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Review

Radon: A likely carcinogen at all exposures

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Summary

Background: Radon is a well-established lung carcinogen that has been extensively studied. Very high concentrations can occur in some underground mines. Concentrations also tend to build up in homes.

Materials and methods: Epidemiological studies of radon-exposed miners and of residential radon and lung cancer are reviewed. Quantitative estimates of the risk of lung cancer, based on the experience of the miners, are applied to residential radon exposures in the United Kingdom. Strategies for the prevention of lung cancer induced by residential radon are discussed.

Results: Estimates are uncertain, but residential radon is probably responsible for about 2000 lung cancer deaths per year in the United Kingdom, or around 6% of the total, making it the second biggest cause after smoking. Over 80% of the deaths are estimated to occur at ages less than 75 and over 80% in smokers or ex-smokers. Around 90% of radon-induced deaths in the United Kingdom probably occur as a result of exposures to radon concentrations below the currently recommended action level of 200 Bq m^{-3}.

Conclusions: Further work is needed to obtain more reliable estimates of the risk of lung cancer associated with residential radon and on the cost-effectiveness of various intervention strategies before the most appropriate policies can be developed for managing exposure to this natural carcinogen.

Key words: lung cancer, prevention, radon, risk assessment

Introduction

Radon-222 is a chemically inert radioactive gas with a half-life of 3.8 days, giving rise to a series of short-lived progeny (Figure 1). It arises from the decay chain of uranium-238 ,which is present throughout the earth's crust, and seeps out of rocks and soil. If radon itself is inhaled, some will be absorbed through the lung, but the majority will be exhaled. However, the progeny are solid and form into small molecular clusters or attach to aerosols in the air and these may be deposited on the bronchial epithelium. Two of the short-lived progeny in the commonest decay chain, polonium-218 and polonium-214, decay by emitting alpha particles. These have a limited range of penetration into tissues but are highly effective at causing genetic damage in the cells they reach.

Radon concentrations in outdoor air are usually very low, but concentrations build up in situations where it is unable to disperse. Some of the highest concentrations occur in underground mines of igneous rocks, especially uranium mines, where it may enter the air directly from the ore, or be brought into the mine dissolved in water. However, appreciable concentrations may also occur in homes, where the principal source is usually the subsoil, although under some circumstances appreciable exposure may occur from building materials or from radon dissolved in water. Residential radon levels are very variable, depending on local conditions and in many countries there is a variation of two orders of magnitude or more in the concentrations commonly observed. In the great majority of countries radon is the principal source of exposure of the general population to ionizing radiation [2], and substantial efforts are being directed towards estimating its effect in the general population. Residential radon concentrations can usually be reduced through relatively simple measures, and several countries now have recommended control strategies.

The BEIR VI Committee published a comprehensive report on the health effects of radon in 1999 [1]. Its effects have been reviewed twice by the International Agency for Research on Cancer [3, 4], which led to its classification as a human carcinogen (group 1), and other reviews have also been published recently [5]. The present article draws on these reports and summarizes the quantitative evidence relating to lung cancer in radon-exposed miners and recent work studying directly the risk of residential radon. It also describes the implications of the risks seen in radon-exposed miners for the general population of the UK and describes current strategies for the prevention of radon-induced lung cancer.

Lung cancer in radon-exposed miners

It was appreciated as early as the 1500s that metal miners in the Erz mountains in central Europe had a very high mortality rate from respiratory disease. How-

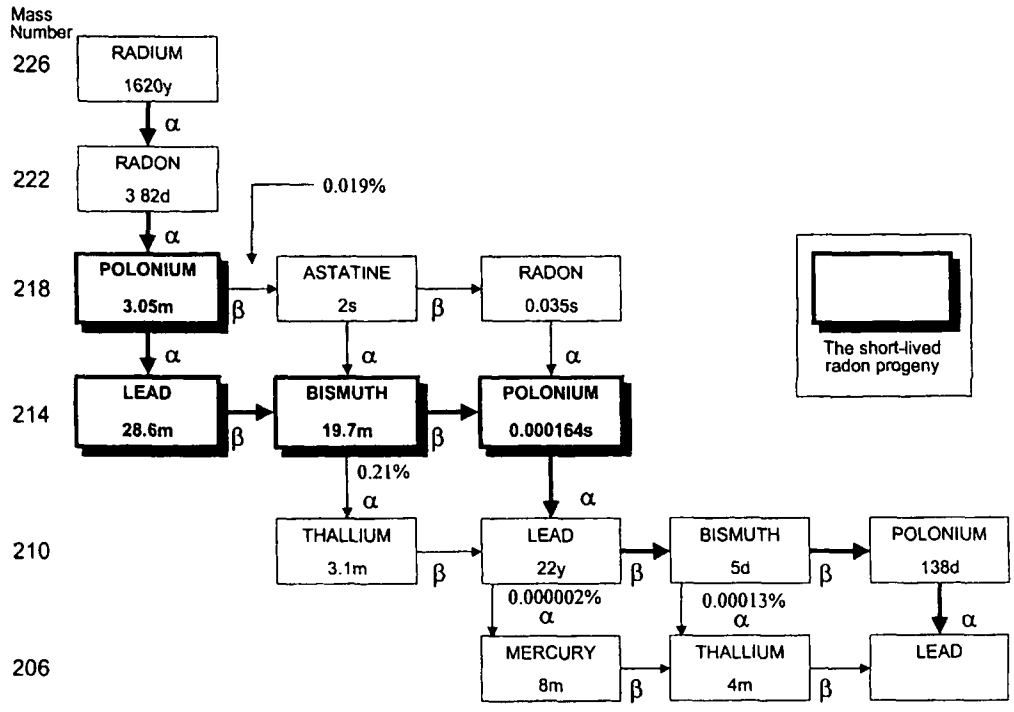

Figure 1 The radon decay chain. An arrow pointing downward indicates decay by alpha-particle emission; an arrow pointing to the right indicates decay by beta-particle emission. Most decay takes place along the chain marked with thick arrows. The small percentage of decay along the chains marked with thin arrows is shown at critical points. The end of the chain, lead-206 is stable, not radioactive. Half-lives of each isotope are shown as seconds (s), minutes (m), days (d) or years (y). Based on [1].

ever, it was not until early in the 20th century that the disease was established as being lung cancer and not until the 1920s, when high levels of radon were identified in these mines, that radon was first postulated as the cause. Support for this postulate was by no means universal, however, and alternative causal theories were also popular, including the effects of dust exposure and metals in the ore, and an increased susceptibility resulting from inbreeding in the small mining communities. It was only in the 1950s and 1960s, when studies of igneous rock miners exposed to high radon levels in other areas also revealed unusually high lung cancer rates, that radon was generally accepted as the cause. Since that time, ventilation and other measures have been used to reduce radon levels in most affected mines that continue to operate. This has reduced the risk of occupationally induced cancer from radon, although it still remains an issue both for those who are currently employed in affected mines and for those who have been employed in the past.

Studies have been carried out of the mortality patterns of several groups of radon-exposed miners and these form the major body of evidence to date concerning the consequences of exposure to radon and its decay products. At present, results have been published of eleven major studies, covering a total of over 60,000 miners in Europe, north America, Asia and Australia, among whom over 2500 deaths from lung cancer have occurred. These studies all include quantitative information on the radon exposures received by the men and many of them also include information on unexposed workers, e.g. surface workers, as an internal comparison group. Eight of the studies are of uranium miners; the remainder are of miners of tin, fluorspar or iron.

In each of the eleven studies, there was a close correlation between radon exposure and lung cancer risk, with miners who had higher exposures experiencing a greater increase in risk than those who had lower exposures, and in every study the relationship was so strong that it was unlikely to be due to chance (Table 1). In contrast to the findings for lung cancer, there is little evidence of any association between radon exposure and mortality from other cancers [6] or other diseases [1] in the same miners.

Although the size of the radon-related increase in lung cancer risk varied by more than an order of magnitude between the different studies, analysis of the information in the individual studies revealed some clear systematic trends in risk. The relative risk of lung cancer (i.e. the proportionate increase in the age-specific risk of lung cancer) rose linearly with increasing cumulative exposure, both overall and in the region < 600 WLM, which is of greatest interest when considering the effects of residential exposures (Figure 2). After allowing for a minimum latent period of around five years between exposure and death, the percentage increase in risk was higher in the period around 10 years after exposure than at 20 or 30 years after exposure. In addition, the percentage increase in lung cancer risk was also greater in individuals who were aged around 50 than in individuals who were aged around 60 or 70. Finally, mines where the radon concentrations were relatively low had a larger

Table 1. Lung cancer mortality in cohort studies of underground miners occupationally exposed to radon.

Study and reference	Type of mine	Number of exposed miners	Mean total WLM	Mean duration of exposure (years)	Number of lung cancer deaths	Percentage increase in age-specific risk of lung cancer per WLM[a]	95% confidence interval
Yunnan, China [7]	Tin	13,649	286.0	12.9	936	0.16	0.1–0.2
W. Bohemia, Czech Republic [8]	Uranium	4,320	196.8	6.7	701	0.34	0.2–0.6
Colorado, USA[b] [9]	Uranium	3,347	578 6	3.9	334	0.42	0.3–0.7
Ontario, Canada[c] [10]	Uranium	21,346	31.0	3.0	285	0.89	0.5–1 5
Newfoundland, Canada [11]	Fluorspar	1,751	388.4	4.8	112	0.76	0.4–1.3
Malmberget, Sweden [12]	Iron	1,294	80.6	18.2	79	0.95	0.1–4.1
New Mexico, USA [13]	Uranium	3,457	110.9	5.6	68	1.72	0.6–6.7
Beaverlodge, Canada [14]	Uranium	6,895	21.2	1.7	56	2.21	0.9–5.6
France [15]	Uranium	1,769	59.4	7.2	45	0.36	0.0–1.2
Port Radium, Canada [16]	Uranium	1,420	243.0	1.2	39	0.19	0.1–0.6
Radium Hill, Australia [17]	Uranium	1,457	7.6	1.1	31	5.06	1.0–12.2
Total[d]		60,606	164.4	5.7	2,674		

[a] The working level (WL) is defined as any combination of the short-lived radon progeny in one litre of air that results in the ultimate release of 1.3×10^5 MeV of potential α-particle energy. Exposure to this concentration for 170 h (or twice this concentration for half as long, etc.) is defined as a working level month (WLM). An individual living in a house with a radon concentration of 20 Bq m^{-3} will be exposed to around 0.08 WLM per year.

[b] Totals given exclude data above 3200 WLM

[c] Values given include all uranium miners, including those with previous gold mining experience.

[d] Totals adjusted for miners and lung cancers included in both Colorado and New Mexico studies.

Source: BEIR VI Committee [1].

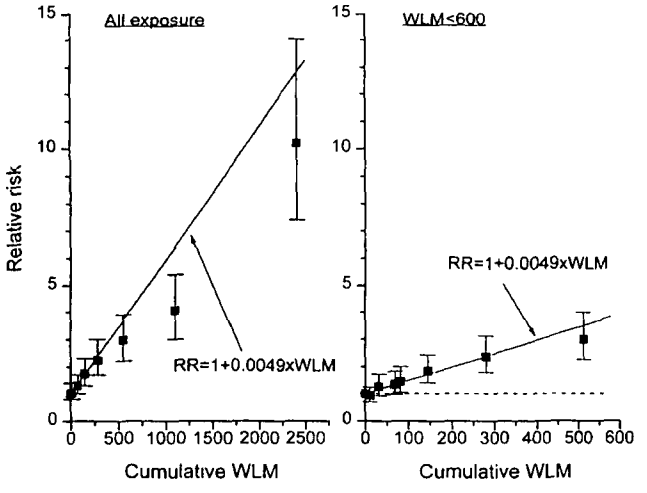

Figure 2. Relative risk (RR) of lung cancer with cumulative radon exposure in the cohort studies of underground miners occupationally exposed to radon (based on [18]).

percentage increase in risk per unit exposure than mines with higher radon concentrations or, equivalently, a given total exposure was associated with a greater increase in risk if it was received over a longer rather than a shorter time period.

In view of the variations in the risk per unit exposure with time since exposure, age, and radon concentration described above, it is difficult to combine the results of the different studies appropriately using just the information published by the individual studies. To overcome this difficulty, the individual data from the eleven studies were collated centrally, and a combined analysis carried out by the BEIR VI Committee [1]. After extensive technical investigations the preferred form of the model relating radon exposure to risk of death from lung cancer was:

$$R = 100\,\beta\, w^*\, \varphi_{age}\gamma_z, \qquad (1)$$

where R is the percentage increase in the risk of death from lung cancer for a person of a certain age with a given history of exposure to radon; β is the parameter relating lung cancer risk to history of radon exposure; w^* represents the radon exposure and takes the form of a weighted average, $w^* = (w_{5-14} + \theta_{15-24} w_{15-24} + \theta_{25+} w_{25+})$, with w_{5-14}, w_{15-24}, and w_{25+} representing the exposure incurred during the periods 5–14, 15–24, and 25+ years prior to the current age. The coefficient of w_{5-14} is equal to one, while θ_{15-24} and θ_{25+} represent the contributions to risk from exposures received 15–24 years and 25+ years previously, compared to exposures received in the period 5–14 years previously. The parameter φ_{age} represents the modifying effect of age, while the parameter γ_z represents the modifying effect of either radon concentration or of exposure duration. Estimates of the parameters β, θ_{15-24}, θ_{25+}, φ_{age}, and γ_z for both the exposure-age-duration and the exposure-age-concentration formulation of the model are given in Table 2.

In most of the miner populations that have been studied the majority of the men would have been cigarette smokers and, in most cases, this will have had an effect on their lung cancer risk that is even greater than

Table 2. Parameter estimates from BEIR VI [1] preferred models in combined analysis of eleven studies of underground miners occupationally exposed to radon. See text for description of models.

Exposure-age-duration model[a]		Exposure-age-concentration model	
$\beta \times 100^{b,c}$	0.55[d]	$\beta \times 100^{b,c}$	7.68[d]
Time-since-exposure windows		Time since-exposure windows	
θ_{5-14}	1 00	θ_{5-14}	1.00
θ_{15-24}	0.72	θ_{15-24}	0.78
θ_{25+}	0 44	θ_{25+}	0.51
Attained age (years)		Attained age (years)	
$\phi_{<55}$	1.00	$\phi_{<55}$	1 00
$\phi_{55\ 64}$	0.52	ϕ_{55-64}	0.57
$\phi_{65\ 74}$	0 28	ϕ_{65-74}	0.29
ϕ_{75+}	0 13	ϕ_{75+}	0.09
Duration of exposure (years)		Concentration of exposure (WL[c])	
$\gamma_{<5}$	1 00	$\gamma_{<0\ 5}$	1 00
γ_{5-14}	2.78	$\gamma_{0\ 5\ 1\ 0}$	0.49
$\gamma_{15\ 24}$	4 42	$\gamma_{1\ 0-3.0}$	0 37
$\gamma_{25\ 34}$	6.62	$\gamma_{3\ 0-5.0}$	0.32
γ_{35+}	10 2	$\gamma_{5\ 0-15\ 0}$	0 17
		$\gamma_{15\ 0+}$	0.11

[a] Risk projections for the US were carried out by the BEIR VI Committee for both models and shown to give very similar results. In the present paper projections based on the exposure-age-concentration model are given

[b] Units are WLM^{-1}

[c] See Table 1 for definition of WLM and WL.

[d] When separate estimates are required for smokers and non-smokers these are 0.50 for ever-smokers and 1.1 for never smokers in the exposure-age-duration model, and 6.90 for ever smokers and 15.3 for never-smokers in the exposure-age-concentration model.

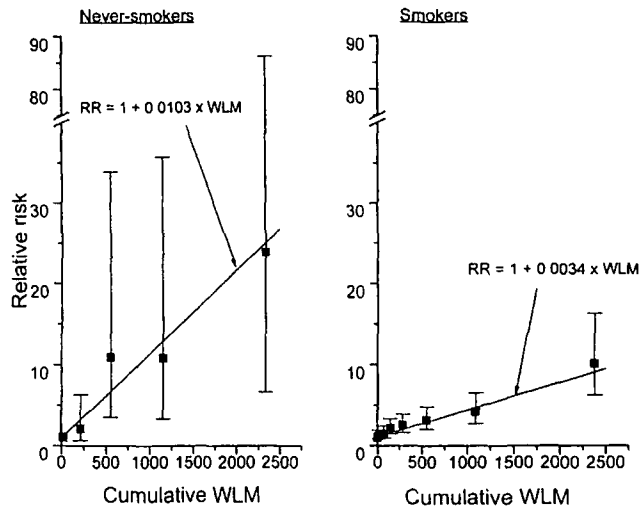

Figure 3. Relative risk (RR) of lung cancer with cumulative radon exposure among lifelong non-smokers and others in the six cohort studies of underground miners for which smoking information was available (based on [18]). Although the increase in relative risk per unit exposure is higher for never smokers than for smokers, the increase in absolute risk will be higher for smokers, as they have much higher rates of lung cancer.

their radon exposure. The effect of radon exposure can be expected to differ between smokers and non-smokers, depending on the way in which the risks of smoking and radon exposure act jointly. Unfortunately, no smoking information is available for five of the miners' studies. Among the six studies for which some smoking information is available, 2798 lifelong non-smokers could be identified, who between them had experienced 64 lung cancers. This was not enough to carry out a full analysis, but the relationship between the relative risk of lung cancer and cumulative radon exposure could be compared in lifelong non-smokers and others. In both groups the relationship between the relative risk of lung cancer and cumulative radon exposure was approximately linear, but the *relative risk* per unit exposure was considerably greater among the lifelong non-smokers than among the current and ex-smokers (Figure 3). To allow for this, a higher value of β was recommended in Equation (1) above for non-smokers and a slightly lower value for smokers, when separate estimates were required for lifelong non-smokers and current or previous smokers (see Table 2 for parameter values).

Lung cancer from residential radon

Average radon exposures received by the miners were an order of magnitude or so greater than average indoor exposures and the average duration of exposure in the miners studies was less than six years (Table 1). The miners were almost all adult males, the information about both their radon exposure and their smoking habits is crude and subject to error, and their conditions of exposure differ substantially from those in homes, with the miners carrying out substantial amounts of heavy work in an atmosphere polluted by dust and fumes. There is, therefore, great uncertainty in applying risk estimates derived from studies in miners to the effects of residential radon, and direct estimates of the risks of residential radon are needed. At the present time, 14 major studies of residential radon have been published (Table 3). These have taken the form of case-control studies in which detailed residential and smoking histories have been gathered for a series of individuals with lung cancer and a series of control subjects who had not developed the disease. The radon concentration has then been measured over a period of several months in the air of the subject's present and previous homes using passive alpha track detectors and the subject's weighted average radon concentration during an appropriate time period has been calculated. To date, most of the studies have had inadequate power to detect a risk on their own, although a weighted average of the published results is indicative of a risk (estimated relative risk at 100 Bq m^{-3} compared with 0 Bq m^{-3} 1.06, 95% confidence interval (95% CI): 1.01–1.10, see Table 3).

Most of the estimates shown in the main body of Table 3 have been obtained using standard statistical methodology for case-control studies in which it is assumed that the average radon concentration to which an individual has been exposed can be assessed without error. However, this assumption is usually violated in two different ways. Firstly, in most of the studies there are

Table 3. Estimates of relative risk (RR) at 100 Bq m^{-3} compared with 0 Bq m^{-3} and 95% confidence intervals (CI) in epidemiological studies of residential radon and lung cancer based on at least 100 cases of lung cancer and direct measurements of radon using α-track monitors.[a]

Study and reference	Number of cases of lung cancer	Number of control subjects	RR	95% CI
New Jersey, USA [19]	480	442	1 49	(0.89–1.89)
Shenyang, China [20]	308	356	0.95	(undefined-1.08)
Stockholm, Sweden [21]	201	378	1.16	(0.89–1 92)
Swedish nationwide [22, 23]	1281	2576	1.10	(1.01–1.22)
			1.17[b]	(1.03–1.37)
Winnipeg, Canada [24]	738	738	0.98	(0.87–1.27)
Missouri, USA I [25]	538	1183	1.08	(0.95–1.24)
South Finland [26]	164	331	1.80	(0.90–3.50)
Finnish nationwide [27]	517	517	1.11	(0.94–1.31)
South-West England [28]	982	3185	1 08	(0.97–1 20)
			1.12[c]	(0 95–1.33)
Missouri, USA II [29]	247[d]	299[d]	0.85[d]	(0 73–1.00)[d]
	372[e]	471[e]	1 63[e]	(1.07–2.93)[e]
Iowa, USA [30]	413	614	1.24	(0.95–1.92)
Western Germany [31]	1449	2297	0 97[f]	(0.82–1.14)
			1.09[g]	(0.86–1.38)
Eastern Germany [32]	1053	1667	1.11[f]	(1.00–1.27)
			1.27[g]	(1.00–1.60)
Swedish never-smokers [33]	258	487	1.28	(0.95–2.05)
Total[h]			1 06	(1.01–1.10)

[a] Based on [4] with additions.
[b] Assuming 50% coefficient of variation in measured radon concentrations.
[c] Assuming 50% coefficient of variation in measured radon concentrations and allowing for uncertainties in estimates of missing values.
[d] Analysis based on air monitors
[e] Analysis based on surface monitors
[f] Entire study.
[g] High radon areas.
[h] Total is weighted average of all studies. Where two estimates are given for a study the first entry has been used

some time-periods for which it is not possible to obtain a radon measurement, for example, because the home previously occupied by the subject had been demolished. Radon concentrations for such missing periods need to be estimated in the analysis. Secondly, even where it has been possible to obtain a measurement, the measured value will be subject to uncertainty in the sense that repeated measurements in the same home vary, with a coefficient of variation of around 50% [23, 28]. These two different sources of uncertainty will have different effects on the results of an analysis that has been carried out using standard techniques [34]. Missing values that have been replaced by estimates will cause confidence intervals to be wider than they would otherwise have been, and are undoubtedly a contributing factor in the low power of the case-control studies in shown in Table 3. In contrast, the presence of uncertainty in measured residential radon concentrations will cause the effect of the radon, as estimated using standard techniques of analysis to be biased towards zero. For two of the case-control studies shown in Table 3, analyses have been carried out that take this bias into account [23, 28]. For these studies, the estimated relative risks of lung cancer at 100 Bq m^{-3} compared with 0 Bq m^{-3} using the standard methods were 1.10 (95% CI: 1.01, 1.22) and 1.08 (0.97, 1.20), while the estimates taking account of measurement uncertainty were somewhat higher, at 1.17 (1.03, 1.37) and 1.12 (0.95, 1.33).

Rather than measuring the current concentration of radon in the air of all the homes of interest, an alternative method of assessing residential radon histories is to estimate an individual's cumulative radon exposure. This can be done by estimating the accumulation of the long-lived radon decay product Pb-210 implanted in the glass surface of an object, such as a picture frame, that has been on display in all the subject's homes for a substantial period of time. The long-lived Pb-210 in turn gives risk to a shorter lived product, Po-210, which can be measured using passive alpha track detectors. From this measurement an estimate can be made of the cumulative radon exposure in the rooms where the glass object has been kept. The uncertainties associated with this method of estimating radon histories have not yet been fully documented, but it should avoid the difficulties caused by missing measurements in subjects' previous home and also the problem that in some countries residential radon concentrations may have changed systematically over time, for example because of a tendency to reduce indoor ventilation rates [35], which would mean that air concentrations measured at the present

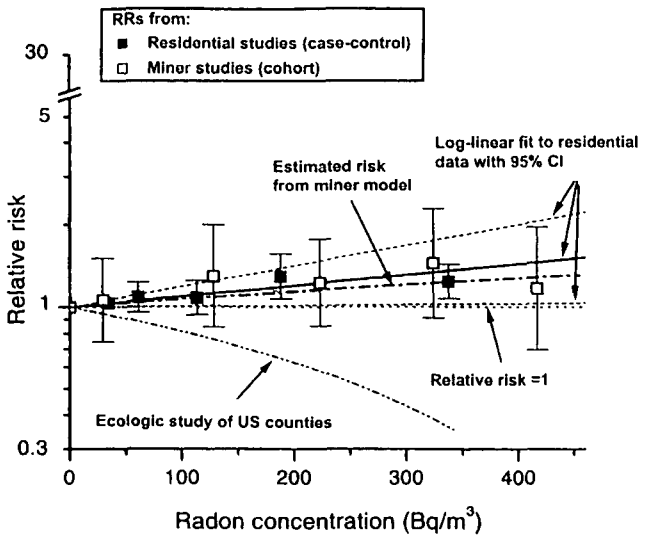

Figure 4. Summary relative risks (RR) from meta-analysis of published data from case-control studies of residential radon [36], pooled analysis of cohort studies of miners occupationally exposed to radon [18], and an ecologic study of 1601 US counties [37]: the negative slope in the ecologic study is approximately 10 times its standard error (figure based on [1]).

time would be systematically biased compared with previous values. To date only one study has published a risk estimate based on cumulative exposure histories from surface monitors [29]: this gave an estimated relative risk at 100 Bq m^{-3} compared with 0 Bq m^{-3} of 1.63 (95% CI: 1.07, 2.93), while the same study failed to show any positive relation using conventional air monitors, with estimated relative risk at 100 Bq m^{-3} compared with 0 Bq m^{-3} 0.85 (0.73, 1.00).

Despite the uncertainties that affect the results of the case-control studies of residential radon, the risks suggested by them are in good agreement with the estimated risks based on the studies of miners (Figure 4). However, public perception of residential radon has been confused, especially in the United States, by the wide publicity given to an ecological study in which average residential radon concentrations in several hundred US counties have been correlated with the county-specific lung cancer incidence rates after adjusting at the county level for various other factors that might affect lung cancer risk, including smoking habits [37]. The results of the ecologic study show a negative association at the county level between residential radon exposure and lung cancer risk, and are clearly out of line with the results of both the miners studies and the case-control studies of residential radon. There is ample evidence to suggest that the results of the ecological study are incorrect, and it can be shown theoretically that adjusting for smoking at the county level will not remove the effect of this factor, which is responsible for the vast majority of cases of lung cancer [38]. For further discussion of this topic see [1, 4, 39, 40].

Implications for the UK of risks seen in radon-exposed miners

Despite the uncertainties in extrapolating from the experience of radon-exposed miners to the effects of residential radon, the most appropriate approach at present to estimating the likely numbers of deaths caused by radon, both alone and in conjunction with smoking, is from the BEIR VI Committee's preferred model. In order to understand the implications of the BEIR VI model for the United Kingdom, it has been combined with UK data on smoking habits, population size, numbers of deaths from lung cancer for males and females in different age groups, and lung cancer rates among lifelong non-smokers. As survival after a diagnosis of lung cancer is very poor [41], projections for lung cancer incidence would be similar to those given here for mortality.

The calculations suggest that of the 34,958 lung cancer deaths that occurred in the UK during 1998, 2275, or 6.5% of the total, were caused by radon (Table 4). Of these, only 349 (1.0% of all lung cancer deaths), can be attributed to radon acting alone, while the remaining 1926 (5.5% of all lung cancer deaths) were caused by both radon and smoking, in the sense that the

Table 4. Causes attributed to the lung cancer deaths occurring each year in the United Kingdom.

Cause	Number of lung cancer deaths	Percentage attributed	
Not caused by active smoking or by residential radon	3,351	9.6	
Caused by radon but not by smoking	349	1.0	6.5 due to residential radon
Caused by smoking and radon (avoidance of either of which would have avoided that particular lung cancer)	1,926	5.5	89.4 due to active smoking
Caused by smoking and not by radon	29,332	83.9	
Total no of lung cancer deaths	34,958	100 0	

Calculation based on 1998 UK national data for numbers of lung cancer deaths, population size and smoking habits [42, 43]. Lung cancer death rate in lifelong non-smokers taken from a US prospective study of mortality [44], adjusted for the lower average radon level in the UK compared with the US. Average radon exposure assumed to be 20 Bq m^{-3} [45]. BEIR VI exposure/age/concentration model with submultiplicative joint effect of smoking and radon assumed for radon risks Additional assumptions are those used in the BEIR VI Committee's projections [1].

Table 5. Lung cancer deaths attributable to residential radon in the United Kingdom each year by age and sex

Age	Males	Females	Total
<35	1.5	1.5	3 (<1%)
35–54	224	161	385 (17%)
55–74	962	554	1516 (67%)
75+	218	153	371 (16%)
All ages	1405 (62%)	869 (38%)	2275 (100%)

Table 6. Effect of various residential radon concentrations on the cumulative risk (%) of death from lung cancer to age 85

	Residential radon concentration (Bq m^{-3})				
	0	20[a]	100	200	400
Lifelong non-smoker	0 7	0.8	1.0	1.4	2.2
Cigarette smoker	29.1	30.4	34.8	40 0	49.3
Whole population:					
Males	9 8	10.4	12 4	14.8	19.6
Females	4.4	4 7	5.7	7.0	9.5

Based on the BEIR VI exposure/age/concentration model for radon risks with submultiplicative joint effect of smoking and radon (lifelong non-smokers and cigarette smokers) or no adjustment for smoking (whole population) [1]. Lung cancer death rates based on 1988 UK national data [42] for whole population, a US prospective study [44] for lifelong non-smokers (with adjustment for the lower average radon concentration in the UK compared with the US), and a recent study of UK lung cancers for cigarette smokers [46]. If, for one particular category, the lung cancer rates per 10^5 in all the five year age groups before age 85 add up to c, then the cumulative risk by age 85 is $1-\exp(-5c/10^5)$. Thus, cumulative risks depend only on age-specific lung cancer rates and not on competing causes of death.
[a] UK average residential radon concentration.

lung cancer could have been avoided by avoiding either smoking or radon exposure. Lung cancer in the UK is primarily attributable to smoking and lung cancer rates are higher in males than in females; as a result, 62% of the deaths attributable to radon are projected to have occurred in males, with only 38% in females (Table 5). It is likely that two thirds of the deaths occurred in individuals between the ages of 55 and 75, with the remainder approximately equally divided between individuals aged 35–54 and 75+. Very few radon-attributable deaths are likely to occur in individuals under the age of 35.

Personal risks will not be uniformly distributed throughout the population and will be determined by both smoking status and residential radon concentration. The likely extent of this variation can be seen when lung cancer mortality rates for individuals with different smoking habits and residential radon concentrations are considered in conjunction with the BEIR VI risk models. The best estimate of lung cancer mortality rates in lifelong non-smokers are those obtained from the American Cancer Society's prospective study [44]. These have been adjusted, according to the BEIR VI model, to take into account the lower average residential radon concentrations of 20 Bq m^{-3} in the UK [45] compared with 46 Bq m^{-3} in the US [2]. At 20 Bq m^{-3} the cumulative risk to age 85 of death from lung cancer in non-smokers is estimated to be 0.8% (Table 6). This would be reduced only very slightly, to 0.7%, if the residential radon concentration were, hypothetically, brought down to zero, and would rise to 1.4% at 200 Bq m^{-3}, the level at which it is recommended in the UK that action be taken to reduce radon levels, and further to 2.2% at 400 Bq m^{-3}. For continuing cigarette smokers, estimates of the age-specific lung cancer death rate are available from a recent study of smoking and lung cancer in the UK [46]. These suggest that the cumulative risk of death from lung cancer by age 85 is around 30% for individuals whose residential radon concentrations are equal to the UK average, but rises substantially, to around 40% for those exposed at 200 Bq m^{-3}, and to 49% for those exposed at 400 Bq m^{-3}. For the entire population, which consists of a mixture of lifelong non-smokers, current smokers and ex-smokers, the estimates lie in between those for lifelong non-smokers and current cigarette smokers (Table 6). Estimates are lower for females than for males, reflecting the fact that in the UK women have smoked less in the past than men, so that

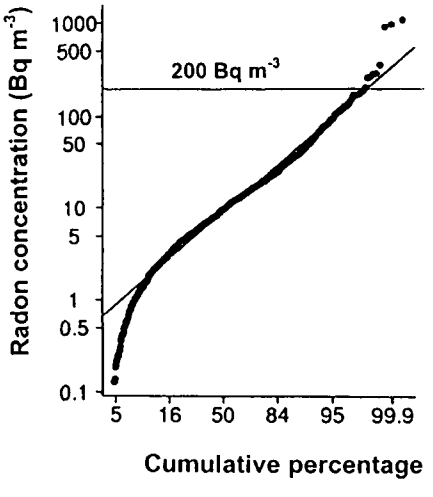

Figure 5 Distribution of residential radon concentrations in the UK based on a national survey [45] and adjusted for the outside air concentration (i.e. concentrations less than or equal to 4.1 Bq m^{-3} are set to 4.1 Bq m^{-3}, and 4 Bq m^{-3} is then subtracted from all concentrations). Solid line indicates best fitting straight line. Figure based on [47].

current lung cancer rates for the country as a whole are lower in women than in men.

The number of deaths attributable to radon at each level of concentration is determined not only by these individual risks, but also by the distribution of residential radon concentrations throughout the country. For the UK, residential radon levels have been shown to follow a log-normal distribution (Figure 5) after allowance for the radon level in outside air, which is around 4 Bq m^{-3} and varies relatively little. These data suggest that less than 1% of homes in the UK have a radon concentration of above 200 Bq m^{-3}. Consequently, the propor-

Table 7. Lung cancer deaths attributable to residential radon in the United Kingdom each year by residential radon concentration.

Range of residential radon concentrations (Bq m^{-3})	Percentage of homes in range	Deaths attributable to residential radon	
		Number	Percent
0–24	75 3	812	35.7
25–49	14.9	492	21.6
50–99	6.8	445	19.6
100–199	2.3	296	13.0
200+	0.7	230	10 1
Total	100.0	2275	100.0

tion of the lung cancer deaths attributable to radon that occur as a result of exposure to residential radon concentrations of 200 Bq m^{-3} or more is only around 10%, with another 13% occurring at concentrations in the range 100–199 Bq m^{-3} (Table 7). In contrast, over one third of radon-attributable deaths are estimated to occur with radon concentrations of less than 25 Bq m^{-3}, and around 20% each at levels in the ranges of 25–49 Bq m^{-3} and 50–99 Bq m^{-3}.

Prevention of lung cancer from residential radon

A recent international survey of radon legislation and national guidelines [48] has shown that most European states and many non-European countries now have recommended procedures for controlling levels of indoor radon. Some countries also have guidelines regarding radon incorporated into their building codes and recommended construction techniques. In most cases the guidelines are formulated in terms of action levels for dwellings and workplaces above which it is advised that remedial measures be taken. These action levels vary from 150–1000 Bq m^{-3} in different countries.

The identification of existing homes with radon concentrations above such action levels is the necessary first step towards reducing the risks that individuals living in them for long periods (Table 6) are likely to experience. With this in mind the UK government has offered free radon measurements for all homes in areas in England estimated to have more than a 5% chance of having a radon concentration above the UK action level for dwellings, which is 200 Bq m^{-3}. Up to a third of those approached took advantage of this policy, and around 400,000 measurements were carried out as a result [49]. However, only around 10%–20% of those living in homes that were identified to be above 200 Bq m^{-3} took any radon remedial action [50, 51]. Current UK government efforts have therefore been directed towards supporting local authority led initiatives that will increase this percentage, such as additional publicity materials and training for local authority staff and local builders, and pilot studies have demonstrated that an approximate doubling of the percentage taking radon remedial measures can be achieved [51].

In considering both the benefits and the costs of radon remediation programmes targeted at homes or other buildings above a specified action level, it is useful to be able to make comparisons with other health related interventions. The methodological framework now considered appropriate in the economic evaluation of health interventions is cost-effectiveness analysis, in which all the direct costs associated with an intervention are divided by the additional health benefits (such as life-years gained) to obtain a cost per unit of health gain, which can then be compared with other interventions [52, 53]. For radon remediation the data needed for such a model include the percent of homes over the action level, the cost of identifying these homes, the percent of these homes where radon remediation is in fact carried out, the cost of radon remediation, the risk of lung cancer per unit radon exposure, the average cost of treatment per lung cancer case, the number of life-years gained per lung cancer case avoided, and also discount rates for ongoing costs associated with radon remediation and for life-years gained.

In the UK, cost-effectiveness studies using this framework have been carried out for interventions to reduce radon concentrations above the action level in both homes and schools for the county of Northamptonshire [54, 55]. For homes, a societal cost-effectiveness ratio of £13,250 ($20,385) per life-year gained was estimated using 1997 prices. The cost of radon remediation is born by the home owner in the UK, and a survey carried out as part of this study found that the average cost of radon remediation in Northamptonshire was £533 ($820). The figure of £13,250 took the percentage of homes above 200 Bq m^{-3} to be 6.3% and the percentage of such households where remediation work was carried out at 11%, as these were the values observed in Northamptonshire. The cost-effectiveness ratio would, however, decrease if either of these values were to increase, and a sensitivity analysis demonstrated that if either the percentage of affected households who actually remediated increased to 30% or the percent of homes over 200 Bq m^{-3} increased to 15% (as is the case for several areas of the UK [56]), then the cost-effectiveness ratio would decrease to around £5000 ($7692) per life-year gained. In the UK, interventions to prevent disease that have cost-effectiveness ratios of less than £10,000 ($15,385) per life year gained have often been adopted as Government funded measures (including the national breast cancer screening programme and the secondary prevention of heart disease using statins) and many interventions with cost-effectiveness ratios in excess of this range are currently in practice [54]. For schools, the cost effectiveness ratio was estimated at £7550 ($11,615) per life-year gained, again at 1997 prices [55]. Once again, this is within the range for health interventions that have often been adopted, although it was found to be less favourable than several of the lung cancer prevention programmes targeted at smoking ces-

sation, which have been shown to have cost-effectiveness ratios per life-year gained of under £1000 ($1539) [55].

If a substantial proportion of the radon attributable deaths are to be avoided, something more will have to be done beyond reducing the levels in homes with concentrations above 200 Bq m^{-3} as these account for only 10% of the total (Table 7). The practicability of reducing radon levels in homes with concentrations at much lower radon concentrations consequently needs to be considered. One way in which this might eventually be achieved would be by requiring radon protection measures in all new dwellings. Several countries, including the UK, have recently adopted legislation regarding protective measures in new dwellings [57]. These are aimed specifically at reducing the probability that a new home will have a radon concentration above the current action level of 200 Bq m^{-3} and have therefore been introduced only in 'radon affected' areas, where more than 3% of measurements in existing dwellings have been found to be above this level. By requiring protection measures for all new homes, this legislation will clearly have an impact on the entire distribution of residential radon concentrations within these areas, as well as on the proportion above the action level. One study [58], again in Northamptonshire, has concluded that the installation of radon protection measures in all new dwellings in the 'radon affected' parts of the county has a cost per unit of radiation exposure avoided which is similar to that for the current policy aimed at remediating existing dwellings above the action level. However, full economic cost-effectiveness analyses have not yet been published, either of the policies recently introduced in the UK or of a policy of requiring low-cost basic radon protection, such as ensuring that the damp-proof barrier is radon-proof and that its edges and any joins in it are appropriately sealed, in all new dwellings.

Discussion

Radon is an established carcinogen and an ubiquitous air pollutant. Evidence from heavily exposed miners and theoretical considerations both suggest that its carcinogenic effects are likely to vary linearly with exposure without any threshold. Quantitative analysis indicates that in the UK, the US [1], and also many other countries, radon is the second most important cause of lung cancer after smoking. Worldwide it probably causes over a hundred thousand lung cancer deaths each year, considerably more than thought to be caused by passive smoking.

Radon deserves attention because residential radon levels can usually be reduced, often by relatively simple and cheap intervention measures, raising, at least in principle, the prospect of reducing the number of radon-induced deaths through preventive measures. However, informed discussion on this topic can take place only if precise, unbiased estimates of the risk associated with a given radon concentration are available, together with good information about factors that modify the risk. Several countries have introduced radon-control strategies, yet at present these are often aimed at the small proportion of the population with extreme exposures. In contrast, relatively little attention has been directed towards the effects of lower radon concentrations, although the typical log-normal distribution of residential radon concentrations means that this is where the bulk of radon-induced deaths are likely to occur. One reason for this may be that direct evidence of the carcinogenic risk attached to residential radon is at present weak, as the majority of studies of the effects of residential radon have low power. At present efforts are underway to pool together the data from the existing studies of residential radon and to explore further the possibilities of deriving estimates of lung cancer risk based on cumulative residential radon exposure as recorded in glass objects, and these should provide firmer evidence. Additional insight may also arise from work aimed at further elucidating the mechanism of radon carcinogenesis (see [1] for a review). To complement these scientific endeavours additional economic work evaluating the cost-effectiveness of various intervention strategies is desirable.

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