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Assessing the Risk from Exposure to Radon in Dwellings

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ASSESSING THE RISK FROM EXPOSURE TO RADON IN DWELLINGS

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ABSTRACT

The factors used to assess the radiation dose and health risks from human exposure to radon in dwellings are critically reviewed in this summary. Sources of indoor radon and determinants of air concentrations and exposure levels are given as well as the uncertainties that exist in their formulation.

Methods of assessing health effects from inhalation of radon and its progeny are discussed with emphasis on dosimetry of radon daughters and formulation of risk per dose values. Finally, methods of assessing risks for general population exposures to indoor radon concentrations are treated.

1. INTRODUCTION

In this chapter, we will consider the determinants of risk of lung cancer due to inhalation of the decay products of the naturally occurring radioactive gas, radon. Radon and its daughters are present in minute quantitites in all of the air that we breathe, with a typical activity of the order of 1 disintegration per minute per liter of air (0.1-1 pCi/L) for the gas and each of the short-lived daughters. The significance of this source of human population radiation exposure is indicated in Table 1, where estimates are given of the annual collective effective dose equivalent to the U. S. population from the main sources of radiation exposure. This quantity, developed by the ICRP,[1] is useful for comparing various types of exposure (e.g., whole-body, partial body, or individual organ exposures) from externally incident radiation or internally deposited radionuclides. It is defined as that whole-body dose equivalent that yields the same overall risk of cancer mortality and hereditary ill-health in the first two generations as the actual dose pattern in the body resulting from the exposure of concern. Thus, the figures given are proportional to risk, as determined using the ICRP risk estimates.[1]

The key point implied by the data in Table 1 is that, under normal conditions, radon is an important source of human radiation exposure, rivalling or even much exceeding in significance other sources that have received considerably more attention. Most of this exposure is received indoors, both because most people spend the bulk of their time there and because indoor radon levels are usually higher than the outdoors levels, a consequence of the fact that indoor-outdoor air exchange

Table 1. U.S. population radiation exposure
(10^6 person-rem per year)

Source	Effective dose equivalent
Cosmic radiation	6
Gamma radiation	6
Radionuclides in body —	
Radon Progeny (0.004 WL)	∿10
All Others	8
Fallout	<1
Medical diagnostic x-rays	10
Nuclear energy	∿0.1

rates are typically low enough so that the radon that enters the indoor air space is effectively confined. Since the degree of confinement, and thus the indoor air concentration, would be expected to increase as the air exchange rate is reduced, this strongly suggests that the now-widespread efforts to improve the efficiency of energy usage in homes and public buildings by tightening them will result in a significant increase in human exposure to radon. Many structures are being built today with air exchange rates that are a small fraction of those typical of older structures.

Another concern derives from the fact that normal radon exposure levels vary from structure to structure by more than an order-of-magnitude, due primarily to differences in radon input rates and in air exchange rates. There is the possibility that a substantial number of structures contain sufficiently high levels that the question of individual risk becomes significant. Related to this is concern about the health impacts of various human activities that increase the radon exposures of individuals and population groups, e.g., underground mining, presence of uranium mill tailings piles, and building construction on reclaimed phosphate land.

These concerns raise important issues that themselves would require a small book to discuss adequately.[2,3] It is not surprising that Federal (notably the Department of Energy, Environmental Protection Agency, and the National Bureau of Standards), state, and local government agencies, utilities, and private industry have all become involved in efforts to assess the nature and extent of these problems, and there are a number of extensive studies being conducted in other countries. The first

results of the enhanced radon research programs conducted in recent years are now becoming available,[4,7] and overall assessments have been carried out by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)[8] and the U. S. National Academy of Sciences.[2] Important new material will be published in the new UNSCEAR report,[9] and in forthcoming special issues of Health Physics[10] and Environment International.[11]

Given this wealth of available information on indoor radon, we will review very briefly only the salient features of radon and radon daughter production and migration, environmental influences on exposure, and lung deposition and dosimetry. We will discuss in somewhat more detail the basis for existing risk estimates and what this suggests concerning future research needs.

2. SOURCES OF INDOOR RADON

Radon-222 and Rn-220 are the decay daughters of Ra-226 and Ra-224, respectively, which are themselves members of the decay chains that originate with the long-lived primordial radionuclides, U-238 and Th-232, respectively. Thus, the initial production of radon is determined by the distribution of radium in the earth's upper crust and in building materials. Some fraction of the radon atoms produced escape (emanate) into the air or water spaces in the medium. They then migrate by diffusion or by air or water transport. The three main contributors to the input of radon atoms into the atmosphere of an inhabited structure are (1) exhalation of radon from the soil under the structure and subsequent diffusion through the foundation or transport through holes and

cracks (2) exhalation of radon produced in building materials from their inner surfaces, and (3) release of radon from the water supply. Radon in outdoor air also enters a structure as the air is exchanged, but this input is usually more than balanced by the loss of radon to the outdoors (since indoor air concentrations are usually higher than those outside the structure). However, any manmade source that contributes to elevated outdoor radon levels (e.g., local coal-fired or geothermal power plants, uranium mill tailings piles, mines, or phosphate fertilizer) can be regarded as indirectly affecting indoor exposures by lessening the degree to which air exchange reduces the indoor levels. In the rare cases where outdoor radon concentrations exceed the indoor, this effect is a more direct one. The only other source of radon input to a building that has been examined is natural gas usage. However, this source is usually very small in comparison with the others. Reviews of these various sources have been made by Travis et al.,[12] and Bruno.[13]

In the United States, the main contributor to the radon input into structures usually seems to be the underlying soil. Any bare soil under the structure provides a direct input, and unvented crawl spaces have been observed to be effective pathways for radon entry. Similar observations have been made with respect to cracks or openings through the foundation. In general, it may be said that radon from the soil seems to have the capability to efficiently find the path of least resistance into a structure. Finally, radon diffuses through the foundation, adding to the exhalation from the inner surfaces derived from radon production in the foundation materials, usually concrete. These phenomena provide a partial explanation for the observed higher

radon levels in basements and on ground floors as compared to higher stories (the other key factor being the normal air circulation patterns).

Building materials can be an important source of indoor radon in some circumstances, particularly when the radium content of these materials is somewhat elevated above normal values (~1 pCi/g). This situation seems to be relatively rare in the United States, but more prevalent in Europe, due partly to different construction practices and materials. A well-known example is the elevated radon levels observed in Swedish houses constructed of aerated concrete consisting of alum shales containing 20-65 pCi/g of Ra-226.[14,15] Some concern has been expressed concerning the use of radium-enriched ash from coal combustion in structural concrete. However, it appears that the effect of enhanced radon production per gram is at least partly compensated for by the low emanation probability from the ash particles.[16]

Water usage can result in significantly elevated indoor radon levels, particularly when the radon concentration of the water supply exceeds 1000 pCi/L. This is a consequence of the fact that radon is readily released into the atmosphere when the water is in a turbulent state, a condition realized in most indoor applications. Gesell and Prichard[17] have studied the quantitative relation between water usage and observed radon levels under typical conditions, and have shown that a value of the order of 1 pCi/L for the increase in indoor radon concentration may be typical for normal usage of water containing 10,000 pci/L of radon. Hess et al.[18] have arrived at a comparable figure. These conversions permit some perspective to be obtained on the fact that, while most water supplies have radon concentrations of <2,000 pCi/L,

there are areas (e.g., in Maine, USA,[18,19] and Finland[20]) where the radon content of drilled well water can exceed 100,000 pCi/L.

3. AIR CONCENTRATIONS AND EXPOSURE LEVELS

Once radon enters a structure, its future history is determined by the competition between the processes of escape to the outdoors and of radioactive decay. Let us consider a rather idealized indoor air space, where the radon input rate from all sources except outdoor air, S ($pCi/L^{-1}h^{-1}$), the radon concentrations in the indoor and outdoor air, R^i and R^o (pCi/L^{-1}), respectively, and the air exchange rate, $\lambda_v(h^{-1})$, are all constant with time. If λ_d is the radon decay constant, by the conservation of radon we have:

$$\frac{dR^i}{dt} = -\lambda_d R^i - \lambda_v R^i + S + \lambda_v R^o = 0 . \tag{1a}$$

$$R^i = \frac{S + \lambda_v R^o}{\lambda_d + \lambda_v} . \tag{1b}$$

$$\simeq R^o + \frac{S}{\lambda_v} \quad (\lambda_v >> \lambda_d = 0.011\ h^{-1}) . \tag{1c}$$

Equation (1c) holds under most circumstances, since λ_v is usually of the order of 1 h^{-1}. From this equation, several important conclusions follow, given an extreme range in values of S from 0.01 to 10 $pCi/L^{-1}\ h^{-1}$, with the bulk of the measured values falling between 0.1 and 1.[21,22] First, under normal circumstances, since $R^o \simeq 0.1\ pCi/L^{-1}$ and $\lambda_v \simeq 1\ h^{-1}$,

R^i is approximately proportional to S and inversely proportional to λ_v, particularly when S is above average. These two factors thus have a profound influence on the observed levels. Second, for typical values of R^o, S (0.5 $pCi/L^{-1}h^{-1}$) and λ_v (1 h^{-1}), R^i is about 0.6 pCi/L^{-1}, a value typical of existing measurements. For very low source terms or very high air exchange rates (e.g., open windows), R^i approaches R^o. Finally, if S is well above 1 $pCi/L^{-1}h^{-1}$ and λ_v is much less than 1 h^{-1}, then R^i can exceed 10 pCi/L^{-1}, a result again consistent with measurements in some structures. Concentrations above 100 pCi/L^{-1} would be very rare, but not impossible, requiring both unusually high rates and very low air exchange rates. These considerations provide a scale of significance for observed radon concentrations, 0.1 pCi/L^{-1} being very low, 1 being typical, 10 being high, and 100 being extremely high. This range of 3 orders-of-magnitude has special significance in considerations of individual risk, since some individuals may be subject to radiation exposures from inhaled radon daughters that are 100 times those that are representative of the whole population.

3.1 RADON DAUGHTERS

Although the radioactive decay of radon has very little influence on its air concentration, it provides the source of radioactive daughter products that, upon inhalation, contribute most of the radiation dose to lung tissue. For the n-th daughter in the decay series, equation (1a) can be modified as follows:

$$\frac{dD_n^i}{dt} = -\lambda d_{1n} D_n^i - (\lambda_v + \lambda_p) D_n^i + \lambda_{d,n} D_{n-1}^i + p\lambda_v D_n^o = 0 . \qquad (2a)$$

$$D_n^i = \frac{\lambda_{d,n} D_{n1}^i + p\lambda_v D_n^o}{\lambda_{d,n} + \lambda_v + \lambda_p} . \qquad (2b)$$

where D^i and D^o are the indoor and outdoor daughter concentrations, respectively, and D_o^i is the same as R^1 in equations (1), i.e., the "zeroth" daughter is radon itself. Equation (2a) differs from (1a) in several important respects. The term corresponding to the radon input rate, S, in equation (1a) is related to the decay of the preceding nuclide in the series within the air space. A factor, $p<1$, is applied to the final term to account for the less-than-complete transfer of the daughter nuclides in the outside air as this air penetrates into the structure (a "plateout" phenomenon). Finally, a factor, λ_p, is added to the second term to represent the loss of daughter atoms from the indoor air as they attach to room surfaces (plateout). This phenomenon has an important influence on the relative concentrations of the various nuclides in the inhaled air.

Special consideration should be given to the first daughter product of radon, polonium-218 (Ra-A), since it is a short-lived alpha emitter that itself strongly contributes to the lung dose and is also the parent of succeeding nuclides in the decay series. For this nuclide, assuming no plateout ($\lambda_p = 0$; $p = 1$) and $\lambda_v = 1\ h^{-1}$, $\lambda_{d,1} = 19.67\ h^{-1}$, and $D_1^o = 0.1\ pCi/L^{-1}$, equation (2b) becomes

$$D_1^i = 0.95\ R^i + 0.005\ . \tag{3}$$

Under these conditions, the Ra-A concentration is close to that of radon, a near-equilibrium situation that is frequently observed. The second term, already very small, is reduced even further by any loss of daughter atoms to plateout in the incoming air. Setting this term to zero, and now including any effect of plateout, equation (2b) becomes

$$D_1^i = R^i(1 + (\frac{\lambda_v + \lambda_p}{\lambda_{d,1}})^{-1} \tag{4}$$

The key point in this analysis is that, since $\lambda_{d,1}$ is large compared to the other λ's, (since $\lambda_p < 1\ h^{-1}$, see below), the Ra-A concentration in inhaled air is largely determined by R^i, and is thus influenced directly by the same factors that determine the radon concentration, notably λ_v and S.

The situation is more complicated for the decay products of Ra-A, mainly because the λ_d's for these nuclides are comparable in value to λ_v and λ_p. Using $\lambda_p = 0.1\ h^{-1}$, $\lambda_{d,2} = 2.24\ h^{-1}$, and $\lambda_{d,3} = 3.05\ h^{-1}$, the appropriate versions of equation (2b) yield values for D_2^i and D_3^i of $0.64\ R^i$ and $0.47\ R^i$, respectively. However, these results are much more sensitive to the values chosen for λ_v and λ_p than was the case of Ra-A. In particular, if $\lambda_v << 1$, the lead-214 (Ra-B) and bismuth-214 (Ra-C) concentrations can exceed $0.8\ R^i$. Thus, reducing the air exchange rate can increase the daughter concentrations by a greater factor than that of radon.

3.2 AEROSOL PROPERTIES

To this point, we have been considering primarily air concentrations of radon and its daughters. However, the magnitude of plateout and, more importantly, the probability and location of ultimate deposition of radon daughters in the lung depend critically on the physical characteristics of the particles to which the daughter atoms become attached. Although the Ra-A atoms produced by radon decay are initially mostly positively charged free atoms, they rapidly neutralize, undergo complex chemical interactions, and attach to atmospheric particulates at a rate which depends on the particulate concentration.[23] Measurements of indoor radon daughter particle size indicate a bimodal distribution, an "unattached" fraction of about 10 percent in the 5-10 nm diameter range and the "attached" fraction mostly in the 50-400 nm range.[8,24] The "unattached" fraction, mostly Ra-A, is in fact mostly attached in some manner not yet entirely understood, so that its designation as the "unattached" or "free atom" fraction is somewhat misleading. This component is significant in that it exhibits a much higher rate of deposition on room surfaces than the attached fraction, and it also appears to be a more efficient dose contributor to critical cells in the lung. The data of Knutson et al.[25] lead to the inference of deposition (plateout) rates of about $\lambda_p \simeq 4\ h^{-1}$ for unattached daughters and $\lambda_p \simeq 0.05\ h^{-1}$ for attached daughters in typical rooms. It follows from these considerations that environmental conditions (e.g., a decline in atmospheric particulate concentration or the operation of an electrostatic precipitator) that simultaneously would tend to lower total radon daughter concentration (by increased plateout) and to increase

the unattached fraction might tend to increase the risk from exposure to these daughters. The complexities of these various processes have been described in detail.[23,25-28]

From this discussion, it is evident that indoor radon daughter concentrations and particle-size distributions in inhaled air are determined primarily by the radon input rate, the indoor-outdoor air exchange rate, and the airborne particulate concentration. Mechanical ventilation can reduce daughter and particulate concentrations by filtration and plateout mechanisms, and also reduce radon concentrations by effectively increasing the air exchange rate through leakage. Environmental parameters such as outdoor wind speed and direction, indoor-outdoor temperature and pressure differences, and soil water content influence the air exchange rate and/or radon input rate. These factors thus indirectly affect radon exposure. Human activity patterns also strongly influence the exposure, e.g., smoking, opening and closing doors and windows, using fans. It is thus not surprising that radon and daughter concentrations within a particular structure exhibit large time variations, typically by factors of 2 to 10. This severely complicates the practical determination of long-term exposures in a particular structure. Such exposure determinations require either a substantial number of instantaneous "grab" samples, appropriately distributed over time-of-day (i.e., activity patterns) and the seasons of the year, or a lesser number of integral measurements of at least a few days duration also distributed over the seasons. Useful discussions of measurement techniques and methodologies are given in references 29-31.

3.3 EXPOSURE UNITS

We have dealt thus far with activity concentrations of the various radionuclides in the indoor air space. However, most of the respiratory tract dose from the inhaled daughters is produced by alpha particles from the decay of Ra-A and polonium-214 (Ra-C'). For each atom of Ra-A deposited in the lung, two alpha particles will be emitted as the various short-lived daughters decay. For each atom of Ra-B or Ra-C deposited, only one such alpha particle will appear. If the activity concentrations of these daughters are weighted by the potential alpha energies to be released per unit activity, then one has a new quantity more closely related to the ultimate lung dose. In investigations of radon exposure in uranium mines, the U. S. Public Health Service developed such a quantity, whose unit is called the working level (WL). This is defined as exposure to an atmosphere that contains any combination of radon daughters such that the total alpha particle emission in a liter of air is 1.3×10^5 MeV in the complete decay through Ra-C'. This particular value corresponds to the potential alpha energy associated with daughter concentrations of 100 pCi/L^{-1} (or, alternatively, with daughters in equilibrium with 100 pCi/L^{-1} of radon). The conversion between the activity concentrations of the daughters, D_n^i, in pCi/L^{-1} and the exposure, W, in working levels is as follows:

$$W = 0.00105\ D_1^i + 0.00516\ D_2^i + 0.00379\ D_3^i\ . \qquad (5)$$

The working level month (WLM) is the unit of cumulative exposure and is defined as exposure to 1 WL for 170 hours (i.e., an occupational month).

In the environmental situation, exposure to 1 WL for a year (8760 h) would correspond to an integrated exposure of 51 WLM.*

This concept of exposure is frequently utilized in health effects assessments. Using previously derived "typical" activity concentrations of 0.60 pCi/L^{-1} for Rn-222, 0.57 pCi/L^{-1} for Ra-A, 0.38 pCi/L^{-1} for Ra-B, and 0.28 pCi/L^{-1} for Ra-C, we infer an exposure of 0.005 WL, a value which is consistent with direct exposure measurements. From the earlier discussion of radon concentrations, exposures near 0.05 WL would be considered high and 0.5 WL very rare and extremely high. Outdoor exposures are generally near 0.001 WL. A representative annual environmental exposure, assuming 80% of the time spent indoors, would be about 0.15 WLM.

An equilibrium factor, F, for the radon daughters is sometimes used, particularly when exposure is estimated from measurements of radon concentrations. This is defined as the ratio of W in equation (5) to the exposure that would pertain if all of the daughters were in equilibrium with the radon, i.e., have the same activity concentration. In the above case, the equilibrium factor would be 0.51. Most measurements in houses yield values for this factor between 0.2 and 0.7, and an average value of 0.5 is often adopted. This would lead to an interpretation of a radon concentration of 1 pCi/L^{-1} as corresponding to an exposure of 0.005 WL. However, substantial deviations from this average

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*Some authors correct this conversion for such factors as differences in breathing rates and volumes between the occupational and environmental situations. Such modifications are not consistent with the definition of "exposure" in working levels.

value for the equilibrium factor can occur, depending primarily on airborne particulate concentrations (i.e., attachment rates) and air circulation patterns and exchange rates.[27,32]

3.4 EXPOSURE MEASUREMENTS

Thus far, the discussion has been restricted to Rn-222 and its daughters. However, very limited data on Rn-220 (thoron) and its daughters in houses indicate that their contribution to lung dose is often not negligible. Measurements of thoron-daughter to radon-daughter working level ratios in 22 Norwegian dwellings by Stranden[33] indicate a range of 0.1 to 0.8, with a mean of 0.5. A few unpublished Environmental Measurements Laboratory (ELM) data[34] in the U.S. give values near 0.1. These data are sufficient to strongly suggest the need for further study of indoor thoron daughter exposure levels and the factors that influence them, particularly when above-average Th-232 concentrations in building materials may be present.

Much of the worldwide data obtained on indoor radon and daughter concentrations is summarized in the UNSCEAR reports.[8,9] A comprehensive review of reported radon and radon progeny exposure conditions in houses and other buildings by Goldsmith et al.[35] has been summarized by Ryan.[36] Measurements were included for buildings on soils considered to contain typical background Ra-226 concentrations (<5 pCi/g and an average of ~1 pCi/g). The measured radon and radon daughter concentrations each appear to be lognormally distributed. The geometric mean radon concentration on main floors was 2.4 pCi/L with a geometric standard deviation of 4.24 (296 measurements). In basements, the geometric mean radon

concentration was 6.46 pCi/L with a geometric standard deviation of 3.69 (296 measurements). Working level concentrations of radon progeny have also been measured and compiled by Goldsmith et al.[35] As summarized by Ryan,[36] the average concentration of radon progeny on main floors was 0.0066 WL with a geometric standard deviation of 3.45 (403 measurements). The mean concentration in basements was 0.0127 WL with a geometric standard deviation of 3.41 (298 measurements). These results illustrate the main phenomena, but there may be inherent biases in this data base due to insufficient sampling periods and the choice of interesting areas of study. A large-scale, carefully planned, survey that would yield a reliable distribution of long-term radon exposure levels in U. S. housing is still lacking. Such surveys are presently being conducted in Sweden and West Germany.

These wide ranges in background concentrations of radon and radon progeny in typical structures indicate the need for measurements in particular situations to determine the degree to which exposures may be elevated above background. It is clear that indoor radon concentrations can often be a factor of 10 or more higher than outdoor concentrations. However, it is difficult to establish whether exposures are atypical for a particular situation such as houses built on reclaimed mining land. For example, the average radon progeny concentrations in structures built on reclaimed lands (including reclaimed phosphate and uranium mining lands) was 0.0124 WL,[36] which is about the same as levels found in basements of typical buildings. It is clear that background exposures to radon and radon progeny need to be better defined for

various population groups in order to provide a basis for assessing the risk associated with a particular action that may increase exposures.

4. HEALTH EFFECTS ASSESSMENT

The output of a health effects assessment is ideally the specification of an exposure-response or dose-response relationship for the toxic materials of concern. The relationships between exposure and dose (dose conversion factors) are central to risk assessments because dose to critical tissues or cells (in which the biological effects arise) can vary widely for a given exposure. Dose conversion factors for radon daughters have been at variance because of differences in assumptions and uncertainties related to aerosol properties, lung models, and critical tissue.[37-42] Such variance has led in some instances to abandonment of dose calculations and reliance on relative exposure estimates and associated epidemiological data in order to suggest guidance for radon daughter exposures. Guidance for general population groups have been based upon risk per WLM derived from data for underground miners, with recognition of the fact that dose per WLM to the general population could differ from that of miners.

The data on lung cancer induction by radon daughters arises from epidemiological studies on underground miners, particularly uranium miners. We will provide a brief discussion of risk estimates from the uranium miner experience, dosimetry for radon daughters, and the uncertainties involved in extrapolation to the general population.

4.1 URANIUM MINING EXPERIENCE

Uranium miners are exposed to a complex atmosphere. Uranium ore dust, silica dust, diesel exhaust fumes, natural aerosols, radon, and radon daughters are present in most mine atmospheres. Although possible effects from one or all of these components in combination are not ruled out, an association between incidence of lung cancer and cumulative exposure to radon daughters has been established. Cumulative exposure to radon daughters may be serving as a surrogate for the complex mixture, and it is possible that the nature of exposure response relationships will vary with the nature of the complex mixture. Thus, it is difficult to extrapolate the results from mining groups to the general population where different complex mixtures are prevalent and different spatial, temporal, age, and sensitivity distributions characterize the exposed population.

Epidemiological data on the induction of lung cancer have been used by Walsh[43] to derive dose conversion factors for radon daughters. After discussing several possible ways of expressing the risk of lung cancer, excess relative risk was selected as an appropriate quantifier. The results for ten different population groups exhibiting radiogenic lung cancer are given in Table 2. The percent increase in excess relative risk per WLM for uranium mining groups varied from 0.4-1.2%/WLM. For all mining groups, the range was 0.4-3.2%/WLM. If radon daughters do not account for all the lung cancer induction, then these risk estimates would overestimate the carcinogenic potency of radon daughters. The higher estimates for nonuranium mining groups where the cumulative exposures to radon daughters were lower and other agents may have been

Table 2. Excess relative risk (in percent) of radiogenic lung cancer in groups exposed to alpha particles, X-rays, gamma-rays, and neutrons[a]

Exposed group	Type of radiation	Percent increase in excess relative risk		
		per WLM	per rad	per rem
Uranium miners				
U.S. (white)	Alpha	0.9		
U.S. (Indian)[b]				
WLM>300	Alpha	0.4		
U.S. (15 years after start), WLM<500[b]	Alpha	0.9		
Canada[c]	Alpha	0.9		
Czechoslovakian[d]	Alpha	1.2		
Fluorspar miners	Alpha	3.0		
Metal miners	Alpha	0.5		
Swedish metal miners	Alpha	3.2		
Thorotrast (Portuguese)	Alpha		1.0	
Atomic bomb survivors	Gamma		0.3	0.3
	Neutron		1.0	0.2
Spondylitics	X-rays		0.2	0.2

[a] Data from BEIR report (56) unless indicated otherwise.

[b] Data from Archer et al. (57).

[c] From data tabulated by Archer, personal communication.

[d] From Sevc et al. (58).

responsible for a greater proportion of lung cancer would indicate that radon daughters become a poor surrogate for total exposure as exposure decreases.

Using the single estimate for thorotrast patients given in Table 2, an epidemiological-based dose conversion factor for radon daughters was calculated[43] to be about 1.4 rad/WLM using the average value of 1.4%/WLM for all mining groups. The range for uranium miners referenced to the thorotrast estimate would be 0.4-1.2 rads/WLM. A dose conversion factor of 6 rem/WLM and an average rem/rad factor of 4 was also derived by using the gamma- and X-ray data in Table 2 as a reference. The dosimetric meaning of these rad and rem values are discussed below.

Evans et al.[44] have recently summarized estimates of risk from environmental exposure to Rn-222 and its decay products. Estimates of lifetime risk from the miner data range from an estimate of 21-54 deaths from lung cancer per million WLM to 1000. Jacobi (cited in Evans et al.[44]) and UNSCEAR[8] propose a range of 100-500 deaths per million WLM. The estimate by Walsh of 0.4-1% increase in excess relative risk per WLM can be converted to lifetime risks in the same range as the UNSCEAR estimates. Extrapolation of such results for miners to the general population is discussed below.

4.2 DOSIMETRY FOR RADON DAUGHTERS

Estimated rad/WLM and rem/WLM conversion factors are important in order to relate the information on uranium miners (exposure in WLM) to the total body of information on radiogenic lung cancer (risk expressed per rad or per rem) and to identify those parameters (and uncertainties)

which are important in extrapolating the results for uranium miners to low exposure rates and to general population groups.

The absorbed dose (rad) is simply a physical energy adsorption (100 ergs/gm) and does not necessarily relate to any biological response. According to the International Commission on Radiological Protection (ICRP),[45] the absorbed dose is to be multiplied by appropriate conversion factors to obtain the dose equivalent. Thus, the dose equivalent (DE) is

$$(DE) = D(QF)\ (DF)\ (OMFs)\ . \qquad (6)$$

where D is the dose in rads and OMFs are other modifying factors such as spatial distribution of dose (DF) or relative biological sensitivity (RBS). The unit of DE is the rem. The DE relates to a given degree of biological response and was developed to enable comparisons of biological effect to be made on a common scale, regardless of the type of ionizing radiation involved. Therefore, the dose in rems will be the same for any type of ionizing radiation producing that degree of a particular biological response. The physical rad doses and conversion factors can and will differ for different types of radiation but when multiplied together, all will converge to the same rem dose. The practical implications of the definition of DE for radon daughter dosimetry have been discussed by Walsh.[43]

Most discussions of the absorbed dose to the respiratory tract have related to the rad per WLM conversion factor and have emphasized lack of sufficient information to estimate the rem per WLM conversion

factor. Specifically, quality factor (QF) and other modifying factors (OMFs) are not quantitative estimators of lung cancer induction in humans by alpha emitters. If these factors were known, then rad per WLM conversion factors could simply be multiplied by them to obtain rem per WLM conversion factors. Having obtained rem per WLM conversion factors, it would make no difference how rad per WLM conversion factors are determined since they would be multiplied by different QFs and OMFs in each case to obtain the rem per WLM conversion factor. This conclusion follows from the definition of DE given in equation (6). Dose calculations have been directed to a determination of dose (rads/WLM) to the critical cells (the cells which become neoplastic). If such calculations could be accomplished accurately, then there would be fewer factors (e.g., distribution factor, DF) involved in determining rem dose. Such calculations are important and necessary for development of mechanistic dose response models. Given the appropriate factors (OF, DF, OMFs), the resulting rem dose should be the same, regardless of the method of physical rad dose calculation.

Given a WLM exposure, calculation of a corresponding rad dose can be made if enough is known about aerosol characteristics, deposition models, clearance of deposited material from the lung, critical tissue or cells and depth-dose curves for the alpha particles. Such dose calculations have been discussed extensively.[6-11,37-50] Formulation of the methods for dose calculation and an expression for the dose per WLM were given by Walsh.[47] Much of the variation in dose per WLM calculations have been due to assumptions regarding aerosol characteristics, lung morphometry and physiology, and the portion of the lung (e.g.,

a particular generation in the tracheobronchial tree versus the whole lung) for which doses were calculated. Some of the more important factors will be discussed further in connection with extrapolation of results for miners to the general population.

A review[47] concluded that a detailed site-by-site (e.g., an area as small as a bifurcation in the tracheobronchitial tree) dose calculation was not possible, and such calculations are still not feasible with any degree of certainty. The average dose to each region (Weibel model, 17 generations) of the tracheobronchial tree was calculated,[47] and showed that the highest doses to particular regions were not much higher than the average dose to the entire tracheobronchial tree. The average dose to the tracheobronchial tree was about 1.4 rads/WLM and the dose to the basal cells of the bronchial epithelium (thought to be the critical cells) located at variable depths below the surface of the bronchial epithelium was estimated to be less than 1 rad/WLM. Later calculations by Harley[52] and by Jacobi[53] have tended to confirm that dose to basal cells is less than 1 rad/WLM. These calculations are in surprising good accord with the dose conversion factors based on the epidemiological data given above.

A calculated dose of less than 1 rad/WLM (say 0.5 rad/WLM), along with the data in Table 2, would indicate that the factor for rem per rad is about 12. Since the risk per rem must be approximately invariant by definition, such results indicate that the basal cells are more sensitive than the entire bronchial epithelium on a rad dose basis, as would be expected. Higher rad/WLM (e.g., 5 rad/WLM) would lead to rem per rad factors less than unity (1); thus, the basal cells would appear

to be less sensitive than the entire bronchial epithelium. Such a result would clearly not be in accord with rem per rad factors for alpha particles. Walsh[43] also showed that the ICRP models can provide an adequate basis for radon progeny dose calculations; and he also concluded that animal toxicological studies tend to support a rem/rad factor less than 10, the value generally used at that time for alpha particles.

4.3 EXTRAPOLATION OF MINING EXPERIENCE TO THE GENERAL POPULATION

The general population is exposed to much lower levels of radon progeny than were the uranium miners. Uranium miners were also exposed to other materials including cigarette smoke that could have influenced lung cancer induction. Other differences relate to work state (e.g., breathing rate), nature of aerosol distribution, population characteristics such as age and sex and relative lung physiology. Thus, extrapolation of the results for uranium miners to the general population is complex and highly uncertain. Only the general features are discussed here.

The influence of potential cocarcinogens, cofactors, or promoters on the induction of lung cancer in uranium miners probably contributes the greatest uncertainty in extrapolations to low level exposures. If these factors are absent in cases of exposure of the general population, then risk estimates based on U miner data will almost certainly overestimate impact on the general population. If these factors were to make a constant contribution over all radon progeny exposure categories, then their relative contribution would increase as exposure to radon

progeny decreased. For example, if risk of lung cancer were doubled (100% increase) at a 100-WLM exposure and 10% of the increase was due to other exposures (chemicals, dusts, cigarettes, promoters, etc.), then at 1 WLM the total increase would be about 11% but only about 1% could be attributed to radon progeny. The risk per WLM for radon daughters would be overestimated by an order of magnitude if these cofactors are not present in another exposure situation. If there is interaction between radiation and these other exposures, then the overestimate would be even greater. If exposures to other materials decline in proportion to radon daughter exposures, there would still be an overestimate, but of lesser magnitude.

Although a linear dose-response relationship for high linear energy transfer radiation (LET) such as alpha radiation is generally assumed,[54] there is no way to confirm such an assumption unless the effects of other potential contributors can be separated out. Stewart and Simpson and Myers and Stewart as cited in Evans et al.[44] have evaluated the American and Czechoslovakian data using various statistical techniques. Their work indicates, according to Evans et al.,[44] that the incidence of lung cancer can be accounted for by a linear relationship with exposure, allowing a constant factor for non-radiogenic lung cancers. They also found that estimates of risk for low-level exposure may include zero as a lower bound. Evans et al.[44] judge from the available epidemiological evidence that an upper bound for the lifetime risk to the general population is about 10^{-4} per WLM.

An approximate relationship for the dose per WLM was given by Walsh.[47,51] The equation provides a basis for summarizing other factors that are important in extrapolation of results from one group to another. The approximate dose per WLM to a region (R) of the respiratory tract is (see Ref. 47 or 50 for a detailed derivation)

$$D_R \simeq \frac{0.354\ f_R\ \nu\ S_2 f_{2C}}{A_R E_2} \quad \text{in rads/WLM .} \tag{7}$$

where

f_R = the fraction deposited in the region (R),

ν = rate of breathing in L/h^{-1},

S_2 = mean stopping power of the Po-214 alpha in tissue at the depth of penetration of interest in $MeV \cdot cm^2/g$,

f_{2C} = the fraction of the Po-214 alphas which reach the depth of penetration of interest,

A_R = the surface area of the region in cm^2, and

E_2 = 7.68 MeV is the Po-214 alpha energy.

The main factors that may differ between uranium miners and the general adult population in terms of the physical dose conversion factor are f_R and ν. The fraction deposited in the respiratory tract, f_R, for the general population may be higher because the particle size distribution may be different than was the case for miners. The breathing rate for the general population will, however, be lower, perhaps by a factor of 2 or more because of a lower level work state.

The factor, f_R, is influenced strongly by the aerosol characteristics. Although radon progeny will attach to a distribution of particle sizes, dose calculations have emphasized a single particle size for the so called "attached" fraction and a different smaller particle size and much higher diffusion coefficient for the "unattached" fraction (the major mode of deposition for radon progeny is by diffusion). The importance of the unattached fraction or "free ion" component of the exposure atmosphere is that the presence or absence of free ions can profoundly affect site and magnitude of deposition in the respiratory tract. The free ions will deposit with virtually 100% efficiency due to their large diffusion coefficient. Raabe[55] has described a method for calculating the unattached fractions of Po-218, Pb-214, and Bi-214 if the aerosol size distribution and particle number concentration are known. When particle number concentrations are $<10^4$ per cc, the fraction of the total potential alpha energy unattached (fraction of WL unattached) can make a significant contribution to the respiratory tract dose. The influence of these factors especially with regard to the differences between mining and nonmining populations need to be investigated further. As a general rule, the particle concentration will be $>10^4$ per cc for reasonable levels of human activity, and the increase in dose per WLM due to greater unattached fractions for nonmining groups will be less than 50%.[49,53]

For children (<12 yrs), deposition in the respiratory tract will also be different due to respiratory tract physiology and morphometry. Although direct data are not available for children on airway dimensions and clearance, scaling down from adult lung dimensions would indicate

that the dose per WLM to children[49,59,60] may be significantly higher than for miners. However, it is not clear whether deposition patterns are bronchial epithelium thicknesses are substantially different in children as compared to adults. This is another area for further research.

4.4 RANGE OF RISK FOR THE GENERAL POPULATION

From the previous discussion, the percent increase in excess relative risk for uranium miners analyzed over several higher exposure categories is about 0.4-1%/WLM. The largest and at present unquantifiable source of uncertainty is associated with the risk/WLM value (%/WLM) and subsequently with the rem/WLM value. The magnitude of the contribution of nonradiogenic carcinogens is not known. Uranium ore dust, diesel engine exhaust, arsenic, nickel, and cigarette smoking are all likely contributors to the total risk of lung cancer. The estimates for uranium miners are likely to be overestimates for the general population. Unfortunately, the degree of conservatism is impossible to estimate. The judgment by Evans et al.[44] of an upper bound lifetime risk for the general population of 10^{-4}/WLM is reasonable but cannot be completely confirmed. A start towards resolution of the problem may be made by application of more rigorous statistical techniques that would test reasonable hypotheses about the relative contribution of the various contributors to risk. A major uncertainty will be exposure estimates for the various possible contributors, including radon progeny. Some better estimates of these exposures might be made on the basis of measurements in experimental mines designed to mimic past exposures to the extent possible.

The 10^{-4}/WLM level of risk would correspond to about 2×10^{-5}/rem which is in accord with ICRP[1] estimates of risk to the lung from external ionizing radiation and is not inconsistent with our analysis of the risk to uranium miners of 0.4-1%/WLM as the percent increase in excess relative risk. The 0.4-1%/WLM range would correspond to a lifetime risk for the miners of about $0.8\text{-}2 \times 10^{-4}$/WLM.

5. RISK ASSESSMENT FOR INDOOR RADON DAUGHTERS

The indoor radon progeny exposure estimates and the risk estimates summarized in Sect. 3.4 may be combined to provide a risk assessment for general population exposures to radon progeny. Working level concentrations on the main floor of buildings averaged about 0.007 WL with a geometric standard deviation of 3.45. The value used by Evans et al.[44] was 0.004 WL but is based on fewer measurements. Under conditions of continuous exposure, the cumulative exposure for a 0.007-WL concentration would be about 0.35 WLM per year or about 25 WLM in 70 years. If, as is commonly reported, about 80-90% of the day is spent indoors, then lifetime exposures would amount to about 20-22 WLM. Outdoor exposure levels would be a factor of 20 or more lower and do not make a significant contribution to total exposure. However, spending more time outdoors or increasing indoor ventilation could significantly lower exposures.

Risk estimates based upon the uranium miner experience are about 0.4-1%/WLM as the percent increase in excess relative risk. Thus, lifetime exposure at mean indoor radon progeny levels may represent an

8-20% increase in risk of lung cancer. This would probably represent a maximum increase for the general population. The numbers take no account of latency period or exposure to nonradiogenic carcinogens as discussed previously.

Exposure estimates for uranium miners did not include background exposures. Background exposures become important for the groups exposed to the lowest levels above background. Thus the risk per WLM becomes more complicated for the lowest exposure categories. For exposure categories at or below 20 WLM, the risk/WLM could be overestimated by at least a factor of 2. For example, the risk/WLM would have been calculated as risk per 20 WLM plus background (up to 20 WLM or more in older miners). This problem with the lowest exposure categories, as well as large uncertainties in exposure estimates for individual cases, the small number of cases involved, and the role of cofactors, requires further study. In the interim, an upper limit of 10% increase in risk due to lifetime exposure to mean indoor radon progeny concentrations may be appropriate when radon progeny exposures are used as a surrogate for the total exposure complex.

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