Estimate of risk from environmental exposure to radon-222 and its decay products

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Consideration of the epidemiological evidence on radiogenic lung cancer in uranium miners and of the incidence of the disease generally leads to an upper estimate for the lifetime risk of 10^{-4} cases per working level month for members of the general population.

IN radiological protection, the emphasis is usually on limiting exposure to artificial radiation. Thus, there is a well-established approach to the protection of staff in hospitals from X rays and to the protection of members of the public from radioactive consumer goods or from effluent from nuclear power plants. How one should deal with exposure to natural radiation is less clear.

Human beings have always been exposed to natural radiation both from within and outside the body. The view adopted by the International Commission on Radiological Protection (ICRP) is that exposure to normal levels of natural radiation cannot usually be subject to control, but it is acknowledged that there may be unusual circumstances in which control is desirable[1]. The Commission has in mind circumstances in which there is a considerable increase in exposure as a result of human practices or choices of environment.

One of the principal ways in which members of the public receive natural irradiation is by breathing the radon-222 decay products in air. Radon gas is emitted by soil, rocks and building materials, all of which contain the parent radium to some extent. Radon can also be present in water, from which it is readily desorbed. Out of doors, the gas is dispersed in the atmosphere, but it concentrates indoors to a degree dictated primarily by the ventilation rate of the dwelling and the rate of radon input. The solid short-lived decay products of radon grow in rapidly[2] and, if attached to an aerosol rather than to walls or furniture, are inhaled and irradiate the lung tissues. Lung cancer may result.

Even in normal circumstances, human exposure to radon decay products is substantial compared with other components of the overall exposure to natural radiation. In abnormal circumstances, radon exposure may be dominant and perhaps also unacceptably high. Such abnormal circumstances stem from the use of building materials with relatively high radium content, for example, or the use of domestic water with exceptionally high radon content.

The basis of dose limitation in radiological protection is risk limitation. For example, the dose equivalent limit for workers is set so that the average risk likely to be incurred is comparable with the average prevailing for other workers in industries regarded as having high standards of safety. For members of the public, the level of acceptability of risk is probably an order of magnitude lower. It is important, therefore, to have some information on risk factors for environmental exposure to radon decay products if a system of control is to be considered.

Direct risk information does not exist for environmental levels of radon decay products. It is therefore necessary either to extrapolate from data for occupational exposure or to infer the risk from the incidence of lung cancer in the wider population. Both approaches are followed here. We have taken care neither to exaggerate the risk nor to overextend the evidence.

Uranium miner data

The only occupational data that can be used for obtaining a risk estimate for lung cancer caused by radon decay products are those from the exposure of underground uranium miners. Although the BEIR report[3] does give an estimate of the risk of lung cancer from whole-body external irradiation, this is insufficient for the present purpose. Of the four sets of data on miners, those from Sweden and Canada are considered to be less useful, and we have therefore concentrated on the data from Czechoslovakia and the United States. Information on exposure is given in terms of working level month (WLM), the unit commonly employed in mining. One WLM is defined as exposure for 1 working month of 170 h to a concentration of one working level (WL), where one WL is any combination of short-lived decay products of radon-222 per litre of air that will result in the emission of 1.3×10^5 MeV of α energy during complete decay[2,4,5].

Several factors cause the data on miners to be more difficult to use than might at first appear. The average concentrations throughout US mines in the 1940s and 1950s were high (7–15 WL) and mean exposures accumulated over the 1950s and 1960s were about 800 WLM (ref. 6); in the last decade, however, levels were lower by one to two orders of magnitude[7,8]. Most of the early information on exposure was obtained by the operators, frequently in areas where ventilation problems were known to exist: on the other hand, there was sometimes a tendency for higher exposures to be under-reported. All the measurements on aerosol characteristics, such as particle size and unattached fraction, have been made under modern mining conditions. These conditions include improved ventilation, the presence of diesel smoke (which probably overwhelms other particulates) and the virtual disappearance of the one-man mine. The evaluation of risk is also complicated by the presence of other carcinogens in mine air as well as by the miners' smoking history and their previous experience of hard-rock mining.

The estimates of lifetime risk that emerge from the data available on miners range from one estimate of 21–54 deaths from lung cancer per 10^6 WLM of collective exposure to one of 1,000 or so. Stewart and Simpson (unpublished) and Myers and

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Stewart[9] have evaluated the American and Czech data using various statistical techniques. Their work indicates that the incidence of lung cancer can be accounted for by a linear relationship with exposure, allowing a constant factor for non-radiogenic lung cancers; their estimate is 21–54 lung cancers per 10^6 WLM from the US data[10], but the Czech data imply a risk about three times as great[11]. The discrepancy is not readily explained. Stewart and Simpson also found a real or apparent threshold in some of the analyses, which suggests that the estimate of risk for low-level exposure may even include zero as a lower bound. (In such cases, Stewart and Simpson used the slopes of the response curves to derive risk factors.) Jacobi[12] also noted the discrepancy between the US and Czech data and proposed a range of 100–500 lung cancers per 10^6 WLM, which is virtually identical with an earlier estimate by UNSCEAR[13]: in neither case is a threshold effect posited. The highest estimate of risk of about 1,000 lung cancers per 10^6 WLM comes from Archer's recent proposal[14].

Whereas it is not possible completely to rule out any of these estimates of the lifetime risk from occupational exposure, our objective is to estimate risk to the general population from exposure to radon decay products in the general environment. In using the data for uranium miners to derive the lifetime probability of fatal lung cancer for members of the public, one must note that there are significant environmental and physiological differences between the two situations. These differences result from the possible contribution to lung cancer induction in mining environments of other dusts and gases, from differences in breathing rates, from differences in equilibrium ratios of the radon decay products, from high as opposed to low concentrations, from differences in smoking habits and from population distributions that differ in age and sex. Although some of these factors may tend in opposite directions, on balance, they suggest that members of the general public may be at a lower risk per unit exposure than miners. For example, breathing rates for a man resting indoors could be two to three times lower than for a man mining, with the concomitant decrease in exposure counteracted to some degree by an increase in the probability of depositing radon decay products in the lung.

We judge from the epidemiological evidence that the most defensible upper bound of the lifetime risk to the general population is 100 deaths from lung cancer for a collective exposure of 10^6 WLM, or 10^{-4} per WLM. This particular factor, although severely rounded to avoid the impression of unwarranted accuracy, can be supported by other indirect epidemiological evidence, which is detailed below, and by an analysis (W. Jacobi, personal communication) of the dosimetry of radon decay products in the manner of Jacobi and Eisfeld[15].

Lung cancer incidence

Most calculations of the exposure of the general population were initially made on the basis of outdoor radon levels. Indoor levels were known to be higher, but it is only recently that an appreciable number of measurements has become available. UNSCEAR[13] summarized the literature appearing before 1976, and more detailed measurements have since been published[16–19]. Information on living habits in the Northern Hemisphere indicates that more than 90% of the day is spent indoors, so that indoor exposure is used here to represent total exposure.

The activity concentration of radon indoors in the Northern Hemisphere lies, on the average, in the range of 0.5–1 pCi per l. If a value of 0.8 pCi l^{-1} is assumed, some measurements in the United States[16] indicate that this can be converted to a decay product concentration of ~0.004 WL. For 168 h per week of indoor occupancy, the exposure would be 0.2 WLM in 1 yr or 12 WLM in 60 yr. The latter period might be shortened somewhat, but this would not change our general conclusion.

Given this mean lifetime exposure of 12 WLM and the risk factor of 10^{-4} per WLM given above, a maximum lifetime risk of 0.12% is obtained by extrapolating linearly and by neglecting latency and accumulation interval. This is a relatively small fraction of the present lifetime risk of lung cancer (about 4% in the United States), which is largely attributable to cigarette smoking.

Making the reasonable assumption that exposure of the general population to radon decay products has been constant over several decades, one can compare the estimate of 0.12% with the incidence of lung cancer before the effects of smoking became pronounced. There are relatively good data[20] on incidence in the United States in 1930 which yield a lifetime risk of 0.1%, roughly 0.15% for males and 0.05% for females. Since not all lung cancers at that time would have been caused by radon, this indirect evidence supports the view that a value of 10^{-4} per WLM is a reasonable upper bound of risk to members of the public.

Quite independently, Cliff and others[17,18] have studied the relationship between the observed exposure to radon decay products and the observed incidence of lung cancer in British women. Taking an average exposure rate of 0.15 WLM per yr, and assuming a risk factor of 2×10^{-4} per WLM (ref. 6) as well as a 20-yr latency period, they predict a higher incidence of lung cancer than is observed from all causes in women below 40 years of age. This supports the evidence from the United States, and indeed an upper bound of 10^{-4} per WLM for the lifetime risk would not be incompatible with the British epidemiological evidence.

The overall evidence therefore points to an upper estimate for the lifetime risk of 10^{-4} cases per WLM for members of the general population.

Risk estimates compared

It is of interest to compare the risk of lung cancer from exposure to radon decay products with that from external penetrating radiation. On the basis of a linear non-threshold model, and purely for protection purposes, ICRP estimates that the risk factor for external irradiation of the lung alone is 2×10^{-3} per Sv or 2×10^{-5} per rem, where Sv and rem are the new and old units of dose equivalent, respectively[1]. Thus, the lifetime risk from an exposure to radon decay products of 1 WLM, namely 10^{-4}, would not exceed that from an external irradiation dose equivalent of 50 mSv (or 5 rem) to the lung alone.

It is difficult, however, to imagine circumstances in the general environment in which the lung alone would be exposed to external radiation: whole-body irradiation is much more likely. The ICRP takes the view that most, if not all, forms of fatal malignant disease and hereditary damage can be induced by exposure to ionizing radiation and that, when all the organs of the body are uniformly exposed, cancer of the lung in particular will account for about 10% of all harmful effects. Thus, the lifetime risk associated with an exposure to radon decay products of 1 WLM would not exceed the overall risk associated with a dose equivalent of about 5 mSv (or 0.5 rem) to the whole body from external radiation.

The annual dose equivalent limit recommended by the ICRP for individual members of the public is in fact 5 mSv.

This review has its origins in an international workshop on radiation protection principles for naturally occurring radionuclides held at Arlington, Virginia, in May 1978 by the Nuclear Energy Agency of the OECD. The authors, who were members of a group considering radon problems, have had a number of subsequent exchanges and discussions on the estimation of risk from this source. Thus the present paper should be attributed to them, even though they had the advantage of the initial group deliberations which included M. Kinoshita. The authors also acknowledge the assistance they received from M. C. O'Riordan.

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ARTICLES

Structure, strength, and polarization changes in radio source SS433

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VLA observations of SS433 at 1,465, 4,885, and 15,035 MHz between 8 September 1979 and 20 June 1980 show structure with size scales between 0.1 and 5 arc s which changes significantly on time scales of 1–2 weeks. Jets with 8–20% linear polarization are ejected on both sides of the central radio source which is coincident with the optical image, along position angles between 80° and 120° with a mean position angle for ejected material of 100°. Features in linear polarized intensity maps can be identified in four successive epochs spanning 196 days, and the resulting proper motions for identifiable clouds are 0.0088 arc s per day. If the feature velocities are equal to that of the optical emission lines, the distance to SS433 is 5.1 kpc. The position angles of identifiable features correlate well with the known precessing optical jets if one extrapolates back to the time of ejection using the observed transverse speeds. The equipartition magnetic field strengths of 0.001–0.01 G indicate synchrotron lifetimes of 10^9–10^{11} s; therefore synchrotron losses are not a dominant factor in the evolution of the extended radio emission. The evolution of radio jets indicates that the relativistic electrons and magnetic fields seen as synchrotron emission may be generated by flows along the rotation axis of the accretion disk of the SS433 star system.

DISCUSSIONS[1–4] of the peculiarities in the optical emission lines of SS433 together with various theories[5–7] have established that SS433 has oppositely directed jets of optically emitting material moving with speeds of $0.26c$. Variations in the apparent Doppler shifts of emission lines indicate an oscillation in the direction of the jets which can be interpreted as precession of an accretion disk responsible for the weak X-ray emission[8] and the high velocity flows perpendicular to the accretion disk which appear as the optically observed jets. The detection of radio emission from SS433 (refs 9, 10) has led us to question whether SS433 has radio jets analogous to those in extragalactic radio sources or double radio sources such as those associated with the strong X-ray source Sco X-1 (ref. 11). We now discuss observations that establish not only that SS433 has radio jets, but that they are linearly polarized with changes in strength, position, polarization, and polarization angle on time scales of 1–2 weeks.

Table 1 SS433 total flux densities at different frequencies and spectral indices

Date	JD − 2444000	1,465 MHz S_ν (Jy)	2,695 MHz S_ν* (Jy)	4,885 MHz S_ν (Jy)	8,085 MHz S_ν* (Jy)	15,035 MHz S_ν (Jy)	α
8 September 1979	125	0.86 ± 0.1		0.41 ± 0.05		0.23 ± 0.05	−0.61
9 September 1979	126			0.41 ± 0.05			
16 September 1979	133			0.45 ± 0.05			
21 September 1979	138	1.0 ± 0.1	0.67 ± 0.06	0.45 ± 0.05	0.33 ± 0.05	0.22 ± 0.05	−0.64
7 December 1979	215	1.75 ± 0.1	1.37 ± 0.13	0.88 ± 0.05	0.65 ± 0.06	0.40 ± 0.05	−0.76
7 March 1980	306	0.62 ± 0.1	0.59 ± 0.06	0.41 ± 0.05	0.31 ± 0.05		−0.57
5 April 1980	335	0.78 ± 0.05	0.55 ± 0.05	0.43 ± 0.05	0.38 ± 0.05	0.20 ± 0.05	−0.54
20 June 1980	411	1.00 ± 0.05	0.70 ± 0.07	0.50 ± 0.05	0.28 ± 0.05	0.20 ± 0.05	−1.0

* Green Bank interferometer data (from ref. 12).

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