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Assessment of the Exposure to and Dose from Radon Decay Products in Normally Occupied Homes

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The exposure to radon decay products has been assessed in seven homes in the northeastern United States and southeastern Canada. In two of the houses, there was a single individual who smoked cigarettes. There were a variety of heating and cooking appliances among these homes. These studies have provide 565 measurements of the activity-weighted size distributions in these houses. The median value for the equilibrium factor was 0.408 as compared with the previously employed value of 0.50. Using the recently adopted ICRP lung deposition and dosimetry model, the hourly equivalent lung dose rate per unit, radon exposure was estimated for each measured size distribution. The mean equivalent dose rate per unit of ^{222}Rn gas concentration was approximately 140 nSv h^{-1} Bq^{-1} m^{-3}. It was found that the equivalent dose was strongly correlated with the ratio of the decay product concentration to that of radon, termed the equilibrium factor, *F*, with a correlation coefficient of 0.785. The correlation coefficient with the ≤2-nm size fraction (the "unattached" fraction) was 0.169, reflecting no significant relationship with the unattached fraction. Differences between houses with smokers present and absent were noted in the exposure conditions, but the resulting dose rate per unit of radon gas concentration was essentially the same for the two groups. Expressed in terms of ICRP's unit of effective dose for members of the public, the mean dose rate conversion coefficient with respect to radon gas concentration found in this study was 3.8 nSv h^{-1} Bq^{-1} m^{-3}.

Introduction

During much of the past decade, there has been considerable attention directed toward the potential problem of the exposure of the general population to radon and its decay products in their homes. In an effort to estimate the potential effects of radon on public health, the U.S. Environmental Protection Agency has estimated that 13 600 lung cancer deaths per year can be attributed to the presence of radon in indoor air (*1*). This estimate is based on the risk assessment developed by the National Research Council's Committee on the Biological Effects of Ionizing Radiation IV (*2*) and a dosimetric extrapolation from exposure conditions to miners in underground mines to the exposure of the general public in their homes (*3*). However, the exposure/risk relationship in that analysis is based on an estimate of the decay product concentrations using single point estimates for the physical characteristics of airborne radon decay products in these different environments, namely, the unattached fraction and median particle size of the attached decay products. It does not include detailed dosimetric assessment based on the complete size distribution of radon decay products. With recent improvements in measurement methods to permit the direct determination of activity-weighted size distributions in normally occupied homes (*4*) and the International Commission on Radiological Protection's (ICRP's) new dosimetric lung model (*5*), it is now possible to measure the distribution of exposures across a number of houses as a function of particle size and from these measurements to obtain a distribution of equivalent lung dose. The purposes of this report are as follows: (1) to summarize the available measurements in houses located in the northeastern United States and southeastern Canada; (2) to provide the resulting distribution of the conversion coefficient between radon gas concentration and equivalent lung dose; and (3) to quantify the dependence of the dose conversion coefficient on aerosol size. We also examine the possible implications of these data for setting a representative conversion coefficient between radon gas concentration and the annual effective dose, ICRP's index of risk.

Experimental Procedures

The measurements of activity-weighted size distributions were made using an automated system with six parallel sampling heads that utilized various types and a number of layers of wire screens to separate the particles on the

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TABLE 1

Summary of Sampling Campaigns and Locations That Provided Activity-Weighted Size Distributions in Normally Occupied Homes

location	house ID	sampling period	no. of samples	smoker	heating system	stove	ref
Northfield, CT		March–April 1990	30	no	hot water baseboard, oil-fired	gas with pilot light	6
Princeton, NJ	31	Nov 1990	61	yes	forced air, gas-fired	gas with pilot light	9
Princeton, NJ	31	Feb 1991	52	yes	forced air, gas-fired	gas with pilot light	9
Princeton, NJ	41	Apr 1991	30	yes	forced air, gas-fired	gas without pilot light	9
Arnprior, ON		May–July 1991	208	no	electric baseboard	electric	7
Parishville, NY		February–March 1992	59	no	forced air, oil-fired		8
Princeton, NJ	51	Mar 1992	46	no	forced air, gas-fired		
Princeton, NJ	51	July 1992	21	no	forced air, gas-fired		
Princeton, NJ	51	Mar 1993	58	no	forced air, gas-fired		

basis of their size over the range of 0.5–500 nm. A detailed description of this automated, semicontinuous, graded screen array system is given by Ramamurthi and Hopke (*4*). A series of measurements were made in six houses. The locations of these houses and some of their characteristics are presented in Table 1. The results for several of these houses have been gathered in order to provide baseline data for a series of studies of the behavior of air cleaners; Northford (*6*), Arnprior (*7*), and Parishville (*8*). Preliminary results including Princeton houses 31 and 41 and 1 week of the Arnprior data have been reported by Wasiolek *et al.* (*9*). Details of these studies are presented in the referenced reports. Three other houses have been studied, but the measurements were made in the basement and may not represent the living areas of these homes. In two other homes, studies were made while they were unoccupied, and particles were produced through various activities such as cooking, vacuuming, and smoking. However, these results are also unlikely to belong to the distribution of activity-weighted size distributions produced in normally occupied homes. Therefore, these results were excluded from the distributions presented in this report. In all cases, radon concentrations were also measured using standard continuous radon monitors.

Background. ^{222}Rn is a gaseous element formed by the decay of ^{226}Ra in the ^{238}U series. Emanating from the earth's surface, radon is present in the air with indoor concentrations usually higher than those observed outdoors. The first four radon decay products (^{18}Po, ^{214}Pb, ^{214}Bi, and ^{214}Po) are referred to as the short-lived decay products because their half-lives are less than 30 min each. ^{218}Po and ^{214}Pb decay with the emission of α-particles, and ^{214}Pb and ^{214}Bi decay by β-particle emission. Disintegration of radioactive atoms is a spontaneous event measured in the number of decays per unit of time. The individual activity concentrations of radon and its decay products in air are expressed in units of becquerel per cubic meter (Bq m^{-3}), where 1 Bq is equal to 1 disintegration/s. In the United States, the airborne concentrations are expressed in picocuries per liter (pCi L^{-1}), where 1 pCi L^{-1} is equal to 37 Bq m^{-3}. The measure of the airborne concentration of the total potential α-energy of the short-lived radon decay products present in a volume of air is called the Potential Alpha Energy Concentration (PAEC) and is calculated from individual activity concentrations by

$$C_p = (5.79C_1 + 28.6C_2 + 21.0C_3) \times 10^{-10} \quad (1)$$

where C_p is the PAEC in J m^{-3}, and C_1, C_2, and C_3 are the activity concentrations of ^{218}Po, ^{214}Pb, and ^{214}Bi, respectively (in Bq m^{-3}).

The newly formed radon decay products can attach to airborne particles or room surfaces. The attachment to aerosols causes changes in the size distribution, and attachment to surfaces removes radon progeny from the air. The fraction of radon progeny in an ultrafine mode (0.5–2.0 nm), not attached to ambient particles, is known as the "unattached" fraction. However, there are uncertainties in the definition of this fraction, and its utility as a specific measure of the size distribution of activity is questionable (*10*).

The losses of radon progeny from the air produce a radioactive nonequilibrium between radon and its decay products. The ratio of the decay product concentration to that of radon, termed the equilibrium factor F, is expressed as

$$F = \frac{1.81 \times 10^8 C_p}{C_0} \quad (2)$$

where C_0 is the radon concentration (in Bq m^{-3}). The exposure to short-lived radon decay products is defined as the integral over time of the potential α-energy concentration (PAEC) to which an individual is exposed (expressed in J m^{-3} h^{-1}).

Radon Progeny Dosimetry. The radioactive decay of inhaled short-lived radon progeny in the respiratory tract results in the deposition of α-energy in the cells of epithelial tissue. This energy deposition may result in the special damage to the nuclear DNA that will lead to the transformation of a normal cell into a malignant cell. The dosimetric quantity that gives the measure of the radiation energy absorbed in tissue is the absorbed dose, D, expressed in gray (Gy). The amount of damage caused to each tissue, T, is represented by the equivalent dose H_T, expressed in sievert (Sv), which is the absorbed dose multiplied by the radiation weighting factor, w_R, of 20 for α-particles (*11*). Deposition of radon progeny in the respiratory tract causes α-irradiation of several epithelial tissues; in the nasal passages, the bronchi, the bronchioles, and the alveolated part of the lungs (*3*). Each of these tissues is potentially at risk for carcinogenesis (*5*).

Dose Calculation. In the various models that have been used to evaluate doses to the lungs from inhaled radon progeny [e.g., Jacobi and Eisfeld (*12*), Harley and Pasternack (*13*), Hofmann (*14*), NEA (*15*), and James (*16*)], a higher dose per unit of exposure was assigned to the unattached fraction. However, as discussed above, the exact range of sizes that are assigned to this fraction in previous measurements have varied widely (*17*). This imprecision in definition has necessitated more detailed assessments in

which the dose conversion coefficients are calculated as continuous functions of particle size. The recent comprehensive evaluation of the conversion factor between exposure to PAEC and absorbed doses was made by a scientific panel of the National Research Council (*3*). Their dosimetry model incorporated up-to-date treatments of lung ventilation, aerosol deposition behavior, and anatomical descriptions of target cells, but these treatments were refined further by the ICRP (*5*). Both of these dosimetry models consider the nuclei of secretory cells as well as those of basal cells as potentially sensitive targets in the respiratory tract. The ICRP's new respiratory tract deposition model also incorporates more accurate values for the filtration efficiency of the nasal passages (*18*) than the NRC model (*3*). As described by James *et al.* (*19*), the new values for nasal filtration efficiency resulted in decreased doses from very small particles (below 10 nm) by a factor of around 2 in comparison with previous estimates. To use the newly adopted ICRP lung model to evaluate the equivalent lung dose as a function of the radon progeny activity-size distribution, the software code LUDEP (*20*), which calculates regional lung deposition per unit exposure, was extended to calculate tissue doses from the short-lived radon progeny as described by Birchall and James (*21*).

The revised conversion coefficients between airborne radon progeny concentration and the equivalent dose rate to the lungs as a whole, calculated as functions of monodisperse particle size, that are given by ICRP's lung dosimetry model are presented in Figure 1a–c), for ^{218}Po, ^{214}Pb, and ^{214}Bi, respectively. A breathing rate of 0.79 m^3 h^{-1} is assumed to represent the average breathing rate of a reference subject (adult male) over the 7000 h yr^{-1} assumed to be spent indoors at home (*22*). This average breathing rate is composed of 55% of the time sleeping (0.45 m^3 h^{-1}), 15% of the time sitting (0.54 m^3 h^{-1}), and 30% of the time doing light exercise (1.5 m^3 h^{-1}).

Results and Discussion

The objective of this work was to combine all of the measurements in the houses in order to estimate the distribution of exposures and resulting doses. A problem arises in that there are differing numbers of measurements in these various houses. As an initial effort, it is assumed that there exists a single distribution to which all of the measurements belong. The results for these measurements are presented as cumulative distribution functions. The distribution of equilibrium factor, *F*, based on the complete set of results (565 samples) is presented in Figure 2. Summary statistics for this distribution are presented in Table 2. The geometric mean value of *F* is 0.374 and the geometric standard deviation is 1.57, while the arithmetic mean is 0.408 with an arithmetic standard deviation of 0.159. Thus, the value of *F* in this sample of North American homes is lower than the previous estimate of 0.50 used by the Environmental Protection Agency in their risk calculations. It is similar to the values observed in German homes by Reineking and Porstendörfer (*23*).

For a given radon gas concentration, and in the absence of any compensating factors, the variation of *F* would translate into a proportional variation in lung dose rate. The observed variation in the hourly equivalent lung dose per unit of radon gas concentration found in this study is presented in Figure 3. Summary statistics for this distribution are presented in Table 2. The variation of lung dose

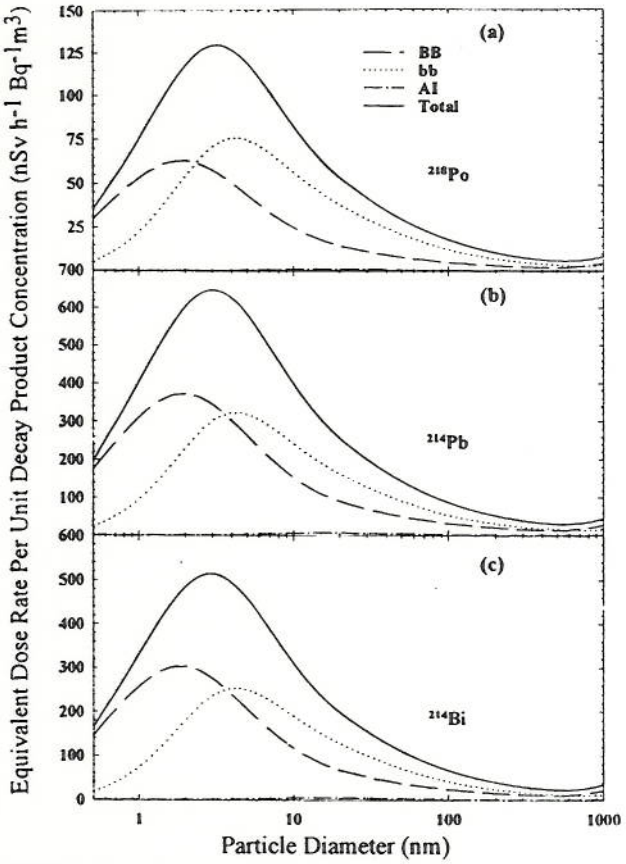

FIGURE 1. Equivalent lung dose conversion factor between airborne concentration of the short-lived radon progeny (in Bq m^{-3}) and equivalent dose rate (in nSv h^{-1}) as a function of carrier particle diameter (in nm). The overall equivalent dose rate to the lungs as a whole is shown together with the contributions from dose received by the bronchial region (BB), the bronchiolar region (bb), and the alveolar-interstitial region (A-I); (a) for ^{218}Po, (b) for ^{214}Pb, and (c) for ^{214}Bi.

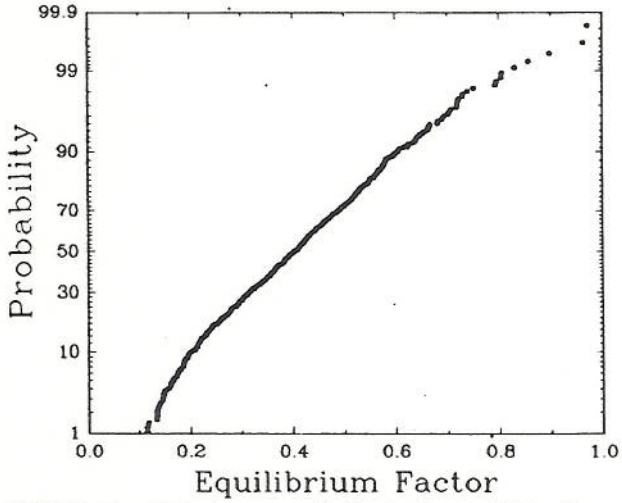

FIGURE 2. Cumulative frequency distribution for equilibrium factor based on the complete set of measurements.

rate normalized to radon gas concentration is found to be almost as great as that of *F*. The geometric standard deviation (gsd) of the dose rate is 1.49, compared to a gsd of 1.57 for *F*. However, it has been thought for several years that variability in *F* is largely compensated by an inverse variability of the so-called unattached fraction, leading to the expectation of a relatively constant dose conversion factor with respect to the radon gas concentra-

TABLE 2

Summary Statistics for Measured or Calculated Distributions

	arithmetic mean	arithmetic standar deviation	geometric mean	geometric standard deviation
all houses				
equilibrium factor	0.408	0.159	0.374	1.6
unattached fraction	0.047	0.032	0.033	3.2
hourly equivalent lung dose per unit of radon concn (nSv h^{-1} Bq^{-1} m^3)	139	51	130	1.5
houses with smokers present				
equilibrium factor	0.481	0.108	0.469	1.3
unattached fraction	0.040	0.022	0.033	2.0
hourly equivalent lung dose per unit of radon concn (nSv h^{-1} Bq^{-1} m^3)	135	29	132	1.2
houses without smokers present				
equilibrium factor	0.383	0.166	0.346	1.2
unattached fraction	0.050	0.035	0.033	3.6
hourly equivalent lung dose per unit of radon concn (nSv h^{-1} Bq^{-1} m^3)	140	57	129	1.5

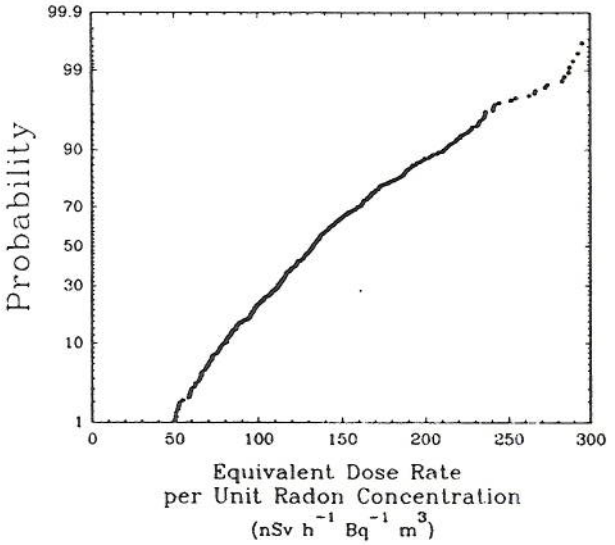

FIGURE 3. Cumulative frequency distribution for hourly equivalent lung dose (in nSv h^{-1}) per unit of radon gas concentration (in Bq m^{-3}) based on the complete set of measurements.

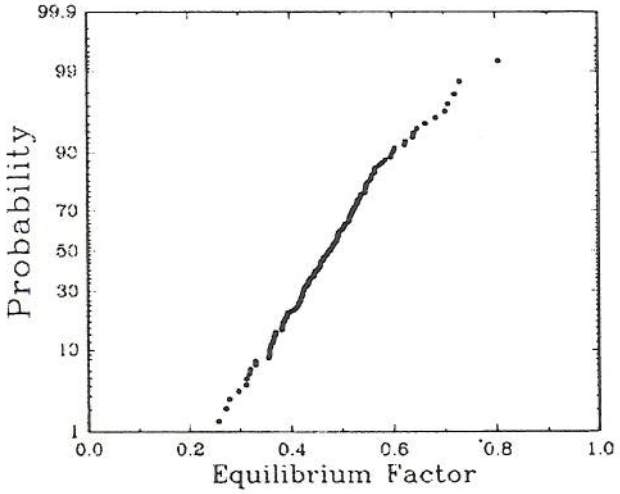

FIGURE 4. Cumulative frequency distribution for equilibrium factor based on the set of measurements for houses with a smoker.

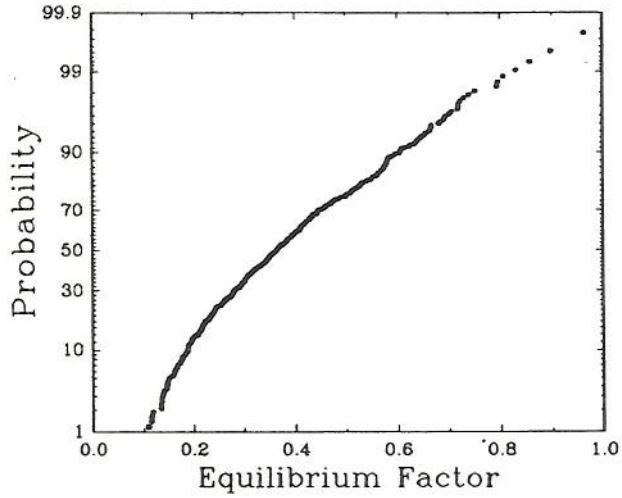

FIGURE 5. Cumulative frequency distribution for hourly equivalent lung dose (in nSv h^{-1}) per unit of radon gas concentration (in Bq m^{-3}) based on the set of measurements for houses with a smoker.

tion (*24, 25*). This concept was adopted by ICRP (*22*) in recommending action levels for protection against exposure at home and at work in terms of just the concentration of radon gas, which is very simple to monitor. We, therefore, examined our results to identify what aerosol size properties had the most influence on the dose conversion coefficient with respect to radon gas concentration (DCC_{Rn}).

Wasiolek et al. (*9*) had earlier reported that the presence of a smoker in a home substantially affected the value of DCC_{Rn}. Thus, we segregated our data into a nonsmoking group (422 samples) and a smoking group (143 samples). The corresponding distributions for the equilibrium factor for the nonsmoking and smoking houses are given in Figures 4 and 5, respectively. Figures 6 and 7 give the distributions of the equivalent lung dose rate normalized to unit concentration of radon gas. The summary statistics for these distributions are also presented in Table 2.

In the case of the separated distributions, the influence of the smoker on the exposure conditions can be observed in the values in this table. The equilibrium factor in the homes with smokers is higher than in the nonsmoker homes, where the arithmetic mean value in the smoker homes is 0.481 and the geometric mean is 0.469, while the nonsmoker values are 0.383 and 0.346. However, the dose rates per unit of radon gas concentration (DCC_{Rn}) for both groups are quite similar. The relationship between DCC_{Rn} and the airborne activity per unit of radon gas concentration, F, can be seen in Figure 8. If DCC_{Rn} is regressed against the equilibrium factor for all homes, the resulting correlation coefficient is 0.785, indicating a strong relationship. The correlation coefficients for the groups separated on the basis of the presence or absence of a smoker are 0.789 and 0.829, respectively. The parameters for the linear fits are given in Table 3.

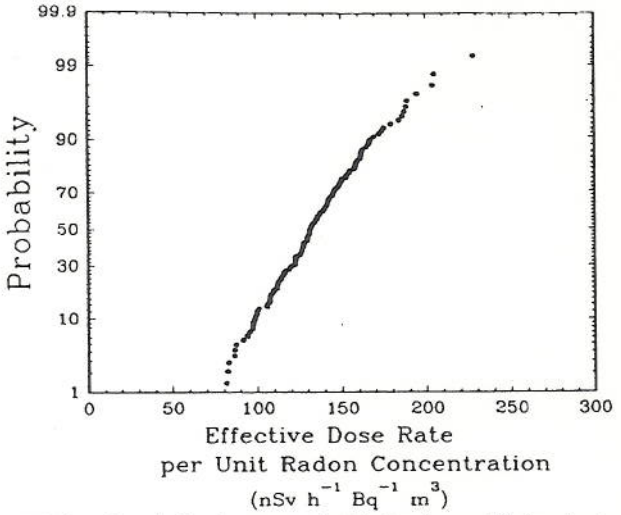

FIGURE 6. Cumulative frequency distribution for equilibrium factor based on the set of measurements for houses without a smoker.

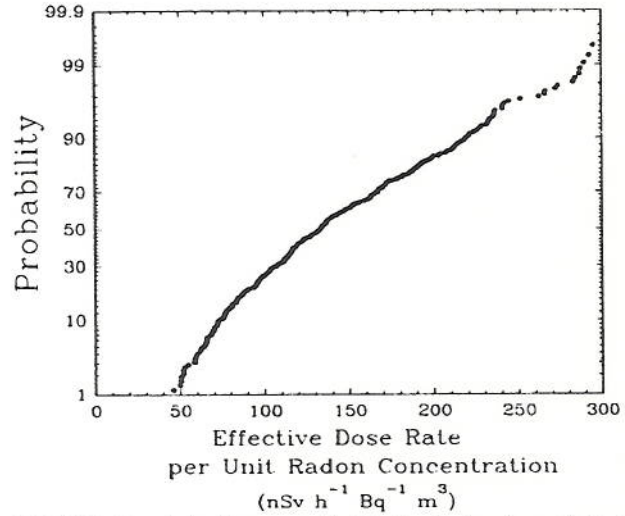

FIGURE 7. Cumulative frequency distribution for hourly equivalent lung dose (in nSv h^{-1}) per unit of radon gas concentration (in Bq m^{-3}) based on the set of measurements for houses without a smoker.

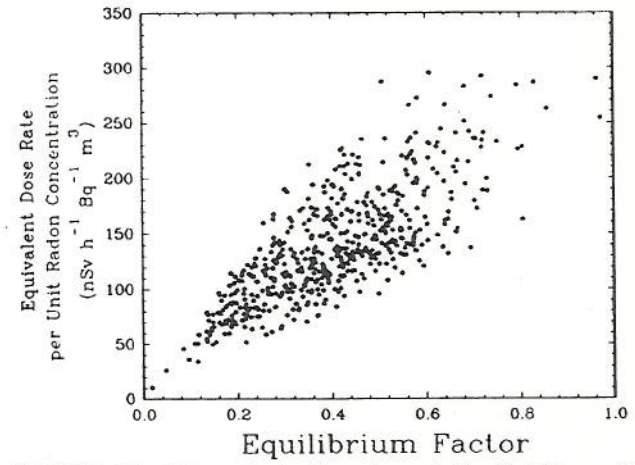

FIGURE 8. Plot of the equivalent lung dose rate (in nSv h^{-1}) per unit of radon gas concentration (in Bq m^{-3}) as a function of *F* for all of the measurements.

It is seen from Table 2 that the mean values of unattached fraction are similar for houses with and without smokers present, *i.e.*, 0.04 and 0.033 for the arithmetic and geometric means, respectively, in houses with smokers present and 0.05 and 0.033 for houses without smokers. However, the variability in the unattached fraction is substantially higher in houses without smokers. Overall, the value of DCC_{Rn} is not related to the unattached fraction. Considering all of the data, the correlation coefficient for DCC_{Rn} as a function of f_p is 0.139, and similar values are found for homes with and without smokers. Thus, the dose per unit of radon is not a strong function of the classical unattached fraction.

TABLE 3

Parameters of Relationships between Hourly Equivalent Dose per Unit of Radon Concentration and *F*

	all homes	smoker present	no smokers present
correlation coeff	0.786	0.790	0.836
slope	252 ± 8	213 ± 14	284 ± 9
intercept	37 ± 32	33 ± 18	32 ± 31

Converting Lung Dose to Risk. The ICRP expresses lifetime risk of cancer and any other effects of radiation in terms of an effective dose, *E*. Like the equivalent tissue dose discussed above, the effective dose is also expressed in sievert (Sv):

$$E = H_T w_T \tag{3}$$

where H_T is the equivalent dose received by tissue, *T*, and w_T is the tissue weighting factor. However, to estimate the risk of lung cancer in the case of exposure to radon progeny, ICRP does not recommend this standard dosimetric approach. Instead, ICRP (*22*) uses a risk estimate that is based directly on the excess incidence of lung cancer found from epidemiological studies of underground miners, which is expressed in terms of lifetime excess risk per unit of exposure to potential α-energy. The recommended value of this risk coefficient is 7.9×10^{-5} mJ^{-1} h^{-1} m^3 (*22*).

To express an amount of radon progeny exposure in terms of effective dose, ICRP recommends a so-called dose conversion convention. Since, for exposure over the "working lifetime" (between the ages of 18 and 64 yr) each 1 mSv of effective dose received by the lungs is considered to increase the total lifetime excess risk of lung cancer by 5.6×10^{-5} (*11*), the reference risk coefficient of 7.9×10^{-5} mJ^{-1} h^{-1} m^3 for an underground miner determines the dose conversion convention as 1.4 mSv mJ^{-1} h^{-1} m^3. However, it can be shown (*21, 26*) using ICRP's new lung dosimetry model, with values of the unattached fraction of potential α-energy and radon progeny aerosol size thought to typify underground mining conditions (*3*), that the calculated dose conversion coefficient given by eq 5 is approximately 5 mSv mJ^{-1} h^{-1} m^3. Therefore, in order to make the modeled effective dose per unit of exposure consistent with the epidemiological risk estimate, it is necessary to introduce a normalizing factor of approximately 0.3. Thus, the effective dose, E_{lung}(mine), from inhalation of radon progeny in an underground mine is given by:

$$E_{lung}(\text{mine}) = H_{lung} w_{lung} \times 0.3 \tag{4}$$

where w_{lung} is the tissue weighting factor of 0.12 for the lungs (*11*).

For exposure in the home, however, ICRP considers the effective dose differently from the "occupational" dose because irradiation in the home can occur over the whole natural lifespan. ICRP prudently assumes that a given amount of dose received by a member of the "general

public" (e.g., in the home) leads to a *higher* lifetime excess risk than the same amount of occupational dose. Thus, the recommended risk factor for the general public is 7.3 $\times 10^{-5}$ mSv^{-1}, i.e., 30% higher than that for 1 mSv received at work. On the other hand, for the specific case of exposure to radon progeny, ICRP assumes explicitly that a given amount of dose received over the whole natural lifetime or over the shorter "working" lifetime bears the same total risk of lung cancer (*22*). In order to imply the correct risk therefore, it follows that the effective dose calculated for exposure in the home must be reduced by the factor of 5.6 $\times 10^{-5}$ / 7.3 $\times 10^{-5}$, i.e., 0.77. Thus, the effective dose, E_{lung}(home), from exposure to radon progeny at home is given by

$$E_{lung}(\text{home}) = H_{lung} w_{lung} \times 0.23 \qquad (5)$$

where the factor of 0.23 is the product of the normalization factor (0.3) from eq 5 and 0.77.

Using this epidemiological normalization factor of approximately one-quarter, from Table 2, the overall arithmetic mean value of the conversion coefficient from radon gas concentration to effective dose rate is 3.8 nSv h^{-1} Bq^{-1} m^3. This is approximately double the value of 2.1–2.4 nSv h^{-1} Bq^{-1} m^3 assumed by ICRP in recommending a range for the action level in dwellings of 200–600 Bq m^{-3}, taken to correspond to 3–10 mSv y^{-1} (*22*).

Conclusions

Activity-weighted size distributions have been measured in seven homes. In several cases, there was a person who lived in the home that smoked. The distributions of airborne radioactivity for each class of homes have been presented. Although there are differences between the smoker and nonsmoker houses in the equilibrium factor, the size dependence of dose as a function of particle size results in similar average dose rates per unit of radon gas concentration for the two groups. In terms of effective dose rate, the overall arithmetic mean value of the dose conversion coefficient was found to be approximately 3.8 nSv h^{-1} Bq^{-1} m^3. This result is approximately double the value assumed by ICRP in recommending a range for the action level in dwellings of 200–600 Bq m^{-3} radon gas concentration. The higher dose conversion coefficient found here for dwellings results primarily from the association of a substantial fraction of potential alpha activity with particles intermediate in size between the classical unattached and attached progeny. Although this study presents the largest body of measured activity-weighted size distributions in homes, it does not represent a statistical sampling of homes, particularly because all of the houses are from the same geographical region and represent similar construction and housing use. Thus, it would be useful to have a systematic study of the exposure to radon decay products across a statistically valid sample of houses.

Acknowledgments

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