

Letter to the Editor

Sir,

Projection of Residential Radon Lung Cancer Risks: The BEIR VI Risk Models

Cavallo⁽¹⁾ suggests that the Committee on the Biological Effects of Ionizing Radiation (BEIR VI) may have invoked an inappropriate K-factor to extrapolate radon lung cancer risks from occupational to residential environments, leading to overstatement of residential radon lung cancer risk. This view arises from certain ambiguities in Appendix B to the Committee's final report⁽²⁾. These ambiguities, relating to comparative dosimetry of radon in mines and homes, have since been clarified by James *et al.*⁽³⁾. The purpose of this communication is to demonstrate that the K-factor used by the BEIR VI Committee for risk assessment purposes is based on the best available scientific data, and that the Committee's estimates of residential radon lung cancer risk are not biased upwards.

Radon-222 (hereafter denoted radon), a naturally occurring noble gas formed by the decay of uranium-238, can migrate from rocks and soils containing uranium into homes. Radon in turn decays into a series of radioactive progeny, which emit alpha particles. The International Agency for Research on Cancer⁽⁴⁾ has identified radon as a cause of human lung cancer, primarily on the basis of well documented excess lung cancer mortality in underground miners exposed to high levels of radon gas in the past (cf. Lubin *et al.*^(5,6)). Although residential exposure levels are generally much lower than those in mines, measurable levels of radon are present in most homes, prompting concerns that indoor radon might be a significant contributor to lung cancer risk in the general population.

The BEIR VI report⁽²⁾ focuses on the health risks of residential radon. The charge to the Committee included the development of risk projection models designed to provide the best possible estimates of lung cancer risk associated with exposure to radon in homes. Building on a comprehensive combined analysis of 11 cohort studies of underground miners conducted by Lubin *et al.*⁽⁵⁾, the BEIR VI Committee developed two preferred risk models. Both models take into account cumulative exposure to radon and attained age, but differ in the way they model an apparent inverse-dose-rate effect (by including either exposure duration or concentration in the model). Notably, the radon exposure measurements in the underground miner studies are

expressed in terms of working level months (WLM), the practical historical measure of exposure to potential alpha-energy of the short-lived progeny of radon. By employing this measure of progeny rather than parent (radon) exposure, the need to take into account (retrospectively) the mine-specific equilibrium fraction (F_{mines}) between radon and its progeny in the BEIR VI risk models is obviated. However, the equilibrium fraction in homes (F_{homes}) is needed to convert from measured radon concentrations (in $\text{Bq}\cdot\text{m}^{-3}$) to progeny concentrations (in WLM) when applying the BEIR VI risk models to predict residential radon lung cancer risks.

In order to use BEIR VI's mining-derived risk models to project lung cancer risks under residential exposure conditions, it is necessary to extrapolate from the relatively high levels of radon exposure experienced in mines to the lower exposures in residential settings. Other differences between occupational and environmental exposure settings, such as differences in respiratory rate (which will affect the rate at which radon and its progeny are inhaled) and particle size distributions (which will affect the fraction of radon progeny attached to particulate matter and subsequent penetration and deposition within the lung), also need to be taken into account since they can affect the amount of alpha-energy absorbed by target cells in the lungs.

Based on the data available at the time of publication of their report a decade earlier⁽⁷⁾, the BEIR IV Committee used a K-factor, which represents differences between occupational and environmental exposure conditions in lung dosimetry, of 1.0, implying that environmental exposures to radon progeny are as effective as occupational exposures with respect to deposition of alpha-energy in the lung. A subsequent review by the Committee on the Comparative Dosimetry of Radon in Mines and Homes⁽⁸⁾ reported values of K largely in the range 0.6–0.8 for normal people without respiratory illnesses, although values of up to 1.0 and higher were also noted, depending on the specific target tissue within the lung, breathing habits (nasal versus oral), and respiratory health status. Recent support for a K-factor of 1.0 is given by James *et al.*⁽³⁾, who provide an up-to-date review of the relevant data, including work by Hopke *et al.*⁽⁹⁾, as summarised in Table 1.

In his commentary on the BEIR VI report, Cavallo⁽¹⁾ concludes that the Committee failed to take into account differences in the equilibrium fractions for mines and homes when estimating residential radon lung cancer risks. The basis for this conclusion lies in the difference between the apparent definition of the K-factor in terms of radon gas concentration (K_{VI}) in the aforementioned

Appendix B⁽²⁾ and the BEIR IV Committee's definition (K_{IV}) of the K-factor in terms of exposure to potential alpha-energy (WLM). The relationship between these two K-factors is

$$K_{VI} = K_{IV} \times \frac{F_{\text{homes}}}{F_{\text{mines}}}$$

In using the BEIR VI risk models to project residential radon lung cancer risks, the BEIR VI Committee in fact used a K-factor of 1.0 with respect to alpha-energy (measured in WLM), corresponding to K_{IV} in the above equation. Cavallo⁽¹⁾ is correct in observing that the value

of K_{VI} of unity with respect to radon gas concentration that was cited without specific derivation in Appendix B to the Committee's final report⁽²⁾ is contrary to the Committee's correct working conclusion that the dose to target cells in the lung per unit exposure to potential alpha-energy from radon progeny in homes is equal to that in mines.

In order to examine the compatibility of residential radon lung cancer risk projections based on the BEIR VI risk models with direct estimates of risk based on epidemiological studies of residential radon exposure, the Committee compared such projections with estimates based on a meta-analysis of 8 residential case-control studies. Since all of these residential studies measured exposure to radon (in $\text{Bq}\cdot\text{m}^{-3}$) instead of radon progeny (in WLM), the Committee used an equilibrium fraction of $F_{\text{homes}} = 0.4$ for homes to express such exposures in WLM. An occupancy factor of 0.7 was also used in this conversion. As indicated in the final report of the BEIR VI Committee (Figure 3-2, p. 89), extrapolation of the miner data using the Committee's risk models produced estimates of residential lung cancer risk close to the direct meta-analytical estimates^(11,12). The projected risks are also in good agreement with a recently completed combined analysis of the primary raw data from 7 large-scale case-control studies completed in North America⁽¹³⁾.

The preceding discussion confirms that the BEIR VI estimates of lung cancer risk were calculated appropriately, and that lung cancer risk estimates based on occupational and residential epidemiological studies of radon appear to be compatible. Quantitative estimates of radon risks are needed not only for the establishment of exposure guidelines for radon in homes⁽¹⁴⁾, but also for estimating the risks of other sources of exposure to radon. In particular, the Committee on Risk Assessment of Exposure to Radon in Drinking Water recently made use of the BEIR VI risk models to evaluate lung cancer risks associated with radon in drinking water⁽¹⁵⁾. Since the completion of the BEIR VI report, further work on uncertainties in estimates of lung cancer risk based on the BEIR VI risk models has been conducted by Krewski *et al.*⁽¹⁶⁾ and Brand *et al.*⁽¹⁷⁾.

Table 1. Dose conversion coefficients and K-factors for typical conditions in mines and homes considered in BEIR VI^(a).

Exposure environment	Dose conversion coefficient ^(b) (mGy.WLM ⁻¹)	K-factor ^(c)
<i>No hygroscopic growth^(d)</i>		
Mine	8.7	—
Home — without cigarette smoke	8.9	1.0
Home — with cigarette smoke	7.5	0.9
<i>× 2 Hygroscopic Growth^(d)</i>		
Mine	7.4	—
Home — without cigarette smoke	8.8	1.2
Home — with cigarette smoke	6.5	0.9

(a) Adapted from James *et al.*⁽³⁾.

(b) Average dose per unit exposure to potential alpha-energy (WLM) for all target cell nuclei in the lungs (bronchial, bronchiolar, and alveolar-interstitial regions of the respiratory tract) calculated as recommended by ICRP⁽¹⁰⁾.

(c) Relative dose to target cells in the lung in homes as compared to mines per unit exposure to potential alpha energy from radon progeny.

(d) Hygroscopic growth refers to the process of particle-size growth resulting from the adsorption of water molecules from the saturated air of the respiratory tract.

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D. Krewski

McLaughlin Centre for Population Health Risk Assessment
University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

J. H. Lubin

Biostatistics Branch, National Cancer Institute
Rockville, Maryland 20852–7368, USA

J. M. Samet

Department of Epidemiology, Johns Hopkins University
Baltimore, Maryland 21205, USA

P. K. Hopke

Department of Chemical Engineering, Clarkson University
Potsdam, New York 13699–5810, USA

A. C. James

ACJ & Associates
Richland, Washington 99352–1618, USA

K. P. Brand

McLaughlin Centre for Population Health Risk Assessment
University of Ottawa, Ottawa, Ontario, Canada K1N 6N5

Response by A. Cavallo

Sir,

*Projection of Residential Radon Lung Cancer Risks:
The BEIR VI Risk Model*

As noted in the abstract of the paper referred to ⁽¹⁾, the BEIR VI Report compared dose to the lung from radon progeny to miners in **diesel-powered mines** with dose

to people in homes. The issue was raised as to whether miners in the epidemiology studies were actually exposed to this type of aerosol, which is basically oil coated soot, and it was concluded that this was emphatically not the case⁽²⁾. Most miners in the epidemiology studies worked in mines with electrical or pneumatic powered machinery, **without diesel engines**. They were not exposed to aerosols that remotely resembled those measured in the New Mexico mines; using these activity-weighted size distributions to determine the

ratio of the lung dose per unit exposure in homes to that in mines (the K factor) is useless, pointless and thoroughly misleading.

Although totally irrelevant for the computation of the K-factor, computation of dose to the lung in diesel powered mines is crucial for regulating occupational exposure. As noted in Table 2 of my paper, the activity median diameter of both mine and home aerosols is about 150 nm, but the geometric standard deviation (GSD) of the aerosol distribution in homes is 2.0 (non-smoking) and 2.5 (smoking), while the equivalent GSD in mines is 4.2. The broad distribution in mines is due to the presence of large amounts of activity on ultrafine particulates ($d_p < 20$ nm); these particles were probably generated by diesel engines. (Such size distributions were also seen in other mines⁽³⁾ with diesel engines.) Also noted were the much higher breathing rates in mines compared to that in homes. No amount of review, re-analysis or re-computation can possibly result in such totally different size distributions and breathing

rates yielding the same dose rate, as is claimed by the BEIR VI Committee.

Finally, there is one additional subtle but important point that must be acknowledged: diesel particles are oil coated soot and are not hygroscopic⁽⁴⁾.

It is clear that in working environments such as mines, where high concentrations of ultrafine particulates and radioactive gas are present, occupational exposure can be much higher than expected⁽⁵⁾. In general, maximum lung exposure levels are set based on outmoded standards that do not take account of particle size distributions; this approach must be changed.

As noted in the conclusion to my paper, additional data on aerosols in mines similar to those in the epidemiology studies are absolutely essential. Particle size distributions from **non-diesel powered mines** must be obtained before the mine-derived radon risk coefficient can be applied to homes.

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A. Cavallo
 US Department of Energy
 Environmental Measurements Laboratory
 201 Varick Street
 New York
 NY 10014, USA