#4252

EPIDEMIOLOGY

Radon as a causative factor in induction of myeloid leukaemia and other cancers

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The international incidence of myeloid leukaemia, cancer of the kidney, melanoma, and certain childhood cancers all show significant correlation with radon exposure in the home. For myeloid leukaemia, analysis suggests that in the UK 6–12% of incidence may be attributed to radon. In Cornwall, where radon levels are higher, the range is 23–43%. For the world average radon exposure of 50 Bq.m^{-3}, 13–25% of myeloid leukaemia at all ages may be caused by radon.

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Introduction

As part of a detailed investigation into the radiation dose from radon inhalation supplied to internal organs of the body other than the lung, we have looked at cancer incidence in countries where radon surveys have been carried out. We looked at acute myeloid leukaemia (AML) first because here a dosimetry model has been developed which predicts that radon could be a causative factor. The study has been extended to other cancers where radon may also be implicated—notably via radiation from its daughter nuclei, although at this stage a detailed model is not yet complete.

Marrow doses from radon

Lucie[1] has reported a correlation between indoor radon concentration and incidence of AML in the UK. Similarly, a correlation between childhood cancer and background indoor gamma-radiation has been observed.[2] It has been assumed, however, that radon exposure leads to a negligible dose contribution to red bone marrow and to the fetus. Therefore, it was assumed previously that there was no a priori reason for an association between radon exposure and leukaemia. For instance, the Black report[3] into the incidence of childhood cancer in the area around the UK's Sellafield nuclear installation paid much attention to the marrow dose received from discharge sources, amounting to about 0·2% of natural background exposure, but the alpha-radiation dose contribution from radon was not taken into account. Only later were dose contributions to red marrow from radon considered[4] but the absolute values have still been deemed to be small. Therefore any correlation between radon exposure and cancer incidence does not by itself implicate radon as an aetiological factor. Indeed, Axelson and Flodin,[5] in reply to Lucie's work, have questioned the role of radon in the incidence of leukaemia in Sweden; radon there occurs in association with background indoor gamma-radiation from building materials. Thus, implication of radon in cancer incidence requires supporting evidence by way of pathways by which it may deliver an appropriate radiation dose to sensitive tissue such as the red bone marrow.

TABLE I—DOSES TO RED MARROW AND FETUS FROM RADON AND ITS DAUGHTER PRODUCTS ASSUMING ALPHA-PARTICLE QUALITY FACTOR OF 20

Age	Annual doses (μSv) after radon exposure of:		
	20 Bq.m^{-3}	400 Bq.m^{-3}	1000 Bq.m^{-3}
Adult	80–100	2360	6920
Child, age 10	60–110	2370	6310
Fetus	15–45	990	2220

We have reported[6] calculations of radiation dose to red bone marrow and the fetus from radon and daughter products, taking into account previously unconsidered factors. These include the fact that red bone marrow contains fat cells in varying proportions depending upon skeletal location and age, and radon is 16 times more soluble in fat cells than in the surrounding marrow. The diameter of these cells is typically 100–150 μm.[7,8] Consequently,

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TABLE II—AGE-STANDARDISED INCIDENCE OF ALL MYELOID LEUKAEMIA, INCIDENCE OF ALL CHILDHOOD CANCER, MEAN RADON CONCENTRATION, AND BACKGROUND INDOOR GAMMA DOSE IN FIFTEEN COUNTRIES

Country	All myeloid leukaemia (per 100 000)	All childhood cancer (per 100 000)	Radon concentration ($Bq.m^{-3}$)	Background gamma dose (μSv/yr)
Canada	2·8	12·4	33	270
Denmark	3·25	12·7	68	450
Finland	2·9	13·5	90	690
France	..	13·1	76	680
France (Alpes Maritime)	2·4	..	34	680
West Germany	2·3	10·9	49	590
Ireland	2·35	..	68	580
Italy	3·35	14·0	55	660
Japan	2·3	10·3	10	440
Norway	3·0	12·2	90	800
Netherlands	2·6	..	31	520
Poland	2·0	9·8	9	370
Switzerland	3·1	12·6	89	950
Sweden	2·8	14·0	100	860
UK	2·35	10·8	20	490
USA	..	13·2	61	..

alpha-particle decays from radon and its short-lived daughter nuclei polonium-214 and polonium-218 within the fat cell deliver some of their energy to surrounding marrow and hence to the haemopoietic cells. Also room air contains not only pure radon but also radon daughters, and these may enter the bloodstream, leading to an additional daughter contribution to the dose to marrow as evidenced by the enhanced radioactivity found in the blood of spa workers.[9]

We have updated our calculations and the resulting doses to marrow for adults, children, and the fetus exposed to given radon levels in room air (table I). The values shown are based on a quality factor of 20 for the alpha-particle.[10] That the doses to adult and child marrow are similar is largely fortuitous. In children radon daughter deposition is at a higher rate but bone marrow fat content is lower than that in adults. At the average radon level in the UK of 20 $Bq.m^{-3}$ a dose range is given. This reflects uncertainties in the transport of radon short-lived daughters within the body. For radon concentrations of 400 and 1000 $Bq.m^{-3}$ an average dose to marrow is given. Note that the increase of marrow dose with radon exposure is non-linear owing to the ingrowth of ^{210}Po in bone.[11] These doses should be seen against an annual background dose from low LET (linear energy transfer) radiation (eg, gamma rays and cosmic rays) of about 1000 μSv. Thus, for example, we calculate that children and adults living in houses at or above the National Radiological Protection Board's (NRPB) new "action limit" of 200 $Bq.m^{-3}$ radon exposure[12] receive a radon-derived dose to marrow similar to or in excess of that from low LET radiation.

If natural radiation is a major causative factor we do have an a priori reason for supposing that radon might be associated with the incidence of leukaemia and other cancers. Lucie[1] investigated the relation between leukaemia and radon concentration within the UK. To include a large number of cancers we have looked at data from up to fifteen countries where useful radon measurements have been published.[13,21] This also helps to alleviate the effects of a shifting population within a given country, with fewer people changing residence across international boundaries.

International survey

For the radon data, the quantity required is the population-averaged arithmetic mean radon concentration. For a given country the results of national surveys have been averaged with a weighting proportional to the number of measurements. The resulting values for fifteen countries are summarised in table II. Data for regions (states and provinces) have been treated similarly, but in the UK

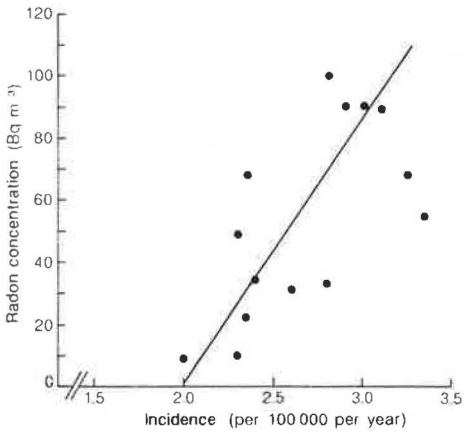

Fig 1—Radon concentration versus international incidence of myeloid leukaemia.

regional averages have been calculated from both the NRPB survey and our own,[21] by combining county values with a weighting proportional to the housing stock sway.

Cancer data have been taken from standard texts.[22-24] In several cases the geographical regions where the cancer and radon data are taken do not coincide exactly. For instance, for Poland the radon data refer to Warsaw only whereas the leukaemia data cover Warsaw, Crakov, and Nowy Sacz. Nevertheless, we have proceeded on the assumption that the population average radon concentration values represent a reasonable measure for the regions covered by the cancer incidence registries.

Fig 1 plots age-standardised incidence for myeloid leukaemia against mean radon concentration for the countries listed in table II, except the USA. The source data[22,23] include all ages and do not distinguish between acute and chronic myeloid leukaemia. The correlation coefficient (r) is 0·65 for the country data alone and 0·62 for the regional data (table III); both values are significant ($p < 0·02$). We have also looked at the correlation of disease incidence with background indoor gamma-radiation using published data.[10-18] The correlation is 0·54 ($p < 0·05$).

To confirm the correlation seen in international data, regions of Canada have been considered in detail. Here, the radon measurements were done in one survey concentrating

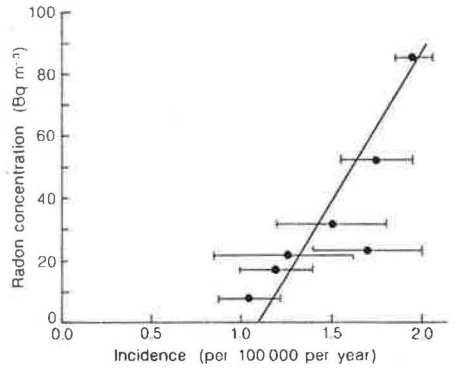

Fig 2—Radon concentration versus incidence of AML in Canadian provinces.

Incidence data was averaged over vol III and V of *Cancer in Five Continents*[17,18] and errors taken from standardised variation. The incidence for Ontario omitted as suggested in vol V. In order of increasing mean radon concentration provinces are: British Columbia, Quebec, Maritime Provinces, Alberta, Newfoundland, Saskatchewan, and Manitoba.

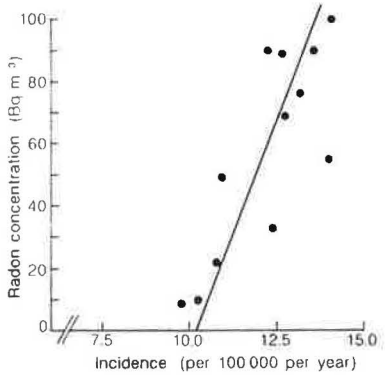

Fig 3—Radon concentration versus international incidence of all childhood cancer.

See table III for breakdown of cancer types. Radon values were taken as for country data in fig 1 and table II.

on the main cities.[20] They coincide, geographically with the source data for cancer incidence[22,23] which in this case provides age-standardised incidence for AML specifically. The data are plotted in fig 2. The correlation of radon concentration with leukaemia incidence is 0·86 ($p < 0·01$).

For childhood cancers the starting point has been the international incidence of all cancers[24] (fig 3). A significant correlation is again observed ($r = 0·78$; $p < 0·01$), and the line of linear regression is well resolved from the vertical, as shown by a Student's *t*-test. In table III we look separately at the correlation of radon with specific childhood cancers. Cancers of the brain and spine, leukaemia, osteosarcoma, and melanoma all show significant correlation with radon, and again the line of linear regression is well resolved from the vertical in all cases. Other cancers show some correlation though the values are not significant at the 95% confidence level.

There are certain cancers in adults where, although detailed dose model predictions are not yet available, radon might be expected to be a factor from irradiation by daughter nuclei. Thus, the kidney is known to act as a filter for radon daughters[9] and the correlation of 0·86 for the incidence of kidney cancer in Canada is highly significant.

Radon and daughter nuclei could be expected to condense or accumulate on the skin, leading to some irradiation of the melanocytes, or these cells could be accessed by radon and daughter nuclei within the epidermis. Therefore, incidence of melanoma might be associated with radon exposure. Here we have considered the data in Ariel's book,[25] which states that melanoma is associated with solar ultraviolet radiation. To establish a correlation with ultraviolet as a function of latitude, countries where the incidence of melanoma is anomalously high (Sweden, Norway, and parts of Canada) are excluded. For associations with radon we have considered only countries and regions in temperate climates at about the same latitude—namely, all countries in table II except Japan and Italy. For the US, the analysis is confined to northern parts where data are available (New York City, New York State, Washington State). From these restricted data there is a significant correlation of incidence with radon exposure ($0·81$; $p < 0·001$).

Finally, we looked at correlations with the incidence of cancers where radon is not expected to be a factor. For example, there is no correlation with radon for stomach cancer. Interestingly, there is also no correlation for lung cancer. Although lung cancer is associated with radon in uranium miners,[26] the incidence in the general population is dominated by the effects of smoking, only 6% being attributed to radon in the UK.[27]

Analysis

If the associations described above are indicative of true cause and effect the gradient and intercept of the best fit line relating cancer incidence to mean radon concentration can be used to estimate what proportion of disease may be attributed to radon exposure in the home. Thus fig 1 suggests that in the UK, where the population-averaged mean radon concentration is 20 $Bq.m^{-3}$, up to 12% of myeloid leukaemia is caused by radon. However, since radon occurs in association with background gamma-radiation, this figure should not be taken at face value; it represents an upper limit to the incidence attributable to radon.

Table II reveals a significant correlation between population averaged mean radon concentration and indoor gamma dose ($r = 0·78$; $p < 0·001$), mainly from the ground and building materials, such that the annual dose from terrestrial gamma-radiation increases by about 4 μSv per $Bq.m^{-3}$ of radon exposure.

To estimate the percentage of leukaemia incidence attributable to radon exposure, we need to know the quality

TABLE III—CORRELATION COEFFICIENTS FOR CANCER INCIDENCE AND RADON AND BACKGROUND GAMMA-RADIATION EXPOSURE

Data set	Cancer	Correlation coefficient (r)		Upper limit to % incidence attributable to radon at:	
		Radon	Indoor gamma	20 $Bq.m^{-3}$	50 $Bq.m^{-3}$
All ages					
14 countries	Myeloid leukaemia	0·65 ($p < 0·02$)	0·54 ($p < 0·05$)	12	25
Regions within countries	Myeloid leukaemia	0·62 ($p < 0·01$)	..	9	19
Canada	AML	0·86 ($p < 0·01$)	..	14	30
Canada	Kidney	0·86 ($p < 0·01$)	..	11	24
14 countries + regions	Melanoma	0·81 ($p < 0·001$)	..	72	86
Childhood cancers, 13 countries					
	All cancer	0·78 ($p < 0·01$)	0·44 (NS)	6	15
	Leukaemias	0·61 ($p < 0·02$)	0·58 ($p < 0·05$)	5	11
	Brain and spinal	0·62 ($p < 0·02$)	0·33 (NS)	11	23
	Osteosarcoma	0·56 ($p < 0·05$)	0·61 ($p < 0·03$)	11	23
	Melanoma	0·56 ($p < 0·05$)	0·27 (NS)	29	50
	Wilms' tumour	0·47 ($p = 0·1$)	0·36 (NS)	8	18
	Soft tissue sarcomas	0·46 ($p = 0·1$)	0·57 ($p < 0·05$)	9	20
	Neuroblastoma	0·43 (NS)	0·32 (NS)	7	15

NS = not significant.

factor of the alpha-particle with respect to gamma-radiation. The graphs of leukaemia incidence versus radon exposure shown in figs 1 and 2 contain sufficient information to deduce either the quality factor of the alpha-particle or the risk factor for leukaemia induction, provided one of these quantities is already known. Although estimates for both of these quantities are available, neither are directly applicable to this situation. The lifetime risk factor is derived from an acute gamma and neutron dose to Japanese bomb victims rather than a chronic low-level radon exposure. The quality factor has been derived from higher level radon exposure in relation to lung cancer in miners and in animal and in-vitro studies in relation to bone marrow.[29] Therefore, we can only proceed by assuming that the currently accepted value for either the risk factor or the quality factor, but not both, is correct. These possibilities are evaluated below.

Radon is causative and the risk for leukaemia induction derived from Japanese bomb data is correct. The lifetime risk factor may be written as (average incidence per year) × (average life expectancy) ÷ (total dose). From the Japanese bomb data, the lifetime risk factor for leukaemia induction by radiation from gamma rays and neutrons is given[4] as $2{\cdot}8 \times 10^{-3}\text{Sv}^{-1}$. We may therefore write $2{\cdot}8 \times 10^{-3}\text{Sv}^{-1}$ as (annual incidence) ÷ (annual dose), both numerator and denominator being per Bq.m^{-3} of radon exposure. The slope of fig 1 gives the annual incidence due to radon as $1{\cdot}25 \times 10^{-7}$ per Bq.m^{-3} giving the annual dose to bone marrow as 45 μSv per Bq.m^{-3}. Since the terrestrial gamma dose to marrow accounts for 4 μSv, radon itself must account for 41 μSv. The dose values in table I suggest the need to increase the alpha-particle quality factor to 180. (Alternatively if an alpha-particle quality factor of 20 is correct the risk factor for adults from Japanese bomb data will be too low by a factor of 9.)

Radon is causative and dose values in table I apply with alpha-particle quality factor of 20. This would mean that the slope in fig 1, and taking into account the contribution from associated gamma-radiation, implies that in the UK 6% of leukaemia is caused by radon. Taking this further, we can add all contributions to the radiation dose received by red bone marrow and extrapolate the line in fig 1 to true zero dose and hence determine the proportion of all leukaemia in the general population caused by radiation. Accordingly, we have added to the radon dose the contribution from background gamma radiation, sea-level cosmic rays, body skeletal burden of natural potassium-40, and burden from medical X-rays. Of these the most difficult to obtain is the "contribution" from medical exposure so this analysis can only be approximate. Nevertheless, extrapolation of the data to zero dose implies that essentially 100% of leukaemia is caused by radiation.

Radon is causative; dose values in table I apply but alpha-particle quality factor is around 50. The simplest way to avoid the prediction that 100% of leukaemia is attributed to radiation exposure is to argue that the alpha-particle quality factor is much more than 20. This has the effect of preferentially increasing the marrow dose in high radon areas which in turn rotates the dose-incidence best fit line anticlockwise with respect to the best fit line in fig 1. A quality factor of around 50 is required to ensure that no more than 60% of leukaemia is caused by radiation in the UK.

These possibilities also follow from analysis of the data in fig 2 for Canada where radon measurements were carried out by one laboratory in areas which coincide with those covered by the cancer registries. In general terms as the alpha-particle quality factor is increased the proportion of leukaemia in the general population attributable to radiation is reduced but the effects of radon exposure itself are increased. At a quality factor of 180 around 30% of leukaemia would be attributed to radiation in the UK with 40% of this fraction due to radon.

The power of the above analysis does not depend on the accuracy of the doses in table I. If the doses are overestimated then the alpha-particle quality factor would need to be still higher than the options above; if they are underestimated then we must seek yet further factors in calculating radon dose to red bone marrow than those considered so far.

The analysis shows that the alpha-particle quality factor is likely to be greater than 20 with a probable upper limit of 180. Although this range is wide it is difficult to suggest a particular value since this would be somewhat arbitrary. It does seem likely, however, that a value much greater than 20 is more appropriate for exposure to alpha-radiation at natural levels. This increases the importance of radon to the radiation dose received by bone marrow. For the UK, if the observed correlations are indicative of cause and effect, the proportion of leukaemia attributable to radon exposure might be in the range 6–12%. In Cornwall, on the other hand, where the mean radon concentration is 110 Bq.m^{-3} the range would be 23–43% and for the world average radon exposure of 50 Bq.m^{-3}, 13–25% of myeloid leukaemia at all ages might be caused by radon. In all cases the higher percentages represent upper limits.

For the significant correlations of radon exposure with other cancers in table III, the absence of precise radon dose values to appropriate organs and tissues means that we cannot separate the contribution from radon and background gamma-radiation. Although these further associations are not explicitly plotted here we can express their slope and intercept in terms of upper limits of the proportion of incidence due to radon exposure. Table III lists these percentages for radon exposures of 20 and 50 Bq.m^{-3} appropriate to the UK and world-wide average radon exposure respectively. Cancers for which the correlation with radon is not significant have been included for completeness. For several cancers around 10% of incidence could be attributed to radon at 20 Bq.m^{-3}, but for melanoma the figure is much higher with up to 72% potentially caused by radon in the UK.

Discussion

Even though we have had to assume that national mean radon concentrations apply to areas where cancer registries have been compiled and acknowledge that the incidence from different countries may not be comparable, we regard the significant correlations of radon exposure with cancer incidence as of special interest. The correlations are even more compelling when account is taken of the data for Canada where the regions covered by the radon surveys and cancer registries properly coincide. For childhood cancers a further factor is the extent to which these are initiated in utero. This addresses directly the extent to which radon reaches the fetus. The observations in general clearly warrant urgent and detailed further investigation to determine whether radon is indeed a causative factor as we hypothesise here. To determine the extent to which radon reduction measures in homes are likely to reduce incidence of these diseases, it will be essential to separate the radon contribution from that of indoor gamma-radiation as we have attempted. In table III we have included, where possible, the correlation coefficients for gamma exposure. The values are generally lower than those for radon exposure. This could be indicative of poorer data on population averaged gamma measurements in the home. On the other hand, the fact that in many cases the correlation for radon is significantly better than that for gamma-radiation, further supports the hypothesis that radon is a causative factor.

It is interesting to note that leukaemia incidence has not been associated with uranium miners exposed to radon,

where extensive data have been gathered on the dominant effect—namely, lung cancer.[26] This, however, may be attributable to the widely different conditions in uranium mines compared with those in the home. Whereas in the case of the lung the dose is dominated by inhalation of radon daughters, radon dose to bone marrow is dominated by the presence of inhaled radon within fat cells in marrow. We estimate that the approximate ratio of radon-related lung cancer to leukaemia in the general population is around 10:1. The lung deposition rate of radon daughters in uranium miners is about five times higher than that for equivalent domestic radon exposure owing to the higher respiratory minute volume of the miners.[8,24] Also, the mine atmosphere typically has an excess of radon daughters leading to an enhancement of lung dose of a further factor of three. Thus we suggest that the ratio of lung to marrow dose in uranium miners could be as much as 15 times higher than that for domestic exposure. Therefore the expected ratio of radon-induced lung cancer to leukaemia in miners could be 150:1.

The sum total of the thirteen representative surveys listed in table IV-16 of the BEIR IV report[26] yields only 548 excess lung cancers. Hence an excess of only 3·6 cases of leukaemia might be expected across all studies. These could easily be missed. Radford[29] has reported an excess of myeloma in Swedish iron miners exposed to a radon atmosphere of 4·8 $WLM.y^{-1}$. This has appeared as a very late effect, being beyond the duration of most currently reported surveys.

This argument does not apply to melanoma and, indeed, cases have been seen in uranium miners, although the incidence has not firmly been attributed to radon.[26]

Finally, the fact that radon hot spots are known to exist may have implications for clustering of childhood cancer in the UK. An increased quality factor for the alpha-particle would increase the likelihood of leukaemia clustering in high radon areas which overlap large areas of population. However, the converse may not be true and not all leukaemia clusters need be explainable in terms of radon exposure.

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From The Lancet

The tax upon imported leeches

SIR:—Permit me to call attention to the propriety of removing the duty on the importation of leeches into this country. Their present high price, as compared with that of other medicinal agents, mainly proceeds from this cause, and, coupled with the mortality that is prevalent amongst them at certain seasons of the year, prevents our reaping the full benefit of so valuable a remedial means. Surgeons of Poor-Law Unions, owing to their low rate of remuneration, and those who practise among the poorer members of the community, are either often compelled to dispense with the use of leeches, when it would be beneficial, or supply them, at a sacrifice, from conscientious motives. It is important, then, that a remedial agent of such general use and value, should be available at as low a rate as possible. . . and Parliament (should be) petitioned for the removal of the impost.

A General Practitioner

**This tax, if it do not make us angry, must make one smile. But, doubtless, it found advocates among the friends of a tax upon wheat,—the supporters of a "home growth,"—and was inflicted, as regarded leeches as well as corn, in order to render us "independent of nations with which we might be at war."

(23 May 1840)