

Radon Exposure Assessment and Dosimetry Applied to Epidemiology and Risk Estimation

Jerome S. Puskin^{a,1} and Anthony C. James^b

^a Office of Radiation and Indoor Air, U.S. Environmental Protection Agency, Washington, DC 20460; and

^b U.S. Transuranium and Uranium Registries, Washington State University, Richland, Washington 95354

Puskin, J. S. and James, A. C. Radon Exposure Assessment and Dosimetry Applied to Epidemiology and Risk Estimation. *Radiat. Res.* 166, 193–208 (2006).

Epidemiological studies of underground miners provide the primary basis for radon risk estimates for indoor exposures as well as mine exposures. A major source of uncertainty in these risk estimates is the uncertainty in radon progeny exposure estimates for the miners. Often the exposure information is very incomplete, and exposure estimation must rely on interpolations, extrapolations and reconstruction of mining conditions decades before, which might differ markedly from those in more recent times. Many of the measurements that were carried out—commonly for health protection purposes—are not likely to be representative of actual exposures. Early monitoring was often of radon gas rather than of the progeny, so that quantifying exposure requires an estimate of the equilibrium fraction under the conditions existing at the time of the reported measurement. In addition to the uncertainty in radon progeny exposure, doses from γ radiation, inhaled radioactive dust, and thoron progeny have historically been neglected. These may induce a systematic bias in risk estimates and add to the overall uncertainty in risk estimates derived from the miner studies. Unlike other radiogenic cancer risk estimates, numerical risk estimates derived for radon from epidemiology are usually expressed as a risk per unit exposure rather than as a risk per unit dose to a target tissue. Nevertheless, dosimetric considerations are important when trying to compare risks under different exposure conditions, e.g. in mines and homes. A recent comparative assessment of exposure conditions indicates that, for equal radon progeny exposures, the dose in homes is about the same as in mines. Thus, neglecting other possible differences, such as the presence in mines of other potential airborne carcinogens, the risk per unit progeny exposure should be about the same for indoor exposures as observed in miners. Results of case-control studies of lung cancer incidence in homes monitored for radon are reasonably consistent with what would be projected from miner studies. Measurements of exposure in these indoor case-control studies rely on different types of detectors than those used in mines, and the estimates of exposure are again a major source of uncertainty in these studies.

© 2006 by Radiation Research Society

OVERVIEW

Radon (^{222}Rn) is a well-established lung carcinogen that occurs at elevated levels in some underground mines, and epidemiological studies of miners have been used to quantify the strength of the association between radon exposure and lung cancer. The carcinogenic mechanism involves the inhalation and deposition of short-lived radon decay products, referred to as radon *progeny*, which emit α particles that irradiate sensitive cells in the bronchial epithelium. (See Fig. 1 for a diagram of the radon decay chain.)

Epidemiological data on numerous underground miner cohorts clearly show an increasing incidence of lung cancer with increasing radon exposure. In 1999, using methodology set forth in an earlier report from the National Cancer Institute (1), the National Research Council (NRC) issued its BEIR VI report, in which a combined analysis of data on 11 miner cohorts was used to develop mathematical models for estimating the risk over a wide range of exposure conditions (2). These models can be used not only to assess the risk to miners, but also to project the risk from indoor exposures, where the exposure conditions differ markedly from those in mines.

Unlike risk estimates for other radiation-induced cancers, the numerical risk coefficients derived for radon from epidemiology are usually expressed simply as a *risk per unit exposure* rather than as a *risk per unit absorbed tissue dose*. Dose is clearly a better measure of the biological damage leading to cancer than exposure, and expressing the risk as a dose-response function facilitates intercomparisons between results of studies involving different exposure conditions (e.g. whole body and partial body, internal and external). Furthermore, when expressed in dosimetric units, risk coefficients can be readily used to project risk for other situations in which the dose to the same tissue can be estimated; e.g., epidemiological data on bone cancer induction by ingested ^{226}Ra might serve as a basis for estimating the bone cancer risk for intake of other bone-seeking α -

¹ Address for correspondence: Office of Radiation and Indoor Air, U.S. Environmental Protection Agency, Washington, DC 20460; e-mail: puskin.jerome@epa.gov.

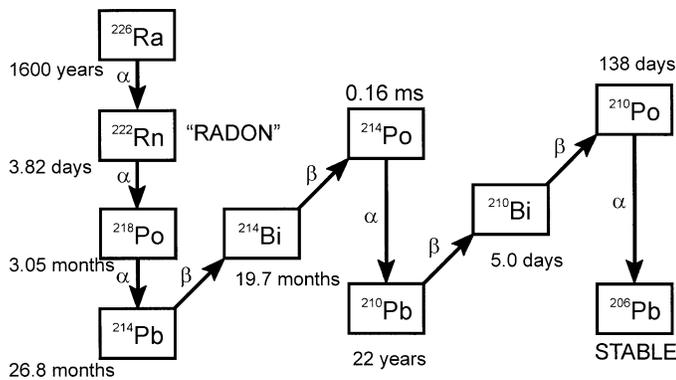


FIG. 1. Radium-226 decay scheme, showing radon-222 (gas) and its radioactive progeny through stable lead-206.

particle emitters for which no direct epidemiological data are available.

Methodology has been developed to calculate the dose to potential target cells in the tracheobronchial tree and lungs due to a defined radon progeny exposure [see review by James (3)]. However, the usefulness of these calculations for the direct estimation of risk, as is done by the International Commission on Radiological Protection (ICRP), for all other inhaled α particles is somewhat limited (4). The dose distribution associated with inhaled radon progeny is highly non-uniform within the respiratory tract, being concentrated in the bronchial and bronchiolar airways, whereas the dose distribution resulting from inhaled long-lived α -particle emitters, such as plutonium, is concentrated in the alveolar region. Hence the risk per unit dose from radon progeny, plutonium or indeed uniform irradiation of the lungs (as occurred in the Japanese atomic bomb survivors) is not directly comparable (5).

A consideration of lung dosimetry is nevertheless invaluable when trying to project estimated risks derived from the miner studies to other radon exposure conditions. In particular, although observed correlations between lung cancer and radon exposure in homes have provided strong confirmatory evidence of a radon-related risk (2, 6), projections from the miner studies still serve as the primary basis for estimating the risk from indoor exposures. Dosimetric comparisons between mine and residential exposure conditions have been used to improve the accuracy and confidence in such projections. In addition, a careful examination of the variability and uncertainties in the dosimetry is a crucial element in a proper characterization of the risk estimates for residential exposures.

Here we discuss many of the problems and uncertainties associated with the estimation of radon exposures in miner cohort studies and in residential case-control studies. In addition, we examine the relationship between radon exposure and radiation doses to the lung, and its use in extrapolating risk estimates derived from the miner cohorts to other populations.

EXPOSURE UNITS AND MEASUREMENTS

In the mine environment, it is customary (7, 8) to measure radon progeny concentrations in terms of the potential α -particle energy concentration (PAEC), which reflects the amount of α -particle energy ultimately released by the short-lived progeny in a liter of air. The PAEC is commonly expressed in terms of the "working level" (WL), where 1 WL is any combination of short-lived progeny in 1 liter of air that will result in the emission 1.3×10^5 MeV of potential α -particle energy (2.08×10^{-5} J m^{-3}). The WL unit is defined in such a way that 1 WL corresponds to 3.7 Bq/liter of radon gas in secular equilibrium with its short-lived progeny (i.e., where each of the progeny is also at a concentration of 3.7 Bq/liter).

From the decay rates and α -particle energies, it can be shown that

$$\text{PAEC (WL)} = 0.00106A_1 + 0.00513A_2 + 0.00381A_3;$$

$$\text{PAEC (J m}^{-3}\text{)} = (5.80C_1 + 28.5C_2 + 21.0C_3) \times 10^{-10};$$

where A_1 , A_2 and A_3 (in Bq/liter) and C_1 , C_2 and C_3 (in Bq/ m^3) are the respective concentrations of ^{218}Po , ^{214}Pb and ^{214}Bi .

The equilibrium fraction (F) is defined to be the ratio of the actual radon progeny PAEC to that which would exist if the short-lived progeny were in secular equilibrium with the radon gas. Thus, for the PAEC in WL and the activity concentration of radon gas (A_0) in Bq/liter, we have

$$F = 100 \text{ PAEC}/A_0.$$

Exposure is usually given in terms of the working level month (WLM), with 1 WLM being equivalent to an exposure at 1 WL for 170 h, a typical number of hours worked by miners per month. Alternatively, the exposure may be expressed in SI units as J h m^{-3} .

The airborne activity concentrations of all three short-lived radon progeny were first measured by the Tsivoglou method (9, 10) by making three separate counts, during a 30-min period, of the α -particle activity remaining on an air-sample filter. However, because of the difficulty in performing multiple α -particle counts under mine conditions, the Tsivoglou method was soon supplanted by the Kuznetz method, which provides a simpler and more direct measure of potential α -particle energy per unit volume of air (10, 11). The procedure involves drawing a known volume of air through a filter, and then a waiting period (most commonly 40 min) followed by a count of the remaining (^{214}Po) α -particle activity on the filter. This method is rather insensitive to the relative concentrations of the individual progeny and yields measurements of WL accurate to within 8% or better over a wide range of mine ventilation and other conditions (10). The Kuznetz method also had the great advantage (for its time) of "robustness". The air sample filters did not have to be α -particle-counted underground, so the counting equipment could be kept in the less hostile environment above ground.

An improvement to the Kusnetz method was developed by Rolle (12). This method makes use of the same equipment as the Kusnetz method but readjusts the waiting and counting periods to gain improved accuracy. Taking into account all sources of error, the method can reduce uncertainties to about $\pm 20\%$. This and other methods for measuring radon progeny PAEC in mine air were discussed in an NEA report (13).

For practical reasons, epidemiological studies of lung cancer associated with radon exposure in homes have generally relied on α -particle track detectors (ATDs) to measure the time-integrated radon gas concentration. The ATD consists of a plastic chip enclosed in a chamber, which allows diffusion of radon gas to the interior but effectively filters out radon progeny and thoron gas (14). At equilibrium, the radon gas concentration in the chamber will be the same as that in the room outside. Once radon gas atoms reach the interior of the chamber, they and the resulting progeny emit α particles that penetrate the surface of the plastic, creating permanent damage (tracks) along their paths. These tracks can then be visualized by etching the plastic with a strong NaOH solution and subsequently counted under a microscope. Such devices are thus often referred to as "track-etch detectors".

Early attempts to measure the PAEC itself in the home environment, using a bare ATD with no diffusional barrier, were largely unsuccessful. This was because the plate-out efficiency of the airborne radon progeny onto the detector surface was highly variable, due to such factors as variable air currents and the buildup of electrostatic charge on the plastic (15).

More recently, the buildup of ^{210}Po α -particle activity resulting from accumulation of the long-lived progeny of ^{210}Pb by α -particle recoil of ^{214}Po atoms into fixed surfaces, such as window glass, has been used as a measure of cumulative past exposure in homes to airborne potential α -particle energy (16). This methodology has also been applied to epidemiological studies, using an ATD to measure the surface density of retained ^{210}Po activity (17).

For the mine environment, ATD technology has also been used to develop a portable personal dosimeter for continuous monitoring of the radon progeny exposure of individual miners (18). In this device, air is pumped continuously through a filter, which traps airborne radioactive particles including radon and thoron progeny. The α particles emitted by the deposited material are then detected by visualizing their tracks in a plastic track-etch detector. Measurements with this method have been used to investigate the reliability of radon progeny exposure estimates used in epidemiological studies of miners, which are usually based on grab samples of radon gas or radon progeny in different locations in the mine coupled with whatever information exists regarding the location(s) of the miner during his work underground (18–20).

EXPOSURE ESTIMATES FOR MINER COHORTS

For the purposes of epidemiology and risk modeling, one would ideally have a detailed time profile of the WL exposure for each miner during his entire work history. This is almost never the case, and miner exposure estimates must be reconstructed from what measurements do exist, based on a series of interpolations, extrapolations and even educated guesses. Table 1 summarizes the methods used to estimate exposures for each of the 11 miner cohorts analyzed in the NRC BEIR VI Report (2).

Measurements of radon gas and radon progeny were usually carried out for the purpose of compliance with occupational health standards. Almost entirely, the compliance monitoring involved grab sample measurements of radon gas or progeny levels in certain mine locations rather than continuous monitoring. The frequency of these measurements increased over time.

Radon/radon progeny levels within a mine may vary greatly, both spatially and temporally. Thus exposures must be assigned by linking each miner's work history with the estimated exposure rate at each location and time in the mines where he worked. Depending on when and where he worked, the exposure information may be quite limited.

Measurements of radon gas and radon progeny were often conducted infrequently and only at certain locations within a mine. For example, if the measurements were being carried out for the purpose of health protection, it was common to measure at locations thought to have the highest radon levels to ensure that miners were not being overly exposed. In addition, incomplete data on work histories may also lead to biases, either downward through a failure to account for some mining activity or upward because a miner was assumed to have worked full time in a mine when actually he had only worked part time. The latter may have been common for some of the smaller mines (21). In addition, measurements may sometimes have been biased downward by mine personnel so as to allow longer exposures.

Historically, radon levels in mines were highest in the earliest years, but information on these exposures is most sketchy. During the period before about 1950, before the hazard was fully recognized, there was generally no artificial ventilation or monitoring of radon and its progeny. In most of the mines of interest, forced ventilation was introduced in the 1950s and early 1960s, along with measurements of radon gas and then radon progeny. Continuing improvements in ventilation led to a sharp drop in radon levels during this period. From the mid-1960s onward, radon levels continued to fall, and detailed radon progeny monitoring was commonly carried out by government agencies. Except for a limited period of the French and Ontario uranium mining, miners were not equipped with personal dosimeters, and the monitoring consisted of frequent grab sample measurements at representative locations.

Thus, at times, no measurements were performed in cer-

TABLE 1
Basis for Exposure Estimates Used in BEIR VI Analysis of 11 Miner Cohorts (2)

Study	Information used in deriving exposure estimates
Colorado Plateau	1940–1950: “Guesstimates” from knowledge about ore bodies, radon emissions from different types of ores, ventilation practices, and later radon or radon progeny measurements 1950–1969: 60–70% based on area averages of measurements in a locality, 10–20% on measurements in other years
New Mexico	1952–1965: Person-weighted averages and limited measurements 1965–1990: State-of-art exposure estimates
Beaverlodge	1949–1961: Limited measurements during 1954–1961 1961–1982: Individual exposure records
Ontario	Pre-1954: Extrapolations, taking into account ventilation changes (22% of estimated collective exposure) 1954–1957: Calculated from radon gas measurements and values of F from estimated air residence times 1958–1967: Mine-wide averaging of WL measurements 1968–: WL data for individual miners
Port Radium	1933–1940: Exposures not accounted for 1940–1960: Sparse measurements of radon gas and estimates of equilibrium fractions using information on mining practices and analogy with Beaverlodge mine
Czechoslovakia	1948–1960: Radon gas measurements and estimates of F under operating mine conditions 1961–: Extensive radon progeny measurements
French	1946–1956: Radon gas measurements begun in 1953 and calculated values of F 1956–1983: Systematic monitoring of radon progeny
Radium Hill	1952–1961: Radon measurements beginning in 1954 and estimated air residence times
China	1900–1953: Retrospective measurements in small pit mines 1953–1972: Retrospective measurements under recreated conditions in tunnels and galleries 1972–: Radon progeny measurements
Newfoundland	1900–1960: Model calculations (much higher exposures during this period) 1960–1969: Mine-wide average of radon progeny 1969–1978: Daily estimates of radon progeny exposures for individual miners
Swedish	1897–1968: Retrospective estimates based on mining conditions and measurements of dust levels 1968–1976: Extensive radon and progeny measurements

tain mines, and the levels are estimated by extrapolations of measurements from other mines taken during the same period. In some of the cohort studies, measurements were made long after the actual exposures to fill in missing information for earlier periods. In these cases, attempts were made either to physically reconstruct the earlier conditions or to mathematically extrapolate exposure levels based on information regarding changes in ventilation and mining practices and conditions (e.g., leakage of radon-containing groundwater).

Measurements in mines were sometimes made of radon gas rather than radon progeny, especially before it was realized that the latter was the hazardous agent. To convert a radon gas concentration to WL, it must be multiplied by the estimated equilibrium factor, F , which under modern mine conditions is typically 18–50% (2). F varies with the ventilation rate, since increasing ventilation shortens the time for the buildup of progeny by radon decay. In earlier years of mining, mines were often poorly ventilated, but ventilation was greatly improved in the 1960s due to the need to reduce fumes from diesel equipment and the increasing concern over the hazard from radon. Therefore, during the period in which radon gas measurements were being made, F was typically considerably higher than

would be found in recent mines and may have even approached unity (22).

For the reasons cited above, the quality of exposure data is very uneven from one study to another, and even from one period to another within a study. For example, in the case of the Colorado Plateau miners, a large study with relatively high exposures, there were no measurements before 1950, and exposures were “guesstimated” using information on the types of ore being mined and on mining practices. Subsequently, mines were monitored, but measurements were spotty, so that exposure estimates are mostly based on extrapolations of measurements made in mines located in the geographic vicinity and on extrapolations from measurements taken in other years. Despite the fact that 43,000 measurements were obtained in these mines, only about 10% of the Colorado Plateau cohort had their exposures estimates based entirely on radon progeny measurements conducted in the same mines concurrent with the exposures (2, 23).

Four cohorts for which there are more complete radon/radon progeny monitoring data upon which to base exposure estimates are the Ontario, New Mexico, Czech and French uranium miners.

Extensive measurements were performed in Ontario

mines during most of the period of mining, but none were made before 1954. Levels were highest during the earlier period, however, so about 22% of the estimated exposure is based on extrapolations from later measurements. For the period between 1954 and 1957, measurements were limited, and the radon progeny levels in each mine were estimated by mining engineers based on information regarding ventilation. Systematic measurements began in 1957, and the exposure estimates for 1957–1967 are based on 4-month averaging of measurements for each mine and an assumed distribution of locations for each miner in that mine. After 1967, exposure assignments were made by the employer based on more detailed information about a worker's daily activity, including time spent in different parts of a mine. A small comparison sample indicated that the methods used prior to and after 1968 yielded similar results (24). One complication is that some Ontario miners also worked as gold miners, during which time they were exposed both to radon and to arsenic, another lung carcinogen. The average radon exposure in gold mines, however, was small compared to that in uranium mines (25). Miners at Elliott Lake were also exposed to appreciable concentrations of ^{220}Rn (thoron) progeny: On average, parallel measurements of radon and thoron progeny indicated that the WL for the latter were typically 50–100% of the former (26). The relative contribution of thoron was likely to have been much lower in earlier periods when ventilation was poorer, however (27). Moreover, a given WL of thoron progeny is expected to cause only about one-third the dose rate to the lung as the same WL of radon (28). Therefore, the thoron exposure is probably not of great concern for epidemiological studies.

Measurements in New Mexico uranium mines were relatively complete, especially after 1968, until mining activity declined in the early 1990s. Individual miner exposures for this period are based on numerous area measurements and detailed personal work records. Annual average exposures levels for this period were low, about 3.8 WLM in 1968 and 1.2 WLM or lower beginning in 1972. During the period 1957 to 1967, there were about 20,000 measurements made, and annual exposures were still only 4–5 WLM, on average. This miner cohort thus provides considerable data on individuals receiving low exposures, although exposures in the New Mexico mines were very high during the early 1950s. In general, the exposure information for this cohort is based on measurements made within the same mine at the time of the exposure, except in the case of some miners who also worked in the Colorado Plateau uranium mines. An important limitation of this study relates to the relatively large sampling errors due to the small size of the cohort and the relatively low exposures.

The Czech uranium miners constitute a large cohort of individuals exposed to moderately high radon levels. Routine monitoring began in 1952, with 100 to 700 measurements per shaft being performed annually prior to 1961, but these measurements were of radon gas concentration

rather than of the PAEC. Aiding in the reconstruction of exposure estimates for the earlier period were radon/radon progeny measurements taken after 1960, including data collected during two accidents, occurring in 1969 and 1973, in which mechanical ventilation was turned off for at least a month. After 1961, radon progeny was monitored, with about three measurements being made per shaft per day, on average. Recently, Czech miner exposures have been re-evaluated with more careful attention to the workers' employment histories to account for other mining experience and the assignment of an individual monthly exposure for each miner based on the particular shaft where he was working (29). As discussed below, with the re-evaluation of exposures, the estimated mean exposure decreased slightly, but the derived risk coefficient increased substantially.

Uranium mining began in France in 1946, and large-scale monitoring of radon progeny began in 1956, at the same time forced ventilation was introduced. Exposure estimates for the period 1946–1955 are based on incomplete measurements of radon gas and consideration of early mining conditions by a group of experts. Radon levels were markedly reduced beginning in 1956, from an estimated median of 11 WLM/year to 3 WLM/year, declining further to 1 WLM/year by the early 1980s. Thus a sizable fraction of the French cohort's cumulative exposure rests on retrospective estimates for the period 1946–1955 rather than on measurements (2).

The French have pioneered the use of track-etch personal dosimeters to monitor radon progeny exposures to individual miners. In one study, conducted in 1978–1979, a comparison was made between exposure estimates based on measurements of ambient radon gas and those based on personal dosimetry (19). For this comparison, an average equilibrium fraction of 0.17 was applied to each radon gas reading to arrive at an ambient PAEC level for each mine location and period. The radon progeny exposure for each miner could then be calculated based on detailed information regarding his work activity. The exposure estimates made in this way tended to underestimate the "true" exposures by 30%, on average, as measured by the personal dosimeters. Closer agreement was found in a comparison between the personal dosimeters and grab sample measurements of PAEC (18). For some miner groups, however, Piechowski *et al.* (19) found that the variance in the exposures determined from ambient monitoring was much lower than indicated by the personal dosimeters, and the overall correlation between the two sets of estimates was poor. At least in part, these discrepancies probably result from a failure in the grab sample method to accurately weight the different work locations for each miner. By providing a time-integrated measure of an individual miner's exposure, the personal dosimeter automatically adjusts for changes in exposure rate due to changes in the miner's location and changes in the mine environment. Such changes cannot be properly accounted for with area measurements. As a result,

uncertainties in exposure estimates will be increased and—perhaps more important—the uncertainties cannot be rigorously quantified.

Much of the exposure assessment in the Chinese tin mines, the Newfoundland fluor spar mines, and the Swedish iron ore mines is retrospective. In the Chinese mines, no measurements were made until 1972. Exposure estimates for the period before 1972 are based on measurements later carried out under conditions thought to mirror the earlier period, but details of the Chinese miner dose reconstruction have not been published. A retrospective study by Corkill and Dory (30) assigned exposures to Newfoundland miners based on a variety of sources of information including mine maps, inspectors' reports, and anecdotal information. The authors employed computer models to try to take into account changes in ventilation and water entry over time. Initial exposure measurements taken in 1960 were found to be highly variable. Ventilation was greatly improved in the 1960s, drastically reducing exposure levels. Thus, whereas radon control and monitoring was introduced in 1960, most of the cohort's exposure was received previously. Hence, despite the strong efforts made to assess past exposures, the uncertainties in exposure remain high for this cohort. In the Swedish mines, radon monitoring began in 1968, and forced ventilation was introduced mainly in 1972. It was assumed, based on consideration of the pattern of natural ventilation and sampling of dust concentrations extending back to the 1930s, that radon levels before 1972 were fairly constant. This assumption has been challenged (31), and the reliability of the exposure estimates for the period before 1968 is now regarded as problematic (2); actual exposures may have been a factor of two or more higher than estimated in some cases, depending on the changing ventilation conditions (31).

Uranium mining at Beaverlodge, in northern Saskatchewan, began in 1949, and large-scale production started in 1953. Some radon and radon progeny measurements were performed in 1954 and 1956, mostly for the purposes of checking ventilation rather than to limit exposures. Regular radon progeny monitoring was initiated at the end of 1961, and daily records were kept of each worker's occupancy. In the first assessment of lung cancer risk based on this cohort by Howe *et al.* (32), the exposure to each miner was assigned on the basis of mine-wide median WL concentrations. These exposure assignments were also used in the BEIR IV and BEIR VI analyses. A reassessment of the Beaverlodge exposure estimates was carried out by SENES (31, 33), which included a detailed reconstruction of mining activity and its correlation with exposure measurements. Means rather than medians of measurements in a given location were used to estimate exposures. In some cases, detailed information on the location of individual miners could be used to gain improved estimates. The estimated average exposure of the Beaverlodge miners increased from 50.6 WLM to 81.3 WLM. Nevertheless, a reanalysis of the earlier case-control data of Howe *et al.* (32), using the new

dosimetry, actually yielded an increase in the estimated risk from 2.70×10^{-4} WLM⁻¹ to 3.25×10^{-4} WLM⁻¹ (34). This increase may have resulted from reduced exposure misclassification. One serious issue with the interpretation of data from this cohort is that many of its members may have accumulated appreciable, unaccounted-for radon exposures in other uranium mines prior to or after their employment at Beaverlodge. In addition, a recent review of the Beaverlodge work histories has uncovered numerous errors: e.g., some surface workers were erroneously classified as underground workers.²

Uranium mining at Radium Hill in Australia started in 1952, and monitoring of radon gas was carried out from 1954 until the mine closed in 1961. Exposures in the Radium Hill mines were low even before mechanical ventilation was introduced in 1955. Radon progeny exposures are based on radon gas measurements coupled with estimates of the equilibrium fraction from information about the ventilation conditions over time (35). For the period before mechanical ventilation, it was assumed that the mine was uniform in radon concentration; for the later period, there was sufficient radon monitoring information to assign estimates of radon gas concentration for three different regions (shaft, drive and stope) on each level of the mine. Ventilation models then yielded radon progeny concentration estimates for each of these regions. Finally, employment records were used to delineate the proportion of time spent in each region and level. Based on these results, an estimate of cumulative radon progeny exposure was derived for each miner. However, due mainly to the very low exposures, the risk coefficient derived from this study has a wide uncertainty range.

Mining at Port Radium in the Northwest Territory of Canada began in 1933, but there are no employment records before 1940. Consequently, exposure received before 1940 cannot be accounted for. Quite limited radon gas measurements were performed between 1945 and 1957. Reported radon concentrations were extremely variable, ranging from 1.85 to 11,100 Bq/liter (36). Radon progeny levels were assigned using the limited radon gas measurements in combination with values for *F*, estimated from information on mine operations and by analogy with the Beaverlodge mine in Saskatchewan (37). For these reasons, exposure projections for Port Radium are highly uncertain. Moreover, the mean duration of exposure at Port Radium was only 1.2 years, and exposures received in other mines, either before or after employment at Port Radium, are not accounted for.

ESTIMATES OF RADON RISK BASED ON ANALYSIS OF MINER COHORT DATA

An up-to-date and comprehensive analysis of lung cancer resulting from underground miner exposures to radon is

² S. E. Frost, personal communication.

TABLE 2
Number of Exposed Miners, Person-Years, and Lung Cancer Deaths, and Mean WLM and WL

Study	Ore	Number of workers	Number of person-years	Number of lung cancers	Mean WLM ^a	Mean duration (years)	Mean WL ^a
China	Tin	13,649	134,842	936	286.0	12.9	1.7
Czechoslovakia	Uranium	4,320	102,650	701	196.8	6.7	2.8
Colorado Plateau	Uranium	3,347	79,556	334	578.6	3.9	11.7
Ontario	Uranium	21,346	300,608	285	31.0	3.0	0.9
Newfoundland	Fluorspar	1,751	33,795	112	388.4	4.8	4.9
Sweden	Iron	1,294	32,452	79	80.6	18.2	0.4
New Mexico	Uranium	3,457	46,800	68	110.9	5.6	1.6
Beaverlodge (Saskatchewan)	Uranium	6,895	67,080	56	21.2	1.7	1.3
Port Radium (Northwest Territories)	Uranium	1,420	31,454	39	243.0	1.2	14.9
Radium Hill (Australia)	Uranium	1,457	24,138	31	7.6	1.1	0.7
France	Uranium	1,769	39,172	45	59.4	7.2	0.8
Total ^b		60,606	888,906	2,674	164.4	5.7	2.9

^a Weighted by person-years; includes 5-year lag period.

^b Totals adjusted for miners and lung cancers included in both the Colorado and New Mexico studies.

contained in the BEIR VI report (2). The main focus of that report was to estimate the risk of radon exposures in homes. Although there have been numerous case-control studies of radon-induced lung cancer in residences, the results of which are generally consistent with risk estimates extrapolated from miner studies (2, 6), the uncertainties in the former are such that the latter must still serve as the primary basis for quantitative estimates of radon risk in homes as well as mines.

Table 2 summarizes information on the 11 miner cohorts that the BEIR VI Committee used in its analysis [see ref. (2), Table D-12, p. 270]. From the combined data on these cohorts, the committee derived two preferred risk models: the “exposure-age-concentration” and “exposure-age-duration” models. Each of these models is of the form:

$$ERR(a) = \beta(w_{5-14} + \theta_{15-24}w_{15-24} + \theta_{25+}w_{25+})\phi_{age}\gamma_z,$$

TABLE 3
BEIR VI Model Parameters

Exposure-age-duration model		Exposure-age-concentration model	
$\beta \times 100$	0.55	$\beta \times 100$	7.68
Time since exposure			
θ_{15-24}	0.72	θ_{15-24}	0.78
θ_{25+}	0.44	θ_{25+}	0.51
Attained age			
$\phi_{<55}$	1.00	$\phi_{<55}$	1.00
ϕ_{55-64}	0.52	ϕ_{55-64}	0.57
ϕ_{65-74}	0.28	ϕ_{65-74}	0.29
ϕ_{75+}	0.13	ϕ_{75+}	0.09
Duration of exposure		Exposure rate (WL)	
$\gamma_{<5}$	1.00	$\gamma_{<0.5}$	1.00
γ_{5-14}	2.78	$\gamma_{0.5-1}$	0.49
γ_{15-24}	4.42	γ_{1-3}	0.37
γ_{25-34}	6.62	γ_{3-5}	0.32
γ_{35+}	10.2	γ_{5-15}	0.17
		γ_{15+}	0.11

where *ERR* is the excess relative risk (i.e., the fractional increase in lung cancer mortality) at age *a*, ϕ_{age} is a parameter depending on *a*, and γ_z is a parameter depending on the radon progeny concentration (exposure rate) or duration of exposure, respectively, for the two models. The quantity in parentheses represents a weighted sum of the exposures w_{5-14} , w_{15-24} , w_{25+} , defining, respectively, the exposures received 5–14, 15–24 and 25+ years prior to the attained age. For each model, values for β , the θ parameters, and ϕ_{age} were derived from fits to the entire data set. The estimates for γ_z , on the other hand, were determined for several restricted categories of data, each reflecting a range of exposure rates or durations. The parameter estimates for the two preferred models are shown in Table 3.

Due to improvements in mining practices and the sharp reduction in uranium mining, current interest centers on the risks to the general public from exposures in homes and in other buildings. These indoor exposure rates are usually lower than even the lowest exposure rates experienced by the 11 miner cohorts. It can be seen from Table 3 that in the exposure-age-concentration model, γ_z tends to increase with decreasing exposure rate. This means that, for a given level of exposure, the risk is maximal at the lowest exposure rates. However, the epidemiological data, supported by results of experimental animal studies, indicate that this “inverse dose-rate” effect saturates, so that as the exposure rate is further reduced, the risk per WLM exposure approaches a constant (38). Consequently, BEIR VI recommends that for residential exposures one should apply the model using the value of γ_z derived for the lowest exposure-rate category, i.e. <0.5 WL (<6 WLM/year). Likewise, since members of the public will be exposed to indoor radon for their whole lives, the report also recommended using the γ_z associated with the longest exposure duration (>35 years) when evaluating risks from residential exposure using the exposure-age-duration model.

Substantial heterogeneity is found in the ERR/WLM es-

imates derived from the 11 cohorts (2). Errors in the miner exposure estimates may contribute significantly to this heterogeneity, but the BEIR VI Committee was unable to make a quantitative estimate of the exposure biases and random errors. It is known from statistical theory that, under very general conditions, random errors tend to bias the exposure–response function toward the null (39). As an illustration of the possible effect of exposure errors, the BEIR VI report noted that an improvement in Czech miner exposure estimates was the major factor in a 65% increase in the estimated ERR/WLM for this cohort, although the estimated average miner exposure changed very little (29). As noted earlier, a reassessment of exposures for the Beaverlodge miners produced about a 60% increase in the average WLM, concomitant with an approximately 20% increase in the estimated risk per WLM (34).

These revised exposure estimates are still, of course, not true measures of the actual exposures, so the associated changes in the estimates of risk per WLM do not necessarily provide a true measure of the magnitude of the errors in the original risk estimates associated with errors in the exposure assignments. However, these changes do illustrate an important point. Although underestimation of exposures by a constant factor would bias the risk coefficient high by the same factor, when the bias is not constant, or when there are large random errors, underestimation of exposures may actually result in an underestimate of the risk coefficient. Thus, while it is likely that miner exposures have been systematically underestimated for some of the 11 cohorts, it is not clear what effect this might have had on the BEIR VI risk estimates.

Another problem in the interpretation of the miner data is the possible influence of other exposures, including tobacco smoke, silica, diesel fumes, γ rays, uranium ore dust, and arsenic. Tobacco use is unlikely to be strongly correlated with radon exposure, so the major uncertainty here relates to its possible synergism with radon progeny in causing lung cancer—it is unlikely to be a strong confounder. Based on limited evidence, the BEIR VI Committee concluded that silica and diesel are probably not strong modifiers of the lung cancer risk (2).

However, a recent assessment indicates that, in the case of uranium mines, exposures to ore dust and γ rays may contribute up to 25–75% of the “effective dose” to the lung (40). These exposures are neglected in most analyses of lung cancer risk in the miner cohorts, where WLM rather than dose is treated as the independent variable. Nevertheless, it is unclear whether or not an appreciable fraction of the lung cancers observed in the miner cohort studies might be attributable to these sources of radiation and whether or not this could produce a significant bias in the BEIR VI estimate of ERR/WLM. These sources would not be significant with respect to the Chinese tin mines, the Newfoundland fluor spar mines, or the Swedish iron ore mines. Moreover, the dose resulting from inhaled ore dust is mostly in the alveolar region of the lung, and it is unclear what

weight to assign to such doses: It has been argued that the alveolar weighting factor of 33% that goes into the ICRP effective dose calculation (41) may be much too high (42). There is also a great deal of uncertainty in γ -ray risk estimates for the lung, which are derived from the atomic bomb survivor data, due mainly to the uncertainty in extrapolating from acute to chronic exposures, and from the Japanese A-bomb survivor population to North American and European miners, who have very different smoking habits and baseline lung cancer rates (43). Finally, much of the exposure for some of the miners was accumulated before the mines were well ventilated; under these conditions, the relative contribution of γ rays to the dose would have been much lower.

Arsenic exposures may be an important source of error in the Ontario and, especially, in the Chinese miner study. Nearly 40% of all the reported deaths among the Chinese miners were from lung cancer, probably mostly due to occupational and environmental exposures to arsenic, and adjustment for arsenic exposure reduced the estimated ERR/WLM for this cohort from 0.61% to 0.16% (44). The difficulties in properly adjusting for arsenic may help to explain why the estimated ERR/WLM for this study lies below the combined uncertainty range for the other studies.

In conclusion, it is often very difficult to quantify the uncertainties in the miner exposures, especially where exposure estimates had to be based on extrapolations from measurements performed at other locations and times. In some cases, groups of miners could have their exposure estimates biased up or down. It would appear that underestimation of the exposure was probably more common, tending to bias risk estimates upward. The presence of other carcinogens in the mine environment may also have led to some overestimation of the ERR/WLM. On the other hand, random misclassification of miner exposures could have produced a substantial underestimation of the risk. This problem is highlighted by the increase in the estimated risk coefficient obtained from the Czech and Beaverlodge miner cohorts based on new, and presumably improved, exposure assessments.

The BEIR VI Committee was unable to quantify the uncertainty in radon risk projection models associated with errors in the exposure estimates for the miner cohorts. Although these errors are likely to be substantial, confidence in the risk projections for indoor exposures is strengthened by the fact that (1) the ERR/WLM appears to be reasonably consistent between the various miner studies, including those for which the exposure measurements are most complete; (2) the estimates are close to what one obtains from a model based solely on the miner data pertaining to exposures below 50–100 WLM, not much higher than cumulative lifetime indoor exposures; and (3) reasonable agreement is found between predictions based on these models and best estimates of risk from epidemiological studies of residential exposures (2, 6).

DOSIMETRY OF RADON PROGENY IN THE RESPIRATORY TRACT

Short-lived progeny, formed by radioactive decay of radon gas, are inhaled and deposited in the lung. Once deposited, they emit α particles that irradiate posited target cells in the lung, which may include both secretory and basal cells lining the bronchial epithelium. Methodology has been developed for estimating the dose to these target cells under specified exposure conditions, which involves mathematical modeling of the lung's anatomy and of physical and physiological processes. These processes include the transport and deposition of particles in the lung as well as the emission of α particles and their subsequent interaction with tissue (2, 28, 41, 45).

Attachment and Deposition Processes

Very rapidly after they are generated by radioactive decay, airborne radon progeny complex with molecular constituents of the atmosphere to form ultrafine particles with a diameter of 0.5–2 nm and a diffusion coefficient of about $0.05 \text{ cm}^2 \text{ s}^{-1}$. The fraction of the PAEC existing in this form is termed the “unattached fraction”. Before decaying, most of the radon progeny attach to ambient aerosol particles, which, in a mine, may be composed of ore dust and diesel fumes. The attached fraction will increase with increasing concentration of aerosol particles and decreasing ventilation rate.

Once inhaled, the radon progeny can be deposited in the lung by three distinct processes. *Diffusion* through random Brownian motion is the dominant deposition mechanism for particles of diameter up to a few hundred nanometers. Indeed, the diffusion process is so efficient for the ultrafine “unattached” progeny that most of these are deposited in the nose or mouth before they reach the tracheobronchial tree. *Gravitational settling* becomes significant for particles larger than about $0.2 \mu\text{m}$ within the peripheral airways and alveoli, where the velocity of airflow is low and residence times are long (41, 46). *Inertial impaction* occurs at places in the respiratory tract where the airflow is forced to change direction. This happens in the nose and oropharynx as well as at central airway bifurcations. If the momentum of the inhaled particle is sufficient, it is unable to follow the airflow pattern as it curves around obstacles, increasing the likelihood of impaction. This process is important for coarse dust particles with an aerodynamic diameter greater than about $2 \mu\text{m}$.

The process of inertial impaction may cause the larger particles to accumulate preferentially at bifurcations. Along with reduced mucociliary transport at bifurcations, this may lead to increased dose at these locations (47). Moreover, deposition of other carcinogens such as cigarette smoke (AMAD $\approx 0.3 \mu\text{m}$) is likely to be enhanced there as well. These regions of enhanced deposition may be particularly important in view of the apparent synergism between radon

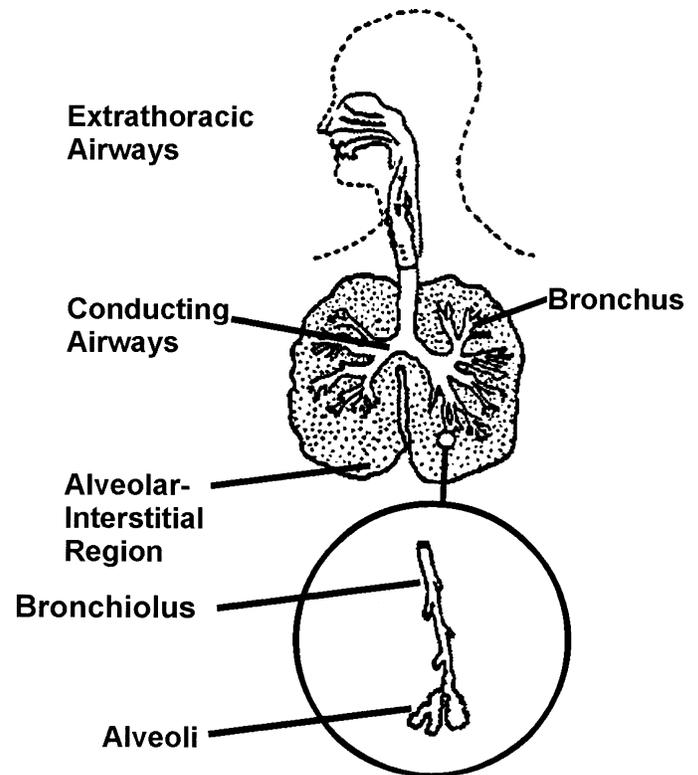


FIG. 2. Functional components of the respiratory tract.

progeny exposure and tobacco smoke in causing lung cancer (47, 48).

In underground mines, the unattached fraction is generally small and contributes only a few percent of the dose (47). The “attached” fraction is mostly in the “accumulation mode” (defined to be particles of $>30 \text{ nm}$ diameter); the accumulation mode includes particles about 200 nm in diameter from diesel exhaust, and coarser particles of dust ($500\text{--}2,000 \text{ nm}$ in diameter). In homes, both the unattached fraction and another component of “attached” progeny, the so-called nucleation mode (consisting of particles $2\text{--}30 \text{ nm}$ in diameter), contribute appreciably to the lung dose. The dose per unit exposure is particularly high for the latter because these particles get past the nose and mouth yet deposit efficiently by diffusion in the bronchial and bronchiolar airways.

Anatomy of the Respiratory System

The human respiratory tract is pictured schematically in Fig. 2. Air is inhaled through the nose or mouth and passes through the larynx and down the trachea, which branches (bifurcates) into a series of airways called bronchi. In turn, the bronchi bifurcate into smaller and smaller airways called bronchioles. The bronchi and bronchioles are lined with cilia and a surface layer of mucus. Synchronized beating of the cilia serves to move the mucous layer proximally, toward the throat, where the mucus is swallowed. Foreign particles, including deposited radon progeny are trapped in

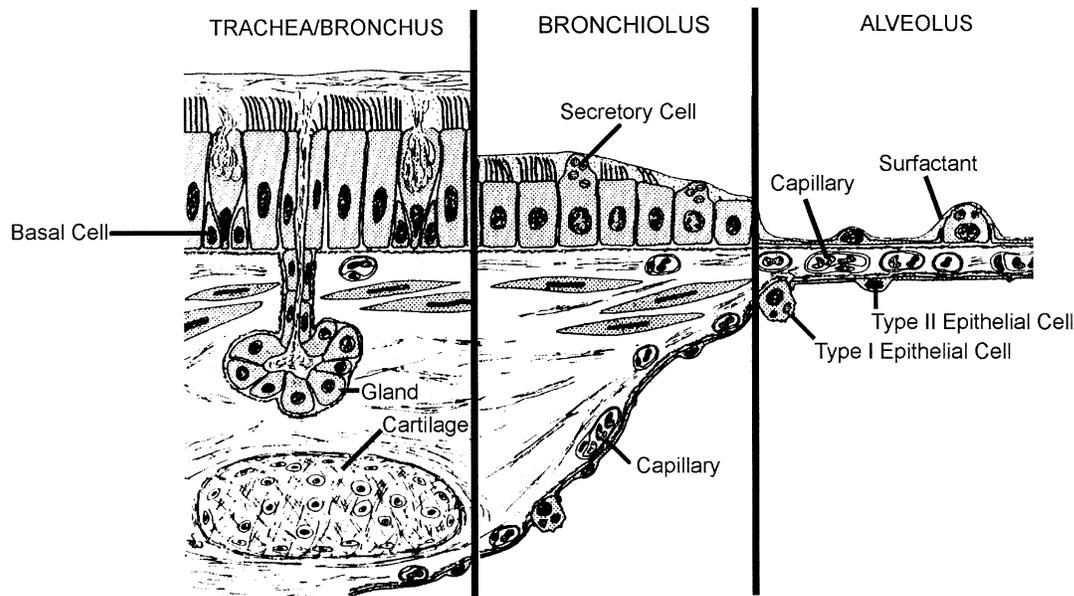


FIG. 3. Structure of the airway walls; adapted from ref. (49).

this mucus, and are thus continually cleared from the bronchi and bronchioles by ciliary action. In the smallest (most distal) bronchioles, mucus is cleared slowly. The rate of clearance increases as the airways become progressively larger (toward the trachea), resulting in a roughly constant thickness of mucus throughout the bronchi and bronchioles (41). Clearance from the bronchi takes place more quickly than from the bronchioles. Overall, bronchial clearance has a relatively small effect in reducing the dose from deposited radon progeny, and bronchiolar clearance very little effect.

The thickness of the bronchial airway lining (bronchial epithelium) is generally greater than the range of the α particles emitted by radon progeny being cleared in mucus, while the thickness of the bronchiolar epithelium is substantially less (Fig. 3). Also, some of the sensitive target cells in the bronchi (basal cells) are located deep below the surface. These factors cause the dose per α -particle disintegration per unit surface area to be less in the bronchi than in the bronchioles. Calculation of these doses is somewhat complex and requires detailed consideration of the airway and target cell geometries (45).

The terminal bronchioles are not ciliated. They serve to conduct air to tiny air sacs, called alveoli, where exchange of oxygen and carbon dioxide with the blood takes place (respiration). To promote respiration, the surface area of the respiratory parts of the lungs is very large, and the tissue membranes are very thin (Fig. 3). Deposited particles are cleared from the alveoli mainly through engulfment by scavenging cells (macrophages). Thus clearance from this part of the respiratory tract is slow and in fact is negligible for the short-lived radon progeny. Calculation of the amount of dose absorbed by the thin alveolar tissue is much simpler than that for the bronchi and bronchioles, since the

alveolar dose can be considered uniform throughout the tissue (target cells).

Pattern of Deposition in the Lung and Calculation of Average Lung Dose

Particle deposition in the various portions of the respiratory tract varies with particle size, breathing rates, relative amounts of oral and nasal breathing, and the dimensions and shapes of the different parts of the respiratory tract.

Figure 4 compares the calculated fractional regional deposition in the nose/oral cavity/larynx, bronchi, bronchioles and alveolar lung as a function of particle size for an adult male exposed in the home or at the typically higher breathing rate of a miner (45). The smallest-size particles deposit efficiently by diffusion and are largely removed in the nose, oral cavity and larynx. Thus the unattached fraction of radon progeny (particles around 1 nm in diameter), long considered as delivering a proportionally greater dose per unit exposure to bronchial target cells, has limited penetration to the bronchi. However, in indoor air, physical and chemical processes can increase the size of radon progeny "particles" to 5 nm or greater diameter. A 5-nm particle can effectively penetrate the nasal and oral passages but will deposit with relatively high efficiency in the bronchial region. Thus the amount of α -particle radiation dose received by bronchial target cells per unit exposure to potential α -particle energy, the so-called dose conversion coefficient (DCC), peaks quite strongly in the 3- to 10-nm particle size range (Fig. 4), the so-called nucleation mode, as illustrated in Figs. 5 and 6.

Figure 5 compares the particle-size distributions associated with radon PAEC that were assumed in the BEIR VI Report to "typify" exposures in homes and mines. Also

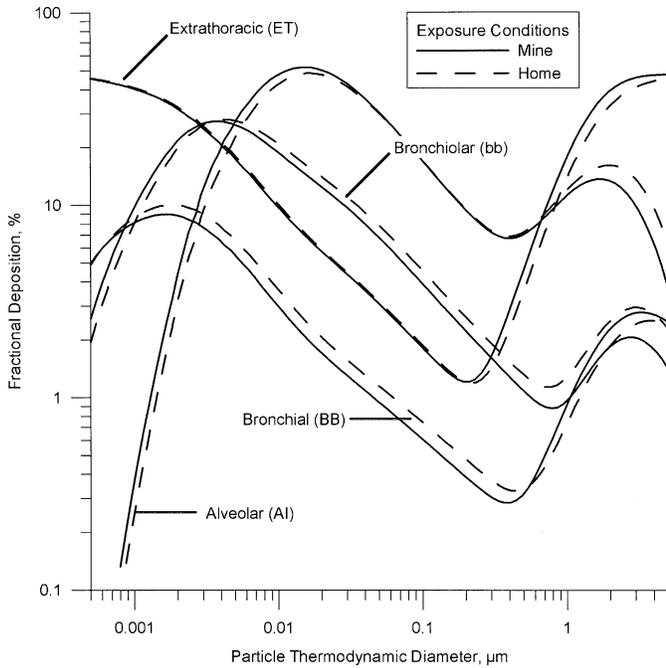


FIG. 4. Fractional deposition in each part of the respiratory tract compared for exposure in mines and homes (assuming particles are unit density spheres).

shown are the DCCs for mines and homes, calculated for varying particle size. These DCCs represent a weighted average of DCCs for bronchial basal cells (1/6), bronchial secretory cells (1/6), bronchiolar secretory cells (1/3), and alveolar cells (1/3), as recommended in ref. (41). The overall dose per unit exposure to potential α -particle energy in each environment is then given by the product of the corresponding DCC distribution and the PAEC distribution, integrated over the whole particle-size range (Fig. 6).

As the size continues to increase toward 100 nm, the effectiveness of the particle deposition decreases because the particle moves less effectively by diffusion. Most of the deposition in the size range above 10 nm occurs in the deep lung (alveolar region). Although the alveolar region contains many more cells than the rest of the lung, deposition in the alveoli is less likely to lead to cancer. The radon progeny deposited in the alveolar region of the lung are spread over a very large area so that the activity per unit mass of tissue—and hence the absorbed dose—is correspondingly small. Moreover, though the alveolar region contains most of the lung’s mass and surface area, the great majority of lung cancers in humans arise in the cells lining the bronchi and bronchioles (28). Thus it would appear that irradiated alveolar cells are much less prone to form tumors.³ For these reasons, irradiation by radon progeny as-

³ This conclusion is implicitly incorporated into the International Commission on Radiological Protection (ICRP) radiation weighting scheme, which presumes that, for a uniformly irradiated lung, only 33% of the cancers will be induced in the alveolar region although it represents well over 90% of the lung’s mass—and almost all of its surface area (41). The 33% value appears, moreover, to be a large overestimate (42).

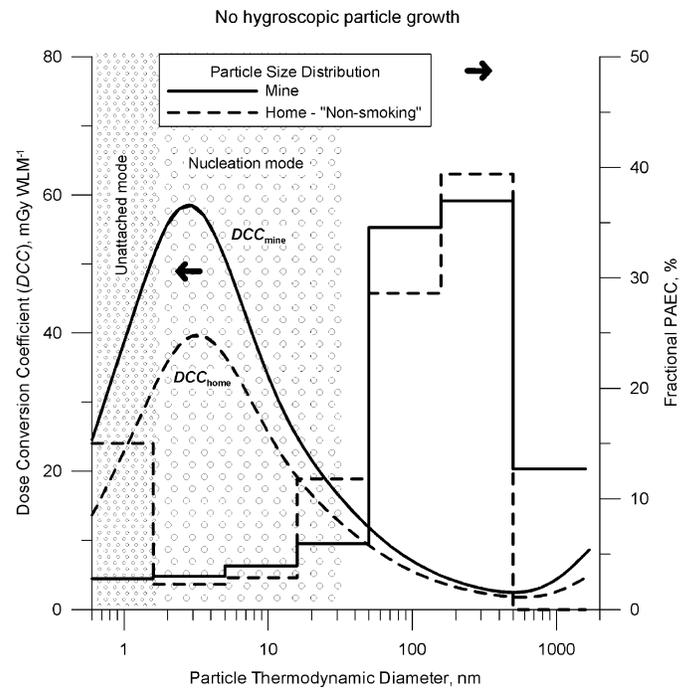


FIG. 5. Activity-size distribution of PAEC, compared for a mine and a “non-smoking” home, with resulting dose conversion coefficients (DCCs).

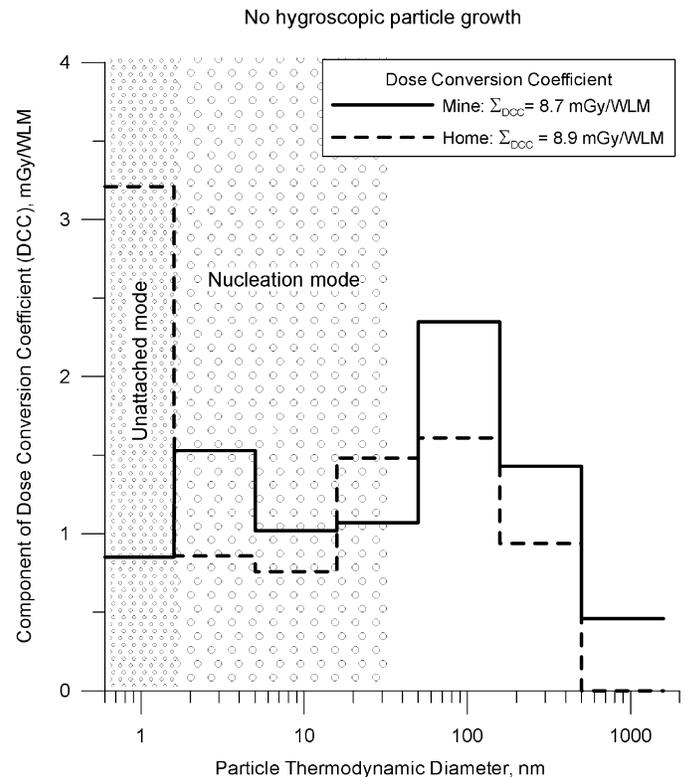


FIG. 6. Distribution of resulting dose per unit exposure as a function of progeny particle size for a mine and a “non-smoking” home.

sociated with particles larger than 10–15 nm in diameter is likely to be relatively unimportant. For larger particles (>2.5 μm), the deposition is mainly in the nose and mouth, and very few of those particles reach the critical target cells of the lung where they could cause cancer.

The dose from short-lived radon progeny depends not only on the amount of aerosol deposited but also on the amount retained in the lung over time. Mucociliary transport in the bronchi and bronchioles carries deposited material out of the lungs and into the gastrointestinal tract. The ^{222}Rn progeny generally decay and cause damage before they can be cleared (41). However, for ^{220}Rn , the second decay product, ^{212}Pb , has a radioactive half-life of about 11 h, allowing much of the ^{212}Pb to be cleared before it decays. As a result, ^{220}Rn decay products are estimated to produce a substantially lower dose for the same exposure to potential α -particle energy than ^{222}Rn progeny (2).

EXTRAPOLATION OF RISK ESTIMATES FROM MINES TO HOMES BASED ON COMPARATIVE DOSIMETRY

The BEIR VI models provide estimates of the excess relative risk per unit radon progeny exposure (ERR/WLM) as a function of age, time since exposure, and exposure rate (or exposure duration), based on a combined analysis of the 11 miner cohorts. Presumably, however, it is the α -particle dose (Gy) to target cells in the lung that determines risk, rather than the exposure (WLM). This presumption serves as a basis for extrapolating the models' predictions from mine to home exposures (50). For home exposures, we can write

$$(\text{ERR}/\text{Gy})_h = (\text{ERR}/\text{WLM})_h(\text{WLM}/\text{Gy})_h,$$

where $(\text{Gy}/\text{WLM})_h$ represents a weighted average of doses to the relevant target cells in the lung per WLM of radon progeny exposure (41, 45). Similarly, for mines,

$$(\text{ERR}/\text{Gy})_m = (\text{ERR}/\text{WLM})_m(\text{WLM}/\text{Gy})_m.$$

Equating these two expressions, we have

$$(\text{ERR}/\text{WLM})_h = K (\text{ERR}/\text{WLM})_m,$$

where the factor K is the ratio of the dose per WLM for a defined residential exposure compared to an exposure in mines; i.e.,

$$K = (\text{DCC})_h/(\text{DCC})_m.$$

Thus, in the absence of information to the contrary, it is assumed that after making the adjustment for the estimated differences in the DCC, the risk models derived for mine exposures of adult males also apply to indoor exposures of the general population, which includes individuals of both genders and all ages. K depends only on differences between mine and home exposures and not on the absolute magnitude of the DCC. As a consequence, K is relatively insensitive to some of the parameters influencing the dosimetry (e.g., the location of target cells in the lung or the

rate of mucus flow in the airways). For this reason, the value of K should be less uncertain than the magnitude of the DCC.

As a follow-on to its BEIR IV report (50) on the risks from radon and other α -particle emitters, the NRC published its *Comparative Dosimetry Report*, which examined radon progeny dosimetry in mines and homes (28). It was concluded in this report that the dose per WLM in homes was about 20% or 30% lower for adult and child exposures in homes, respectively, than for adult male exposures in mines ($K = 0.7$ – 0.8). A re-evaluation of K was performed in the BEIR VI study (2) based on modified assumptions. These changes included (1) lower breathing rates in mines, (2) increased nasal deposition of unattached progeny, and (3) a more complex consideration of the full particle-size spectrum of radon progeny in both mine and home atmospheres. The latter included introduction of the new, dosimetrically important nucleation mode occupying the intermediate particle-size range between the classical "unattached" and "attached" aerosol modes considered in BEIR IV. Based on these more comprehensive data, the best estimate for K was re-evaluated as being very close to 1. Hence the risk models derived for miners were used in BEIR VI without adjustment to project risk in homes.

However, as pointed out by Cavallo (51), BEIR VI had apparently redefined K in terms of the dose per unit gas exposure instead of the dose per WLM. Cavallo inferred that, as a result, the ERR/WLM in homes had been overestimated by about a factor of 2.2. This issue has recently been re-examined, and it now appears that unspecified, nearly offsetting, errors were made in the BEIR VI calculations (45, 52). Revised calculations, incorporating the BEIR VI input parameters, yield values of K close to 1 over a range of residential exposure conditions and dosimetric assumptions (45), as illustrated earlier (Fig. 6). Thus the BEIR VI projections of risks to the general public from indoor exposures do not require modification (53).

In conclusion, despite the differences in mine and residential exposure characteristics, the DCC is estimated to be similar in the two cases. Largely, this reflects a higher breathing (intake) rate in mines counterbalancing an increased efficiency of lung deposition in homes associated with the higher prevalence of unattached and nucleation-mode progeny in the indoor environment (45).

Questions nevertheless remain about the dosimetric extrapolation from mines to homes. Although the data used by the BEIR VI Committee to characterize the mine aerosol were drawn from numerous and detailed measurements in four different underground mines, these mines were all operated in the 1970s, and all with diesel equipment (51). It has not been established how well these mining conditions represent those in non-diesel mines or those in earlier mines, generally, where diesel was not employed and ventilation was often poor. Likewise, the data used by the BEIR VI Committee to characterize the home aerosols were drawn from a very large number of individual samples tak-

en from just four homes (three in the northeastern U.S. and one in Ontario, Canada), and it is again unclear how representative the conditions in these homes are for the entire U.S.

EXPOSURE ESTIMATES FOR RESIDENTIAL STUDIES

A large number of case-control studies have now been carried out in which estimated residential radon exposures of lung cancer cases and controls are compared. By necessity, the radon exposure assessment is retrospective. Typically, investigators try to gather information on residences that subjects inhabited in some period, most commonly a 5–30-year time window, prior to diagnosis of the cancer. This often requires the placement of detectors in houses now occupied by new residents.

To obtain a seasonal and spatial average of the radon exposure, enclosed ATDs (radon pots) are usually placed in various locations in the homes for 1 year. This method provides a measure of the yearly average radon gas concentration at the location where the detector is placed. The calibration and use of this technique have been reviewed (54, 55).

To compare results from the residential studies with predictions made on the basis of models derived from the miner studies, the radon gas measurements in homes are converted to WLM. This requires an estimate of the equilibrium fraction, F , and the fraction of time subjects spend in their homes (occupancy factor). Measurements indicate that F is highly variable under residential exposure conditions, from one house to another, and within the same house across time. As a best estimate of the median value in homes, the NRC BEIR VI Committee recommended a value of 40% for homes without a smoker (2). F is expected to be somewhat higher in homes with a smoker since the radon decay products attach to airborne smoke particles, slowing down their plate-out by diffusion. BEIR VI adopted a value of 70% for the occupancy factor.

There are serious limitations to the method used to estimate radon progeny exposure in homes aside from the fact that it is based on measurements of radon gas exposure rather than radon progeny exposure or actual dose to the lung. Subjects may have moved one or more times during the exposure period of interest, and it may not be practicable to make measurements in all the houses where the person had resided. Changes in building configuration, ventilation, heating or lifestyle can produce significant changes in radon levels. In addition, greater than 20% year-to-year variations in indoor radon levels have been found, related perhaps to variations in precipitation (54, 56, 57). Hence even a 1-year cumulative measurement may not provide an accurate estimate of the average radon concentration in the house over the exposure period of interest. Finally, diurnal variations in radon concentration may occur, which could be correlated with the presence or absence of the occupants in the home. As a consequence, the average radon concen-

tration, as measured by the detector, may not accurately reflect the average level to which residents are exposed.

Another approach to the estimation of retrospective exposures has been developed using pieces of glass associated with a particular house (e.g. from a window) or an individual (e.g. from a picture frame) for a known length of time (16, 17). The basis of the method is that the decay of the short-lived radon decay product ^{214}Pb plated onto the glass will result in a recoil ^{210}Pb nucleus, which can penetrate a short distance into the glass, where it remains until it decays with a half-life of 22 years to the subsequent shorter-lived α -particle emitter ^{210}Po . Thus, for a constant concentration of radon progeny in room air, the buildup of $^{210}\text{Pb}/^{210}\text{Po}$ activity in the surface of an openly exposed piece of glass is reasonably linear over several decades. The activity of ^{210}Po that has built up in the glass surface can be measured with high sensitivity (and low background), and this will reflect the radon progeny concentration in the ambient air integrated over the previous several decades. Alpha particles emitted by decay of ^{210}Po in the glass surface can be counted readily and economically with solid-state α -particle track detectors placed in close contact with the glass for a week or so.

This methodology can be used to complement the standard ATD method described above. In particular, it can be used to assess “missing exposures” in past residences not accessible to the investigators by measuring ^{210}Po activity on a “personal” item that has been in the study subject’s possession for several decades. Although the glass-based measurements can provide useful complementary information, it is unclear whether exposure estimates made by this technique are as reliable as those obtained with the enclosed ATDs. However, good agreement between results from the glass-based method and from long-term measurements in homes has been reported (58, 59). By exposing picture glass for a known time in a variety of home environments, and also obtaining contemporary measurements of the average radon gas concentration (using ATDs), Fitzgerald and Hopke (59) found a $\pm 36\%$ standard deviation in the ratio of the radon gas concentration predicted by the ^{210}Po activity on glass to the ATD measurements. For the majority of the data, the ratio was within $\pm 20\%$. However, some individual values were found to be too high or too low by more than a factor of two. By analyzing these data further, in terms of a model of the underlying production rate of ^{210}Po on the exposed glass surface, Fitzgerald and Hopke showed that the “outliers” represented unusually high or low values of the plate-out rate per unit airborne concentration of short-lived radon progeny. These corresponded to uncommon combinations of ventilation rate, ambient aerosol particle concentration in room air, and the available surface-to-volume ratio in the room studied. The home environments studied were all in the northeastern U.S. or Ontario, Canada. Fitzgerald and Hopke pointed out that, whereas the majority of the data obtained in these homes (from ^{210}Po on glass) gave an estimated radon ex-

posure close to that measured with contemporary ATDs, this relationship may be systematically different in homes from other regions of the U.S.

Three case-control studies have employed the glass technique in combination with the standard radon pots. In two of these studies, conducted in Missouri (60) and in Sweden (61), a higher excess risk per WLM was found when the exposure was quantified based on the glass detectors. Glass detectors were also employed in the Iowa study (62), but in this case reasonably good agreement was found between results obtained with the two techniques.⁴

The plate-out of radon progeny onto the glass surface is a complex function of the activity size distribution and air movement in the room. Moreover, the response will be a nonlinear function of the long-term cumulative exposure, depending on the detailed temporal pattern of radon progeny concentration. The usefulness of this technique therefore has not been fully evaluated.

DISCUSSION AND CONCLUSIONS

The epidemiological data for underground miner cohorts currently serve as the primary basis for estimating the risks from radon exposures, including indoor exposures. The assessment of exposure for individual miners in these studies is often very incomplete, so that exposure estimates are sometimes based upon spatial averages within a mine or upon extrapolations from measurements taken at a later time or even in another mine. The errors in exposure estimates are likely to be largest for early periods of mining, when measurements were absent or less frequent, and exposures tended to be higher during that period. It is difficult to assess the importance of such errors on model projections for low-level exposures.

Although there is reasonable concordance in the ERR/WLM estimates from the various miner studies, there are statistically significant discrepancies among these estimates. Some of these discrepancies may relate to inaccurate adjustments for the dependence of risk on exposure rate and temporal factors; some may result from confounding by the effects of tobacco, arsenic, or other mine exposures; still others may be related to genetic and lifestyle differences between the various mining cohorts. Nevertheless, errors in the assignment of miner exposures are probably a major factor underlying the discrepancies. It is unlikely that many of these exposure assignments can be greatly improved. Although additional follow-up of miners with lower, and better quantified, exposures might be helpful in reducing uncertainties, there is little support for such efforts.

The extrapolation of risk estimates based on epidemiological studies of underground miners to the case of residential exposures rests on a comparison between the mine and homes of the estimated dose, per unit radon progeny exposure, received by presumptive target cells in the lung

(K factor). The value of K is determined primarily by the potential α -particle activity-weighted particle-size distributions that are assumed to characterize exposure in mines and homes (45). These distributions are uncertain: In mines, measurements of these distributions were performed only in the 1970s, and all with diesel equipment; in homes, the measurements are limited and are not necessarily representative (2, 45). Further characterization of historical conditions in mines applicable to the miner cohorts, as well as a more complete assessment of exposure conditions in the U.S. housing stock, could substantially increase the accuracy and reliability of our projections of residential radon risk based on the models derived from the miner data.

Since the concern about radon risk now centers upon exposures in homes rather than in mines, current research focuses on the collection and analysis of data on residential studies of radon-induced lung cancer. Exposure assessment for these studies poses different challenges than for the miner cohort studies. In particular, the much lower levels of radon risk associated with residential exposures implies that a higher level of accuracy in exposure assessment is required to be able to detect and quantify risk. Recent efforts to integrate the information from all the published residential studies point strongly to a risk at residential exposure levels, the magnitude of which is consistent with model projections from the miner studies (6). The accumulation of additional residential data, coupled in some cases, perhaps, with improved retrospective dosimetry based on glass, may strengthen confidence in the models, or even provide a direct quantitative basis for estimates of the risk in homes that will complement or supersede the model projections derived from the miner cohorts.

ACKNOWLEDGMENT

The authors gratefully acknowledge Dr. Philippe Duport for many useful insights concerning the measurements of radon exposures in underground mines.

REFERENCES

1. J. H. Lubin, J. D. Boice, Jr., C. Edling, R. W. Hornung, G. Howe, E. Kunz, R. A. Kusiak, H. I. Morrison, E. P. Radford and D. A. Pierce, *Radon and Lung Cancer Risk: A Joint Analysis of 11 Underground Miners Studies*. Publication No. 94-3644, National Institutes of Health, Washington, DC, 1994.
2. National Research Council, Committee on the Biological Effects of Ionizing Radiation, *Health Effects of Exposure to Radon (BEIR VI)*. National Academy Press, Washington, DC, 1999.
3. A. C. James. Lung dosimetry. In *Radon and Its Decay Products in Indoor Air* (W. W. Nazaroff and A. V. Nero, Eds.). Wiley, New York, 1988.
4. ICRP, *Protection Against Radon-222 at Home and at Work*. Publication 65, *Annals of the ICRP*, Vol. 23, No. 2, Pergamon Press, Oxford, 1993.
5. A. C. James and A. Birchall, New ICRP lung dosimetry and its risk implications for alpha emitters. *Radiat. Prot. Dosim.* **60**, 321-326 (1995).
6. J. Lubin, Discussion: Indoor radon and risk of lung cancer. *Radiat. Res.* **151**, 105-107 (1999).

⁴ R. W. Field, personal communication.

7. W. F. Bale, Memorandum to the Files, March 14, 1951: Hazards associated with radon and thoron. *Reprinted in Health Phys.* **38**, 1061 (1980).
8. D. A. Holaday, History of exposure of miners to radon. *Health Phys.* **16**, 547–552 (1969).
9. E. C. Tsvigolou, H. E. Ayer and D. A. Holaday, Occurrence of non-equilibrium atmospheric mixtures of radon and its daughters. *Nucleonics* **11**, 40–45 (1953).
10. NCRP, *Measurement of Radon and Radon Daughters in Air*. Report No. 97, National Council on Radiation Protection and Measurements, Bethesda, MD, 1988.
11. H. L. Kuznetz, Radon daughters in mine atmospheres: a field method for determining concentrations. *Am. Ind. Hyg. Assoc. Q.* **17**, 85–88 (1956).
12. R. Rolle, Rapid working level monitoring. *Health Phys.* **22**, 233–238 (1972).
13. Nuclear Energy Agency, *Metrology and Monitoring of Radon, Thoron and Their Daughter Products*. Organization for Economic Co-operation and Development, Paris, 1985.
14. B. M. R. Green, L. Brown, K. D. Cliff, C. M. H. Driscoll, J. C. H. Miles and A. D. Wrixon, Surveys of natural radiation exposure in UK dwellings with passive and active measurement techniques. *Sci. Total Environ.* **45**, 459–466 (1985).
15. A. Frank and E. Benton, Some plateau problems in passive radon dosimetry. In *Conference on Radon Daughters Plateout Phenomena*. University of Illinois, Urbana, IL, 1979.
16. C. Samuelsson, Retrospective determination of radon in houses. *Nature* **334**, 338–340 (1988).
17. J. A. Mahaffey, M. A. Parkhurst, A. C. James, F. T. Cross, M. C. R. Alavanja, J. D. Boice, S. Ezrine, P. Henderson and R. C. Brownson, Estimating past exposure to indoor radon from household glass. *Health Phys.* **64**, 381–391 (1993).
18. A. M. Chapuis, P. Duport and P. Zettwoog, Individual dosimeter for radon and thoron daughters. In *Proceedings of the Specialist Meeting on Personal Dosimetry and Area Monitoring Suitable for Radon and Daughter Products (Paris 20–22 Nov. 1978)*, pp. 87–95. Nuclear Energy Agency, Organization for Economic Co-operation and Development, Paris, 1979.
19. J. W. Piechowski, L. E. Gac, J. Brenot, J. C. Nenot and P. Zettwoog, Exposure to short-lived radon daughters: Comparison of individual and ambient monitoring in a French uranium mine. In *Proceedings of the International Conference on Radiation Hazards in Mining: Control, Measurement and Medical Aspects*, pp. 539–548. Society of Mining Engineers of American Institute of Mining, Metallurgical and Petroleum Engineers, Inc., New York, 1981.
20. S. Bernhard, J. F. Pineau, A. Rannou and P. Zettwoog, 1983: One year of individual dosimetry in French miners. In *Occupational Radiation Safety in Mining, Proceedings of the International Conference, Vol. 2* (H. Stocker, Ed.), pp. 526–540. Canadian Nuclear Association, Toronto, 1984.
21. R. G. Beverly, A comparison of mine exposures with regulatory standards and radon daughter concentrations. In *Proceedings of the International Conference on Radiation Hazards in Mining: Control, Measurement and Medical Aspects*, pp. 549–558. Society of Mining Engineers of American Institute of Mining, Metallurgical, and Petroleum Engineers, Inc., New York, 1981.
22. L. D. Hamilton, L. W. Swent and D. B. Chambers, *Visit to the Centre of Radiation Hygiene, Institute of Hygiene and Epidemiology, Prague Czechoslovakia*. Trip report to Division of Environmental Health, World Health Organization, Geneva, 1990.
23. F. E. Lundin, J. K. Wagoner and V. E. Archer, *Radon Daughter and Respiratory Cancer, Quantitative and Temporal Aspects*. NIOSH-NIEHS Joint Monograph No. 1, U.S. Department of Health, Education and Welfare, Washington, DC, 1971.
24. J. Muller, R. Kusiak and A. C. Ritchie, *Factors Modifying Lung Cancer Risk in Ontario Uranium Miners 1955–1981*. Ontario Ministry of Labour, Ontario Workers' Compensation Board of Ontario, Atomic Energy Control Board of Canada, Toronto, 1989.
25. R. A. Kusiak, A. C. Ritchie, J. Muller and J. Springer, Mortality from lung cancer in Ontario uranium miners. *Br. J. Indust. Med.* **50**, 920–928 (1993).
26. J. Bigu, Mine models and the thoron problem in underground uranium mines. In *Proceedings of the International Conference on Radiation Hazards in Mining: Control, Measurement and Medical Aspects*, pp. 243–255. Society of Mining Engineers of American Institute of Mining, Metallurgical, and Petroleum Engineers, Inc., New York, 1981.
27. J. R. Johnson, The relative effect of ventilation on the potential alpha energy from ^{222}Rn and ^{220}Rn progeny. *Health Phys.* **49**, 996–998 (1985).
28. National Research Council, *Comparative Dosimetry of Radon in Mines and Homes*. National Academy Press, Washington, DC, 1991.
29. L. Tomásek, S. C. Darby, T. Fearn, A. J. Swerdlow, V. Placek and E. Kunz, Patterns of lung cancer mortality among uranium miners in West Bohemia with varying rates of exposure to radon and its progeny. *Radiat. Res.* **137**, 251–261 (1994).
30. D. A. Corkill and A. B. Dory, *A Retrospective Study of Radon Daughter Concentrations in the Workplace in the Fluorspar Mines of St. Lawrence, NFLD*. Report No. INFO0127, Atomic Energy Control Board, Ottawa, 1984.
31. SENES Consultants, Ltd., *Uncertainty in Exposure of Underground Miners to Radon Daughters and the Effect of Uncertainty on Risk Estimates*. Report to the Atomic Energy Control Board, Ottawa, 1989.
32. G. R. Howe, R. C. Nair, H. B. Newcombe, A. B. Miller and J. D. Abbatt, Lung cancer mortality (1950–1980) in relation to radon daughter exposure in a cohort of workers at Eldorado Beaverlodge Uranium Mine. *J. Natl. Cancer Inst.* **77**, 357–362 (1986).
33. SENES Consultants, Ltd., *Detailed Reconstruction of Radon Daughter Exposures of Eldorado Beaverlodge Uranium Mine Employees*. Report to the Atomic Energy Control Board, Ottawa, 1991.
34. G. R. Howe and R. H. Stager, Risk of lung cancer mortality after exposure to radon decay products in the Beaverlodge cohort based on revised exposure estimates. *Radiat. Res.* **146**, 37–42 (1996).
35. A. Woodward, D. Roder, A. J. McMichael, P. Crouch and A. Mylvaganam, Radon daughter exposures at the Radium Hill uranium mine and lung cancer rates among former workers, 1952–1987. *Cancer Causes Control* **2**, 213–220 (1991).
36. G. R. Howe, R. C. Nair, H. B. Newcombe, A. B. Miller, J. D. Burch and J. D. Abbatt, Lung cancer mortality (1950–1980) in relation to radon daughter exposure in a cohort of workers at Eldorado Port Radium Uranium Mine: possible modification of risk by exposure rate. *J. Natl. Cancer Inst.* **79**, 1255–1260 (1987).
37. SENES Consultants, Ltd., *A Re-evaluation of Radon Decay Product Exposures to Underground Workers at the Port Radium Mine*. Report to the Atomic Energy Control Board, Ottawa, 1996.
38. D. J. Brenner and E. J. Hall, The inverse dose-rate effect for oncogenic transformation by neutrons and charged particles. A plausible interpretation consistent with published data. *Int. J. Radiat. Biol.* **58**, 745–758 (1990).
39. W. A. Fuller, *Measurement Error Models*. Wiley, New York, 1987.
40. P. Duport, Is the radon risk overestimated? Neglected doses in the estimation of the risk of lung cancer in uranium underground miners. *Radiat. Prot. Dosim.* **98**, 329–338 (2002).
41. ICRP, *Human Respiratory Tract Model for Radiological Protection*. Publication 66, *Annals of the ICRP*, Vol. 24, No. 1/4, Pergamon Press, Oxford, 1994.
42. W. J. Bair, Overview of ICRP respiratory tract model. *Radiat. Prot. Dosim.* **38**, 147–152 (1991).
43. EPA, *Estimating Radiogenic Cancer Risks. Addendum: Uncertainty Analysis*. EPA 402-R-99-003, U.S. Environmental Protection Agency, Washington, DC, 1999.
44. X. Z. Xuan, J. H. Lubin, J. Y. Li, L. F. Yang, Q. S. Luo, Y. Lan, J. Z. Wang and W. J. Blot, A cohort study in southern China of tin miners exposed to radon and radon decay products. *Health Phys.* **64**, 120–131 (1993).

45. A. C. James, A. Birchall and G. H. Akabani, Comparative dosimetry of BEIR VI revisited. *Radiat. Prot. Dosim.* **108**, 3–26 (2004).
46. J. Heyder, G. Rudolf, C. F. Schiller and W. Stahlhofen, Deposition of particles in the human respiratory tract in the size range 0.0015–15 μm . *J. Aerosol Sci.* **17**, 811–825 (1986).
47. W. Hoffman, D. J. Crawford-Brown, M. G. Ménache and T. B. Martonen, Carcinogenic risk of non-uniform alpha particle irradiation in the lungs: Radon progeny effects at bronchial bifurcations. *Radiat. Prot. Dosim.* **38**, 91–97 (1991).
48. T. B. Martonen, W. Hoffman and J. E. Lowe, Cigarette smoke and lung cancer. *Health Phys.* **52**, 213–217 (1987).
49. P. H. Burri and E. R. Weibel, Funktionelle aspekte der lungenmorphologie. In *Röntgendiagnostik der Lunge*. Aktuelle Probleme der Röntgendiagnostik 2 (W. A. Fuchs and E. Vögeli, Eds.). Huber, Bern, 1973.
50. National Research Council, Committee on the Biological Effects of Ionizing Radiations, *Health Risks of Radon and Other Internally Deposited Alpha-Emitters (BEIR IV)*. National Academy Press, Washington, DC, 1988.
51. A. Cavallo, The radon equilibrium factor and comparative dosimetry in homes and mines. *Radiat. Prot. Dosim.* **92**, 295–298 (2000).
52. D. Krewski, J. Lubin, J. Samet, P. Hopke, A. C. James and K. P. Brand, Projection of residential radon lung cancer risks: The BEIR VI risk models. *Radiat. Prot. Dosim.* **102**, 371–373 (2002).
53. J. W. Marsh, A. Birchall and K. Davis, Comparative dosimetry in homes and mines: Evaluation of K-factors. In *The Natural Radiation Environment VII* (J. P. McLaughlin, E. S. Simopoulos and F. Steinhäusler, Eds.). Elsevier, Amsterdam, 2005.
54. J. C. H. Miles, Temporal variation of radon levels in houses and implications for radon measurement strategies. *Radiat. Prot. Dosim.* **93**, 369–375 (2001).
55. J. C. H. Miles, G. M. Kendall, Z-F Ibrahim and C. B. Howarth, Practical procedures for a radon etched track dosimetry service. *J. Radiol. Prot.* **24**, 165–171 (2004).
56. D. J. Steck, Spatial and temporal indoor radon variations. *Health Phys.* **62**, 351–355 (1992).
57. L. M. Hubbard, H. Mellander and G. A. Swedjemark, Studies on temporal variations of radon in Swedish single-family houses. In *Sixth International Symposium on the Natural Environment (NRE-VI)* (P. K. Hopke, Ed.). *Environ. Int.* **22** (Suppl.), (1996).
58. D. J. Steck, M. C. R. Alavanja, R. W. Field, M. A. Parkhurst, D. J. Bates and J. A. Mahaffey, ^{210}Po implanted in glass surfaces by long term exposure to indoor radon. *Health Phys.* **83**, 261–271 (2002).
59. B. Fitzgerald and P. K. Hopke, A prospective assessment of the ^{210}Po surface collection for estimating ^{222}Rn exposure. *J. Environ. Radioact.* **51**, 79–98 (2000).
60. M. C. Alavanja, J. H. Lubin, J. A. Mahaffey and R. C. Brownson, Residential radon exposure and risk of lung cancer in Missouri. *Am. J. Public Health* **89**, 1042–1048 (1999).
61. F. Lagarde, R. Falk, K. Almrén, F. Nyberg, H. Svensson and G. Pershagen, Glass-based radon-exposure assessment and lung cancer risk. *J. Expos. Anal. Environ. Epidemiol.* **12**, 344–354 (2002).
62. R. W. Field, D. J. Steck, B. J. Smith, C. P. Brus, J. S. Neuberger, E. F. Fisher, C. E. Platz, R. A. Robinson, R. F. Woolson and C. F. Lynch, Residential radon gas exposure and lung cancer: The Iowa radon lung cancer study. *Am. J. Epidemiol.* **151**, 1091–1102 (2000).