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RADON AND LUNG CANCER: INCREMENTAL RISKS ASSOCIATED WITH RESIDENTIAL WEATHERIZATION

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Abstract—Energy conservation measures for residences that decrease air-exchange rates between the indoors and outdoors are shown, in the absence of substantial pressure-driven sources of radon, to increase substantially the concentrations of short-lived radionuclides in indoor air that are produced by the decay of naturally occurring radon. The associated incremental risk of lung cancer, although small compared to the risk attributable to tobacco smoking, is probably large enough to concern a substantial fraction of the population. For an average location in the U.S., the risk of fatal lung cancer attributable to radon may be increased by 115% by retrofitting that reduces the air-exchange rate from 1 to ½ air changes per hour. The model used in this study to estimate risk includes the relationship between removal processes and deviations from radioactive equilibrium. Results are given for selected changes in the air-exchange rate resulting from weatherization for several representative situations that are characterized by outdoor conditions, mineral content of the soil, and indoor radon source strength.

1. INTRODUCTION

Indoor-outdoor air exchange and the resulting heat losses from buildings account for about one-third of total heating and cooling loads in the United States.[1] In response to rapidly increasing real energy prices during the last decade, insulation, weatherstripping and other conservation devices have been installed in residences throughout the U.S. Between 1980 and 1982, approximately 716,000 houses were weatherized.[2] Although decreased infiltration leads to lower heating costs, it may also cause the buildup of air pollutants that originate indoors. Radon, which is produced by the decay of radium-226, emanates from soil, ground water, and many building materials, and tends to concentrate in indoor air. Although most homes in the U.S. have airborne radon levels of 0.2–4.0 pCi/l, some homes in mineralized† regions have been found to have concentrations of 50–100 pCi/l.[3]

Weatherization of dwellings that decreases air exchange may lead to higher concentrations of pollutants in indoor air. A clear relationship between energy efficiency and indoor radon levels has not been established experimentally.[4] However, data from some field studies show higher concentrations in energy-efficient homes. In a study of residences in New York, Fleischer[5] found radon concentrations three times greater in a sample of energy efficient homes than in a sample of conventional homes. In a study of indoor air quality in conjunction with a weatherization project, Offerman *et al.*[6] measured radon concentrations 42% greater, on average, after retrofits were completed that decreased infiltration by about 23%.

Radon exposure has been linked to lung cancer in uranium miners and is now generally considered a risk factor for the disease. This study concerns the incremental risk of lung cancer associated with increased radon concentrations in indoor air resulting from decreased air infiltration caused by weatherization of dwellings. Inhaber[7] gives a preliminary estimate of this risk. We greatly expand on his estimate, showing sensitivity of our estimates to several parameters, many which have significant uncertainty.

2. AIR INFILTRATION AND INDOOR RADON CONCENTRATIONS

A number of studies have involved measurement of air infiltration rates for both conventional and energy-efficient homes. In a survey[8] of 312 recently constructed houses throughout the U.S. and Canada, the mean measured infiltration rate during the months

† Mineralized regions are characterized by soil with substantial mineral-matter (as opposed to organic-matter) content that usually contains low-grade uranium. Examples include the Florida phosphate lands and parts of the northwestern U.S. not covered by basalt.[3]

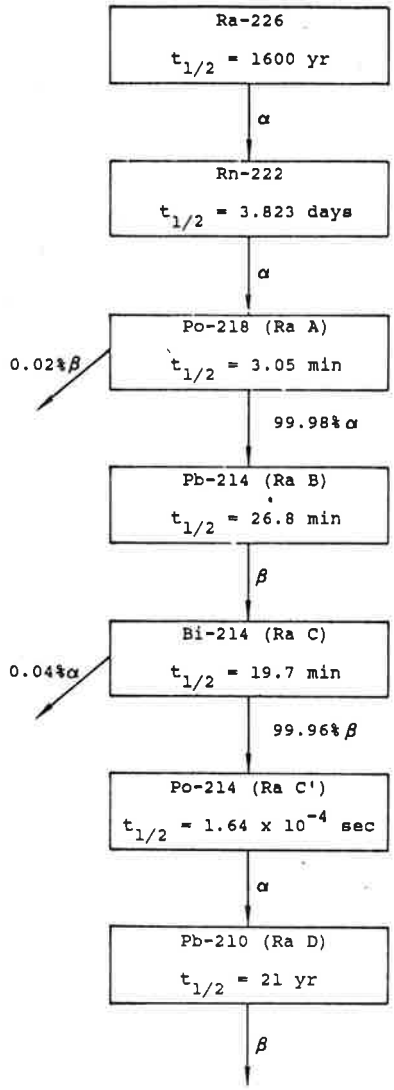

Fig. 1. The part of the decay pathway of the Uranium-238 series from Radium-226 to Lead-210 is shown together with the corresponding half life ($t_{1/2}$) of each product. The decays of Radon-222 and two of its daughter products, Polonium-218 and Polonium-214, result in alpha emissions and can contribute to radiation damage of the lung. Only the principal pathway is shown.

of November through March was 0.63 ach.† In a reevaluation of existing data, Nero[9] estimates average infiltration rates in the U.S. to be 0.7–0.8 ach. Although rare, air-exchange rates as great as 4.0 and lower than 0.25 ach have been measured. Generally, an infiltration rate of 0.5 ach is considered representative of recently built well-sealed homes.[10]

Several measures are presently used to reduce heat losses from residences associated with infiltration, including installation of plastic sheeting (which also serves as a vapor barrier) in walls on the indoor side of insulation in new construction, use of foam sealants around plumbing and electrical outlets, use of caulking and weatherstripping around doors and windows, sealing of fireplaces, and the installation of storm windows and doors. Measurements[11–13] of effective leakage areas before and after installation of weatherstripping and caulking have shown average reductions of effective leakage area of 20–30%, with a range of 0–60%. Such reductions can result in increased indoor concentrations of radon and other pollutants which originate indoors.

Decay of radium-226, which is ubiquitous, results in the production of radon-222 which has a half-life of 3.8 days (see Fig. 1). Radon has four progeny, Po-218, Pb-214, Bi-214, and

† 1 ach = 1 air change per hour = air exchange equivalent to one room (or building) volume per hour (in hr^{-1}).

Table 1. The cumulative radiation exposure for the U.S. population is shown by source (after P. J. Walsh and W. M. Lowder, Ref. 14).

Source	Effective dose equivalent, 10^6 p-rem/yr
Cosmic radiation	6
Terrestrial gamma radiation	6
Radionuclides in body	
Radon progeny	~10
All others	8
Fallout	<1
Medical diagnostic x-rays	10
Nuclear energy	~0.1
Total	~40

Po-214 (Ra A, B, C, and C′, respectively), each with a half-life of less than 30 minutes. Any substance containing radium-226 is a potential source of radon. Trace amounts of radium are found in soil, groundwater, and many building materials. Radium decay and the subsequent emanation of radon from soil beneath the building and from building materials contribute to elevated concentrations in indoor air.

Upon inhalation, unattached radon progeny deposit directly on surfaces, primarily in the upper respiratory system and tracheobronchial region. Progeny attached to fine particulates may be retained in the pulmonary region of the respiratory system with the particles. Subsequent decay results in a radiation dose to the lung from within the body. Alpha emissions, in particular, are readily absorbed by solid materials, and, therefore, are absorbed completely in a thin layer of the sensitive lining of the respiratory system, where cancers tend to originate. As the result, alpha emissions from the decay of polonium-218 and polonium-214 represent the primary health hazard associated with airborne radon progeny.

Under average environmental conditions, radon is responsible for a significant fraction of human radiation exposure (see Table 1). The effective dose equivalent† of radon for the U.S. population is about 10×10^6 person-rem/year, roughly equal to the dose equivalent from medical diagnostic X-rays.[14]

Outdoor radon concentrations are ordinarily between 100 and 400 pCi/m^3 with an average value of 250 pCi/m^3.[15,16] The main contributor of radon to outdoor air is soil. The emanation rate from soil is dependent primarily on the composition of the soil and its condition, i.e. the moisture content, temperature and porosity. Wind speed and thermal stability of the air slightly above the ground also affect the emanation rate.[17] For a given location, higher concentrations are ordinarily associated with lower wind speeds. Measurements[15] of diurnal variations in the emanation rate usually show maxima at night and early morning and minima in the afternoon. Thermal inversion and low wind speed at night and during early morning cause local ground-air stability and therefore higher radon concentrations.

Emanation is reduced by snow cover, heavy rainfall and increased atmospheric pressure. Precipitation causes the surface layers of soil to become saturated with moisture, reducing the vertical porosity for transport of radon. For extremely moist soil, radon concentrations within 36 inches of the surface may increase and remain high until the soil dries. Frozen ground decreases the vertical flux by about 40% compared to average summertime values.[18]

† A dose equivalent is that dose to the whole body that yields the same overall risk of cancer mortality and hereditary ill health in the first two generations as the actual dose pattern on the body resulting from the exposure of concern.[14]

Gesell's review of experimental results[15] shows that seasonal variations of atmospheric radon concentrations also can be substantial. Minimum concentrations generally occur between January and March, and maximum levels between August and October.

The primary sources of radon for indoor environments are soil (and geological substrate), building materials, the water supply and outdoor air. All of these show some natural variability with geographic factors (e.g. the degree of mineralization of the soil). Gaseous radon diffuses through the soil and building foundation into indoor spaces. Solid materials, like concrete which is commonly used for building foundations, in general, serve as radon barriers. A concrete slab, for example, can reduce radon emanation from soil to 4–20% of rates for uncovered soil.[19] However, radon still enters a building through cracks in the foundation, and emanates from concrete itself.

Besides concrete, other building materials containing minerals, including stone, brick, and gypsum wallboard, represent sources of radon. Concentrations of Ra-226 between 0.5 and 3.0 pCi/g have been measured in these materials.[19] Emanation rates of radon per unit activity concentration of Ra-226 in the material range from about 0.0001 pCi/m^2-s per pCi/g for some 13-mm thick gypsum ceilings to 0.02 pCi/m^2-s per pCi/g for 20-cm thick light concrete.[20] The emanation rate for average non-mineralized soil is about 0.5 pCi/m^2-s per pCi/g; the rate for uranium mine tailings is approximately 1.6.[21] As a result, compared to soil, building materials usually contribute only a small fraction to the total radon source strength for an indoor space. However, where industrial residues have been incorporated into building materials (as in Grand Junction, Colorado, where α and γ radiation levels 40 times greater than the rate for most concrete have been observed[22]), building materials may represent the primary source of radon.

Although surface water contains very low concentrations of radon (usually $\sim$0.001 pCi/l, on average, at the surface of large lakes and oceans), ground water often contains significant amounts of radon. Hess *et al.*[23] found wells in New England to have concentrations averaging 22,000 pCi/l.[23] Drury *et al.*,[24] in a study examining 90,000 water samples throughout the U.S., 28,000 of which were domestic sources, found that domestic samples had an average activity concentration of 1.7 pCi/l. The population-weighted mean concentration was 0.8 pCi/l. Partridge, Horton and Sensintaffar[24] found experimentally that an average of 50% of the radon in water is released into the air during normal household activities. Generally, water supplies with concentrations less than about 500 pCi/l are considered insignificant sources of radon for indoor air.[25]

The activity concentration in indoor air depends on several factors: the emanation rate of radon from the parent material, the decay rate of each species, the rate of transport into indoor spaces, the rate of removal by ventilation, and the rate of removal by deposition on solid surfaces (e.g., on the walls of the room). In the sections that follow, a model is developed for estimating concentrations of radon and radon progeny in indoor air. The model is used to determine incremental changes in alpha activity associated with weatherization of dwellings. These changes in activity concentrations are then used to estimate the incremental risks of fatal lung cancer.

3. INDOOR RADON CONCENTRATIONS

A number of models have been used to predict the behavior of radon and its progeny in indoor environments.[14,26,27] The model used in this study is similar to that of Kusuda, Silberstein and McNall.[16]

Indoor concentrations of radon and radon progeny are determined by the rates of input and removal. The activity concentration $A_{j,i}$ of species j in indoor air is governed by the species activity balance (see Appendix)

$$dA_{j,i}/dt = Q_j - (\lambda_j + \dot{V})A_{j,i} + \dot{V}A_{j,o} - qA_{j,i}. \quad (1)$$

Here, $A_{j,i}$ and $A_{j,o}$ represent respectively, the indoor and outdoor activity concentrations of the jth radionuclide (for j = 0, 1, 2, 3, 4, numbered sequentially from Rn-222 to Po-214), Q_j is the source strength of species j, $\dot{V}$ is the rate (in dimensionless air changes per hour) at which outside air enters the building (which is also equal to the rate at which indoor air leaves), λ_j is the decay constant for radionuclide j (see Table 2), and q is the deposition rate.

Table 2. Radioactive decay constants and potential alpha energies for Rn-222 and its progeny (cf. Refs. 16 and 28).

Radionuclide	Radioactive Decay Constant, λ_j[a] (hr^{-1})	Atoms/pCi[b]	Potential Alpha Energy Per Atom (MeV)	Potential Alpha Emission Per Unit Activity, C_j	
				MeV/pCi	WL/(pCi/l)
Rn-222	7.548×10^{-3}	17488	19.16[c]	c	c
Po-218	13.632	9.77	13.68	134	0.00103
Pb-214	1.5516	85.3	7.68	659	0.00507
Bi-214	2.1108	63.1	7.68	485	0.00373
Po-214	1.52×10^{7}	8.7×10^{-6}	7.68	6.6×10^{-5}	5.1×10^{-10}

[a] $\lambda_j = (\ln 2)/t_{1/2}$, where $t_{1/2}$ is the half life of radionuclide j.

[b] Atoms/pCi = (3.7×10^{-2} atomic distinegrations/sec/pCi)/(λ_j hr^{-1} x 1 hr/3600 sec).

[c] The alpha energy associated with decay of radon to Po-218 has little relevance to health risk because the half life of Rn is relatively long (about 3.8 days).

The source strength for radon (Q_0) is the total input of radon from building materials, water, and soil, in pCi/l. Values of Q_0 in residences range from 0.01–10 pCi/l-hr, with most measurements between 0.1 and 1 pCi/l-hr.[29] For radon progeny, the source strength Q_j is associated with decay of the previous daughter (see Appendix), i.e.

$$Q_j = \lambda_j A_{j-1}. \tag{2}$$

The deposition rate q has values[30,31] ranging from 0.2–200 hr^{-1} and depends on the size distribution of aerosols and on variations of static electricity charge separation caused by changes in meteorological conditions.[31]

In the steady state, the net accumulation of activity concentration is zero; therefore,

$$Q_j - (\lambda_j + \dot{V})A_{j,i} + \dot{V}A_{j,o} - qA_{j,i} = 0. \tag{3}$$

It follows that the activity concentrations of radon ($A_{0,i}$) and radon daughters ($A_{j,i}$ for $j = 1, 2, 3, 4$) in a room are given, for slowly varying conditions, by the relations

$$A_{0,i} = (Q_0 + \dot{V}A_{0,o})/(\lambda_0 + \dot{V} + q) \tag{4}$$

and

$$A_{j,i} = (\lambda_j A_{j-1,i} + \dot{V}A_{j,o})/(\lambda_j + \dot{V} + q), \tag{5}$$

respectively.

Alpha-activity concentrations† in air are often expressed in terms of working levels (WL). One working level is any combination of radon daughters in one liter of air that upon complete decay to lead-210 results in the emission of 1.3×10^5 MeV of alpha energy. The corresponding unit of exposure is the working level month (WLM), which is an exposure to 1 WL for the equivalent of a working month of 173 hours.‡

The total potential alpha energy concentration, E (in WL or MeV/l) attributable to the radon decay chain is given by

$$E = \sum_{j=1}^{4} C_j A_{j,i}, \tag{6}$$

where C_j represents the potential alpha energy emission per unit activity concentration for complete decay of species j to Pb-210. Values of C_j are given in Table 2.

For radon and the first four progeny in equilibrium, $A_k = A_j$ for all k and j (see the Appendix) and the potential alpha energy emission per unit activity (E_{equil}) is

$$E_{equil} = A_{0,i} \sum_{j=1}^{4} C_j = 0.00983 A_{0,i}. \tag{7}$$

Therefore, an activity of about 102 pCi/l at radioactive equilibrium is equivalent to 1 WL. If the radon and progeny are not in equilibrium, e.g. when the ventilation rate is high, less than 1 WL is produced by concentrations of 102 pCi/l.

The equilibrium factor (F) indicates deviation from equilibrium and is defined as the ratio of the actual total potential alpha energy to the total potential alpha energy for the progeny in equilibrium, i.e.,

$$F = E/E_{equil} = 101.73 \text{ (pCi/l)/WL} \times \sum_{j=1}^{4} C_j A_{j,i}/A_{0,i}. \tag{8}$$

For example, if $F = 0.5$, a radon activity concentration of about 200 pCi/l is equivalent to 1 WL. The deviation from equilibrium in indoor air is affected by the ventilation rate, rate of deposition on walls, and by attachment to aerosols. Because the decay of radon is much

† The activity concentration of a species is the rate of radioactive decay of that species by alpha emission per unit volume, e.g. in pCi/l of air.

‡ 30.4 days/average month × 5 working days/7 days × 8 hours/working day = 174 working hours/month. By convention, a working month is set equal to 173 hours.

slower than the decay of the progeny, the extent to which equilibrium is approached is insensitive to the concentration of radon. Measurements in homes in New York and Sweden show that indoor equilibrium factors are usually between 0.3 and 0.7, with an average of about 0.5.[32,33] Deviations from radioactive equilibrium are substantial because other removal processes with characteristic times comparable to those for radioactive decay are present (e.g. removal by air exchange and deposition on walls).

Outdoor radon and progeny concentrations are governed by the relation[16]

$$\lambda_j A_{j-1,o} - (\lambda_j + I_o)A_{j,o} = 0, \tag{9}$$

where I_o represents the clearance factor and the subscript o identifies outdoor values. The first term represents the production of activity concentration attributable to species j; the second term represents removal of species-j activity associated with radioactive decay and clearance caused by air motion. The clearance factor (I_o) accounts for the removal of radon from the local outdoor space by processes other than radioactive decay, e.g. by deposition and by air flow. Solution of Eq. (7) yields the relation for the outdoor activity concentration of the progeny, viz.

$$A_{j,o} = A_{0,o} \sum_{k=1}^{j} \lambda_k/(\lambda_k + I_o) \qquad \text{for} \qquad j = 1, 2, 3, 4. \tag{10}$$

For specific values of outdoor concentrations of radon $A_{0,o}$ and clearance factors I_o, the concentrations of the progeny can be determined by using Eq. (10).

The equilibrium factor F_o for outdoor air is [see Eq. (8)]

$$F_o = 101.73E_o/A_{0,o}, \tag{11}$$

where the potential alpha activity concentration outdoors (E_o) is given by the relation

$$E_o = A_{0,o} \sum_{j=1}^{4} C_j = [0.00983 \text{ WL/(pCi/l)}]A_{0,o}. \tag{12}$$

Values of F_o between 0.6 and 0.9 have been measured, with an average value of about 0.8.[22]

Given the outdoor equilibrium factor F_o, the clearance factor may be determined by using Eqs. (8)–(10), and outdoor activity concentrations for the progeny may then be determined from Eq. (8). Values of the clearance factor for several values of F_o are shown in Table 3.

4. LUNG-CANCER RISK

Lung cancer is a disease with highest incidence in people aged 55–75 years. The latent period for cancer induction after the initiating event is about 20 years,[34] and lung cancer is rarely diagnosed before 40 years of age. Currently, about 139,000 new cases of lung cancer are diagnosed each year in the U.S.,[35] representing an incidence of about 590 cases annually per million people. Cigarette smoking is responsible for over 75% of these lung cancers, and causes a cancer death rate for smokers close to double that for non-smokers.[35] Radon exposure may be partially responsible for lung cancers in smokers and may be responsible for an even greater fraction of the lung cancers in non-smoking persons.

Respiration of air containing radon progeny results in a radon dose to both primary regions of the lung. The dose to the tracheobronchial region is caused mainly by deposition

Table 3. Clearance factors (I_o) associated with various outdoor equilibrium factors, F_o.

F_o	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
I_o (hr^{-1})	2.64	1.71	1.15	0.77	0.50	0.30	0.13	0.0

and subsequent decay of unattached daughters.[14] The dose to the pulmonary region, that area where gas exchange occurs, is associated with deposition of particles less than 0.17 μm and decay of attached radon progeny.[36]

Epidemiological studies[36–41] have shown an increased incidence of lung cancer in uranium miners associated with exposure to radon. However, these miners were exposed to much higher levels of radon progeny than the general public usually encounters. The dose to the respiratory system is affected by several factors, including the radon concentration, the distribution of progeny between the attached and unattached states, the type of respiration (e.g. nose or mouth breathing, depth and rate) and the actual morphology of the lung. Given the concentration in the air, these factors and the dose to the lung depend primarily on age and physical activity. Exposure duration, dose magnitude, possible synergistic effects of smoking, and latency periods for disease development also must be accounted for in estimating risk for the general public from incidence data for miners.

Most epidemiological evidence concerning the relationship between lung cancer and radon exposure is for uranium miners. Because of greater ventilation and lower aerosol concentrations in residences, more unattached radon daughters are present in dwellings than in uranium mines. Thus, the dose per WLM of exposure, to the tracheobronchial region, that area where bronchogenic lung cancer attributable to radon originates, is greater in residences than in mines.[14] The alpha energy originating in the tracheobronchial region is absorbed in the mucous sheet, ciliated cells and goblet cells of the bronchial epithelium, and may cause lung cancer to eventually begin in the basal cells of the bronchial epithelium.[34] This difference in dose per unit of exposure between mines and residences is not included in the linear model used in this study, so that the estimated risks are probably conservatively low.

In order to assess lung cancer risk associated with radon exposure in residences, the number of hours spent inside the home is required. Time spent at home varies widely and depends on personal habits and circumstances. However, in the well-known study of time budgets, the Multinational Comparative Time Budget Research Project[42] completed in the early 1970's and in which 30,000 people from 12 countries participated, persons between the ages of 18 and 65 years were found to spend, on average, 72.8% of the day (i.e. 17.5 hours per day) inside the home.

Given the radon and progeny concentrations that characterize a particular environment and the time of exposure to that environment, the annual exposure to radon progeny, X (in WLM/yr), can be determined from the relation

$$X = H \times F \times 0.00983 \times A_{0,i}/173, \tag{13}$$

Table 4. Estimates of fatal lung-cancer risk per WLM for exposure to radon.

Study	Risk		Reference
	10^{-6} fatal lung cancers/WLM	10^{-6} fatal lung cancers/WLM-yr	
ICRP (1981)	100-500	---	43
Evans et al (1981)	100	---	39
Jacobi (1977)	200	---	44
UNSCEAR (1977)	200-450	---	22
BEIR (1980)	---	6-47	37
Myers, et al (1981)	100-500	---	45
Cohen (1980)	200-450	---	40
Harley and Pasternack (1981)	---	10	38

where H is the number of hours/yr of exposure to the specified environment. F is the equilibrium factor, and $A_{0,i}$ is the indoor radon concentration (in pCi/l). The term $F \times 0.00983 \times A_{0,i}$ represents the potential alpha energy emission per unit activity, E (in WL), associated with the particular indoor concentrations of radon progeny, and 173 is the total number of hours per working month.

Assuming that the dose-response relation is linear with no threshold, the number of lung cancer deaths (D) per 10^6 people per year can be estimated by using the relation

$$D \text{ (in deaths/}10^6 \text{ p-yr)} = RX \times 10^6, \tag{14}$$

where R is the estimated risk of fatal lung cancer in fatalities/WLM.

Epidemiological data for uranium miners have been used in conjunction with lung-dosimetry models to estimate the risk of fatal lung cancer for the general public per unit of exposure to radon progeny (i.e., per WLM). These estimates vary (see Table 4) by as much as a factor of five from 100×10^{-6} fatal lung cancers per WLM to 500×10^{-6} fatal lung cancers per WLM.

Using an average risk from the BEIR report[37] of 25 fatal lung cancers per year/WLM-10^6 people, 70 years of an average annual environmental exposure of 0.25 WLM, with a 20-year latency period for development of fatal lung cancer (i.e., 50 years of exposure that could result in lung-cancer death) results in a predicted incidence attributable to radon of about 310 fatal lung cancers/yr-10^6 people. This estimate would unrealistically account for about 60% of the present 500 fatal lung cancers/10^6 people per year in the U.S., most of which are actually attributable to cigarette smoking.

A more plausible estimate of the incidence of fatal lung cancer associated with radon exposure is obtained by using a lifetime risk per unit of exposure of 100×10^{-6} fatal lung cancers/WLM which is the lower bound given by three of the studies cited in Table 4. For an average dose of 0.25 WLM/yr, this value of risk per unit of exposure attributes about 5% of the observed number of fatal lung cancers in the U.S. to radon, which is not an unreasonable estimate in view of the significance of cigarette smoking. Therefore, $R = 100 \times 10^{-6}$ fatal lung cancers/WLM is used in this study. Although the resulting estimates of risk contain significant uncertainty, because dose response is assumed linear, the fractional incremental risks of weatherization are unaffected by the specific value of R used.

5. RESULTS

The risk associated with radon in indoor air generally increases with reductions in the air-exchange rate, $\dot{V}$. This risk is illustrated in Fig. 2 for a representative outdoor concentration $A_{0,o} = 0.2$ pCi/l (slightly lower than the average for the U.S.) and an estimated outdoor equilibrium factor $F_o = 0.8$. The observed increase in risk is caused by higher concentrations of radon progeny at lower ventilation rates. Some radon is transported indoors with entering outdoor air; however, because outdoor concentrations are lower than indoor concentrations, the net effect of ventilation and infiltration is to decrease alpha activity in indoor air. For example, a decrease in the air-exchange rate from 1.0 to 0.5 hr^{-1} increases the alpha activity concentration for $Q_0 = 1$ pCi/l-hr from 1.19 to 2.17 pCi/l and correspondingly increases the risk of fatal lung cancer by 27 fatalities per million. An increase in source strength, Q_0, also substantially increases risk. An increase from $Q_0 = 1.0$ to 3.0 pCi/l-hr increases the risk of fatal lung cancer by about 140% to 59/10^6 p-yr, for $\dot{V} = 1$ hr^{-1}, showing the significance of local geology and building construction.

The indoor equilibrium factor F also varies substantially with $\dot{V}$ (see Fig. 3). For air-exchange rates less than 1.0 hr^{-1}, equilibrium factors generally increase with decreasing infiltration rate. At low air-exchange rates, radon and progeny have more time during which to decay indoors, and consequently, equilibrium is more closely approached. At air-exchange rates greater than 1, minimum values of F are observed. These minima occur at the ventilation rate for which the decrease in indoor activity associated with ventilation is exactly compensated by the increase associated with introduction of outdoor air with higher equilibrium factor. Except for low source strengths (e.g., $Q_0 = 0.06$ and 0.3 pCi/l-hr), these minima do not occur for values of $\dot{V}$ that usually occur in residences, i.e., for $\dot{V}$ less than

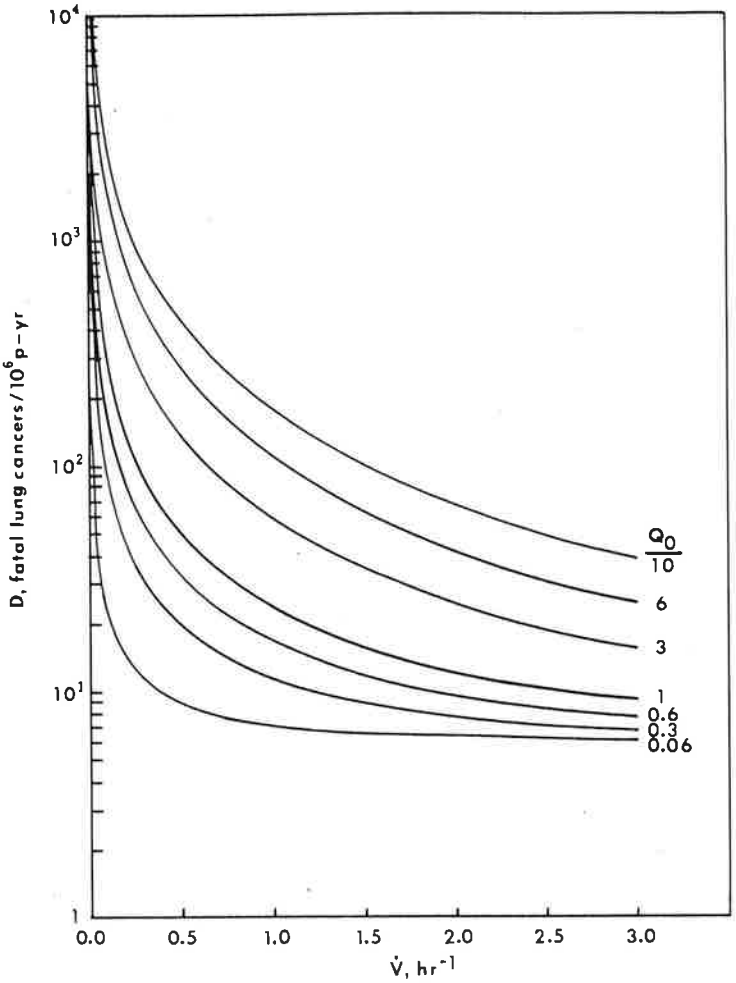

Fig. 2. Risks of fatal lung cancer (D) associated with radon in indoor air in residences are shown as functions of air-exchange rate, $\dot{V}$, for selected indoor source strengths of radon, Q_0 (in pCi/l-hr). Results correspond to 17.5 hrs/day spent in the residence, an outdoor radon concentration $A_{0,o}$ = 0.2 pCi/l and an outdoor equilibrium factor F_o = 0.8.

about 2.0 hr^{-1}. In all cases, as $\dot{V} \rightarrow \infty$, the indoor equilibrium factor approaches the outdoor value F_o, because the composition of the indoor air is then essentially equivalent to that outdoors. Although much less common, air infiltration rates greater than 2 hr^{-1} have been measured in some low-income residences.[5]

The effect of equilibrium factor on the risk of fatal lung cancer is shown in Fig. 4. Risk decreases with decreasing equilibrium factor for most air-exchange rates of interest. However, at high air-exchange rates, F increases with increasing $\dot{V}$, but the risk continues to decrease. This decrease in risk is the result of lower concentrations of radon and all progeny at large $\dot{V}$.

Outdoor equilibrium factors and concentrations of radon also affect the risk of fatal lung cancer. This is shown in Fig. 5 for $\dot{V}$ = 1.0 hr^{-1}. For example, for $A_{0,o}$ = 1.0 pCi/l, F_o = 0.9 and Q_0 = 1.0 pCi/l-hr, D is about 48 fatal lung cancers/10^6 p-yr for $\dot{V}$ = 1.0 hr^{-1}; D is approximately 63 fatal lung cancers/10^6 p-yr for $A_{0,o}$ = 1.5 pCi/l and F_o = 0.9 at the same source strength. The influence of outdoor conditions is even more substantial at higher air-exchange rates.

Our model does not account for the influence of the air-exchange rate on the source strength. Nazaroff et al.[47] have recently shown that the source strength can be highly time dependent and a function of air-exchange rate. This relationship apparently depends on the specific pathways by which radon enters the building, and therefore, is very building specific. The source strength should decrease with decreasing air-exchange rates for houses

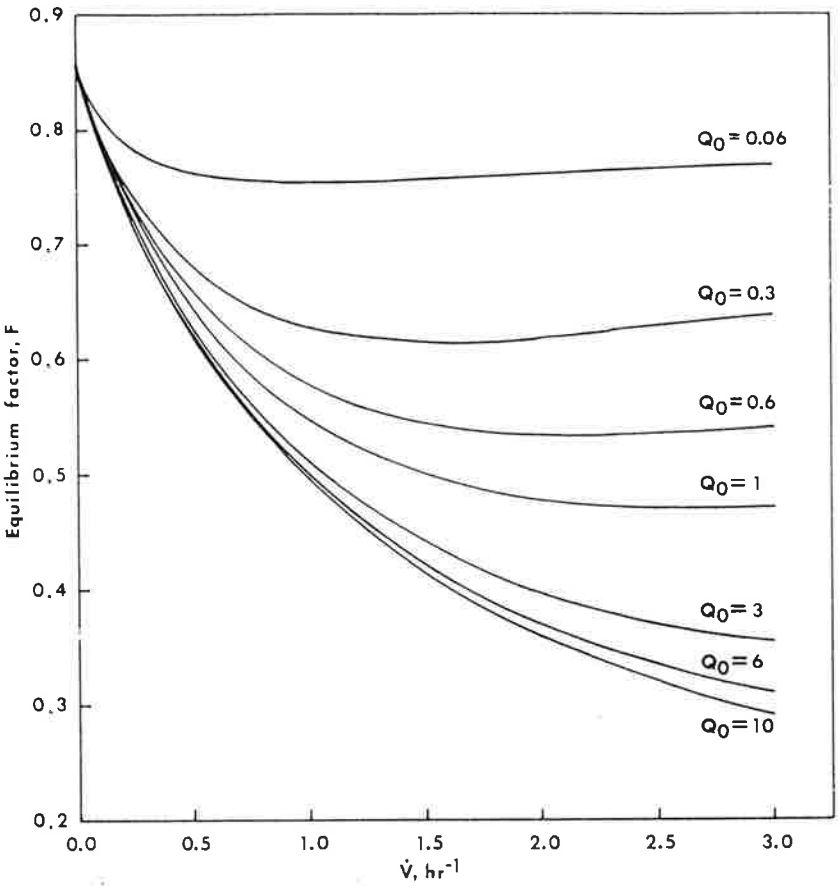

Fig. 3. Equilibrium factors, F, for radon and radon progeny are shown as functions of $\dot{V}$, the infiltration rate for the residence, for selected values of the source strength of radon, Q_0 (in pCi/l-hr).

having significant pressure-driven radon sources. This time dependence is not included here, but deserves further study.

6. INCREMENTAL RISKS ASSOCIATED WITH WEATHERIZATION

Risks for three representative regions are shown in Table 5 for selected air-exchange rates. For a representative house with an air-exchange rate of 1.0 hr^{-1}, outdoor conditions of $F_o = 0.8$ and $A_{0,o} = 0.2$ pCi/l, and a source strength for the indoor space $Q_0 = 1.0$ pCi/l-hr, the estimated risk of lung cancer attributable to radon exposure is 23.6 fatal lung cancers/10^6 p-yr (see Fig. 2).

Weatherization of an existing residence that decreases infiltration by 25% from 1.0 to 0.75 hr^{-1} increases the risk of fatal lung cancer by 37%. For an air-exchange rate of 0.5 hr^{-1}, which is representative of recently built, moderately energy-efficient homes, the risk is 51 fatalities/10^6 p-yr, which is more than double the risk at $\dot{V} = 1$ hr^{-1}.

Geography is an important determinant of both outdoor activity concentrations and indoor source strengths. Health risks are increased substantially in mineralized regions, where outdoor radon concentrations and source strengths are greater. For example, for an outdoor radon concentration of 1.0 pCi/l with $F_o = 0.9$, an air-exchange rate $\dot{V} = 1$ hr^{-1}, and a source strength $Q_0 = 3$ pCi/l-hr, the estimated risk is 82.9 fatalities/10^6 p-yr, about 350% of the risk in an average dwelling. For energy-efficient homes, with ventilation rates of 0.5 hr^{-1}, this risk is approximately doubled. Substantial risks ($D \simeq 48$ fatalities/10^6 p-yr) are found in mineralized regions even for relatively leaky homes with $\dot{V} = 2.0$ hr^{-1} because both radon concentrations in outdoor air and source strengths are significant.

In nonmineralized regions with a representative source strength $Q_0 = 1$ pCi/l-hr, reducing infiltration from 2 to 1 hr^{-1} results in a 95% increase in risk of fatal lung cancer attributable

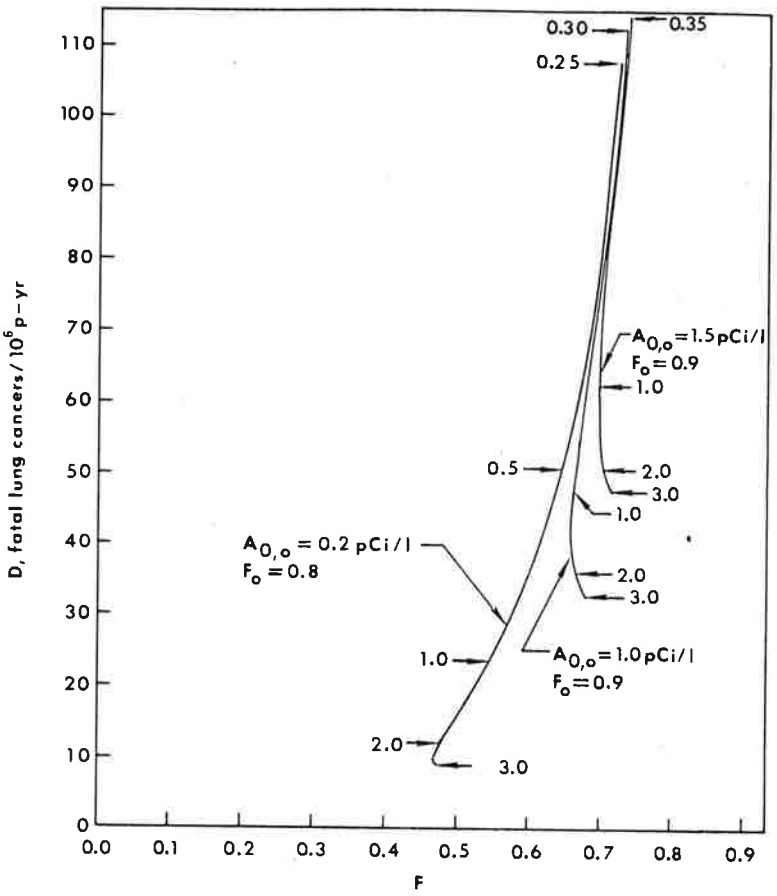

Fig. 4. Risks of fatal lung cancer (D) are shown as functions of the indoor equilibrium factor (F). Results correspond to outdoor radon concentrations $A_{0,o}$ = 0.2, 1.0, and 1.5 pCi/l and a source strength Q_0 = 1.0 pCi/l-hr. Values of $\dot{V}$ (in hr^{-1}) are identified at selected points along each curve.

to radon decay. A reduction from 1 to 0.5 hr^{-1} results in a 115% increase in risk (from 23.6 to 50.7 fatalities/10^6 p-yr). For average outdoor conditions with a low source strength Q_0 = 0.06 pCi/l-hr, reducing infiltration from 2 to 1 hr^{-1} results in an 11.8% increase in risk of fatal lung cancer. An infiltration reduction from 1 to 0.5 hr^{-1} increases risk of death from lung cancer caused by radon by 23.2%.

For homes in mineralized regions, risks are increased by 72% and 98% for the same reductions in infiltration (2 to 1 hr^{-1} and 1 to 0.5 hr^{-1}, respectively); however, the absolute risks are much greater in the mineralized regions (83 and 164 fatalities/10^6 p-yr for 1 and 0.5 hr^{-1}, respectively). Although most of the U.S. can be classified as non-mineralized, outdoor concentrations at some locations are substantial; for example, concentrations as high as 1.5 pCi/l and source strengths of 10 pCi/l-hr have been measured in the mineralized Florida phosphate lands.

Health risks also vary with age of the occupants because breathing rates, activity levels and time spent in the dwelling depend on age. For example, an adult working eight hr/day, five days/week, might spend only about 12 hr/day on average inside the home (after accounting for employment, commuting time and recreational activities outside of the home). In this case, the risk associated with radon exposure in the residence is reduced by about 46%, compared to the previous estimates that are based on an average of 17.5 hr/d in the residence. For a representative home with $\dot{V}$ = 1 hr^{-1} in a non-mineralized area, where $A_{0,o}$ = 0.2 pCi/l, F_o = 0.8 and Q_0 = 1.0 pCi/l-hr, the estimated risk of fatal lung cancer attributable to radon is 16.2/10^6 p-yr compared to 23.6/10^6 p-yr for those who spend 17.5 hr/d inside the home.

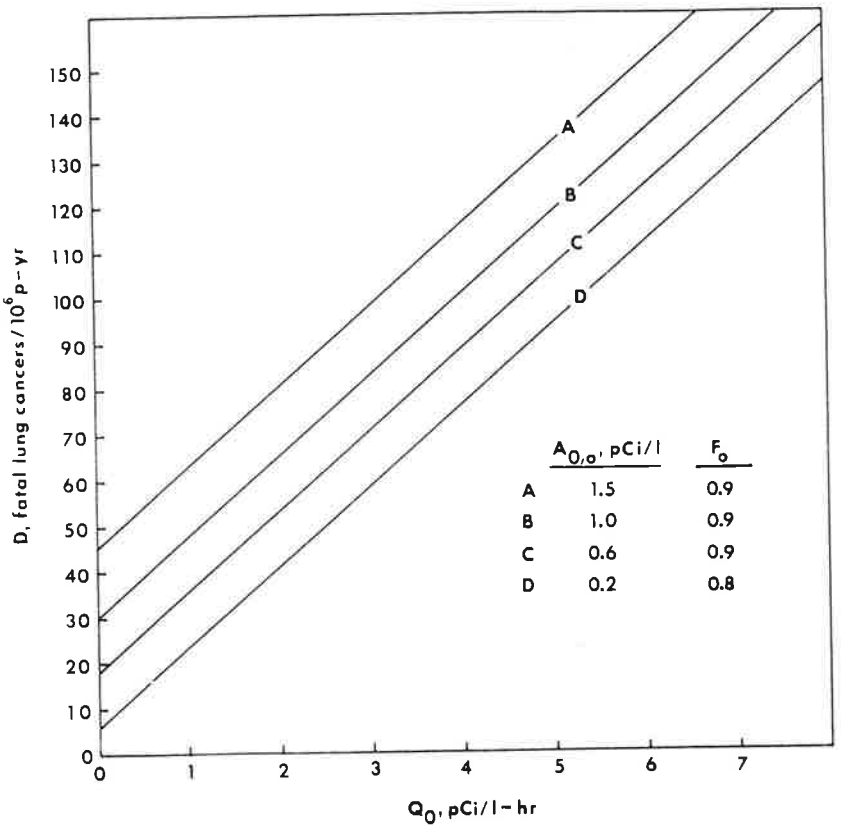

Fig. 5. Risks of fatal lung cancer (D) associated with radon decay products are shown plotted against the indoor radon source strength, Q_0, for selected outdoor radon concentrations and outdoor equilibrium factors, for $\dot{V} = 1\ hr^{-1}$.

Table 5. Risk estimates (in fatal lung cancers/10^6 p-yr) for selected environmental conditions and infiltration rates.

Type of location	Conditions	Risk (fatal lung cancers/10^6 p-yr) for $\dot{V}$ (in hr^{-1}) of:				
		2.00	1.00	0.75	0.50	0.25
Heavily mineralized region; high source strength	Q_0 = 3 pCi/l-hr $A_{0,o}$ = 1.0 pCi/l F_o = 0.9	48.3	82.9	109.0	164.2	335.7
Average nonmineralized region; moderate source strength	Q_0 = 1.0 pCi/l-hr $A_{0,o}$ = 0.2 pCi/l F_o = 0.8	12.1	23.6	32.3	50.7	107.9
Average region; low source strength	Q_0 = 0.06 pCi/l-hr $A_{0,o}$ = 0.2 pCi/l F_o = 0.8	6.3	7.1	7.6	8.7	12.1

Risks associated with radon also depend on the prevalence of the use of natural ventilation for cooling. Risks have been estimated here assuming that air-exchange rates are decreased year-round, which is a reasonable assumption for air-conditioned residences. However, for dwellings without air conditioning, windows are opened during summer months, and consequently the incremental risk associated with weatherization may be as low as 50% of the estimated values.

To accurately estimate the total national health risk attributable to radon, the geographic distributions of radon sources, housing characteristics, and population are required. However, a rough preliminary estimate can be made using average conditions. Using a representative home with an average air-exchange rate $\dot{V} = 1\ \text{hr}^{-1}$, in a non-mineralized region where the outside concentration of radon $A_{0,o} = 0.2$ pCi/l with $F_o = 0.8$, and $Q_0 = 1$ pCi/l-hr, the estimated number of excess lung-cancer fatalities attributable to radon for the entire U.S. population is found to be about 6130 annually. If U.S. residences were retrofitted to reduce average air-exchange rates to 0.5 ach, the number of fatal lung cancers associated with radon would increase by 7040 per year (i.e. by 115%).

Several other factors increase the variation in actual indoor activity concentrations (including the characteristics of the indoor space), the details of which are not included in the model used for this study. The fate of radon decay products indoors is largely dependent on the concentrations and size distribution of aerosols in the space, and the surface/volume ratio of the room. Unattached radon progeny may decay to the next daughter product, attach to particles in the air (in the size range 0.05–0.5 μm), or plate out (i.e. deposit) on surfaces. If decay occurs while a daughter is attached or already deposited, recoil energy from alpha decay may cause the new daughter product to revert to the free state. Attachment, plate out, and recoil may be characterized by rate constants and probabilities of occurrence.[30] For example, the rate constant for the decay of radium-A accompanied by recoil detachment is equal to the decay constant, λ_1, multiplied by the probability that a free ion will be created as a result of the recoil.[27] The attachment rate is proportional to the concentration of aerosols in the space. The constant of proportionality is the attachment coefficient. Porstendorfer and Mercer[48] give an average value for the attachment coefficient of 1.25×10^{-6} cm^3/sec in indoor air. When the indoor concentration of aerosols is between 0.5×10^4 and 2×10^4 particles/cm^3, the average attachment rate is between 22 and 100 particles/hr. In a review, Bruno[30] gives a range of attachment rates of 20–280/hr. The attachment coefficient for outdoor air is about half the indoor value because the fraction of larger particles is greater indoors. Therefore, increased ventilation decreases the value of the attachment coefficient, and, consequently, the attachment rate.

The deposition (plate out) rate constant, q, is dependent on the deposition velocity and the size of the enclosed space. The deposition velocity is the rate at which atoms deposit per unit area (i.e., the flux density) divided by the number of atoms in the space. Airflow is a major determinant of the deposition velocity; however, detailed characterization of airflow in an indoor space is difficult because of its spatial and temporal variability. Thus, deposition velocities, and consequently q, have wide and uncertain ranges. Bruno[30] gives estimates for q of 1–200 hr^{-1} without distinguishing between attached and unattached fractions. Swedjemark[31] reports that attached particles have a deposition rate of approximately 0.2 hr^{-1} while the unattached fraction deposits at a rate of 30 hr^{-1}.[29] Measurements of the fraction of radium A in outdoor air that is unattached show a mean value of 0.07, with 0.2 as an upper limit.[49] The unattached fraction consists mostly of radium A. As the result, because most radon progeny in a representative indoor environment are attached, the deposition rate for unattached particles contributes negligibly to the weighted average deposition rate. Therefore, we use the value for attached progeny of 0.2 hr^{-1} as the average for all daughters. When more suspended particulates are present, e.g. in rooms with smokers, the attachment rate increases somewhat, and the deposition rate would be expected to decrease, increasing both the indoor activity concentration and equilibrium factor F. Increased concentrations of fine respirable particulates also may result in retention of more particles in the respiratory system and increase the dose to the lung associated with attached progeny. These interactions between radon progeny and particulates, and the relationships between activity concentrations, particle-size distributions and number densities, and radiation dose

to the lung, particularly in environments with smokers, require further study. These factors then could be used to determine the risk of specific indoor environments.

The incremental risk of fatal lung cancer attributable to increased exposure to radon associated with weatherization of residences can be related to the energy saved as the result of decreased infiltration (see Table 6). For average climatic conditions for St. Louis (about average for the U.S.) and representative radon concentrations for a non-mineralized region with a moderate radon source strength, a reduction of infiltration from 1 ach to 0.25 ach for a representative air-conditioned residence with two occupants and a total volume of 1200 ft^3 (340 m^3) results in an incremental risk of 6.15×10^{-6} deaths/GJ. Using a simpler procedure to estimate the incremental change in activity concentration associated with weatherization, and an estimate of 300 fatal lung cancers/10^6 p-WLM (an estimate we consider high), Inhaber[7] estimated a risk per unit of energy saved of 1.15×10^{-5} deaths/GJ (1.17×10^{-5} deaths/10^6 p) for weatherization of residences which decreases air exchange from 1 to 0.2 ach. For a reduction in $\dot{V}$ from 1 ach to 0.25 ach for a residence in a mineralized region with a high source strength our risk estimate is 18.4×10^{-6} deaths/GJ, an estimate that is comparable in magnitude to Inhaber's. Although the estimated lung-cancer risk is small compared to that of cigarette smoking, the results show that except for dwellings with low source strengths in non-mineralized regions, decreased air-exchange rates can lead to significant increases in the risk of fatal lung cancer from radon exposure.

Because energy conserving trends will undoubtedly continue when energy prices increase again, care should be exercised to limit the detrimental effects on human health of radon in indoor air. Although smaller than Inhaber's[7] estimate (except for mineralized regions with high source strengths), our risk estimates show that in the absence of special treatments, increases in radon concentrations associated with reducing infiltration rates can represent significant health risks. The incremental incidence of lung-cancer fatalities attributable to weatherization will not become apparent immediately because of the long latency periods before actual development of cancer. Statistical measurement, if possible at all, will not be possible until decades after weatherization is completed on a large scale. At a minimum, prudence should be exercised in implementing conservation measures that decrease air-exchange rates, especially in regions where radon source strengths are known to be high. Better understanding of radon transport to indoor spaces, the relationships of air-exchange rate to source strength and removal processes, and the effectiveness of control measures is required to select options that will ultimately limit long-term risk.

Table 6. The incremental risk ($\Delta D/\Delta E$) of fatal lung cancer per unit of energy saved for incremental reductions in $\dot{V}$ of 0.25, 0.50 and 0.75 hr^{-1} from 1.0 hr^{-1}.

Type of location	Conditions	$\Delta D/\Delta E$ (fatal lung cancers/GJ) for $\Delta\dot{V}$ of:		
		0.25	0.50	0.75
Heavily mineralized region; high source strength	Q_0 = 3 pCi/l-hr $A_{0,o}$ = 1.0 pCi/l F_o = 0.9	5.71×10^{-6}	8.89×10^{-6}	1.84×10^{-5}
Average nonmineralized region; moderate source strength	Q_0 = 1 pCi/l-hr $A_{0,o}$ = 0.2 pCi/l F_o = 0.8	1.90×10^{-6}	2.96×10^{-6}	6.15×10^{-6}
Average region; low source strength	Q_0 = 0.06 pCi/l-hr $A_{0,o}$ = 0.2 pCi/l F_o = 0.8	1.16×10^{-7}	1.79×10^{-7}	3.66×10^{-7}

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APPENDIX

Relationship between species mass balances and species activity balances

The mass balance for species j in indoor air for a perfectly mixed room may be expressed as

$$dN_{j,i}/dt = \dot{N}_j - (\lambda_j + \dot{V})N_{j,i} + \dot{V}N_{j,o}, \tag{A-1}$$

where $N_{j,i}$ and $N_{j,o}$ are the number densities of radionuclide j in indoor and outdoor air, respectively, $\dot{N}_j$ is the rate at which species j is produced in the room or injected into it, λ_j is the decay rate (i.e., the fraction of j-atoms that decay per unit time), and $\dot{V}$ is the air-exchange rate in dimensionless volumes per unit time. Because no significant radon is produced in the air, the source strength for radon, Q_0, is the result of emanation from building materials and soil beneath the building. Because the half-life of radon is almost four days, and the half-lives of the four progeny are all less than 30 minutes (see Fig. 1), the primary source of radon progeny is the decay of the parent radionuclide, i.e.

$$\dot{N}_j = \lambda_{j-1}N_{j-1,i} \qquad \text{for} \qquad j = 1, 2, 3, 4, \tag{A-2}$$

and, therefore, Eq. (A-1) becomes

$$dN_{j,i}/dt = \lambda_{j-1}N_{j-1,i} - (\lambda_j + \dot{V})N_{j,i} + \dot{V}N_{j,o}. \tag{A-3}$$

The number density is related to the activity concentration by

$$\begin{aligned} N_{j,i}(j \text{ atoms/l}) &= A_{j,i}(\text{pCi of } j/\text{l}) \times K_j(j \text{ atoms/pCi}) \\ &= A_{j,i}K/\lambda_j, \end{aligned} \tag{A-4}$$

where $A_{j,i}$ is the indoor activity concentration of species j, $K_j = K/\lambda_j$, and $K = 3.7 \times 10^{-2}$ atom disintegrations/sec per pCi. Upon substitution of Eq. (A-4) into the Eq. (A-1), we obtain the species activity balance,

$$\begin{aligned} dA_{j,i}/dt &= \lambda_j A_{j-1,i} - (\lambda_j + \dot{V})A_{j,i} + \dot{V}A_{j,o} \\ &= Q_j - (\lambda_j + \dot{V})A_{j,i} + \dot{V}A_{j,o}, \end{aligned} \tag{A-5}$$

where $Q_j = \lambda_j A_{j-1,i}$ is the source strength for species j.

At equilibrium, the net rate of production of all species is zero, i.e., $dN_{j,i}/dt = 0$ for all species j. Equilibrium can only be reached when the rates of injection and removal by ventilation are very slow compared to the rates of radioactive decay. Therefore, from Eq. (A-1) with $\dot{V} \simeq 0$, the condition for equilibrium is

$$dN_{j,i}/dt = \lambda_{j-1}N_{j-1,i} - \lambda_j N_{j,i} = 0.$$

Using Eq. (A-4) to express the number concentrations in terms of activity concentrations, we obtain the relation for equilibrium

$$A_{j-1,i} = A_{j,i} \qquad \text{for} \qquad j = 1, 2, 3, 4$$

or

$$A_{1,i} = A_{2,i} = A_{3,i} = A_{4,i} = A_{0,i}. \tag{A-6}$$