[b] | 0.008–0.011[b] | BEIR III (*4*) |
| Czechoslovakian uranium, 1950–75 | 2530 | 25 | 18–21[c] | 0.023[c] | Sevc et al. (*5*) Kunz et al. (*6*) Thomas and McNeill (*7*) |
| Ontario uranium, 1959–77 | 14558 | 18 | 6–15[c] | 0.017–0.038[c] | Muller et al. (*8*) |
| Newfoundland fluorspar, 1951–71 | 2414 | 20 | 18 | 0.024[d] | Thomas and McNeill (*7*) |

[a] Coefficients derived from linear fit to exposures below 600 WLM. Values are for smokers and nonsmokers combined except for the Radford study (*3*).

[b] Range due to uncertainty in dose estimates. Upper value based on new dose re-evaluation (R. J. Waxweiler, personal communication).

[c] In these studies correction for latent period has been made, with reduction of expected cases by assuming that the lung cancer rate during the first ten years after start of mining was ½ the rate present in the subsequent 8–15 years of follow-up. The expected cases thus deleted were also subtracted from the observed cases. Absolute risk calculations based on person-years after 10 years from start of underground work.

[d] Relative risk has been adjusted for cigarette smoking by a method described by Thomas and McNeill (*7*) from data provided by A. J. de Villiers.

foundation slabs, basements or crawl spaces have cracks in them or open sump pumps, radon can enter through these pathways. Radon in basements is generally two to three times higher than elsewhere in houses, and Cohen (*16*) has shown in a survey in Pittsburgh that if basements are not separated from upper floors by a closed door, the values on first floors are higher than when a closed door is present.

A most important factor determining the concentration of radon in homes is the ventilation of living spaces. Normally a home is considered to have about one complete air change per hour, but recent measurements show that the ventilation rate is highly variable (*17*). Values below 0.1 air change per hour (said to be a lower limit for adequate odor control) were found even in houses that were not "energy efficient," and the mean value found was about 0.5 air changes per hour. The less "drafty" the house, the higher the indoor radon will be relative to outdoor values. Insulating walls and use of storm windows in winter and air conditioning in summer will reduce air changes and result in higher radon values. Meteorological factors such as wind velocity can also affect home ventilation.

Ventilation and air movement in homes also influences the degree of equilibrium of radon daughters with their parent radon. Equilibrium is present when each airborne short-lived daughter is decaying at the same rate as the radon in the enclosed space, but because the daughters, especially $^{218}$Po, can quickly "plate out" on surfaces in the room, equilibrium in the air is never present. Usually the daughters are at about 50% of equilibrium, with this fraction dependent to some degree on ventilation (*18*). Another factor of importance is air movement, which mixes room air and gives better opportunity for the daughters to plate out on walls and furnishings. Greater air movement during winter by convection or forced-air systems may account for the fact that in cold climates indoor radon daughters in winter are usually not strikingly higher than in summer, when windows and doors are often open all the time.

Dust or other aerosols in the air will provide a large surface area for daughter ions to attach to and retard plate-out, and this mechanism accounts for most of the daughters remaining airborne. The presence of cigarette smoke can increase the equilibrium fraction by providing additional surface for attachment. A small fraction, less than 10% of the total daughters, will be present as unattached ions. This unattached fraction is important because of the efficiency with which they will be deposited in the upper respiratory tract when they are inhaled. This efficiency is higher than for the dust particles to which most daughters are attached, and for this reason the unattached fraction is more effective in delivering an alpha radiation dose to the proximal regions of the bronchial tree than is the bound fraction.

## Surveys of Radon and Radon Daughters in Homes

As indicated already, systematic random surveys of radon in dwellings have not yet been done for all types of housing in any country at present. For example, there have been few measurements in apartments. From the evidence available we may, however, get an idea of the magnitude of the problem. Surveys up to 1981 in the U.S. have been summarized by Nero (*11*). Except for special problem areas such as Grand Junction, CO or central Florida, the results in these small surveys do not indicate that many houses in most areas exceed the NRC remedial action level of 0.015 WL (or about 3pCi/L of radon) or the EPA action level of 0.02 WL (or about 4 pCi/L of radon). Cohen (*16*) has recently completed a survey of 169 houses in the Pittsburgh area, with annual average values determined by track etch dosimeters that measure radon-222 only. A number of his conclu-

**Table 3. Fraction of Canadian houses sampled by McGregor et al. (*19*) exceeding particular values of working levels of radon daughters.**

| City | No. houses sampled | Geometric mean concentration WL | Exceeding 0.015 WL | | Exceeding 0.02 WL | | Exceeding 0.05 WL | |
|---|---|---|---|---|---|---|---|---|
| | | | No. | % | No. | % | No. | % |
| Calgary, Alta. | 900 | 0.0019 | 7 | 0.8 | 2 | 0.2 | 0 | 0 |
| Charlottetown, P.E.I. | 814 | 0.0018 | 10 | 1.2 | 7 | 0.8 | 0 | 0 |
| Fredericton, N.B. | 455 | 0.0032 | 30 | 6.6 | 15 | 3.3 | 1 | 0.2 |
| Halifax, N.S. | 381 | 0.0031 | 82 | 9.3 | 45 | 5.1 | 3 | 0.3 |
| Montreal, P.Q. | 600 | 0.0014 | 13 | 2.2 | 6 | 1.0 | 1 | 0.2 |
| Quebec, P.Q. | 584 | 0.0013 | 18 | 3.1 | 12 | 2.1 | 1 | 0.2 |
| St. John, N.B. | 867 | 0.0018 | 36 | 4.2 | 24 | 2.8 | 5 | 0.6 |
| Sherbrooke, P.Q. | 905 | 0.0023 | 75 | 8.3 | 57 | 6.3 | 15 | 1.7 |
| St. John's, Nfld. | 585 | 0.0015 | 12 | 2.1 | 4 | 0.7 | 1 | 0.2 |
| St. Lawrence, Nfld. | 435 | 0.0017 | 26 | 6.0 | 18 | 4.1 | 8 | 1.8 |
| Sudbury, Ont. | 772 | 0.0036 | 87 | 11.3 | 53 | 6.9 | 3 | 0.4 |
| Thunder Bay, Ont. | 627 | 0.0025 | 21 | 3.3 | 14 | 2.2 | 1 | 0.2 |
| Toronto, Ont. | 751 | 0.0018 | 13 | 1.7 | 7 | 0.9 | 1 | 0.1 |
| Vancouver, B.C. | 823 | 0.0009 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total | 9,999 | | 430 | 4.3 | 264 | 2.6 | 40 | 0.4 |

sions are of interest. About 18% of these houses had average radon values over 3 pCi/L on the first floor, with second floor values slightly lower. Basement measurements were over twice as high. Drafty houses had lower levels than less drafty ones but there was no correlation with use of natural gas or well water supplies. The conclusion is that there could be a significant proportion of houses in the Pittsburgh area in which high radon levels (3 pCi/L radon means about 0.015 WL at 50% equilibrium) may require remedial action. Obviously comprehensive random sampling in many regions of the U.S. is needed to determine the extent of this problem nationwide, and apartment buildings should be included.

Such a random nationwide survey in Canada was carried out by McGregor et al. (*19*), in which grab sample measurements of both radon and radon daughters were made by the Lucas and Kusnetz methods. Houses were sampled in census tracts, every tenth house on the longest street being chosen. Nearly 10,000 houses in 14 cities and towns across the country were sampled, with samples taken during the summer in basements or on first floors if no basements were present. It is likely that these measurement circumstances mean that the values found are reasonably applicable to inhabited areas of the houses throughout the year. Scott (*20*), in a study in Elliott Lake, Ontario, has shown that grab sampling is an adequate survey method compared with long-term integrated measurements, except the dispersion of grab sample results is greater than for integrated data because of sample variability due to short sampling time.

McGregor et al. found that their results varied substantially from city to city but the variation within any city was even greater. In each city the distribution was approximately log-normal. Some of this variation occurred because windows were sometimes open during measurements. Table 3 shows the results for these cities, with the fraction of houses exceeding a particular level of radon daughters given. Even with allowance for the greater variation present because of the method of sampling, it is evident that a significant fraction of the houses exceed the NRC action level of 0.015 WL, and the Canadian and EPA action levels of 0.02 WL. In Elliott Lake, Ontario, adjacent to uranium mining operations, about 20% of houses were found to exceed 0.02 WL, and about 30% to exceed 0.015 WL (*20*).

Extensive surveys in Sweden have been carried out though these have not been random. It is evident from these surveys (*12*) that in parts of Sweden with uranium-bearing shales near the ground surface, nearly all the houses are above a value of radon of 3 pCi/L, or equivalent to 0.015 WL. In Britain, a small national survey (*21*) gave only a few percent of homes above 0.015 WL, similar to the Canadian results, but in houses built on igneous rock geology with uranium present, nearly half showed values over 0.015 WL (*21*), similar to the Swedish results.

## Cumulative Exposures to Radon Daughters

In applying lung cancer risk evaluation to exposures at home, we need to have some estimate of the average concentration of radon daughters to which whole populations are exposed. It is evident from the results cited above that it is not yet possible to obtain such an average value with any certainty. But the U.S. and Canadian results suggest that an average daughter indoor concentration of 0.004 WL, about three times average outdoor concentrations, is appropriate. To convert this to an annual cumulative dose in WLM we need only multiply by the number of hours of exposure per year. On the assumptions that people spend two-thirds of their time at home, and most of the rest of the time at work or at school, where we may assume their exposure is half the concentration at home, this would be equivalent to exposure 83% of the time at the home concentration. On this basis the average cumulative working level months would be

$$0.004 \times 0.833 \times 8766 \text{ (hr/yr)} /170 = 0.17 \text{ WLM}$$

This exposure cannot be directly compared to the equivalent Working Level Months for miners, however, because the miners do heavy work and breathe more rapidly and deeply as a result. The generally accepted average level of breathing among miners is 1.2 $m^3$/hr, a value with which I concur. In contrast, adult men at home would breathe no more than 0.4 $m^3$/hr, for light activity and when account is taken of lower pulmonary ventilation rates for sleeping. Harley (personal communication) believes that the rad dose to the epithelium from daughter alpha decays per volume of air inhaled is higher for quiet breathing than for deeper more rapid breathing, thus the tissue dose would not be directly proportional to the volume breathed. From her estimates, at the same Working Level value the dose per unit time for the miners would be twice the dose for a person at home breathing at one-third the volume per hour. In terms of average annual radiation exposure at home the result would, therefore, be equivalent to about 0.085 WLM in comparison to mine exposures.

## Application of Lung Cancer Risk Estimates from Miners to General Population

The risk estimates per WLM given in Table 2 apply to men exposed at higher dose rates and during their working lifetime. There remains the question of the applicability of these risk estimates to women and children exposed for a lifetime at lower dose rates. With regard to the latter, the results in the miners indicate that risks are generally independent of dose rate over the range of doses up to 600 WLM (*3*). With respect to

sex, the results in the Japanese A-bomb survivors exposed to gamma rays show similar lung cancer effects for both men and women (*22*), and thus it is likely that alpha radiation would also have the same effect in both sexes. It is possible that irradiation during childhood may lead to a somewhat greater lifetime risk of cancer than irradiation in later life, but the evidence on this point for lung cancer is still uncertain.

Finally, miners are exposed to a dusty environment, with specific constituents in the dust possibly also contributing to the cancer risk. In the Swedish miners we have studied (*3*), however, other dust or aerosol constituents could be ruled out, and the concordance of results for miners exposed to widely different ores and working conditions argues strongly that the radon daughters are the principal determinants of lung cancer risk. The presence of mine dust could decrease the unattached fraction of daughter ions (*23*), and thus reduce the risk per WLM compared to the home situation, but the larger aerodynamic diameter of mine dust compared to dust in homes could increase particle impaction at bronchial bifurcations somewhat, and thereby increase the risk per WLM for the miners. For these reasons, the differences in dust characteristics between the mines and in homes does not appear likely to lead to much difference in risk.

From Table 2 we apply for nonsmokers a relative risk coefficient of 0.10 per WLM, and for smokers about 0.02 per WLM. The results in the Swedish iron miners exposed at relatively low dose rates indicated an average time from initial exposure to death from cancer of at least 30 years (*3*), so this latent period should be allowed for in determining lifetime risks for the general public. From the relation which gives relative risk at age $A$ as $DR(A-30)$, where $D$ is the average annual dose (0.085 WLM) and $R$ is the relative risk coefficient given above for smokers and nonsmokers, we note that at age 60 for nonsmokers, 25% of lung cancers would be attributable to radon daughters, with a somewhat higher fraction at older ages. For smokers, the proportion would be 5% or more. This result indicates that several thousand lung cancers per year in the general population may be attributed to radon daughter exposures.

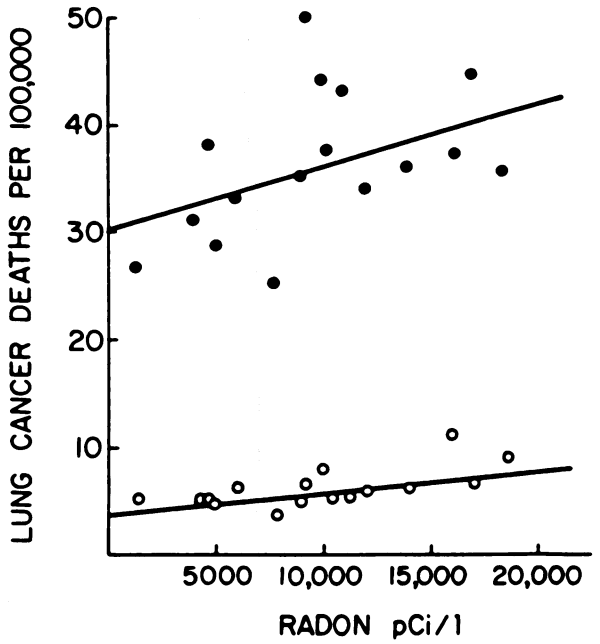


FIGURE 1. Age-adjusted lung cancer mortality rates 1950–69 vs. estimated radon concentrations in water by county in Maine: (•) men; (○) women. The lines are fitted by least squares. Data of Hess et al. (*15*) reproduced by permission of the Health Physics Society.

Epidemiologic studies are under way to determine whether this conclusion can be supported by direct evidence from populations exposed to varying levels of radon daughters at home. One preliminary study in Maine by Hess et al. (*15*) indicates that the above result is roughly confirmed. These authors noted that the 16 counties in Maine had widely differing geology and as a result differences in radon in water supplies. They also showed that indoor air radon correlated fairly well with radon in water, which would be expected because the source of both is the same, radium in the bedrock, and radon in water is a significant contributor to indoor radon in its own right. From the National Cancer Institute survey of deaths from cancer by county in the U.S. for 1950–1969, they could plot lung cancer rates against the estimated average ground water radon concentration. This plot is shown in Figure 1 for men and women.

The linear correlation for women was statistically significant at the 0.01 level but only at the 0.10 level for men. From the lung cancer rates shown, the men obviously had a much higher percentage of smokers in the relevant period of 1925–1945 (indeed the rates for women are so low that there must have been few smokers among them), and variations in the proportions of men smoking by county could have obscured the radon effect somewhat. From the correlation of air radon with water radon given by Hess et al., with an increase of radon of 6200 pCi/L in water, the indoor air level would increase by 0.8 pCi/L of radon or 0.004 WL. From the slope of the line in Figure 1, for women a rise of 6200 pCi/L of water (equivalent to an increase in air of 0.004 WL) would indicate a 33% increase in cancer risk. For men the result is 12%. Thus the results for women, if taken as applicable to nonsmokers, are in good agreement with the estimates derived above from the miner data. If the men are assumed to have been a mixture of about half smokers and half nonsmokers, their results are also reasonably consistent. This is obviously a very tentative set of results that should be confirmed, but the agreement is surprising nevertheless.

## Remedial Action

The first question is obviously: at what level of radon daughters in the home should there be concern? Because we are dealing here with alpha radiation, there is little

disagreement that the linear no-threshold dose–response curve applies, and thus the higher the radon daughter exposure the greater the lung cancer risk. On this basis, any elevation could be significant, especially for exposures beginning in childhood. Obviously there are practical limitations on an action level. The NRC limit of 0.015 WL is about four times the normal average. From the estimates given above, this would mean that exposure for most of one's life to this concentration would increase the risk of lung cancer in nonsmokers by 75% or more and by about 15% in smokers, compared to being exposed to 0.004 WL. Is this acceptable? I believe that I would advise a family with children to take remedial action even at 0.015 WL, though with only adults living in the home, I probably would not. If one recommends remedial action down to, say, 0.01 WL, however, a very large number of houses would require such action, and it could be important how costly the remedies were.

The methods of reducing radon daughters are simple in concept, though perhaps not so simple in carrying out: reduce influx of radon, increase house ventilation, and remove radon daughters by increasing plate out and filtering out airborne dust. Radon influx can be reduced by sealing off cracks or other pathways through which radon can enter foundations, prohibiting use of building materials containing excessive radium, and exposing well water high in radon to outdoor air prior to use, perhaps difficult in freezing weather. Improved house ventilation in the face of energy conservation may require heat-exchanging ventilators.

It is evident that the problem of radon in homes is a significant public health issue. Prevention of these cancers is possible, but it will be interesting to see how vigorously preventive strategies are applied in the near future. This is also a matter that should be properly addressed in any new construction.

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