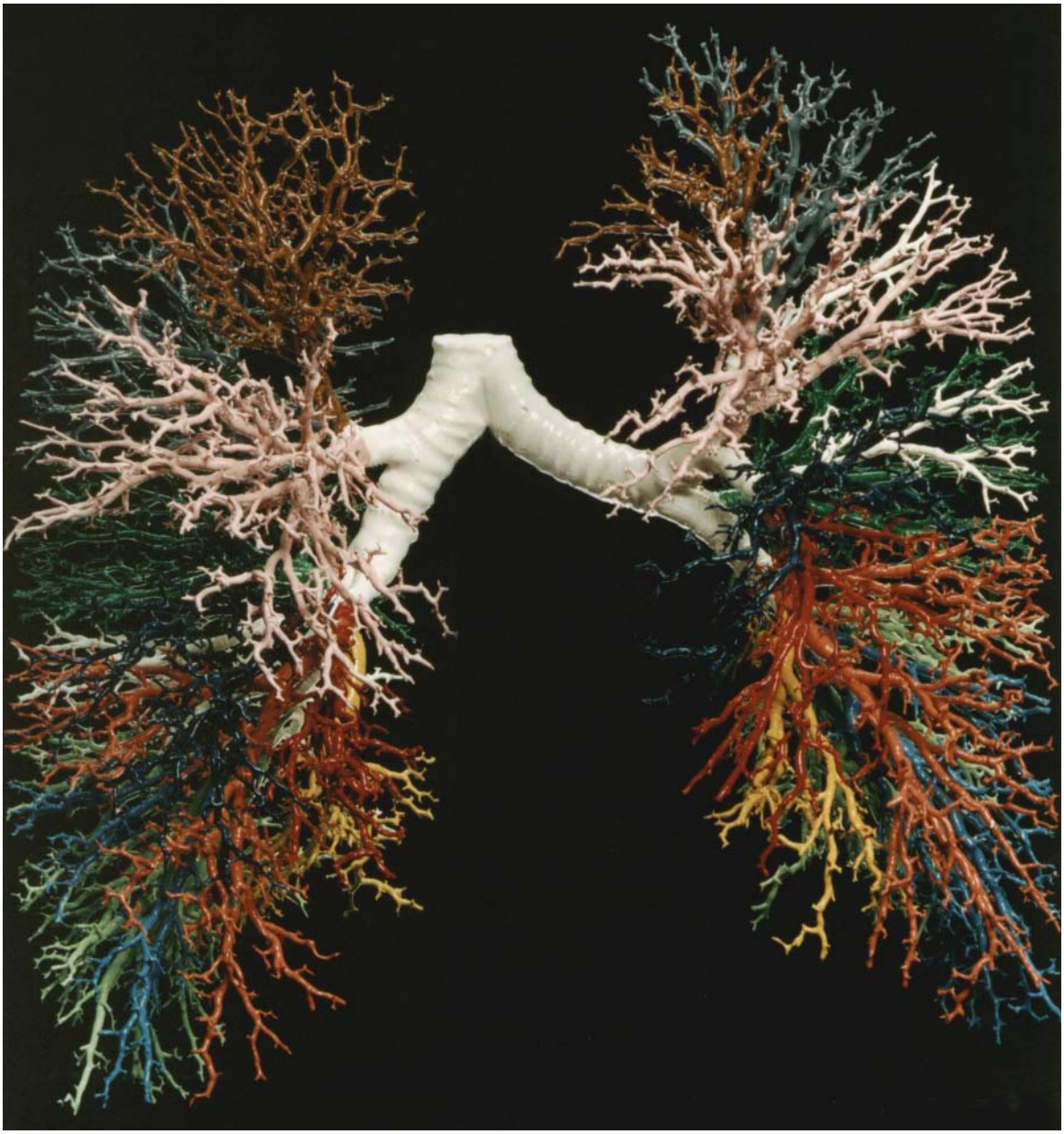
Health Protection Agency

Radon and Public Health

Report of the independent Advisory Group on Ionising Radiation



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Radon and Public Health

Report of the independent Advisory Group on Ionising Radiation

Documents of the Health Protection Agency
Radiation, Chemical and Environmental Hazards
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Contents

Foreword

The Radiation Protection Division of the Health Protection Agency (HPA) undertakes research to advance knowledge about protection from the risks of ionising and non-ionising radiations. It provides laboratory and technical services, runs training courses, and provides expert information. It also has a statutory responsibility for advising UK government departments and those with regulatory responsibilities for ionising and non-ionising radiations in the fields of medical, public and occupational exposure.

The HPA Radiation Protection Division was formed when the National Radiological Protection Board (NRPB) merged with the HPA on 1 April 2005. In 1995 the Director of the NRPB had set up the Advisory Group on Ionising Radiation (AGIR) that had as its terms of reference:

> 'to review work on the biological and medical effects of ionising radiation relevant to human health in the occupational, public health, medical and environmental fields and advise on research priorities'

In addition, the AGIR was given the task of helping the NRPB, where appropriate, to deal with any urgent request for advice or work from the Department of Health or other government departments. The AGIR was reconstituted in 1999 as an independent body and reported directly to the Board of the NRPB; since April 2005 it reports to the HPA Board Subcommittee on Radiation, Chemical and Environmental Hazards. The remit of the AGIR is restricted to the provision of scientific judgements and does not include the development of specific recommendations relating to radiation protection policy. These are matters for the HPA and its Board. For details of the current work of the AGIR, see the website at www.hpa.org.uk.

The AGIR has to date issued six reports that consider

a heterogeneity in response to radiation,

b guidance on promotion of further optimisation of medical exposures,

c epidemiology of second cancers,

d UK population risks for leukaemia,

e review of risks from tritium,

f high dose radiation effects and tissue injury.

In 2000 the AGIR set up a Subgroup on Radon Epidemiology, with a remit to review the evidence of the effects of exposure to radon and its decay products on health, as relevant to the population of the UK. The present report prepared by the Subgroup also considers the available strategies for remediation of radon affected buildings and includes a health economics analysis of radon remediation.

Radon and Public Health

HAS BEEN PREPARED BY THE

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Radon and Public Health

Report prepared by the Subgroup on Radon Epidemiology of the independent Advisory Group on Ionising Radiation

Chairman of Subgroup: Professor J Little
Chairman of Advisory Group: Professor B A Bridges OBE

This report from the independent Advisory Group on Ionising Radiation reflects understanding and evaluation of the current scientific evidence as presented and referenced in this document.

Executive Summary

The Advisory Group on Ionising Radiation advises the Health Protection Agency on the biological and medical effects of ionising radiation relevant to human health. The Advisory Group set up a Subgroup on Radon Epidemiology, with a remit to review the evidence of the effects of exposure to radon and its decay products on health, as relevant to the population of the UK. This is the report prepared by the Subgroup on Radon Epidemiology.

Radon is a natural radioactive gas which is emitted in varying quantities from all rocks and soils. If it escapes from the ground to the open air, it is quickly diluted to low concentrations. However, radon can reach high concentrations in enclosed spaces such as caves and mines and also in buildings. Radon in the home delivers larger doses to the UK public than any other natural source of ionising radiation.

Epidemiological studies of miners established many years ago that exposure to high radon concentrations leads to an increased risk of lung cancer. Most of the risk comes not from radon gas itself, but from its short-lived decay products. However, 'radon' is used as a convenient shorthand.

It was recognised more than 20 years ago that radon in homes and other buildings in the UK might reach concentrations high enough to lead to a material risk of lung cancer. The magnitude of the risk from radon in homes was estimated from the results of studies of miners, supported by calculations of radiation dose to different tissues and laboratory studies on animals. On the basis of this evidence, the UK introduced a radon control policy through the provision of guidance in 1987 (revised in 1990), and was one of the first countries in the world to do so.

Since this advice was issued, considerable additional evidence has become available. In particular, direct evidence is now available on the effects of residential exposures. We have reviewed the risk of lung cancer from exposure to radon in homes using all sources of information. The data from all these sources are interpreted in the light of the general understanding of the biological, chemical and physical events that take place when ionising radiation interacts with cells in the human body.

Particularly important information comes from analyses of data from a number of studies in which measured radon concentrations in the homes of people who developed lung cancer were compared with similar measurements in the homes of people who did not develop lung cancer, whilst at the same time taking each person's smoking history into account. The data were brought together in a common format and then an analysis of the pooled data was carried out. Pooling the data in this way allows for more accurate assessment of the risks than was possible in any of the individual studies. In analysing these data, it is important to take into account the fact that radon concentrations vary greatly with time, leading to uncertainties in estimates of long-term exposure to radon. Allowance has been made for these uncertainties in the pooled analyses.

All the sources of information paint a broadly similar picture of the risks of radon exposure, but we conclude that the pooled analyses provide the most important and relevant evidence on the risks of residential exposures. We conclude that there is clear evidence of a risk from indoor radon, both overall and at concentrations below the current UK Action Level for radon of 200 Bq m^{-3}.

The results of the pooled analyses are expressed as a proportional (percentage) increase in risk of lung cancer per unit exposure to radon. The best current estimate of this risk is based on the pooling of European studies, taking measurement errors into account. This analysis estimated that an increase of 100 Bq m^{-3} in the long-term average radon concentration in the home would cause an increase in the risk of lung cancer of between 5 and 31% with a central risk estimate of around 16%. Various factors mean that the true risk is likely to be somewhat higher than this central value.

This risk appears to vary linearly with the radon concentration, with no threshold below which the risk is zero. There is no strong evidence from the pooled analyses that the proportional increase in risk varies with age or between men and women, and it is similar for non-smokers, ex-smokers and current smokers. Because of this, a particular radon exposure that doubled the low risk of lung cancer in a non-smoker would also double the high risk of lung cancer in a smoker. Cigarette smoking remains the most important cause of lung cancer. At the national average residential radon concentration of 21 Bq m^{-3} the cumulative risk of death from lung cancer by the age of 75 years is 0.4% for a lifelong non-smoker and 15% for a continuing cigarette smoker. At 200 Bq m^{-3} these risks rise to 0.5 and 19%, respectively.

Overall, these analyses indicate that about 1100 deaths from lung cancer in the UK are caused by exposure to radon in homes each year – that is, just over 1 in 500 from all causes. For most of these deaths, lung cancer was caused jointly by radon and smoking in the sense that the disease could have been prevented either by avoiding smoking or by avoiding exposure to radon. Over 40% of deaths caused jointly by radon and smoking are likely to occur in people who have already given up smoking.

Radon concentrations in homes are determined by various factors including the geology of the ground beneath a house, details of the way the house is constructed, and factors such as the method of heating and of ventilation. Broad patterns in the way radon concentrations vary across the country can be identified, but concentrations can vary greatly between adjacent and apparently similar homes. The long-term average radon concentration in a house can be determined only by long-term measurements in the house in question made in several different years.

Many measurements of radon concentrations in homes have been undertaken in the UK. The distribution of these measurements shows that in most homes concentrations are below 50 Bq m^{-3}, but in a small proportion concentrations exceed 200 Bq m^{-3}. Because only small numbers of people are exposed to such concentrations, the majority (over 95%) of the radon-induced deaths occur among those who are exposed at radon concentrations below 200 Bq m^{-3}, the current Action Level.

Homes can be modified in a number of ways to reduce the entry of air carrying radon from the soil underneath them. The UK and several other countries already have programmes to reduce high radon concentrations in existing homes, and to install radon preventive measures in new homes in areas where high indoor radon concentrations are likely. As current and alternative radon policies all have different

costs and likely benefits, it is useful to compare them using cost-effectiveness analysis, and this report presents estimates of the cost-effectiveness of a number of alternative policies. This has not previously been part of the justification of radon programmes, but is supported by the view that radon reduction programmes can be seen as a medical screening procedure, with cost-effectiveness analysis providing useful guidance on the targeting of areas of the country in which to implement these programmes, and other aspects of implementation on which attention is most needed. Now that a health economic model has been developed, further cost-effectiveness analyses can be performed to take account of new information and also to investigate the potential consequences of specific aspects of the implementation of radon control policies.

The cost-effectiveness analyses suggest that radon preventive measures, such as a sealed membrane under the ground floor, in new homes, would be justified throughout the entire country rather than just in specific areas, as at present. Programmes of measurement and remediation in existing homes are not cost-effective at present, but might become so in areas with mean indoor radon concentrations of 60 Bq m^{-3} or above if the Action Level were reduced from 200 Bq m^{-3} to 100 Bq m^{-3}. The results of the cost-effectiveness analysis of radon remediation in existing homes are sensitive to a number of factors including the cost of identifying homes with high radon concentrations and the proportion of homes identified as having high radon concentrations in which remedial action is undertaken. New approaches might change these factors and improve the cost-effectiveness of radon campaigns. Results for existing homes are also highly dependent on the smoking status of inhabitants: remediation of homes where measurements are above 100 Bq m^{-3} is likely to be very cost-effective for households consisting of current cigarette smokers, but not for households consisting of lifelong non-smokers. However, there is some evidence that current and ex-smokers are less likely to comply with remedial programmes than lifelong non-smokers, for whom radon remediation is not cost-effective in any but the areas with the highest radon concentrations found in the UK.

We note that the cost-effectiveness of preventive radon programmes in new homes is likely to be more favourable than the figures given here imply since the true risks from radon are likely to be higher than those assumed. This cannot be quantified at present, but the cost-effectiveness estimates can be revised as new evidence appears.

We have focused our attention in this report on residential radon exposures because of the recent availability of powerful epidemiological and other information relevant to these circumstances. We are, however, aware that our findings are likely to have public health implications for exposures in the workplace and in public places such as schools.

In addition to the review of epidemiological evidence of lung cancer, this report reviews all the sources of information on the risks of other cancers and of other possible harmful effects. Calculations of doses from radon and its decay products to all tissues and organs of the body suggest that there may be some risk of other cancers, particularly when ingestion of radon in water and deposition of radon decay products on skin are considered. However, such risks are calculated to be small compared with that of lung cancer, and there is no consistent epidemiological evidence of elevated levels of other cancers or other harmful effects as a result of exposure to radon.

In conclusion, the available evidence indicates a causal association between lung cancer and radon at the concentrations encountered indoors in ordinary homes and other buildings in many parts of the UK. Around 1100 radon-induced lung cancer deaths occur each year in the UK, the majority as a result of exposure at concentrations well below 200 Bq m^{-3}, the current Action Level. It now seems appropriate to move to a population-based approach that aims to reduce the collective dose from radon exposure progressively, thereby reducing progressively the number of radon-induced lung cancers that occur in the UK each year. Health economic evaluation shows that several options for such a move would be cost-effective.

1 Introduction

Key Points

1 Radon enters homes from the ground underneath them. Radon concentrations vary widely from home to home, with high concentrations in some homes.

2 The decay products of radon can be inhaled and can irradiate sensitive cells in the lungs with alpha particles, thus increasing the risk of lung cancer.

3 Under the current UK radon control programme, radon preventive requirements for new homes are specified in the building regulations in some areas.

4 The current UK radon control programme specifies an 'Action Level' for radon. Those living in a home where the radon concentration has been measured which, after adjustment for seasonal variation, has been found to be above this level, are advised to remediate at their own expense. Some areas have been designated as 'radon Affected Areas' and government-funded measurement campaigns directed towards finding homes above the Action Level have focused on parts of these areas with particularly high radon concentrations.

5 This report reviews the evidence on the risk of lung cancer arising from residential radon exposure. It also presents estimates of the cost-effectiveness of different strategies for reducing radon exposures.

6 We have focused our attention on radon in the home because of the availability of a substantial amount of new epidemiological data on the risk of lung cancer from radon in the home.

7 Homes are not the only places in the UK where exposure to radon occurs and our findings may well have implications for exposures in workplaces and public places, such as schools.

Radon is a natural radioactive gas, which has no taste, smell or colour. It is produced by the radioactive decay of uranium and thorium, present in all soils and rocks in small quantities. There are a number of isotopes of radon but the most important are radon-222 (derived from uranium-238) and radon-220 (derived from thorium-232). Radon-220, generally known as thoron because of its parent radionuclide, delivers much smaller doses to the UK public than radon-222, and will not be discussed here. In the rest of this report, the term radon will refer to radon-222.

Because radon has a 3.82-day half-life, it can diffuse in the ground before decay. If it escapes from the ground to the outdoor air, it is quickly diluted to low concentrations. However, radon concentrations indoors can reach high levels, usually not by diffusion of radon out of the ground but by a flow of air carrying radon from the ground into buildings. This occurs because, as a result of warm indoor air rising, the air pressure at ground level in most buildings is slightly lower than the pressure in the air beneath them. This causes a flow of soil air to be drawn into buildings, carrying radon with it.

Radon is soluble in water and under some circumstances high concentrations can be found in groundwater – for example, from wells or boreholes. If the water is used for domestic purposes this is another route of exposure to radon. In the UK this is thought to be a very small problem and one which can usually be remedied easily. Building materials can also be a source of indoor radon, although they are usually a much smaller source than the ground.

1.1 Risks of radon exposure

When radon gas decays it gives rise to isotopes of the solid elements lead, bismuth and polonium, as shown in Figure 1.1. These short-lived decay products are also radioactive, and attach themselves to natural aerosol particles in the atmosphere. Both unattached decay products and decay products attached to particles may be inhaled, and may then stick to the walls of the lungs and other parts of the respiratory system. As these radon decay products undergo further decay, they emit alpha particles which irradiate the cells lining the walls of the respiratory system. It should be noted that the term 'exposure to radon' is used as shorthand for 'exposure to radiation released by radon and its decay products'.

It has been appreciated since the 1500s that metal miners in the Erz mountains of central Europe had a very high mortality rate. However, the disease accounting for this was not identified as lung cancer until 1879 (Harting and Hesse, 1879) and the aetiological role of radon was not established until the 20th century. In 1951 Bale pointed out that the hazard was due to the radiation dose from the decay products of radon rather than radon itself (Bale, 1951).

Since Bale's observation, many studies of the risks of exposure to radon among miners of igneous rocks have been carried out, and these studies have demonstrated an increasing risk with increasing exposure (see Appendix H). In 1988, the International Agency for Research on Cancer (IARC) classified radon as a human carcinogen (IARC, 1988). There is also considerable evidence from laboratory studies of animals on the effects of exposure to radon (see Appendix G).

The range of exposures to radon in homes overlaps with the range of exposures found to cause lung cancer in miners. This suggests that radon in the home may increase the risk of lung cancer for those exposed at the highest concentrations. However, the conditions of exposure in mines are usually different from those in homes, so it is important to evaluate the risk of exposure to radon in homes directly. A number of studies designed to examine this question have been carried out, and these are discussed in Chapter 3. Combined ('pooled') analyses of the individual data from these studies have recently been published. These analyses now provide the most important body of evidence on the risks of exposure to radon in the home.

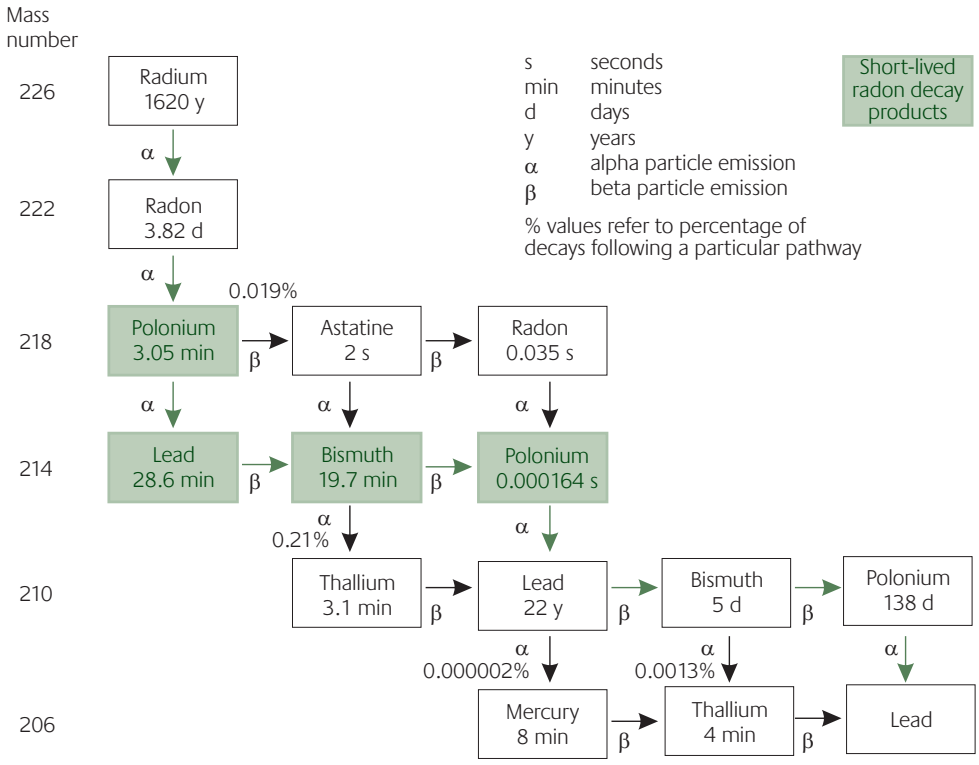

FIGURE 1.1 Radium-226 decay chain

1.2 Measures to limit human exposure to radon in UK homes

A number of physical quantities are used to describe the concentration of radon gas and of its decay products: the concentration of radon in becquerels per cubic metre (Bq m^{-3}) is an example. In order to compare radon exposures with exposures from other types of radiation, some other quantities, usually involving certain assumptions, are also sometimes used. Effective dose, sometimes abbreviated to dose, is an example. All these quantities are described in Appendix A. Ways in which radon concentrations can be assessed and quantified are outlined in Appendix B.

Many measurements of indoor radon concentration have been undertaken in the UK. These are described in more detail in the next chapter. The results imply that radon in the home delivers larger doses to the UK public than any other natural source of ionising radiation. In 1984, the need for reduction of radon exposures was recognised by the Royal Commission on Environmental Pollution (RCEP, 1984) and the International Commission on Radiological Protection (ICRP, 1984). The National Radiological Protection Board (NRPB, now the Radiation Protection Division of the Health Protection Agency, HPA) first issued guidance on this matter in 1987 (NRPB, 1987). This advice suggested the

remediation of existing homes with high radon concentrations and also preventive measures to reduce radon concentrations in new buildings by means of changes in the building regulations in some areas. In 1990 the NRPB reviewed the evidence on radon risk and recommended an 'Action Level' of 200 Bq m^{-3} for homes (NRPB, 1990a). Where measurements above the Action Level were found, emphasis was placed on reducing radon concentrations as far as possible, not just reducing them below the Action Level. The NRPB advice was considered and endorsed by the Committee on Medical Aspects of Radiation in the Environment (COMARE) and accepted by government.

Further details of the development of radon policy and programmes in the UK are given in Appendix D. Programmes funded by government have allowed large numbers of measurements to be carried out with the aim of identifying homes above the Action Level. These measurements have been used to produce maps indicating the proportion of homes with seasonally adjusted measurements above the Action Level in different areas (see Chapter 2). Indoor radon concentrations can be reduced by reducing the entry of soil air carrying radon in a number of ways and these are described in Appendix L.

1.3 Policies on control of domestic exposures to radon in other countries

The UK is not alone in having introduced controls on domestic exposures to radon. Control measures in countries within and outside the (then) European Community were reviewed by Åkerblom (1999) and the review was updated by Synnott and Fenton (2005) for the expanded European Union as it then existed. Table 1.1 summarises the reference levels for radon in various countries as reported by these authors. The table distinguishes between reference levels for existing dwellings and those for new buildings. These measures are usually obligatory in new homes and advisory in existing ones. Where countries have both advisory and enforced levels for existing homes, only the former is quoted in the table.

1.4 Remit of current report

In this report we review recent research on the risks of exposure to radon and its decay products, taking into account the available data from epidemiological, laboratory and mechanistic studies. Our main focus is on the risk of lung cancer from radon exposure in the home (Chapters 3 and 4), but we have also considered the effects of radon exposure on tissues other than the lung (Chapter 5).

We have also examined the cost-effectiveness of a variety of approaches to reducing radon concentrations in homes (Chapter 6).

Finally, we draw attention to the factors we judge to be important in helping to refine UK policy on reducing the risk to the general public from exposure to radon.

The main text of the report is relatively short. Reference is made to detailed appendices which are grouped into three broad categories: basic information (Appendices A–F), effects of exposure (Appendices G–K) and countermeasures (Appendices L–N).

TABLE 1.1 Radon controls in other countries (after Åkerblom, 1999, and Synnott and Fenton, 2005)

Country	Reference level (Bq m^{-3})	
	New homes	Existing homes
From Synnott and Fenton, 2005		
Austria	200	400
Belgium	400	400
Czech Republic	200	400
Denmark	200	200
Finland	200	400
Germany	200	400
Greece	200	400
Ireland	200	200
Slovenia	400	400
Spain	200	200
Sweden	200	200
Switzerland	400	400
UK	200	200
From Åkerblom, 1999		
Belarus	200	200
Estonia	200	400
Latvia	300	300
Lithuania	200	400
Luxembourg	150	150
Norway	200	400
Poland	200	400
Russia	200	400
Slovak Republic	250	500
Yugoslavia	200	200

Note: Where the information reported by Åkerblom has been updated by Synnott and Fenton, only the latter has been reported. Radon reference levels are radon concentrations above which control measures are recommended.

2 Exposures to Radon in the UK

Key Points

1 The radon concentration within any dwelling varies substantially with time. There is usually both diurnal and seasonal variation, and also appreciable variation from year to year.

2 Measured radon concentrations vary widely between dwellings, depending not only on temporal variability, but also on the local geology, on details of building construction and on the habits of the occupants.

3 Surveys of indoor radon concentrations have been carried out to determine the overall distribution of concentrations, to identify dwellings with high radon concentrations, and to allow radon mapping. The HPA has produced detailed maps of the percentage of homes exceeding 200 Bq m^{-3} (the UK Action Level since 1990).

4 High radon concentrations can also be found in workplaces. There is some existing legislation controlling exposures of workers to radon.

5 Radon in water supplies can also contribute to exposures, although it is rare for this to be a significant contributor to doses to the UK public.

2.1 Radon concentrations in homes

Radon in homes can originate from a number of sources, including the building materials and the water supply (Kendall et al, 1994). However, in practice in the UK, the ground underneath the building contributes most of the indoor radon. This is particularly true in buildings with high radon concentrations. No UK house with a measured radon concentration over 200 Bq m^{-3} has been found to have an appreciable radon contribution from sources other than the ground beneath it. However, where radon concentrations are very low, such as on the upper floors of blocks of flats, the main source may be radon in the outdoor air or emissions from building materials.

In considering how much radon enters a building from the ground beneath it, it is useful to consider two factors: the concentration of radon in the soil gas beneath the building, and the transfer of soil gas into the building. Concentrations of radon in soil gas are determined by geology and high concentrations of radon are more likely if the rocks and soils contain high concentrations of uranium. However, if the ground is not permeable, then the radon will remain trapped in the ground. In most cases, the radon entering a house from the ground has originated within a few metres, but if the ground is particularly permeable or fissured, it may come from a greater distance. If the underlying rock is covered with clay,

then radon is unlikely to be able to escape to the surface as it could if the covering was of a more porous material. Granites tend to have high concentrations of uranium and, in consequence, indoor radon concentrations are high in some granite areas. High radon concentrations are not found in all granite areas, however, and conversely high radon concentrations can sometimes be found elsewhere (for example, on limestones, sandstones and some other geological formations).

The transport of radon from soil gas to room air depends on the details of construction of the house (particularly on the presence of cracks or openings in the floor) and on the way in which it is occupied (for example, the indoor temperature relative to outdoors and the ventilation).

Atmospheric pressure is usually lower indoors than outdoors, owing to the warm indoor air rising. This creates a gentle suction at ground level in the building, in the same way as warm air rising up a chimney creates a suction. Wind blowing across chimneys and windows can also create an underpressure indoors. The typical ventilation rate of a house is about one air change an hour. Most of the inflowing air comes through doors and windows, but perhaps 1% or so comes from the ground, drawn in by the lower indoor air pressure. In an average house, the air flow from the ground amounts to a couple of cubic metres of soil gas entering the house each hour. It is this pressure driven flow, rather than diffusion, which is responsible for most of the radon entering homes.

Concrete floors often have cracks around the edges and gaps around service entries such as mains water supply, electricity or sewage pipes. It is these cracks and gaps through which soil gas enters. If homes have suspended timber floors the gaps between the floorboards are the major route of entry. Pathways for soil gas to enter homes are often not identifiable without careful investigation, and vary between apparently identical homes.

The radon concentration in a building depends on the rate of entry of the radon and the rate at which it is removed by ventilation. Increasing the ventilation rate will not, however, always decrease the radon concentration. This is because ventilation rate and underpressure are related, and some ways of increasing ventilation, such as the use of extractor fans or opening upstairs windows, can also increase the underpressure.

The factors described above vary greatly from one dwelling to another and lead to large differences in radon concentrations. The underpressure and ventilation rate also vary with time in all buildings. Underpressure tends to be highest in cold weather and at night because then the difference in temperature between indoors and outdoors is greatest. At these times, ventilation routes such as windows and doors are generally closed, so a higher proportion of the air drawn in by underpressure comes from the soil, thus increasing indoor radon concentrations.

The variation of indoor radon concentrations with time can be very substantial (Miles, 2001). This has implications not only for the practicability of modelling radon concentrations, but also for how radon concentrations are measured. A short-term measurement taken over a few hours or even a few days cannot be expected to give an accurate indication of the long-term average radon concentration. A measurement over several months will give a more stable estimate, although even here, allowance for the season of the year or for average temperature during the measurement period is needed (Pinel et al, 1995). Finally, radon concentrations within a home have been shown to vary substantially from year to

year, even when no alterations have been made to the home, and when the occupiers have not changed. In the UK the coefficient of variation of measurements made in the same home in different years, when each measurement is made over several months with seasonal correction, was found to be 51% in one study (Lomas and Green, 1994) and 44% in another (Hunter et al, 2004).

2.2 Distribution of residential exposures to radon in the UK

A large-scale representative survey of concentrations of radon in UK dwellings was carried out in the 1980s (Wrixon et al, 1988). The purpose was to determine both the average radon concentration in UK homes and the distribution of concentrations from home to home. A formal random sampling procedure was used to select a representative sample of dwellings from the UK housing stock. The occupiers of the selected dwellings were invited to take part in the survey, and sent two further reminder letters if they did not reply. Overall, 54% of householders agreed to take part in the survey. The survey produced complete measurement results in 2093 dwellings.

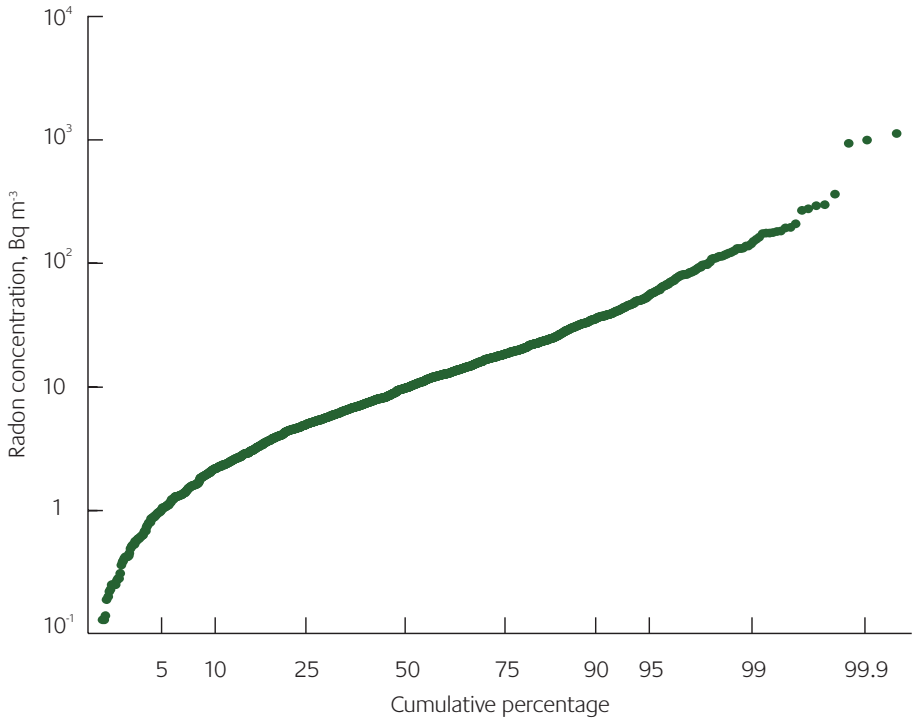

FIGURE 2.1 Residential radon concentrations from the UK national survey (Wrixon et al, 1988), adjusted for outside air concentration (ie concentrations less than or equal to 4.1 Bq m^{-3} are set to 4.1 Bq m^{-3}, and 4 Bq m^{-3} is then subtracted from all concentrations) (based on Gunby et al, 1993)

The radon measurements were made using passive radon detectors sent to the participants by post. Detectors were placed in the main living area and an occupied bedroom. In order to estimate the annual average radon concentration, measurements were made over two consecutive six-month periods.

On the basis of the results, the mean measured radon concentration taken over a period of a year in UK dwellings was estimated to be around 20 Bq m^{-3} (Wrixon et al, 1988), and the median measured radon concentration was lower, at around 10 Bq m^{-3}. The distribution of measured radon concentrations was found to be approximately log-normal (Gunby et al, 1993), as in most other large surveys of indoor radon concentrations (see Figure 2.1). The distribution implied that the measured radon concentration would be above 200 Bq m^{-3} in 0.4% of the UK housing stock, or approximately 100,000 houses.

In the years following the initial national survey, several substantial programmes to measure indoor radon concentrations in homes were undertaken in the UK. Most of these have been funded by national government, and have been directed towards finding dwellings in which the measured radon concentration, taken over a period of a few months and adjusted for seasonal variation, is above 200 Bq m^{-3}, the current UK Action Level for radon (NRPB, 1990a). The results are summarised in Table 2.1.

TABLE 2.1 Summary data provided in 2007 by the HPA on radon in dwellings for the UK

Parameter	England	Scotland	Wales	Northern Ireland	UK (rounded)
Housing stock	22,300,000	2,400,000	1,300,000	700,000	27,000,000
Population weighted average measured radon concentration (Bq m^{-3})	21	16	20	19	20
Number of dwellings measured	456,500	18,400	16,800	22,900	515,000
Number of dwellings with measurements at or above the present UK Action Level of 200 Bq m^{-3}, after adjustment for seasonal variation	51,100	370	1,780	1,150	54,400
Estimated total number of dwellings that would have seasonally adjusted measurements at or above the Action Level if the entire UK housing stock were measured	100,000	2,000	10,000	4,000	100,000
Dwellings found to have measurements at or above the Action Level as a percentage of the estimated total number	51%	19%	18%	29%	54%

2.3 Maps of radon concentrations in UK homes

The current UK radon programme has made extensive use of maps produced by the NRPB/HPA indicating the proportion of homes with seasonally adjusted measurements exceeding 200 Bq m^{-3}. With these maps national and local government could identify areas with a high proportion of such homes, and the proportion of homes likely to have measurements exceeding the Action Level in each area could be estimated.

Initially, radon measurements in UK homes were made by the NRPB for the national survey described above and for regional surveys. Subsequently, the NRPB/HPA made some further measurements to achieve an even geographical spread of results, so as to allow mapping of the geographical variation of radon concentrations. However, by far the majority of measurements were carried out by them in programmes that targeted high radon areas and were designed to identify homes where radon concentrations exceeded the Action Level of 200 Bq m^{-3}. In these programmes, the detectors were normally left in place for three months. The results were then corrected for seasonal variation, depending on the average outdoor temperature during the measurement (Miles, 1998).

Radon mapping of the UK has progressed in stages as more homes were measured and mapping methods developed:

a mapping first parts, then all, of England, Wales and Northern Ireland, and parts of Scotland at 5-km resolution (see, for example, NRPB, 1990a,b),

b mapping the parts of England with the highest radon concentrations at 1-km resolution (Green et al, 2002),

c mapping the variation in the potential for high indoor radon concentrations both between and within geological units over all of England and Wales to a typical accuracy of about 50 m (Miles and Appleton, 2005),

d most recently, a set of maps has been published that has included a correction for year-to-year variations in radon concentrations in homes (Miles et al, 2007).

All of these maps have concentrated on estimating the percentage of homes with radon concentrations above 200 Bq m^{-3}. In doing this they have relied upon the fact that the distribution of radon concentrations in UK homes (after subtraction of outdoor radon) is well represented by a log-normal distribution, both for the UK as a whole and for small areas within it (Gunby et al, 1993; Miles, 1994, 1998). The geometric mean (GM) and geometric standard deviation (GSD) of a group of measurements can be used to estimate the mean radon concentration, the percentiles of the distribution and the proportion above any threshold. The uncertainty in the estimate depends on the number of measurements available and the quantity being estimated. For example, for a given number of measurements, the proportional uncertainty in the estimates of the median and mean are much smaller than the proportional uncertainty in the upper 5% or 1% of the distribution. For further details see Appendix C.

Figure 2.2 shows a recent HPA map with the estimated percentages of homes with seasonally adjusted radon measurements above 200 Bq m^{-3}. Radon concentrations vary substantially from place to place.

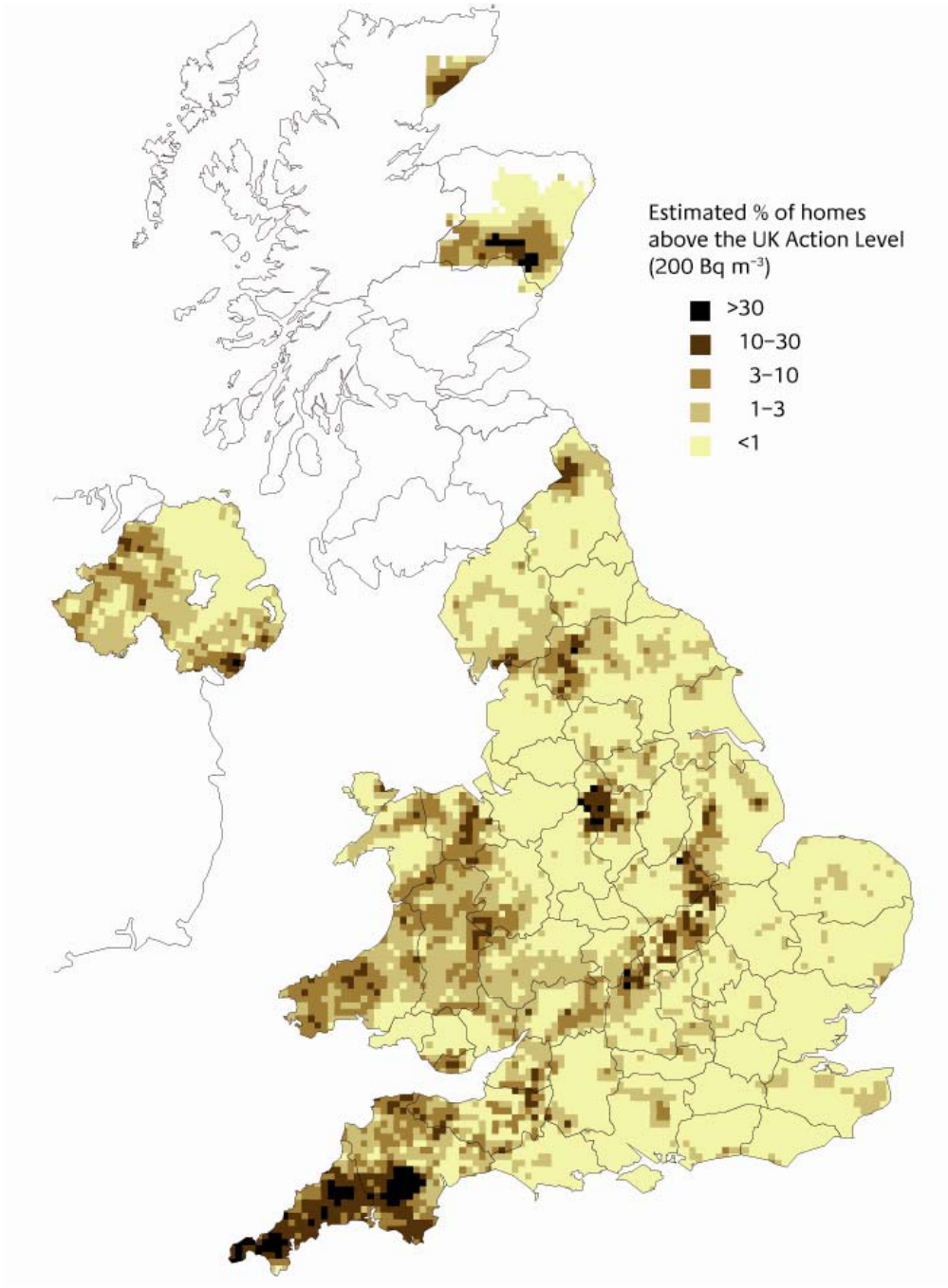

FIGURE 2.2 Map produced by the HPA of the UK showing the percentage of homes with seasonally adjusted measurements above 200 Bq m^{-3}, the current UK Action Level for radon

As a consequence, radiation doses to the public from radon are much more variable than doses from other natural sources such as terrestrial gamma rays. It can be seen that the highest concentrations are in granite areas of the southwest peninsula, but that elevated concentrations are also found elsewhere (for example, on the Derbyshire limestone). Nevertheless, it must be remembered that radon concentrations are variable and that maps indicate only where high concentrations are likely. Adjacent and apparently similar homes can have quite different radon concentrations which can be determined only by measurement.

2.4 Occupational exposures to radon in the UK

Miners are the workers most obviously liable to be exposed to radon. This applies in particular to non-coal mines. For a number of reasons, including the requirement for good ventilation in order to control methane, radon concentrations in coal mines are generally lower. Table 2.2 shows that, in the UK, the mean annual dose to coal miners was 0.6 mSv, while that to non-coal miners was 3.5 mSv. Since radon is a radioactive decay product of uranium, the highest concentrations are likely to be found where uranium-containing minerals occur. Such minerals are found in southwest England, where relatively high concentrations of radon have been found in tin mines. However, there are no longer active tin mines in the UK, and therefore average miner doses have fallen in recent years. Details of doses received by miners are given in Table 2.2 (further details can be found in Hughes, 1999). There is legislation to control doses to miners and to those who work in show caves and mines (Parliament, 1999).

High radon concentrations can be found in above-ground workplaces, just as they can in homes. Generally speaking, large workplaces such as factories have ventilation systems which tend to result in lower radon concentrations than in some smaller workplaces such as shops and offices, which have similar radon concentrations to those in homes. Legal controls apply in the UK where long-term radon concentrations in workplaces measured over a 24-hour period exceed 400 Bq m^{-3}. It has been estimated, on the basis of the results of measurements in dwellings and in workplaces, that about 50,000 workers in 5,000 workplaces are subject to exposures that exceed this concentration. Such workplaces can be identified only by direct measurements and most of them have not yet been identified (Table 2.3). Where high radon concentrations are found in an above-ground workplace, remedial building works can reduce them.

2.5 Radon exposures in caves and abandoned mines

High radon concentrations can be found in natural caves and in abandoned mines. This topic has been reviewed by Dixon (1996). Generally, there is unlikely to be a health risk to members of the public who make brief visits underground, but high doses might be incurred by those who spend long periods there. Occupational exposures in such places are controlled by legislation. There is a voluntary code of conduct for those who visit caves recreationally (Kendall and Dixon, 1997).

TABLE 2.2 Estimated radon exposures in UK mines in 1997/98 (numbers rounded). Data provided by the HPA

Type of mine	Number of miners in effective dose range (mSv)			Total number of miners	Collective effective dose (man Sv)	Average effective dose (man Sv)
	0–5	5–15	>15			
Large coal	10,000	0	0	10,000	6.0	0.6
Small coal	700	0	0	700	0.7	1.0
All coal mining	10,700	0	0	10,700	7.0	0.6
Non-coal mining	1,200	100	0	1,300	4.4	3.5
Total	11,900	100	0	12,000	11.0	0.9

TABLE 2.3 Radon in UK workplaces where the Ionising Radiations Regulations (1999) apply. Data provided by the HPA

	Dose range (mSv)				
	0–5	5–10	10–20	>20	Total
Estimated total number of workplaces	4,400	400	150	50	5,000
Number found	1,169	92	28	12	1,300

Note: The data are to May 2001; they exclude mines and underground workplaces.

2.6 Radon concentrations in outside air

The review by Wrixon et al (1988) considered concentrations of radon and its decay products in the UK outdoor air. Several authors have presented data which indicate that the mean outdoor radon concentration in the UK is about 4 Bq m^{-3}. This is lower than that in many other countries. The United Nations Scientific Committee on the Effects of Atomic Radiation quotes a world average of 10 Bq m^{-3} but with a wide range from 1–100 Bq m^{-3} (UNSCEAR, 2000). In general, more radon decay products are associated with a given radon gas concentration outdoors than indoors.

Outdoor radon concentrations can also be quite variable and higher concentrations will be found in still conditions, when radon escaping from the soil tends to be held close to the surface. In the UK, a few tens of becquerels per cubic metre might arise in such circumstances, and even higher concentrations in sheltered areas such as valleys. Much higher outdoor radon concentrations have been reported in other countries (see, for example, Robe et al, 1992, and Streck et al, 1999).

2.7 Radon in water

Radon is soluble in water, so high concentrations can arise where water comes into contact with uranium-containing minerals. As a result, high concentrations of radon can occur in domestic water supplies in some areas. Radon in domestic water could present a hazard in two ways. The radon can de-gas from the water, giving rise to exposure by inhalation. This can be a problem in some waterworks as well as in homes (Schmitz and Nickels, 2001). Alternatively, if the water is drunk before the radon de-gasses, it can give rise to doses to the gastrointestinal tract and to other body organs.

The concentration of radon in room air is generally about one ten-thousandth of the concentration of radon in tap water where water is the radon source (UNSCEAR, 1993; BEIR VI Committee, 1999). This means that to give rise to radon in room air at the UK average value of 20 Bq m^{-3} the concentration of radon in water would need to be about 200 Bq L^{-1} (becquerels per litre). This is much larger than the concentrations of radon normally encountered in water in the UK, so radon in water does not normally contribute materially to radon in indoor air in the UK.

Recommendations from the European Union (European Commission, 2001) propose a maximum concentration of 100 Bq L^{-1} for public water supplies and 1000 Bq L^{-1} for private supplies. Under typical circumstances, the latter will give rise to an air concentration of about 100 Bq m^{-3}. Controls on radon in drinking water in the UK have been summarised by Kendall (2004). A distinction is made between private and public supplies, with more stringent controls on the latter.

Radon concentrations are very low in most UK public water supplies (Henshaw et al, 1993). Two-thirds of UK water comes from reservoirs or rivers where radon concentrations are likely to be of the order of 1 Bq L^{-1} (Hesketh, 1980). Groundwater can contain a few tens of becquerels per litre and in isolated cases where higher concentrations have been found, water supplies have been aerated before being supplied to the public so as to remove radon and other gases.

Radon concentrations may be higher in private water supplies, particularly in the case of wells drilled into rock. A survey in West Devon examined water from 118 private supplies. Nine of these (8%) were found to have radon concentrations which exceeded the guideline level of 1000 Bq L^{-1} described above (BGS/DETR, 2000).

It should be noted that in some other countries – for example, Finland, Sweden and parts of the USA – much higher concentrations of radon in drinking water can be found.

3 Risks of Lung Cancer from Radon

Key Points

1 There is compelling evidence from epidemiological studies of radon-exposed miners, from animal experiments, and from dosimetric calculations that exposure to radon can lead to lung cancer. Based on this information, the International Agency for Research on Cancer classified radon as a class I carcinogen, ie that there was sufficient evidence of carcinogenicity in humans, in 1988.

2 Studies of people in their own homes now provide confirmation that radon is acting as a cause of lung cancer in the general population.

3 These studies provide powerful new evidence to help quantify the risk of residential radon exposure. The dose–response relationship appears linear, with no evidence of any threshold radon concentration below which there is no risk. There is also substantial evidence that there is a risk below 200 Bq m^{-3}, the current Action Level in the UK.

4 The association between the long-term average residential radon concentration and the risk of lung cancer found in a pooled analysis of individual data from 13 European studies is the best current basis for risk estimation. This analysis estimated that an increase of 100 Bq m^{-3} in the long-term average radon concentration in the home would cause an increase in the risk of lung cancer of between 5 and 31% with a central estimate of 16%.

5 A number of factors could not be taken into account in the recent pooled analysis of European studies. As a result, it is likely that the true risk from radon is, if anything, somewhat higher than the central risk estimate of 16% per 100 Bq m^{-3}.

6 The available evidence suggests that the same percentage increase in lung cancer risk per 100 Bq m^{-3} increase in radon concentration applies for men and women, across all age groups and for current smokers, ex-smokers and lifelong non-smokers.

The first human evidence that exposure to radon gas and its short-lived radon decay products could cause lung cancer came from groups of underground miners of uranium and some other igneous rocks who were occupationally exposed to substantial concentrations of radon (Appendix H). Analysis of 11 such studies based on more than 2000 lung cancer deaths (BEIR VI Committee, 1999) showed that radon had caused many hundreds of deaths from lung cancer among those studied, with an

approximately linear dose–response relationship both in smokers and in non-smokers. However, while the studies of miners have certain strengths, for reasons described below there is substantial uncertainty in extrapolating from these studies to obtain a quantitative assessment of the risk of lung cancer from radon in the home. Nevertheless, estimates of the probable doses in the home suggested indirectly that a material risk of lung cancer was likely. Dosimetric considerations also helped to indicate how quantitative information on risks, originally derived from occupational studies, might be applied to residential exposures (Appendix F).

Recently, epidemiological studies of residential exposure to radon and lung cancer have provided powerful new direct evidence on the public health hazard from radon. More than 20 separate studies of residential radon and lung cancer have been published (see Appendix I). Individually none of these studies was large enough to provide an estimate of the risk that was sufficiently precise to be useful. Much more precise, and therefore much more useful, estimates have been obtained recently by a series of pooled analyses, in which the individual data from several studies were collated centrally and then a single analysis of the combined data carried out. These pooled analyses provide the best and most direct evidence on the risks of exposure to residential radon. For this reason, and because they have been published relatively recently, we discuss them in this chapter and give a more detailed account in Appendix I. Other evidence comes from animal studies (Appendix G) and investigations of radiobiological mechanisms (Appendix E).

Three pooled analyses of the risk of lung cancer from residential exposure to radon have been undertaken: of thirteen European studies (Darby et al, 2005, 2006), of seven North American studies (Krewski et al, 2005, 2006) and of two Chinese studies (Lubin et al, 2004). These three major pooling studies, which all made use of the individual data from the original studies, are summarised in Table 3.1. For all three, results were reported in terms of the percentage increase in the risk of lung cancer per 100 Bq m^{-3} increase in residential radon exposure.

The exposure quantity of interest in the epidemiological studies was the *long-term average radon concentration* in the homes where an individual lived during the reference exposure window of 25 or 30 years where, for each home, the radon concentration has been averaged over all the years that the individual lived in it. This quantity is not directly available. Instead the quantity that is available for each individual is the *measured radon concentration*. This is usually obtained by taking measurements of a few months' duration in as many as possible of the homes in which the individual lived during the 25 or 30 years' reference exposure window and adjusting them for seasonal variation. If there are any homes for which it is not possible to obtain a measurement, an estimate needs to be constructed for that home. A weighted average can then be calculated of the measurements and estimates for each home, with weights proportional to the length of time that the individual lived there. Measured radon concentrations calculated in this way do not take into account the year-to-year variability that has been observed in several studies. As a result, analyses of data from the radon case–control studies that are based simply on measured radon concentrations, and ignoring this year-to-year variability, will underestimate the risk of radon-induced lung cancer, and special methods need to be employed to take it into account (see Appendix C for further discussion).

TABLE 3.1 Summary of estimates of the risk of lung cancer associated with residential radon in pooling studies that have combined individual data from a number of case–control studies

					% increase in risk of lung cancer per 100 Bq m^{-3} increase in radon concentration (with 95% CI)		
					Based on measured radon		Based on long-term average radon [(ii)]
Study	Number of studies included	Number of cases of lung cancer	Number of controls	Exposure window (years)	Including all subjects	Limited [(i)]	
European	13	7,148	14,208	5–34	8 (3–16)	9 (3–18)	16 (5–31)
North American	7	3,662	4,966	5–30	11 (0–28)	18 (2–43)	–
Chinese	2	1,050	1,995	5–30	13 (1–36)	32 (7–91) [(iii)]	–
Weighted average of above results of pooled analyses	22	11,860	21,169		10 (8–12)		~20 [(iv)]
Miners exposed at <0.5 WL [(vi)]	11	2,787	n/a		19 30 [(v)]		–

Notes

(i) Considering only individuals with one or two homes during the period of interest and for whom there was information on exposure for a period of at least 20 years.

(ii) Allowing for year-to-year random variation in residential radon concentrations.

(iii) Considering only individuals who had a single home during the period of interest.

(iv) Informal estimate, indicating the likely effect of removing the bias induced by random year-to-year variation in radon concentrations (see text).

(v) Considering only miners with cumulative exposures <50 WLM.

(vi) See Appendix A for definitions of WL and WLM.

3.1 European pooling study

The largest of the pooling studies to date is that based on the European studies (Darby et al, 2005, 2006). A total of more than 7,000 lung cancer cases and over 14,000 controls were included. The study examined the effect on lung cancer risk of exposures to radon during the 30-year period ending 5 years prior to the diagnosis of lung cancer (or prior to a comparable reference date for control individuals). Two features of the analysis were the very fine stratification used to allow for the effect of smoking and the allowance for year-to-year random variability that is present when the radon concentration in a home is measured in different years.

The European pooling study showed a clear association between increasing exposure to radon and lung cancer. There was no significant variation between the relative risks (ie the proportional increases in age-specific risk per unit increase in radon concentration) estimated for the component studies, and the results were not dominated by any single study. The proportional increase in risk per 100 Bq m^{-3} did not vary significantly with age, sex or smoking status. The risk appeared to be approximately linear with no evidence for a threshold below which there was no risk (Figure 3.1). Models in which there was no effect up to a threshold did not fit the data significantly better than a linear model; the upper 95% confidence limit for any possible threshold was 150 Bq m^{-3}. Furthermore, the investigators found a statistically significant association between radon concentrations and lung cancer even when analysis was restricted to people in homes with measured radon concentrations below 200 Bq m^{-3} ($p = 0.04$). When individuals with measured radon in the range 100–199 Bq m^{-3} (mean 136 Bq m^{-3}) were compared with those with measured radon below 100 Bq m^{-3} (mean 52 Bq m^{-3}), their risk of lung cancer was increased by 20% (95% confidence interval, CI, 3–30%, $p = 0.01$).

When the analysis was conducted in terms of measured radon concentrations, it was estimated that the risk of lung cancer increased by 8% per 100 Bq m^{-3} increase in measured radon concentration (95% CI 3–16%).

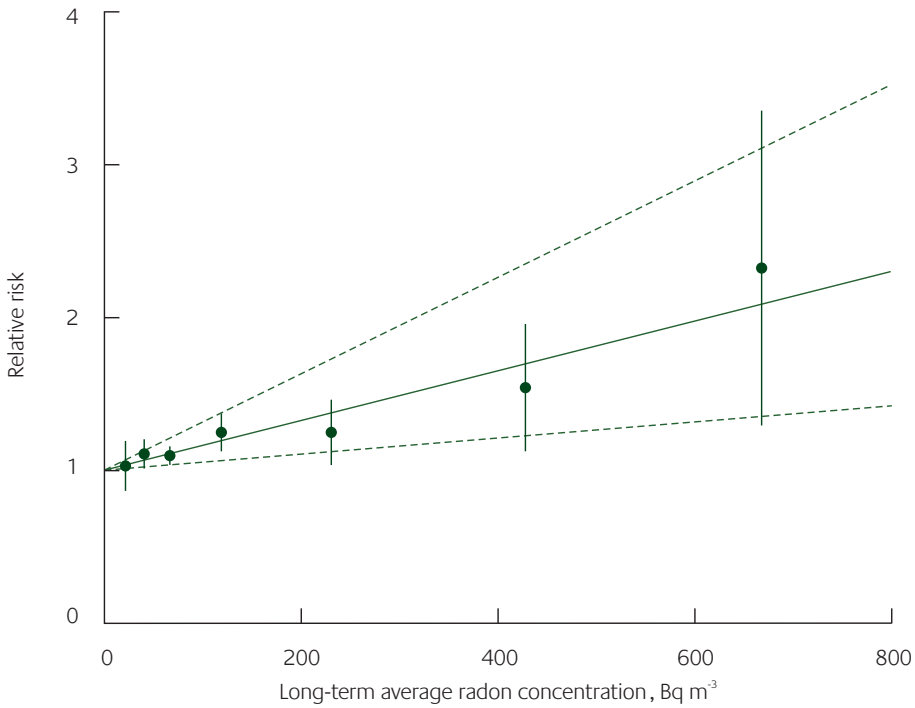

FIGURE 3.1 Relative risk of lung cancer versus long-term average residential radon concentration in the European pooling study, based on Darby et al (2005, 2006). Relative risks and 95% confidence intervals are shown for categorical analyses and also the best-fitting straight line. Risks are relative to that at 0 Bq m^{-3}

However, when the analysis was repeated using long-term average radon concentrations (ie taking into account the random year-to-year variability in residential radon concentrations), the final estimated risk was higher, at 16% (95% CI 5–31%).

When the risk estimate was calculated considering only individuals with complete exposure histories (ie measurements for all the homes where the individual had lived during the 30-year reference period), the value was very similar to that based on all the individuals in the study.

3.2 North American pooling study

Krewski and co-workers (2005, 2006) published a pooled analysis which involved 3662 cases and 4966 controls from seven studies in Canada and the USA. The reference period considered was the 25-year period ending 5 years before the diagnosis of lung cancer, slightly shorter than in the European pooling. In this analysis there was also detailed allowance for smoking, although the stratification was not as fine as that used in the European pooling.

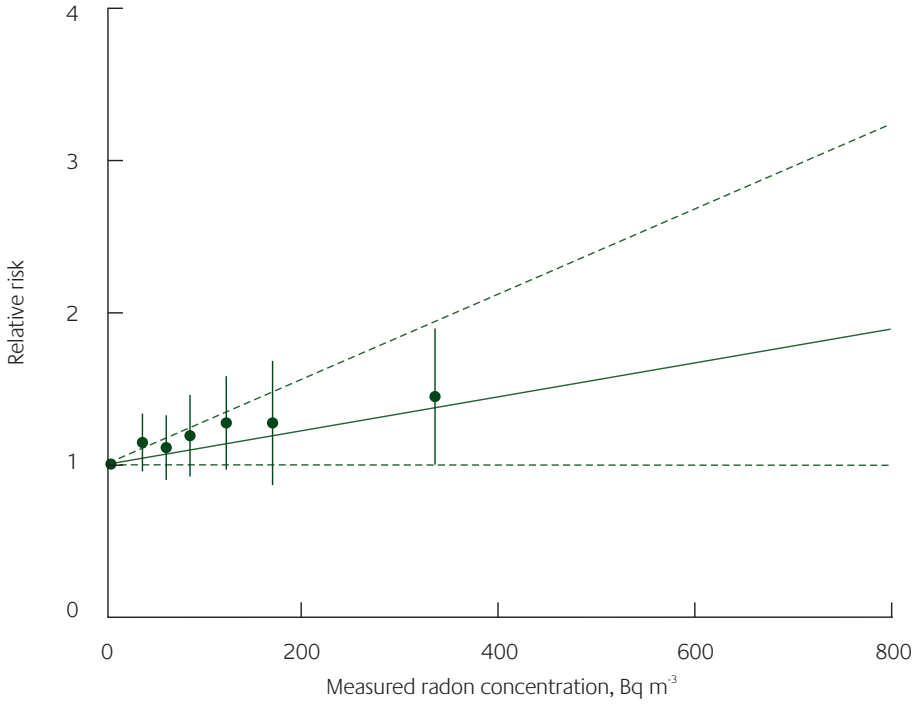

FIGURE 3.2 Relative risk of lung cancer versus measured residential radon concentration in the North American pooling study, based on Krewski et al (2005). Relative risks and 95% confidence intervals are shown for categorical analyses and also the best-fitting straight line. Risks are relative to those in the category <25 Bq m^{-3}

When compared with individuals whose measured radon concentrations were below 25 Bq m^{-3}, individuals in various categories of higher measured radon concentration had increased risks of lung cancer, but for no individual category was the increase statistically significant (see Figure 3.2). The estimated increase in risk per 100 Bq m^{-3} measured radon concentration was 11% (95% CI 0–28%). There was no significant heterogeneity between the risk coefficients estimated for the component studies, and the overall estimate did not change substantially when any of the studies was excluded from the analysis.

Krewski et al also considered analyses restricted to various subgroups. When the analysis included only individuals for whom radon exposure histories were more complete, in that monitoring data were available for at least 20 years out of the 25-year exposure time window, the increase in risk per 100 Bq m^{-3} measured radon concentration was 14% (95% CI 1–35%). A similar result was obtained by restricting attention to those who had lived in only one or two homes. This differs from the finding in the larger European pooling, where the risk estimate for a subgroup analysis including only individuals with at least 20 years' coverage was similar to the overall estimate.

There was no significant variation in the estimated proportional increase in risk per 100 Bq m^{-3} with smoking status, sex, age or educational level.

3.3 Chinese pooling study

Lubin and co-workers (2004) published a study which involved 1050 cases and 1996 controls from studies in two areas in China: Gansu and Shenyang. The first of these studies was much the larger. As with the North American pooling, the reference period considered was 5–30 years before diagnosis. Four smoking categories were considered in the analysis.

For the pooled data, the increase in risk per 100 Bq m^{-3} was 13.3% (95% CI 1–36%). This positive slope was chiefly due to the Gansu data, although the results of the two component studies were compatible with each other. For subjects resident in only one home for the whole of the 25-year reference period, the increase in risk per 100 Bq m^{-3} measured radon was 32% (95% CI 7–91%) based on the pooled data.

As with the European and North American poolings, there was no significant difference between the estimated proportional increase in risk per 100 Bq m^{-3} calculated for the different smoking categories.

The risk estimate was higher in analyses restricted to those study subjects who had personally provided information on other possible risk factors such as smoking, excluding subjects for whom the information had been obtained from a surrogate (ie somebody other than the study subjects themselves).

3.4 Overall appraisal of risk of residential exposure to radon based on the three pooling studies

The best evidence of the risks of residential radon exposure would come from a pooled analysis of individual data from all the residential studies, including all those in the three pooling studies described above. Such a world pooling is currently under way, but its results will not be available for some time.

The three major pooling studies reported increased risks of lung cancer based on measured radon concentrations of 8% (95% CI 3–16%), 11% (0–28%) and 13% (1–36%) per 100 Bq m^{-3} (Table 3.1). As these three estimates are statistically compatible with each other, a weighted average of them, with weights proportional to their variances, can be calculated. This gives a joint estimate from the three pooling studies, based on measured radon concentrations, of 10% per 100 Bq m^{-3}.

In each of the three major pooling studies the investigators also reported alternative analyses. All the studies considered subsets of the data for individuals for whom measurements were more complete in the exposure time window or who had lived in only one or two homes. In the case of the North American and Chinese poolings, these restricted analyses resulted in higher risk estimates than did the analysis including the entire data, but in the case of the larger European pooling the results of the restricted analysis were very similar to those of the analysis including the entire data. The authors of the North American and Chinese poolings argued that these restricted analyses gave more reliable risk estimates. This was on the basis that exposure estimates based on direct measurements in the homes in question are better than those which include imputed values and that it is easier to make reliable estimates of exposure for individuals who have lived in few homes. However, it should be noted that those who respond to requests for measurements in their homes may differ in, for example, socioeconomic status from those who do not.

As described above, estimates based on measured radon concentration will underestimate the true risks associated with residential radon exposure, due to the year-to-year random variation in radon concentrations in a home. The only pooling study that has to date carried out a detailed analysis of the risks of residential radon based on long-term average, as opposed to measured, radon concentrations, is the European pooling. In this study, the estimate based on long-term average concentrations was double the estimate based on measured radon concentrations. Data from repeated radon measurements made in separate years in the same home in China show a similar amount of variation to that seen in the European studies (Darby et al, 2005; Lubin et al, 2005), suggesting that an estimate from the Chinese study based on long-term average radon concentration would also be approximately double the estimate based on measured radon. No data from North America on repeated measurements made on different occasions in the same home are available at present, but it seems likely that the association with measured radon there also underestimates the relationship between lung cancer and long-term average radon concentration.

Darby et al (2005, 2006) noted a number of factors that could not be included in the analysis of the pooling studies. In particular, there would have been errors in the assignment of individuals to smoking categories, variation in the radon concentration between the different rooms in a home and, in some countries, there may have been systematic changes in the radon concentrations over the last few decades, due to increased energy efficiency. There may also be some element of risk resulting from exposure to radon outside the 25- or 30-year exposure time window considered. The overall effect of these factors may mean that the true effect of radon is somewhat higher than the estimated risk in the European pooled analysis, even after correction for year-to-year random variation in measured radon concentrations, and also higher than the risks reported in the other two pooling studies.

The three pooling studies present very similar estimates of the proportionate risk of lung cancer from residential exposure to radon. They provide overwhelming evidence that radon is acting as a cause of lung cancer in the general population and at concentrations found in ordinary homes. In particular, there is substantial evidence that there is a risk even below 200 Bq m^{-3}, the concentration at which action is currently advocated in many countries. In the European pooling the dose–response relationship appeared linear, with no evidence of a threshold dose (Figure 3.1). The results of the North American pooling were also consistent with a linear dose–response relationship with no threshold, although greater uncertainty was involved (Figure 3.2).

On the basis of the pooled analyses of residential studies, the relative risk of radon exposure appears to be similar between sexes, across age groups and between current smokers, ex-smokers and lifelong non-smokers.

3.5 Summary of all evidence on risks of lung cancer

In addition to the studies of residential radon exposure described above, there is overwhelming evidence from epidemiological studies of radon-exposed miners and from animal experiments (reviewed in Appendix G) that exposure to radon and its decay products leads to an increased risk of lung cancer. Evidence from radiobiology considered in Appendix E and dosimetric calculations described in Appendix F support this conclusion and assist in interpreting the implications of the occupational studies for the risks of environmental exposure to radon. In this chapter we consider how quantitative estimates of lung cancer risks may be derived in the light of all these sources of information in order to help determine policies for control of radon as a public health hazard.

As described in Appendix H, a major review of the data on miners was undertaken by the US Committee on the Biological Effects of Ionizing Radiation. Its report, published in 1999, is known as BEIR VI Report on the health effects of radon (BEIR VI Committee, 1999). The BEIR VI Committee considered 11 major studies, covering a total of over 60,000 miners in Europe, North America, Asia and Australia, among whom over 2,500 deaths from lung cancer had occurred. These studies all included quantitative information on the radon exposures received by the men and many of them also included information on unexposed workers, such as surface workers, as an internal comparison group. The BEIR VI Committee presented two models to quantify the induction of lung cancer by exposure to radon and these are described in Appendix H. In general, the exposures received in mines have been higher and received over a shorter period than those received in homes. However, it should be noted that more recent research has focused on groups of European uranium miners who received comparatively low exposures at relatively low exposure rates when compared with other groups of miners (Tirmarche et al, 2003). Work is continuing to assess the risks of lung cancer and of other types of cancer amongst European uranium miners, whilst taking account of smoking habits and of other exposures in these mines (eg from uranium dust and from gamma radiation).

Dosimetric calculations described in Appendix F also indicate that inhaled radon decay products, attached to particles in the atmosphere, will largely be deposited on the internal surfaces of the lung and other parts of the respiratory tract. The decay products have radioactive half-lives of a few tens of

minutes and a large majority of them will decay before they can be cleared from the lung. The respiratory tract therefore receives much the largest dose of any organ or tissue. The calculation of doses to the lung from radon decay products is complex, but estimates of dose to the lung correspond to a level of risk broadly comparable with that observed in epidemiological studies. Calculations for different conditions indicate that the absorbed dose to the lung per unit exposure to radon decay products is similar in mines and homes. This gives some confidence that measured risks due to radon exposure in mines may be a guide to the risks of residential exposure, although the miners were in many cases also exposed to other occupational lung carcinogens, such as arsenic, which complicates extrapolation from the miner studies to the effects of residential exposures. In addition, the smoking habits and tobacco-related risks in the published studies of miners would be substantially different from present-day habits and risks.

All the sources of information listed above have their strengths. It is also true that each paints a broadly similar picture. Nevertheless, in deciding upon a quantitative estimate of the risks of residential exposure to radon, we have depended little on the data from animal experiments. While these involved carefully measured exposures and allowed investigation of the possible effects of co-carcinogens, in particular tobacco smoke, many of these studies were conducted at radon concentrations higher than those of interest in a residential context. In addition, the relevance of tobacco-related risks in laboratory animals to those in human populations is unclear. Moreover, the differences observed between different animal species and strains emphasise the difficulties in extrapolating these data to humans (for further details, see Appendix G).

There is no doubt that the best and most direct quantitative risk estimates are based on epidemiological studies of humans, rather than on animal studies. Nevertheless, it is inevitable that there will be a low dose region, where the statistical uncertainties are too large for epidemiology to provide direct evidence. As we have noted above, direct evidence now extends down to radon concentrations below 200 Bq m^{-3}. The usual assumption in radiological protection is that radiation risks are proportional to the dose, decreasing as doses decrease, but without any threshold below which risks are zero. We are convinced by the evidence from radiobiology outlined in Appendix E that this assumption is appropriate for radon exposures and, if it is true, then the majority of radon-induced deaths will occur at concentrations below 200 Bq m^{-3} (see Chapter 4).

For reasons detailed in Appendix I, we have also leant more heavily on the new pooled analyses of the residential studies than on the studies of miners which have so far been published. Particularly important factors in this judgement were the detailed allowance for the effects of tobacco smoking in the residential studies and the fact that exposure conditions in mines differ from those in homes. Allowance for uncertainties in the measurement of radon concentrations is also a very important matter and we note that the fullest allowance for this is made in the European pooling.

In summary, we judge that the magnitude of the risk from radon is such that exposure to 100 Bq m^{-3} for 30 years increases the risk of lung cancer by 5 to 31% with a central risk estimate of around 16%. The latter is more likely to be an underestimate than an overestimate.

The residential studies suggest that this relative risk does not vary with age, sex or smoking status. Despite the evidence from the miner studies for a higher relative risk in non-smokers and for a relative risk which declines after the age of 50 years, on balance we prefer to follow the residential studies.

For a given radon concentration, the absolute risk of lung cancer is much higher in smokers than in lifelong non-smokers so the absolute magnitude of the additional risk caused by radon is much larger in smokers than in non-smokers. The evidence from epidemiological studies of miners exposed to radon suggests that the main part of the risk is expressed within 30 or 40 years following exposure.

At long-term average radon concentrations of 0, 100, 200, 400 and 800 Bq m^{-3}, the cumulative *absolute* risks of lung cancer by age 75 years would be about 0.4, 0.5, 0.5, 0.7 and 0.9%, respectively, in lifelong non-smokers, and 15, 17, 19, 23 and 30%, respectively, in current cigarette smokers in the UK. For recent ex-smokers, the risks would be somewhat lower than those for current smokers, while for long-term ex-smokers, the risks would be close to those for lifelong non-smokers.

4 Number of Radon-induced Lung Cancers in the UK

Key Points

1 It is estimated that 3.3% of lung cancer deaths in the UK are attributable to residential radon exposure. This corresponds to around 1100 deaths each year out of the annual total of around 34,000 lung cancer deaths.

2 Of the 3.3% of lung cancer deaths attributable to residential radon exposure, only 0.5% are due to radon acting alone. The remaining 2.8% are caused both by radon and by active smoking in the sense that the lung cancer would have been avoided if the person concerned either had never smoked or had never been exposed to radon. Nearly half of the deaths caused both by radon and by smoking are likely to occur in people who have already given up smoking.

3 At the national average long-term residential radon concentration of 21 Bq m^{-3}, the cumulative risk of death from lung cancer by the age of 75 years is 0.4% for a lifelong non-smoker and 15% for a cigarette smoker. At 200 Bq m^{-3}, these risks rise to 0.5 and 19%, respectively.

4 For smokers, much of the risk of radon-induced lung cancer as well as of smoking-induced lung cancer can be avoided by giving up smoking. For an individual who stops at age 30 years, the risks are 1.6 and 2.1% at 21 and 200 Bq m^{-3}, respectively, while for an individual who stops at age 50 years they are 6 and 7% at 21 and 200 Bq m^{-3}, respectively.

5 Only around 0.4% of homes in the UK are thought to have measured radon concentrations of 200 Bq m^{-3} or more. Consequently, only around 4% of the radon-attributable lung cancer deaths that occur in the UK each year are likely to occur in association with measured radon concentrations of 200 Bq m^{-3} or more.

6 The overwhelming majority of the population live in homes where the measured radon concentrations would be much less than 200 Bq m^{-3}. As a consequence, about 70% of radon-attributable deaths are estimated to occur with residential radon concentrations of less than 50 Bq m^{-3}, around 17% at concentrations in the range 50–99 Bq m^{-3} and a further 9% in the range 100–199 Bq m^{-3}. Thus the vast majority of radon-induced lung cancer deaths occur in areas not currently designated as 'radon Affected Areas'.

We consider that the most appropriate approach to calculating the probable numbers of deaths caused in the general population of the UK by exposure to radon at home is to use the risk estimate obtained in the analysis in which the data from the European studies of residential radon and lung cancer have been pooled (see Chapter 3). This amounts to assuming that the risk of lung cancer increases by 16% (95% confidence interval, CI, 5–31%) for each 100 Bq m^{-3} increase in the long-term average residential radon concentration. This percentage increase is applicable regardless of smoking history. There are reasons to believe that the true risk may be somewhat higher than that indicated by the European pooling study, but a better estimate cannot yet be made from the studies of residential radon. The risk estimate was, however, somewhat higher in the studies of radon-exposed miners than in the European pooling study. We therefore also present results where the risk of lung cancer from residential radon has been estimated indirectly, from the studies of radon-exposed miners (BEIR VI Committee, 1999). Based on the NRPB representative national study of the distribution of radon concentrations (see Chapter 2), we consider that the best estimate of the mean long-term average residential radon concentration is 21 Bq m^{-3}.

In order to understand the implications of these estimates for the number of deaths attributable to radon in the UK each year, we have considered them in conjunction with the numbers of people and the numbers of deaths from lung cancer for males and females in different age groups in the UK (Cancer Research, 2008), together with the estimated ratios of the lung cancer death rate in current and in ex-smokers in the UK compared with lifelong non-smokers (Peto et al, 2000), and lung cancer death rates among lifelong non-smokers for males and females of different ages. The best estimates of the lung cancer death rates in lifelong non-smokers are those obtained from the American Cancer Society's prospective study (Thun et al, 2006). These have been adjusted to take into account the lower average residential radon concentrations of 21 Bq m^{-3} in the UK compared with over 40 Bq m^{-3} in the USA (UNSCEAR, 2000). As survival after a diagnosis of lung cancer is very poor, with only around 5% of those diagnosed with the disease surviving for five years, projections for lung cancer incidence would be very similar to those given here for mortality.

These calculations suggest that out of the total of 34,150 lung cancer deaths that occurred in the UK during 2006, an estimated 1110, or 3.3%, were caused by residential radon exposure (see Table 4.1). Of these, only 157 (0.5% of all lung cancer deaths) can be attributed to radon acting alone, while the remaining 953 (2.8% of all lung cancer deaths) were caused by both radon and smoking in the sense that the lung cancer could have been avoided by avoiding either smoking or radon exposure. When this calculation is repeated using the radon risks estimated indirectly from the studies in miners, the estimated number of radon-induced lung cancer deaths is somewhat larger, at 2044 or 6.0% of the total. However, once again, the estimates suggest that the majority of these deaths were caused by radon acting in conjunction with active smoking.

Lung cancer in the UK is primarily attributable to cigarette smoking. In the past, men have smoked more than women, and so lung cancer death rates at present are higher in males than in females; as a result, nearly 60% of the deaths attributable to radon are estimated to have occurred in males, with just over 40% in females (see Table 4.2). It is likely that about half of the radon-induced deaths occurred in individuals between the ages of 55 and 75 years, with most of the remainder occurring in individuals over age 75 years. Only a small proportion of radon-induced deaths are likely to occur at ages less than 55 years, and very few in individuals under 35 years.

TABLE 4.1 Causes attributable to the lung cancer deaths in the UK in 2006 [i]

(a) Based on direct risk estimate [ii]

Cause	Number of deaths	% attributed
Not caused by active smoking or by residential radon	4,664	13.6
Caused by radon but not by active smoking	157	0.5
Caused both by active smoking and by radon [iv]:		
in current smokers	532	1.6
in ex-smokers	421	1.2
Caused by active smoking and not by radon	28,376	83.1
Total UK lung cancer deaths in 2006	34,150	100.0

↑ *3.3% due to radon* ↓

↑ *85.9% due to active smoking* ↓

↑ *86.4% due to active smoking or radon* ↓

(b) Based on indirect risk estimate [iii]

Cause	Number of deaths	% attributed
Not caused by active smoking or by residential radon	3,627	10.6
Caused by radon but not by active smoking	370	1.1
Caused both by active smoking and by radon [iv]:		
in current smokers	948	2.8
in ex-smokers	726	2.1
Caused by active smoking and not by radon	28,479	83.4
Total UK lung cancer deaths in 2006	34,150	100.0

↑ *6.0% due to radon* ↓

↑ *88.3% due to active smoking* ↓

↑ *89.4% due to active smoking or radon* ↓

Notes

(i) Calculation based on UK 2006 national data for numbers of lung cancer deaths and population size (Cancer Research, 2008). Lung cancer death rate in lifelong non-smokers taken from an American prospective study of mortality (Peto et al, 1992), adjusted for the lower residential radon concentrations in the UK.

(ii) Using risk coefficient from studies of residential radon (Darby et al, 2005, 2006, and Appendix I).

(iii) Using risk coefficient from studies of radon-exposed miners (BEIR VI Committee, 1999, and Appendix I).

(iv) That is, avoidance of either active smoking or exposure to radon would have avoided that particular lung cancer.

TABLE 4.2 Lung cancer deaths attributable to residential radon exposure in the UK in 2006 by age and sex

	Method of estimation					
	Based on direct risk estimate [(i)]			Based on indirect risk estimate [(ii)]		
Age group	Males	Females	Total	Males	Females	Total
<35	~0.5	~0.5	1 (<0.1%)	~2	~1	~3 (<0.2%)
35–54	35	29	64 (6%)	172	148	320 (16%)
55–74	312	216	526 (48%)	769	559	1328 (65%)
75+	290	227	517 (46%)	213	179	393 (19%)
All ages	637 (58%)	473 (42%)	1110 (100%)	1156 (57%)	888 (43%)	2044 (100%)

Notes
(i) Using risk coefficient from studies of residential radon (Darby et al, 2005, 2006, and Appendix I).
(ii) Using risk coefficient from studies of radon-exposed miners (BEIR VI Committee, 1999, and Appendix I).

TABLE 4.3 Cumulative absolute risk of death from lung cancer to age 75 years in the UK by smoking history and long-term average residential radon concentration

Long-term average radon concentration (Bq m^{-3})	Cumulative risk of death from lung cancer to age 75 years (%) [(ii,iii)]			
	Lifelong non-smoker	Ex-cigarette smoker: stopped at age 30	Ex-cigarette smoker: stopped at age 50	Continuing cigarette smoker
0	0.41	1.57	5.5	14.7
21 [(i)]	0.42	1.62	5.7	15.2
100	0.47	1.8	6.4	16.9
200	0.53	2.1	7.2	19.0
400	0.66	2.6	8.9	23.0
800	0.92	3.5	12.2	30.5

Notes
(i) Mean UK long-term average residential radon concentration.
(ii) Radon risk estimated directly from European pooling study (Darby et al, 2005, 2006, and Appendix I). Lung cancer death rate in male lifelong non-smokers from American Cancer Society CPS-II (Thun et al, 2006). Lung cancer risks in continuing and ex-cigarette smokers from males in the UK case–control study of lung cancer (Peto et al, 2000) and the UK 2006 national lung cancer death rate (Cancer Research UK, 2008).
(iii) Cumulative risks ignore the risk of deaths from other causes. If, for one particular category, the lung cancer rates per 100,000 people in all the five-year age groups before age 75 years add up to c, then the cumulative risk by age 75 is $1 - \exp(-5c/100{,}000)$. Thus, cumulative risks depend only on age-specific lung cancer rates and not on competing causes of death.

For any individual, the risk of dying from lung cancer will be determined both by smoking history and by past exposure to residential radon. The way in which these two factors influence the risk can be seen when lung cancer mortality rates for individuals with different smoking histories and residential radon concentrations are considered. For lifelong non-smokers, the cumulative risk to age 75 years of death from lung cancer for an individual who has lived all their life in a home with a radon concentration equal to the national long-term average of 21 Bq m^{-3} is estimated to be 0.42% (see Table 4.3). This would be reduced slightly, to 0.41%, if the residential radon concentration were, hypothetically, brought down to zero, and would rise to 0.53% for a lifelong non-smoker who had lived for a long time in a home with a radon concentration of 200 Bq m^{-3}, and further to 0.66 and 0.92% for lifelong non-smokers who had lived for a long time in homes with radon concentrations at 400 and 800 Bq m^{-3}, respectively. For cigarette smokers, the cumulative risk of death from lung cancer by age 75 years is around 15% when the residential radon concentration is equal to the UK average, but rises to around 19% for cigarette smokers exposed at 200 Bq m^{-3}, and to 23 and 30% for continuing smokers exposed at 400 and 800 Bq m^{-3}, respectively. For those who have successfully stopped smoking, the risks are substantially lower than for those who continue to smoke, but they remain considerably above the risks for lifelong non-smokers (see Table 4.3 and Figure 4.1).

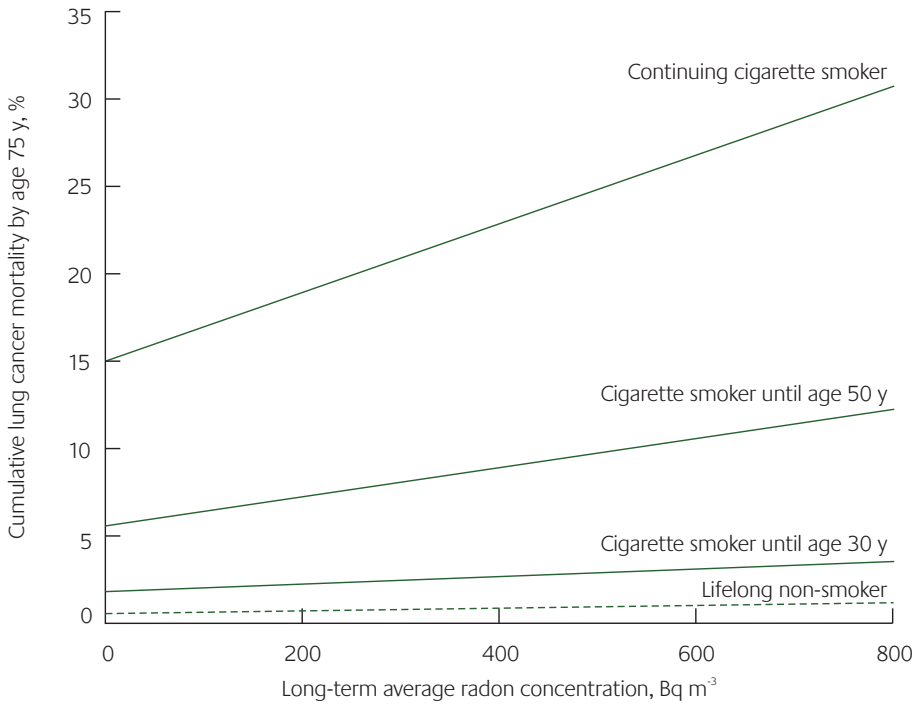

FIGURE 4.1 Cumulative absolute risk of death from lung cancer by age 75 years versus long-term average radon concentration at home for continuing cigarette smokers, ex-smokers and lifelong non-smokers in the UK (see Table 4.3 for sources of data and method of calculation)

The number of deaths attributable to residential radon at each level of concentration is determined not only by the risk associated with a given concentration, but also by the number of homes with concentrations at that level. For the UK, residential radon concentrations have been shown to follow a log-normal distribution after allowance for the radon concentration in outside air, which is around 4 Bq m^{-3} and varies relatively little (see Figure 2.1 and Appendix C). These data suggest that around 0.2% of homes in the UK have a long-term average radon concentration of 200 Bq m^{-3} or more. Consequently, the proportion 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 3%, with another 8% occurring at concentrations in the range 100–199 Bq m^{-3} – see Table 4.4(a). In contrast, around 70% of radon-attributable deaths are estimated to occur following exposure to radon concentrations of less than

TABLE 4.4 Lung cancer deaths attributable to radon in the home in the UK in 2006

Range of long-term average radon concentrations (Bq m^{-3})	% of homes in range	Deaths attributable to residential radon		
		Number		
		Direct [i]	Indirect [ii]	Percentage [iii]
(a) By long-term average radon concentration [iv]				
0–49	93.0	790	1455	71.2
50–99	5.5	197	362	17.7
100–199	1.3	89	164	8.0
200+	0.2	34	63	3.1
All UK homes	100.0	1110	2044	100.0
(b) By measured radon concentration				
0–49	91.2	776	1428	69.9
50–99	6.5	191	354	17.3
100–199	1.9	97	178	8.7
200+	0.4	46	84	4.1
All UK homes	100.0	1110	2044	100.0

Notes
(i) Using risk coefficient from studies of residential radon (Darby et al, 2005, 2006, and Appendix I).
(ii) Using risk coefficient from studies of radon-exposed miners (BEIR VI Committee, 1999, and Appendix I).
(iii) Percentages for direct and indirect calculations are identical.
(iv) Based on the national representative survey of radon concentrations (see Appendix C, Section C4, for further details).

50 Bq m^{-3}, and nearly 20% following exposure to concentrations in the range 50–99 Bq m^{-3}. These percentages are based on long-term average radon concentrations (ie they take into account the year-to-year variation in measured radon concentrations). If they are re-calculated based on radon measurements taken in a single year, they change only slightly – see Table 4.4(b). Just over 4% of radon-induced deaths are estimated to occur in homes with a measured radon concentration of 200 Bq m^{-3} or above, while over two-thirds occur in homes with measured radon concentrations below 50 Bq m^{-3}.

Only around one-quarter of radon-induced deaths are likely to occur in areas currently designated as 'radon Affected Areas' (see Table 4.5). The vast majority of radon-induced lung cancers are likely to occur in other areas and to be widely scattered throughout the country. Even within the areas currently designated as radon Affected Areas, it is likely that only around one in eight of radon-induced deaths occurs in homes where the measured radon concentrations would be above 200 Bq m^{-3}, with the remainder occurring as a result of concentrations below 200 Bq m^{-3} (see Table 4.5).

TABLE 4.5 Estimated numbers of radon-induced lung cancers occurring each year according to whether or not they are likely to be in a radon Affected Area and, for those that are, whether or not they are likely to have been identified by a radon measurement above the current Action Level of 200 Bq m^{-3}. (See Appendix C, Section C4, for details of assumptions in the calculation)

Area	Number of households	Estimated number of radon-induced deaths each year [(i)]
Whole UK	24,900,000	1,110 (100%)
Not in radon Affected Areas	22,300,000	833 (75%)
In radon Affected Areas	2,600,000	277 (25%)
In radon Affected Areas with measurement <200 Bq m^{-3}	2,500,000	241 (22%)
In radon Affected Areas with measurement ≥200 Bq m^{-3}	80,000	36 (3%)

Note: (i) Based on direct estimate of the risk of radon-induced lung cancer and the national representative survey of radon concentrations (see Appendix C, Section C4, for further details).

5 Effects of Radon Exposures Other than Lung Cancer

Key Points

1 The doses from both inhaled radon decay products and radon gas are overwhelmingly to the respiratory tract with doses from the former outweighing the latter. Doses to other organs are very small in comparison.

2 Ingestion of radon gas and its decay products can lead to radiation exposure. Ingested radon gas, dissolved in water, gives a higher dose than ingested decay products. In both cases the stomach receives the largest dose. There is little evidence from epidemiology to demonstrate any risk of stomach cancer that is attributable to radon.

3 Deposition of radon decay products on intact skin may possibly lead to skin cancers. However, calculated doses to skin are critically dependent on the position of the sensitive cells and further work is required before any firm conclusions can be reached. Cancer registries generally have incomplete coverage of non-melanoma skin cancers, which makes such cancers difficult to investigate in epidemiological studies.

4 If radon and its decay products do have effects on organs other than the lung, the effect is so weak as to be undetectable in the published epidemiological studies.

The main risk caused by exposure to radon and its decay products is lung cancer, as discussed in Chapter 3. However, other organs and tissues also receive radiation doses from radon and its decay products, and it is possible that cancers or other harm may be induced. This chapter discusses the evidence for such additional risks. The evidence comes from estimation of risks based on calculated doses, from epidemiological studies and from studies of animals. Fuller discussions of these matters can be found in Appendices J and K. The growing body of understanding about the mechanisms by which radiation causes malignant and other diseases is also relevant and is discussed in Appendix E.

5.1 Evidence from dose calculations

It is usual in radiological protection to estimate the harm caused by radiation in two stages: first to calculate the radiation doses to organs or tissues of interest, and then to estimate the probable effects using standard risk factors. These risk factors come from several sources of information, notably studies of the survivors of the atomic bombings in Japan, which are reviewed by agencies such as the ICRP and UNSCEAR.

TABLE 5.1 Annual doses (mSv) to organs and tissues from inhalation and from ingestion of radon and its decay products. The doses shown are committed equivalent doses to organs and committed effective doses. Details of the calculation are in Appendix J

	Exposure to air containing radon at 200 Bq m^{-3} with radon decay products (F = 0.41)			Ingestion of water containing 1000 Bq L^{-1} of radon with decay products	
Organ/tissue	**Decay products Type F**	**Decay products Type M**	**Radon gas**	**Decay products**	**Radon gas**
Lung	35.8	159	1.2	0.01	1.26
ET airways (i)	44.5	70.9	0.42	0.02	0.04
Stomach	0.19	0.08	0.06	1.15	50.4
Small intestine	0.17	0.05	0.06	0.51	2.6
Colon	0.16	0.02	0.05	0.13	0.1
Red bone marrow	0.28	0.03	0.65	0.03	0.66
Bone surfaces	1.48	0.17	0.03	0.12	0.03
Liver	0.43	0.05	0.09	0.04	0.57
Breast	0.15	0.02	0.42	0.01	0.44
Kidney	5.20	0.54	0.05	0.25	0.05
Gonads	0.15	0.02	0.05	0.02	0.05
Brain	0.15	0.02	0.06	0.01	0.06
Bladder	0.21	0.02	0.05	0.02	0.05
Muscle	0.15	0.02	0.05	0.02	0.05
Effective dose	**5.30**	**19.7**	**0.28**	**0.17**	**6.00**
Fetus	0.06	0.01	0.04	0.01	0.06
Skin	25	25	–	–	–

Note: (i) ET airways are the extrathoracic airways, ie nose, mouth and throat.

The assessment of doses from radon and its decay products is discussed in detail in Appendices G and J. Appendix G deals with modelling doses to the respiratory tract and Appendix J with doses to other organs and tissues. The main routes of intake are through inhalation or ingestion of radon and its decay products, and the main results of the calculations are shown in Table 5.1. This table gives doses for inhalation and ingestion of radon at the recommended Action Levels for radon in air and water; most exposures will be smaller, particularly so far as ingestion is concerned. Calculations of doses from inhalation of short-lived radon decay products are shown for two standard assumptions about the solubility of the decay

products. These assumptions are known as Type M and Type F behaviour, the names coming from Moderate or Fast solubility. The range of doses for Type F and Type M gives an indicative spectrum of doses from radon decay products. Further details of the calculations are in Appendix J.

Much the largest doses in Table 5.1 are from inhalation of the decay products, confirming that lung cancer is the largest risk. Inhaled radon decay products can enter the bloodstream, although the rate at which this occurs is uncertain. Doses to other organs and tissues are nevertheless relatively small. Inhaled radon gas is mostly expelled with exhaled air, but some dissolves in the bloodstream and is carried round the body. Because radon has a relatively high solubility in fat, it gives significant radiation doses to tissues with a high fat content, including red bone marrow.

Radon is soluble in water, and under some circumstances relatively high concentrations can be found in drinking water. The calculations indicate that, both for radon gas and for its decay products, it is the lining of the stomach that receives the highest dose from ingested radon. In contrast to the situation for inhalation, ingested radon gas is retained in the stomach rather than being immediately lost, and radon gas gives bigger doses than its decay products.

Another way in which people receive radiation doses from radon is as a result of the deposition of decay products on the skin. Because the important radiation consists of short-range alpha particles, much of the radiation will be absorbed in the dead outer layers of the skin, but some may reach the cells in which skin cancers originate. There is some uncertainty about the depth at which these sensitive cells lie, and it is therefore not possible to make reliable estimates of the doses delivered by this route. Factors such as air movement and electromagnetic fields affect the deposition of radon decay products on the skin, and so affect skin doses.

These calculations give an indication of organs and tissues which may be at risk. However, it should be remembered that the results of such calculations are approximate, particularly where short-range radiation delivers doses to sensitive cells which form a small target, a circumstance which often applies to radon decay products. It is important to look for confirmation of any risk from other sources.

5.2 Evidence from epidemiology

The epidemiological evidence for possible effects of radon apart from lung cancer is discussed in detail in Appendix K.

There have been several studies of radon and childhood cancer. Some ecological studies (ie studies based on geographical correlations) have reported positive associations between childhood cancers and radon (see, for example, Alexander, 1990, Henshaw et al, 1990a,b, and Evrard et al, 2005). However, as discussed in Appendix I, ecological studies have substantial drawbacks as a tool for the study of radon-induced cancer and we generally give little weight to them. In particular, for studies of radon and childhood cancer the characteristics of individual dwellings have a substantial effect on radon concentrations in homes, so average concentrations in an area are poor indicators of individual exposure (Kendall et al, 1994). In addition, there is evidence that geographical confounding factors can influence the direction of the relation between childhood cancer and area averages of radon concentration (Muirhead et al, 1991, 1992).

Large case–control studies of individuals have been carried out in Great Britain (UK Childhood Cancer Study (UKCCS) Investigators, 2002) and in the USA (Lubin et al, 1998; Steinbuch et al, 1999) and a somewhat smaller study in Germany (Kaletsch et al, 1999). The UK Childhood Cancer Study was the largest of these studies with 2226 cases of childhood cancer and 3773 controls. Taken together, these three case–control studies do not suggest a relationship between indoor radon concentrations and childhood leukaemia overall or with acute lymphoblastic leukaemia. The British study did not suggest any relationship between radon and other forms of childhood cancer, and in particular did not find an increased risk for central nervous system tumours, as had been observed in the earlier small German study. A recent study in Denmark by Raaschou-Nielsen et al (2008) studied various childhood cancers and found a positive dose–response only for acute lymphoblastic leukaemia. The methodology for ascertaining radon exposure differed from that in all previous studies in this area. More details are given in Appendix K.

Laurier et al (2001) reviewed epidemiological studies of adult haematological malignancies published from 1988 to 2001, including miners' cohorts as well as nineteen ecological studies and eight case–control studies. Laurier et al concluded that the available data did 'not provide evidence for an association between radon exposure and leukaemia'. We agree with this conclusion.

Results from epidemiological studies of gastric cancers have been mixed. Despite excesses seen in some studies (see, for example, Kjellberg and Wiseman, 1995), other studies have been negative (see, for example, Mifune et al, 1992, and Auvinen et al, 2005). Overall, convincing evidence of a causal link between radon exposure and gastric cancer is lacking.

There have been a number of studies of radon and skin cancer. Malignant melanoma does not appear to be associated with radon exposure (Darby et al, 1995). A link between radon exposure and non-melanoma skin cancer has been reported in an ecological study (Etherington et al, 1990) and in a case–control study of miners (Sevcova et al, 1978). Mortality is generally not an appropriate endpoint to consider in studies of non-melanoma skin cancer, due to its low case-fatality rate. However, incidence studies are complicated by the fact that many cases are not reported to central cancer registries as would normally be the case with other types of cancer. No definite conclusions on the risk of radon-related non-melanoma skin cancer can be drawn from the published studies.

Cohort studies have the advantage that they cover all types of cancer. In general, such studies show no excess risk of any malignancy, with a few exceptions. The exceptions – for example, leukaemia – were based on very small case numbers and have not been supported by other work. In the case of gastric cancer a significant excess in one study showed no dose–response, nor did an excess of primary liver cancer, casting doubt on whether radon was the cause of these cancers.

The epidemiological evidence for an association between radon exposure and other cancers is very weak.

We also considered the published evidence for links between radon exposure and non-malignant conditions. Some ecological studies have suggested a link between multiple sclerosis and radon exposure (see, for example, Bølviken et al, 1997, 2003). However, to date, the published studies are weak and no conclusions can be drawn from them. No epidemiological evidence exists to support any teratological or reproductive effects in humans.

Overall there is no convincing epidemiological evidence to suggest that radon exposure contributes directly to excess disease or mortality other than of lung cancer.

5.3 Evidence from animal studies

The evidence on radon risks from animal studies is discussed in Appendix G. The principal finding has been that of lung tumour induction, as in humans. In addition, at high exposure levels there is evidence for non-malignant lung damage (lung fibrosis and emphysema) resulting in reduced lifespan.

Most animal studies reported all tumours and other health effects, whether occurring in the lung or not. In unexposed animals, the incidence of non-lung tumours was generally considerably higher than that of lung tumours, and this reduces the power of the studies.

Kidney tumours have shown increased incidence following radon exposure, significantly so for some studies. No increase in leukaemia incidence following exposure to radon and its decay products has been observed in experimental animals. Various other tumours have been observed at slightly elevated frequencies, but not consistently between different laboratories. However, combined analysis of data from different laboratories has indicated that exposure to radon and its decay product results in significant lethal effects in addition to lung tumours (Kaiser et al, 2004).

There is also some evidence from animal experiments to suggest that at very high exposures there may be some damage to the immune system.

On balance we conclude that animals studies do not provide strong evidence of any risk from radon and its decay products other than of lung cancer, except at very high levels of exposure.

5.4 Summary

On the basis of calculated doses from radon and its decay products, there is a small theoretical risk of malignant and other damage to a variety of organs other than the lung. Some animal experiments tend to support this view, but such studies involve high radon exposure levels.

The human epidemiological evidence on cancers other than lung cancer is mixed. Some positive associations between radon and diseases have been reported in ecological (geographical) studies. However, it is now recognised that the average residential radon concentration in an area is a poor surrogate for individual dose. Results based on such studies are thus potentially unreliable. Most case–control studies in which individual radon measurements were available found little or no risk of any conditions, including childhood cancers, adult haematological malignant diseases, gastric cancer and skin cancer. Cohort studies lead to a similar conclusion with the possible exception of some leukaemias. No epidemiological evidence exists to support any teratological or reproductive effects in humans.

Our overall conclusion is that if radon and its decay products do have effects on organs other than the lung, then the effect is so weak as to be generally undetectable in the published epidemiological studies.

6 Cost-effectiveness of Radon Preventive and Remedial Programmes in UK Homes

Key Points

1 Health economics calculations have been carried out to evaluate the cost-effectiveness of current and possible future radon control policies for the UK.

2 The results of these calculations, which are summarised below, are expressed in terms of costs per quality adjusted life-year gained with discounting and at 2007 prices from a societal perspective (ie costs per QALY) and are based on HPA UK radon maps in use to November 2007. These costs per QALY have then been compared with the maximum values considered acceptable across government departments, which at the moment are typically in the range £20,000–£30,000.

3 For new homes, the government policy in England and Wales of requiring the installation of basic preventive measures such as a sealed membrane in all new homes in areas of the country where the mean long-term radon concentration is 52 Bq m^{-3} or above (ie areas where 3% or more of measurements are above 200 Bq m^{-3}) is likely to have a cost per QALY of about £8,000. This is well below the typical maximum acceptable value in government departments. Extending this policy across the entire country is likely to have a cost per QALY of £11,400. This is still well below the typical maximum acceptable value in government departments.

4 Low-cost ways of further improving the effectiveness of the basic measures that are currently available, eg more resistant membranes or more rigorous inspection regimes, are also likely to be very cost-effective. If the average reduction in radon concentration that they achieve could be improved by 10% (ie from 50 to 60%) for a 50% increase in the cost (ie from £100 to £150), or by 20% for a 100% increase in the cost, then the cost per QALY for a national policy would still remain below £20,000.

5 A policy of finding new homes that continue to have high radon concentrations after basic measures have been installed, and installing active measures such as electric fans in them, is likely to be cost-effective only if the Action Level is reduced substantially from its current value of 200 Bq m^{-3} and even then only in high-radon areas. For an area with a mean long-term radon concentration of 90 Bq m^{-3} prior to the installation of any radon preventive measures, such a policy would have a cost per QALY of £28,800 for an Action Level of 100 Bq m^{-3}, and a cost per QALY of £29,000 for an Action Level of 50 Bq m^{-3}. The costs per QALY of this policy would be lower for areas with higher radon concentrations and higher for areas with lower radon concentrations.

6 The HPA recommendation for existing homes is that people living in areas where the mean long-term radon concentration is 36 Bq m^{-3} or higher (ie at least 1% of homes have measurements above 200 Bq m^{-3}) should measure the radon concentration in their home, and remediate if the measurement is above the current Action Level of 200 Bq m^{-3}, while government policy in England consists of targeting areas of the country where the mean long-term radon concentration is 64 Bq m^{-3} or above (ie at least 5% of homes have measurements above 200 Bq m^{-3}), offering free radon measurements, and then encouraging householders to remediate at their own expense if the measurement is above 200 Bq m^{-3}.

7 For areas with a mean long-term radon concentration of 36 Bq m^{-3} remediation of existing homes has a cost per QALY of £154,700 which is over five times the maximum considered acceptable, while for areas with a mean long-term average radon concentration of 64 Bq m^{-3}, the cost per QALY of remediation in existing homes is £36,800 per QALY, which is also above the maximum considered acceptable. For existing homes, the costs per QALY of remediation decrease as the mean radon concentration in the area increases and vice versa.

8 Reducing the radon Action Level from its current value of 200 Bq m^{-3} is likely to improve the cost-effectiveness of policies directed at existing homes. Assuming that the willingness of householders to remediate was unchanged by such a reduction, an Action Level of 100 Bq m^{-3} would have a cost per QALY of £29,800 in areas with a mean long-term radon concentration of 60 Bq m^{-3}. Low-cost ways of improving testing and remediation rates would also be likely to improve the cost-effectiveness of remediation in existing homes.

9 Where a radon measurement already exists for a particular home, eg because the householder has already purchased a measurement or has already accepted an invitation to have a free measurement, carrying out remedial work becomes cost-effective at a measured radon concentration of about 100 Bq m^{-3}, where the cost per QALY is £25,300. At higher measured concentrations, the costs per QALY are lower.

10 The above calculations assume that radon policies are equally likely to reduce radon concentrations for current smokers, ex-smokers and lifelong non-smokers. For basic preventive measures in new homes this is likely to be true, but recent evidence suggests that remediation rates among lifelong non-smokers may be higher than rates among smokers. If this is true, then the cost-effectiveness of remediation in existing homes would, in practice, be less favourable than indicated by our analyses.

11 The results of the calculations depend on assumptions about the risk of lung cancer from radon exposure, the discount rate, the costs and effectiveness of interventions and the willingness of householders to follow advice. Sensitivity analyses have been carried out to indicate the extent to which the costs per QALY would change under different assumptions.

Radon remediation and control have been the subject of a number of previous economic evaluations (see Appendix M for selected references). In this chapter we present a new analysis undertaken with the intention of informing discussion about current and future policy in this area, illustrating the techniques and using the most up-to-date information available for the UK. Sensitivity analyses have been used to assess the potential effect of differing assumptions and altered circumstances on the results. However, not all such future conditions can be anticipated, and programme techniques may well change. The numerical results presented here – in particular, the costs per quality adjusted life-year (QALY) gained for the UK radon programme and for a number of potential future programmes for the UK – are indicative of the cost-effectiveness of radon reduction programmes in general, but our approach could be used to make specific estimates for other programmes with their own attributes, including programmes for other countries. We note that a review of the UK radon programme is currently under way (J Meara, HPA: personal communication).

We evaluate two main types of programme. Firstly, we consider the cost-effectiveness of radon prevention in new homes across a range of area-specific mean radon concentrations. Secondly, we consider the cost-effectiveness of policy concerning existing homes. By evaluating these programmes in similar terms to other health interventions, comparisons can be made based on outcomes and costs per life-year gained.

The calculations use two estimates of radon risk: one derived from studying directly the lung cancer risk from radon exposure in the home, and another derived indirectly from studies of radon-exposed miners (see Chapter 3 for further details). These are referred to as direct and indirect risk estimates, respectively. Although many of the calculations have been carried out for both risk estimates, in drawing conclusions greater emphasis has been placed on results based on the direct risk estimates, in line with the evidence presented in Chapter 3 which concludes that they form the most appropriate basis for risk estimation. Full details of the methods and input values for the parameters used are in Appendix M. Parameters used in the calculations are given to full precision to allow the calculation to be reproduced. However, this does not imply that all the data required for the calculations are established to this precision.

6.1 Radon prevention in new homes

Table 6.1 reports the central estimates of the cost-effectiveness model concerning new homes, with all new homes having 'basic preventive measures' against radon ingress, such as a sealed radon-proof membrane fitted during construction in all areas where the mean long-term radon concentration is 52 Bq m^{-3} (ie 3% of homes have measured radon concentrations above 200 Bq m^{-3}).

Using the direct risk estimate, the cost-effectiveness model predicts that the cumulative lifetime risk of lung cancer at pre-remediation radon concentrations is 6.38%. Post-remediation, the lifetime risk falls to 6.14%. This is equivalent to a reduction of 0.006 lung cancer cases in a household of average size (2.4 people), which in turn is equivalent to 0.08 life-years gained, or 0.04 discounted life-years gained.

TABLE 6.1 New homes: main estimates of effects, costs and cost-effectiveness for basic preventive action in all new homes in an area where 3% of homes have measured radon concentrations above 200 Bq m^{-3}, based on the HPA methodology of considering one radon measurement of three months' duration with seasonal adjustment. This corresponds to an area with a mean long-term radon concentration of 52 Bq m^{-3}

Government policy in England and Wales requires basic preventive measures in new homes in areas with mean long-term radon concentrations at or above this level

	Radon risk estimate	
	Direct [(i)]	**Indirect** [(ii)]
Initial		
Lifetime cumulative risk of death from lung cancer (% per person)	6.38	6.62
Post-preventive action		
Lifetime cumulative risk of death from lung cancer (% per person)	6.14	6.18
Health gain per household		
Lung cancer cases averted	0.006	0.010
Total life-years gained	0.08	0.14
Total life-years gained – discounted	0.04	0.07
Total QALYs gained	0.06	0.11
Total QALYs gained – discounted	0.03	0.06
Resource use and costs per household		
Radon prevention cost – discounted	£100	£100
NHS lung cancer treatment costs averted – discounted	£29	£52
Other NHS costs incurred by added life expectancy – discounted	£177	£322
Net cost – discounted – societal	£248	£369
Net cost – discounted – NHS	*£148*	*£269*
Net cost – discounted – households	*£100*	*£100*
Cost-effectiveness		
Cost per life-year gained – discounted – societal	£6,226	£5,101
Cost per QALY gained – discounted – societal	**£7,953**	**£6,516** [(iii)]
Cost per QALY gained (discounted) – NHS	*£4,752*	*£4,752*
Cost per QALY gained (discounted) – households	*£3,201*	*£1,764*

Notes

(i) From studies of residential radon and lung cancer in Europe (Darby et al, 2005, 2006).

(ii) From studies of radon-exposed miners (BEIR VI Committee, 1999).

(iii) Shaded value corresponds to current policy in England and Wales.

In this case the only costs are the radon prevention costs of £100 per home. Discounted savings from lung cancer treatment costs averted are approximately £29, while added NHS costs as a result of longer life expectancy are £177. The net cost is therefore £248, of which £148 is incurred by the NHS and £100 by the householder.

Combining the outcomes and costs reported above, the cost per quality adjusted life-year gained of radon prevention in areas where the mean long-term radon concentration is 52 Bq m^{-3}, is £7,953. This figure is well below the maximum level that might be considered cost-effective either by reimbursement or by agencies such as the National Institute for Health and Clinical Excellence (NICE), or by other government departments.

Table 6.2 gives more detail on how widely these basic preventive measures could be applied cost-effectively. As the mean radon concentration falls, the cost per QALY gained increases, but even at a mean long-term radon concentration of 10 Bq m^{-3}, the cost per QALY gained is still only just over £20,000. When the whole country is considered, the cost per QALY gained remains well below the accepted maximum at approximately £11,400 per QALY gained using direct risk estimates and only £8,400 using indirect risk estimates. Hence, a policy of fitting basic preventive measures, such as sealed membranes, could be extended to all new homes across the UK and remain highly cost-effective.

These results depend on a number of the assumptions, including the cost of fitting effective radon barriers, which are discussed in Appendix M. However, the policy has a high probability of being cost-effective under all the scenarios considered.

Using the cost-effectiveness model, the outcomes, costs and cost-effectiveness of a 'full preventive measures' policy are also considered in areas where the mean long-term radon concentration is 87 Bq m^{-3} (ie 10% of homes have measured radon concentrations above 200 Bq m^{-3}). In this scenario a means of under-floor ventilation, such as a sump and pipework, is fitted in addition to the basic preventive measures, then all homes are tested once completed and occupied to find those that still have radon levels above 200 Bq m^{-3}, and electric fans are installed in those homes. Using the direct risk estimates, we estimate that the incremental cost per QALY gained of this policy is approximately £54,000. This figure is well above the level (£20,000–£30,000 per QALY gained) that might typically be considered the maximum willingness to pay when assessing alternative ways of improving health. The relatively poor cost-effectiveness of this policy arises partly from the high lifetime costs of active measures such as electric fans, with both running and replacement costs, and partly from the fact that basic preventive measures will already have reduced radon levels by 50% and substantially reduced the numbers of homes likely to be over the Action Level, from 10% over 200 Bq m^{-3} to under 2% in the example given above. This increases the costs of detecting the remaining houses above the Action Level, and also results in large numbers of houses having full preventive measures installed during construction for every one that will ultimately require further radon lowering actions. Reducing the Action Level from its current value of 200 Bq m^{-3} would make a full preventive measures policy more effective. For an area with a mean long-term radon concentration of 90 Bq m^{-3} prior to the installation of any radon preventive measures, such a policy would have a cost per QALY of £28,800 for an Action Level of 100 Bq m^{-3}, and a cost per QALY of £29,000 for an Action Level of 50 Bq m^{-3}.

TABLE 6.2 New homes: effect on cost per quality adjusted life-year (QALY) gained of varying the definition of areas where basic preventive measures are required in all new homes

Mean long-term radon concentration in area (Bq m^{-3})	Cost per QALY gained [i]		% of national housing stock in areas with mean above this value
	Direct risk estimate	Indirect risk estimate	
10	£21,406	£13,909	87.5
15	£15,858	£10,860	60.9
20	£13,084	£9,335	39.6
25	£11,419	£8,421	25.6
30	£10,310	£7,811	16.7
35	£9,518	£7,376	11.2
36 [ii]	£9,383	£7,301	10.3
40	£8,923	£7,049	7.6
45	£8,461	£6,795	5.2
50	£8,091	£6,592	3.7
52 [iii]	**£7,953**	**£6,516** [vi]	3.2
55	£7,788	£6,425	2.6
60	£7,536	£6,287	1.9
64 [iv]	£7,372	£6,196	1.5
65	£7,323	£6,170	1.4
70	£7,140	£6,069	1.0
75	£6,982	£5,982	0.8
80	£6,843	£5,906	0.6
85	£6,720	£5,838	0.5
87 [v]	£6,667	£5,809	0.4
90	£6,612	£5,779	0.4
Whole country			
21	£11,427	£8,425	100

Notes
(i) Discounted – societal.
(ii) That is, 1% of measurements >200 Bq m^{-3}.
(iii) That is, 3% >200 Bq m^{-3}.
(iv) That is, 5% >200 Bq m^{-3}.
(v) That is, 10% >200 Bq m^{-3}.
(vi) Shaded value corresponds to current policy in England and Wales.

6.2 Radon remediation in existing homes

Table 6.3 shows the results of the cost-effectiveness analysis concerning existing homes, when invitations are targeted on areas with a mean long-term radon concentration of 64 Bq m^{-3} (ie 5% of homes have radon concentrations above the current Action Level of 200 Bq m^{-3}). Government policy in England is to target radon remediation programmes at areas with mean radon concentrations at or above this level.

Using the direct risk estimate based on the European pooling study, the model predicts a cumulative lifetime risk of lung cancer at pre-remediation radon concentrations in households above the current Action Level of 7.82%. Post-remediation, the lifetime risk falls to 6.19%. This is equivalent to a reduction of 0.04 lung cancer cases in a household of average size, which in turn is equivalent to 0.52 life-years gained, or 0.27 discounted life-years gained.

The prevailing mean radon concentration in an area influences the number of householders who need to be invited to test for each household that remediates. The proportion of householders invited to test who agree to do so is taken to be 30%, and the proportion who take remedial action when advised to do so is set at 20%, based on previous experience (see Appendix D). At a mean long-term radon concentration of 64 Bq m^{-3}, 333 invitations to test will result in 100 homes tested, five found to be above the current Action Level, and one remediated. The cost of the invitations is £550 and the cost of testing is £4,200. These costs, together with remediation costs, come to a discounted total of £6,801. Around £195 is saved from the averted lung cancer treatment costs, but the additional life expectancy has NHS costs of £1,203. Consequently the net cost is £7,809 per household remediating. Of this, £1,088 falls on the NHS, £4,750 on the HPA and £2,051 on households.

Combining the outcomes and costs reported above, the cost per life-year gained is £28,833 and the cost per QALY based on the direct estimate of risk is £36,828. This result depends on a number of assumptions, as discussed in Appendix M.

Table 6.4 shows how the costs per QALY gained change as the Action Level is varied from its current value of 200 Bq m^{-3}, and the targeted area is also varied from 64 Bq m^{-3}. It is assumed that other parameters do not change: for example, the proportion of householders with homes over each Action Level that decide to remediate is held constant, as are the assumed cost and efficacy of remediating. These assumptions may not hold in practice – for example, propensity to remediate could either increase or decline at lower Action Levels – but there is little evidence on this. As the radon level in the targeted area decreases, cost-effectiveness deteriorates, as a smaller proportion of homes have measurements above any specified Action Level.

The effect of lowering the Action Level is more complex. The proportion of homes over the Action Level, the numbers remediating and the total QALYs gained, all increase. Initially, net costs rise less rapidly than health benefits, and so cost-effectiveness improves as the Action Level decreases. For a targeted area with a mean long-term radon concentration of 64 Bq m^{-3}, cost-effectiveness continues to improve down to an Action Level of 100 Bq m^{-3}. Below this value, benefits increase less rapidly than costs and the cost-effectiveness starts to deteriorate, although at an Action Level of 50 Bq m^{-3} the cost-effectiveness compared with no action is still more favourable than it was at 200 Bq m^{-3}. For an Action Level of 100 Bq m^{-3}, a policy of remediating existing homes would have a cost per QALY of £29,800 in areas with

TABLE 6.3 Existing homes: main estimates of effects, costs and cost-effectiveness for a policy of inviting householders to test in areas where 5% of homes are likely to have measured radon concentrations above 200 Bq m^{-3} and recommending remediation if the measurement is above the current Action Level of 200 Bq m^{-3} (assuming 30% of householders accept the invitation to test and 20% found to be over the Action Level agree to remediate)

This reflects government policy in England. Measured radon levels based on the HPA methodology of one radon measurement of three months' duration with seasonal adjustment. Areas where 5% of homes have measurements over 200 Bq m^{-3} have a mean long-term radon concentration of 64 Bq m^{-3}

	Radon risk estimate	
	Direct [(i)]	Indirect [(ii)]
Initial		
Lifetime cumulative risk of death from lung cancer (% per person)	7.82	9.20
Post-remediation		
Lifetime cumulative risk of death from lung cancer (% per person)	6.19	6.27
Health gain per household remediating		
Lung cancer cases averted	0.04	0.07
Total life-years gained	0.52	0.93
Total life-years gained – discounted	0.27	0.49
Total QALYs gained	0.40	0.73
Total QALYs gained – discounted	0.21	0.38
Resource use and costs per household remediating		
Number of invitations to test	333	333
Invitation costs	£550	£550
Number of radon tests	100	100
Radon testing cost	£4,200	£4,200
Radon remediation cost – discounted	£2,051	£2,051
Subtotal: invitation, testing and remediation costs – discounted	£6,801	£6,801
NHS lung cancer treatment costs averted	£656	£1,185
NHS lung cancer treatment costs averted – discounted	£195	£353
Other NHS costs incurred by added life expectancy – discounted	£1,203	£2,171
Net cost – discounted – societal	£7,809	£8,620
Net cost – discounted – NHS	*£1,008*	*£1,819*
Net cost – discounted – HPA	*£4,750*	*£4,750*
Net cost – discounted – households	*£2,051*	*£2,051*
Cost-effectiveness		
Cost per life-year gained – discounted – societal	£28,833	£17,633
Cost per QALY gained – discounted – societal	**£36,828**	**£22,523** [(iii)]
Cost per QALY gained – discounted – NHS	*£4,752*	*£4,752*
Cost per QALY gained – discounted – HPA	*£22,404*	*£12,412*
Cost per QALY gained – discounted – households	*£9,673*	*£5,359*

Notes
(i) From the European study of residential radon and lung cancer (Darby et al, 2005, 2006).
(ii) From studies of radon-exposed miners (BEIR VI Committee, 1999).
(iii) Shaded value corresponds to current government policy in England.

TABLE 6.4 Existing homes: effect on cost per quality adjusted life-year (QALY) gained of varying both the targeted area and the Action Level within targeted areas. Results shown for direct estimate of radon risk

Targeted area (Bq m^{-3} long-term mean)		Cost per QALY gained (discounted – societal)						
		Action Level (Bq m^{-3} measured value) [i,ii]						
		25 Bq m^{-3}	50 Bq m^{-3}	100 Bq m^{-3}	150 Bq m^{-3}	200 Bq m^{-3}	300 Bq m^{-3}	400 Bq m^{-3}
20	[iii]	**£85,200**	£105,600	£285,200	£744,300	£1,682,500	£6,271,900	£17,840,700
25		**£69,800**	£72,000	£138,100	£300,100	£608,500	£1,980,300	£5,145,100
30		£60,600	**£56,900**	£86,100	£159,700	£293,800	£851,800	£2,056,100
35		£54,100	**£48,500**	£62,200	£100,900	£170,100	£444,300	£1,005,900
36 [vii]		£53,100	**£47,200**	£58,900	£93,400	£154,700 [iv]	£395,900	£885,400
40		£49,300	**£43,000**	£49,200	£71,600	£111,500	£264,300	£564,600
45		£45,400	**£39,100**	£41,400	£55,100	£79,900	£173,000	£350,000
50		£42,200	£36,200	**£36,200**	£44,900	£61,200	£121,700	£233,900
52 [viii]		£41,000	£35,100	**£34,400**	£41,600	£55,400	£106,500	£200,200
55		£39,500	£33,800	**£32,500**	£38,100	£49,300	£90,600	£165,800
60		£37,200	£31,900	**£29,800**	£33,400	£41,300	£70,600	£123,300
64 [ix]		£35,600	£30,700	**£28,200**	£30,700	£36,800 [vi]	£60,000	£101,100
65		£35,200	£30,300	**£27,700**	£30,000	£35,600	£57,100	£95,300
70		£33,400	£28,900	**£26,000**	£27,400	£31,500	£47,600	£76,100
75		£31,800	£27,700	**£24,600**	£25,300	£28,400	£40,700	£62,500
80		£30,400	£26,600	**£23,500**	£23,700	£25,900	£35,500	£52,500
85	[v]	£29,100	£25,600	£22,500	**£22,400**	£24,000	£31,500	£45,000
87 [x]		£28,500	£25,200	£22,100	**£21,800**	£23,200	£29,900	£42,000
90		£27,900	£24,700	£21,700	**£21,300**	£22,400	£28,400	£39,200
95		£26,800	£23,900	£20,900	**£20,300**	£21,100	£25,900	£34,700
100	[v]	£25,900	£23,200	£20,300	**£19,500**	£20,100	£23,900	£31,200

Notes

(i) Based on the HPA methodology of considering one three-month radon measurement with seasonal adjustment.

(ii) There is little information available at present on the percentage reduction in radon concentration that would result from remediating homes with pre-remediation concentrations of 25 or 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher pre-remediation concentrations, ie 85%.

(iii) Entries in bold denote the most cost-effective Action Level for each targeted area.

(iv) The HPA recommendation for the UK is that people living in areas where 1% of more of homes have radon measurements above 200 Bq m^{-3} measure the radon in their home and carry out remediation if the measurement is above 200 Bq m^{-3}.

(v) Entries between the lines have a cost per QALY gained between £20,000 and £30,000.

(vi) Government policy in England targets areas with 5% or more of radon measurements >200 Bq m^{-3} and has an Action Level of 200 Bq m^{-3}.

(vii) That is, 1% of measured values >200 Bq m^{-3}.

(viii) That is, 3% >200 Bq m^{-3}.

(ix) That is, 5% >200 Bq m^{-3}.

(x) That is, 10% >200 Bq m^{-3}.

a mean radon concentration of 60 Bq m^{-3}. As noted above, these calculations assume that all other parameter values are unchanged, which may not be the case in practice.

Table 6.5 shows different cost-effectiveness estimates when it is assumed, hypothetically, that a household consists entirely of never-smokers or of current cigarette smokers, compared to the baseline case where smoking-related risks are assumed to be the same as in the population as a whole. It is assumed in this scenario that the smoking status of the household is held constant over time. It is evident that cost-effectiveness is closely related to smoking status: cost-effectiveness varies from £13,727 per QALY gained among current cigarette smokers to £173,720 amongst never-smokers.

In circumstances where a radon measurement already exists for a particular home, eg from a previous invitation or because the householder has already purchased a measurement, carrying out remedial work

TABLE 6.5 Existing homes: lung cancer risk, remediation costs and cost per life-year and quality adjusted life-year gained for the total population, and for households consisting entirely of never-smokers or current cigarette smokers. Based on invitations to existing householders in areas with a mean long-term radon concentration of 60 Bq m^{-3} to test and remediate if the measured radon level is over an Action Level of 100 Bq m^{-3}, ie results shown for direct estimate of radon risk (assuming 30% of householders accept the invitation to test and 20% found to be over 100 Bq m^{-3} agree to remediate)

	Household of:		
	Population prevalence of smoking [(i)]	Never-smokers only [(i)]	Current cigarette smokers only [(i)]
Lifetime cumulative lung cancer risk (% per person)			
Initial	7.13	0.94	28.52
Post-remediation	6.09	0.80	24.81
Health gain per household remediating			
Lung cancer cases averted	0.03	0.003	0.09
Total QALYs gained [(ii)]	0.14	0.02	0.35
Resource use and costs per household remediating			
Invitation, testing and remediation costs [(ii)]	£3,414	£3,414	£3,414
NHS lung cancer treatment costs averted [(ii)]	£126	£17	£447
Other NHS costs incurred by added life expectancy [(ii)]	£774	£110	£1,870
Net cost	£4,062	£3,506	£4,836
Cost-effectiveness			
Cost per QALY gained – societal	£29,789	£173,720	£13,727
Cost per QALY gained – discounted – NHS	*£4,752*	*£4,590*	*£4,037*
Cost per QALY gained – discounted – HPA	*£9,995*	*£67,514*	*£3,868*
Cost per QALY gained – discounted – households	*£15,043*	*£101,616*	*£5,822*

Notes
(i) See Appendix M for details of calculations.
(ii) Discounted.

becomes cost-effective at lower radon concentrations than indicated in Table 6.4, as no further invitation or testing cost is involved. Where smoking-related risks are the same as for the population as a whole, then remediation has a cost per QALY of £25,300 for a measured radon concentration of 100 Bq m^{-3} and has more favourable cost-effectiveness at higher measured values.

6.3 Discussion and conclusions

Our analysis indicates that installing effective radon barriers in all new homes is highly cost-effective and that the policy could be extended to all new homes in the UK. There may also be other potential benefits of adopting such measures for all new homes. A single, consistent standard is more easily applied and so may be more effectively implemented, and radon preventive measures would also reduce the entry of vapour and other indoor pollutants from the ground. Finding and dealing with new homes where the measured radon concentration is still high after these measures have been implemented is expensive, due both to detection costs and to the running costs of active remediation, and may only be cost-effective in a few areas with high radon levels.

The cost-effectiveness of a policy that relies upon identifying existing homes with measured radon concentrations above 200 Bq m^{-3} and inviting householders to remediate at their own expense has a cost-effectiveness ratio that is above the current maximum willingness to pay ceiling of between £20,000 and £30,000 per QALY gained for areas with mean long-term average radon concentrations of 70 Bq m^{-3} or lower, mainly due to the low proportions (historically around 20%) undertaking remediation in homes over the Action Level. The analyses showed that lowering the Action Level below the current value of 200 Bq m^{-3} may improve the cost-effectiveness of remediation policies which require the identification of homes with high radon concentrations, as the invitation and testing costs involved in finding homes above the Action Level can be spread over more remediating homes if the Action Level is lower.

Increasing the proportion of identified homes that undertake remediation would substantially improve the cost-effectiveness of programmes to remediate existing homes. It has long been evident that an individual's likelihood of taking remedial action is affected by the way in which risk information on radon is presented (Smith and Desvousges, 1990; Smith et al, 1995; Kendall et al, 2005). At present the link between risk information and remedial action is poorly understood or researched, although it has been shown that the level of advice and support is important in determining the proportion of householders who remediate (Appendix D). Further work in this area may result in higher remediation rates and improved cost-effectiveness.

These estimates assumed that all other parameters remained unchanged: for example, that the proportion of householders in homes over the Action Level who decide to remediate is the same whether that level is set at 200, 100 or 50 Bq m^{-3}, as is the assumed average efficacy of remediating and the average cost of remediation. Estimates of the effect of altering these assumptions are given in Appendix M, and more research is needed to test them.

The analysis presented here has been performed primarily from a societal perspective, and includes all direct costs incurred by individuals, local and central government departments and the NHS.

Tables 6.1 and 6.3 also show cost and cost-effectiveness data from the perspective of the NHS alone, households alone, and the HPA. The NHS would bear the majority of costs for the new homes policy, mainly because the additional health care costs of added life expectancy are greater than the savings from fewer lung cancer cases. However, the policy would be highly cost-effective from the NHS perspective alone, as well as societally. Concerning the existing homes policy, most of the costs are related to identifying homes with high radon concentration and the costs for this are currently incurred by the HPA. Householders almost always bear the cost of taking remedial action, and this is likely to be one factor influencing remediation rates. Transferring some or all of these costs to another party might increase remediation rates and could, as a result, improve the overall cost-effectiveness ratio substantially. Such cost transfers do occur in other policy areas: health and local authorities, for instance, in certain circumstances pay towards the costs of adapting homes for people with disabilities, and local authorities will, in fact, pay the costs of radon remediation in council houses where a need is demonstrated. However, a difference is that radon remediation has benefits not only to the existing private home occupier but to future occupiers which may be reflected in the house price, supporting an argument that homeowners should at least share the costs.

The fact that both radon and smoking increase the risk of lung cancer has frequently resulted in the cost-effectiveness of radon remediation programmes and smoking cessation programmes being compared. Smoking cessation interventions typically have very favourable cost-effectiveness ratios, and so should be fully funded if a cost-effectiveness framework is used. However, assuming such interventions are indeed being provided, there is no reason why such a low cost-effectiveness ratio should be the benchmark for radon remediation or other interventions relevant to smoking-related diseases: instead, we have adhered to the approach of identifying a maximum willingness to pay for health gains, whether in the health sector or in other government departments. Details of this are set out in Appendix M, but the indication is that as long as the cost per QALY gained is no greater than £20,000–£30,000, interventions are unlikely to be rejected on cost-effectiveness grounds.

The analysis presented here assumes that those in whom lung cancers are averted by prevention or remediation have smoking habits, and therefore lung cancer risks, that are typical of the entire population. This is likely to be the case for prevention in new homes. However, a recent study has shown that remediation rates among householders who are lifelong non-smokers are about twice that of householders who are current smokers (Dr Y Chow, HPA: personal communication). The consequence of this is that the cost-effectiveness of remediation in existing homes would, in practice, be less favourable than indicated by our analyses, possibly by a substantial amount.

The results presented here are also sensitive to whichever discount rate is selected. The baseline analysis uses Department of Health and Department of Transport approved annual discount rates of 3.5% for costs and 1.5% for health benefits (HM Treasury, 2003). The long periods over which the health benefits from reduced radon-induced lung cancer deaths are realised mean that the choice of discount rate will strongly affect the results.

Finally, the results depend on the radon risk estimate assumed. The calculations in this chapter mainly use radon risk estimates derived from the European pooling study in which the risk of lung cancer from indoor radon was studied directly (Darby et al, 2005). This study estimated that an increase of 100 Bq m^{-3} in

long-term average radon concentration would cause a 16% increase in the risk of lung cancer (95% confidence interval 5–31%). As noted in Chapter 3, there are reasons to believe that the true risk may be somewhat higher than that estimated by this study. Therefore we have also presented some parallel calculations based on risks estimated indirectly from studies of radon-exposed miners (BEIR VI Committee, 1999), which give somewhat higher risks than those derived from the European pooling study, and consequently somewhat lower cost-effectiveness ratios. We have, however, based our conclusions on the direct estimate of risk.

7 Conclusions

Radon in the home delivers larger doses to the UK public than any other natural source of ionising radiation. The UK was one of the first countries in the world to introduce a radon control policy, with the issue of guidance in 1987 (NRPB, 1987) which was revised in 1990 (NRPB, 1990a). On the basis of the limited evidence of the risk of lung cancer associated with radon at that time, the guidance suggested the remediation of existing homes with high radon concentrations and also preventive measures to reduce radon concentrations in new buildings by means of changes in the building regulations in some parts of the country. A programme of measurement of radon levels was instituted, and when homes with high radon levels were found, emphasis was placed on reducing radon concentrations as far as possible, not just reducing them below a threshold.

Since this advice was issued, considerable additional evidence has become available. In particular, there is now direct and conclusive evidence that indoor radon in ordinary homes causes lung cancer in the general population. The dose–response relationship appears linear, with no evidence of a concentration below which there is no risk, and there is substantial evidence that the majority of radon-induced lung cancers occur following exposure at concentrations below 200 Bq m^{-3}, the concentration at which action is currently advocated in the UK.

We judge that the current best estimate of the risk of residential exposure to radon is based on the results of the recent pooled analysis of European studies of residential radon and lung cancer. The central estimate of risk obtained in this analysis is that living in a home with a long-term average concentration of radon gas of 100 Bq m^{-3} increases the annual risk of lung cancer by 16% in non-smokers, ex-smokers and current smokers alike, with a 95% confidence interval of 5 to 31%. There were, however, a number of factors that could not be taken into account in this analysis and it is likely that the true risk is somewhat higher than this estimate. The estimated risk of radon-induced lung cancer based on studies of underground miners exposed to high concentrations of radon also indicates a somewhat higher risk than 16% per 100 Bq m^{-3}. In our view, the evidence indicates that any other hazards of exposure to radon are much less important than lung cancer in terms of their implications for public health.

Radon is an important cause of radiation-induced cancer in the general population. Based on the studies of residential radon, it is estimated that 3.3% of the approximately 34,000 deaths from lung cancer each year in the UK are caused by indoor radon: that is, about 1100 deaths every year, or just over 1 in 500 deaths from all causes. Only 0.5% of the lung cancer deaths (ie about 160 per year) are likely to be attributable to residential radon acting alone, and the remaining 2.8% (ie about 950 per year) are caused both by residential radon and by smoking in the sense that the lung cancer could have been avoided by avoiding either smoking or radon exposure. Over 40% of the deaths caused both by radon and by smoking are likely to occur in people who have already given up smoking.

At the UK mean long-term residential radon concentration of 21 Bq m^{-3}, the cumulative risk of death from lung cancer by the age of 75 years is 0.4% for a lifelong non-smoker and 15% for a continuing cigarette smoker. At 200 Bq m^{-3}, these risks rise to 0.5 and 19%, respectively. For recent ex-smokers, the risks are somewhat lower than those for continuing smokers, while for long-term ex-smokers, the risks are substantially lower than those for continuing smokers.

Indoor radon concentrations vary widely from home to home and the distribution of measured concentrations in a given area is usually very close to log-normal. In characterising the exposure of the UK population to indoor radon at home, attention to date has focused on the upper tails of the distribution of radon concentrations and, for example, radon maps of the country show only the estimated percentage of dwellings with radon concentrations above 200 Bq m^{-3} in small areas. However, the upper tails of the distribution can be estimated only with considerable uncertainty so that, for a given number of radon measurements, the estimate will often differ substantially from the population value. In contrast, the mean and median radon concentration for a given area can be estimated much more precisely from the same number of measurements. Furthermore, central estimates such as the mean or median are directly related to the typical risk from indoor radon to an individual living in that area.

Characterisation of the upper percentiles of the distribution of indoor radon concentrations is further complicated by the fact that repeated radon measurements made in the same home in different years exhibit substantial year-to-year variation and this variation is larger in homes with higher long-term average radon concentrations. This means, for example, that in any set of radon measurements the highest values will usually be substantial overestimates of the long-term average concentration in the homes concerned. Central measures, such as the mean and median, are affected much less by year-to-year variation.

Current UK policies for radon in homes focus on high radon concentrations. They have two components designed, firstly, to identify those at highest risk so that they can be advised to take remedial action and, secondly, to reduce radon levels in new homes. Thus, the components comprise:

a identification and remediation of existing homes in which the radon concentration is thought to exceed 200 Bq m^{-3},

b installation of radon preventive measures in new buildings in certain areas. Areas where more than 1% of dwellings have radon concentrations above 200 Bq m^{-3} have been designated as radon Affected Areas. In Scotland and Northern Ireland radon preventive measures are required in all new homes in radon Affected Areas unless it can be shown that the radon concentration in the building is unlikely to exceed 200 Bq m^{-3}, while in England and Wales radon preventive measures are required in areas where more than 3% of dwellings have radon concentrations above 200 Bq m^{-3}, unless it can be shown that the radon concentration in the building is unlikely to exceed 200 Bq m^{-3} in which case no preventive measures are required.

We have shown that more than two-thirds of radon-attributable deaths are estimated to occur as a result of living in a home where the measured radon concentration would be less than 50 Bq m^{-3}, around 20% in homes with concentrations in the range 50–99 Bq m^{-3}, and a further 10% in homes with concentrations in the range 100–199 Bq m^{-3}. Less than 5% of radon-induced lung cancers are likely to

occur as a result of exposure in homes where the measured radon concentration would be above 200 Bq m^{-3}. Furthermore only about 25% of radon-induced deaths are estimated to occur in areas designated as radon Affected Areas. Thus current policies do not address the vast majority of radon-induced deaths, which are caused by radon concentrations below 200 Bq m^{-3} and in areas other than those currently designated as radon Affected Areas.

Health economics analysis can provide a useful input to guide revisions to radon policy by evaluating the cost-effectiveness of different options. The results of these calculations can be expressed in terms of costs per quality adjusted life-year gained (ie costs per QALY). This enables different policies for reducing radon-induced lung cancer to be compared with each other, and it also enables the cost-effectiveness of radon policies to be evaluated according to the criteria used for other health interventions including, for example, those used by the National Institute for Health and Clinical Excellence for the NHS. At the present time the maximum cost per QALY considered acceptable across government departments is typically in the range £20,000–£30,000.

Our economic analysis suggests that the current government policy in England and Wales of requiring the installation of basic radon preventive measures, such as a sealed membrane in all new homes in areas of the country where the mean long-term radon concentration is 52 Bq m^{-3} or above, has a very favourable cost-effectiveness ratio compared with the maximum value considered acceptable in government departments. We estimate that, after allowing for a lag period, each year that this policy is in effect averts an additional 0.5 lung cancers in every future year (see Table 7.1).

An alternative policy of requiring basic radon preventive measures in all new homes throughout the entire country would also have a very favourable cost-effectiveness ratio, with a cost per QALY of around £11,400. We estimate that it would also avert considerably more lung cancers than the current policy, with 4.4 lung cancers averted in every future year for each year that the policy is in effect (see Table 7.1). Such a policy would also, in the longer term, progressively reduce the average radon exposure of the population as a whole, as the housing stock is gradually replaced.

There may, in addition, be other potential benefits of adopting a policy requiring basic radon preventive measures for all new homes throughout the UK: a single consistent standard is more easily applied and so may be more effectively implemented, and radon preventive measures would also reduce the entry of vapour and other indoor pollutants from the ground. Low-cost ways of further improving the effectiveness of the basic measures than are currently available, eg more resistant membranes and better sealing, or more rigorous inspection regimes, could also be very cost-effective.

The current 'full protective measures' policy of requiring, at construction, the installation of a means of under-floor ventilation such as a sump and pipework, in addition to the basic preventive measures, in all new homes in areas with mean long-term radon concentrations of 87 Bq m^{-3} and above, but with no requirement either to measure the subsequent radon concentration or to activate the extra measures, has effectiveness similar to that of the installation of basic measures only, but it has a higher cost. It is therefore not cost-effective in its present form. An alternative full protection measures policy that requires the builder to measure the radon concentration in the new home on occupation and to activate the extra measures if the radon concentration is found to remain above the Action Level could be cost-effective in high radon areas, but only if the Action Level is reduced substantially from its present value of 200 Bq m^{-3}.

TABLE 7.1 New homes: numbers of lung cancers averted [(i)] from various radon prevention policies

The calculations assume that new homes would, in the absence of any radon preventive measures, have radon concentrations similar to those seen in the national survey of indoor radon concentrations (Wrixon et al, 1988). Other assumptions in the calculations are listed in Section C3 and Table M1 [(ii)]

(a) Current government policy in England and Wales, ie installation of basic radon preventive measures in areas with mean radon concentration of 52 Bq m^{-3} or higher (ie at least 3% of measurements above 200 Bq m^{-3})	
Number of lung cancers averted every year by 1 year of policy	0.5
Number of lung cancers averted every year by 2 years of policy	2 x 0.5
Total number of lung cancers averted every year by 10 years of policy	5, increasing by 0.5 each year
(b) Basic radon preventive measures throughout the UK	
Number of lung cancers averted every year by 1 year of policy	4.4
Number of lung cancers averted every year by 2 years of policy	2 x 4.4
Total number of lung cancers averted every year by 10 years of policy	44, increasing by 4.4 each year
(c) Potential policy for high radon areas, requiring basic preventive measures plus fitting a means of under-floor ventilation such as a radon sump and pipe, together with measurement of the radon concentration after occupation and installation of fan where measurement exceeds the Action Level. Results shown for areas with mean radon concentration of 90 Bq m^{-3} or higher, Action Level of 100 Bq m^{-3} and assuming 100% compliance [(iii,iv)]	
Number of lung cancers averted every year by 1 year of policy	0.03
Number of lung cancers averted every year by 2 years of policy	2 x 0.03
Total number of lung cancers averted every year by 10 years of policy	0.3, increasing by 0.03 each year
(d) Same as (c) above but with Action Level of 50 Bq m^{-3} [(iv,v)]	
Number of lung cancers averted every year by 1 year of policy	0.05
Number of lung cancers averted every year by 2 years of policy	2 x 0.05
Total number of lung cancers averted every year by 10 years of policy	0.5, increasing by 0.05 each year

Notes

(i) Installing radon preventive measures in a new home will, after a lag period, avert radon-induced lung cancers in every future year.

(ii) Calculations based on estimate of radon-related lung cancer risk from pooled analysis of studies of residential radon and lung cancer in Europe (Darby et al, 2005, 2006).

(iii) Current government policy in England and Wales also requires provision for under-floor ventilation, such as a sump, in homes in areas with mean radon concentrations of 87 Bq m^{-3} or higher (ie at least 10% of measurements above 200 Bq m^{-3}). However, there is at present no requirement either to measure the subsequent radon concentration or to activate the extra measures. Therefore, such a policy has effectiveness similar to the installation of basic measures only, but it has a higher cost and it is not cost-effective in its present form.

(iv) Lung cancers averted in (c) and (d) are additional to those in (a).

(v) There is little information available at present on the percentage reduction in radon concentration that would result from installing a fan in homes with radon concentrations as low as 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher concentrations, ie 90%.

For example, in an area with a mean long-term radon concentration of 90 Bq m^{-3} prior to the installation of any radon preventive measures, such a policy would have a cost per QALY of £28,800 for an Action Level of 100 Bq m^{-3}, and a cost per QALY of £29,000 for an Action Level of 50 Bq m^{-3}, while for areas with higher mean long-term radon concentrations the costs per QALY would be lower. We estimate that a policy of requiring such measures in areas where the mean long-term radon concentration is at least 90 Bq m^{-3}, with an Action Level of 100 Bq m^{-3} and assuming 100% compliance by the occupants, would result in active measures being installed in approximately 120 homes each year, and would avert an additional 0.03 lung cancers every year for each year that it is in place, in addition to the lung cancers averted by a policy of basic measures alone. For a similar policy but with an Action Level of 50 Bq m^{-3} we estimate that active measures would be installed in approximately 300 homes each year. If the percentage reduction in radon concentration in the range 50–99 Bq m^{-3} were the same as at higher levels, this policy would avert an additional 0.05 lung cancers every year for each year that it is in place, in addition to the lung cancers averted by a policy of basic measures alone (see Table 7.1).

Turning to existing homes, the current government policy in England consists of targeting areas of the country with mean long-term radon concentrations of at least 64 Bq m^{-3} or above (ie 5% or more of homes with measurements above 200 Bq m^{-3}). At its boundary (ie in areas where the mean long-term radon concentration is 64 Bq m^{-3}), this policy has a cost-effectiveness ratio of £36,800 per QALY, which is above the maximum acceptable range in other contexts. However, we estimate that if the policy were fully implemented nationally in all areas where the mean long-term radon concentration were 64 Bq m^{-3} or higher, it would, after a lag period, avert 0.9 lung cancers each year (Table 7.2).

TABLE 7.2 Existing homes: numbers of lung cancers averted [(i)] from various radon reduction policies. Other assumptions in the calculations are listed in Section C3 and Table M1 [(ii)]

Policy	Total number of lung cancers averted every year after policy fully implemented
(a) Current government policy in England [ie targeting invitations to test in areas with a mean radon concentration of 64 Bq m^{-3} or higher (ie at least 5% of homes with measurements above 200 Bq m^{-3})] assuming a 30% acceptance rate and a remediation rate of 20% of those above the Action Level	0.9
(b) Effect of targeting invitations to test at areas with a mean long-term radon concentration of 60 Bq m^{-3} and recommending remediation when measurement is above 100 Bq m^{-3}, assuming a 30% acceptance rate and a 20% remediation rate	2.1
(c) As (b) but with acceptance rate increased from 30 to 60% and remediation rate increased from 20 to 50%	10.4

Notes
(i) Reducing the radon concentration in a home will avert radon-induced lung cancers in every future year.
(ii) Calculations based on estimate of radon-related lung cancer risk from studies of residential radon and lung cancer in Europe (Darby et al, 2005, 2006).

Reducing the Action Level below 200 Bq m^{-3} would increase the cost-effectiveness of policies directed at existing homes and, if the willingness of householders to remediate were unchanged by such a reduction, an Action Level of 100 Bq m^{-3} would be more cost-effective than the present one of 200 Bq m^{-3}, with a cost per QALY of £28,200 in areas with a mean long-term radon concentration of 64 Bq m^{-3} and £29,800 in areas with a mean long-term radon concentration of 60 Bq m^{-3}. Lowering the Action Level would, in addition, avert more lung cancers each year. We estimate that when it had been fully implemented, and after a lag period, a policy of targeting invitations to test in areas with a mean long-term radon concentration of 60 Bq m^{-3} or higher with an Action Level of 100 Bq m^{-3} would avert 2.1 lung cancers each year, which is considerably more than the number that would be averted by the current policy. Lowering the costs of identifying existing homes with high radon concentrations, or increasing the proportion of identified homes that undertake remediation, would further improve cost-effectiveness and potentially provide cost-effective programmes for existing homes in additional areas with lower mean radon concentrations. For example, if the percentage of householders accepting the invitation to measure the radon concentration in their home increased from 30 to 60% and the percentage remediating following a measurement above the Action Level increased from 20 to 50%, then a policy that targeted areas of the country with a mean radon concentration of 60 Bq m^{-3} or higher with an Action Level of 100 Bq m^{-3} would, after full implementation, avert 10.4 lung cancers each year (Table 7.2).

Where a radon measurement already exists for a particular home, eg from a previous offer of a free measurement or because the householder has already purchased a measurement, carrying out remedial work has a cost per QALY of £25,300 in a population with the smoking habits of the general population at a measured radon concentration of 100 Bq m^{-3}, and lower costs per QALY at higher values. If radon remediation were carried out in all homes that had already been measured by the HPA and found to have radon concentrations of 100 Bq m^{-3} or above, then 31 lung cancers would be averted each year if the population of those measuring had smoking status similar to that of the whole country (see Table 7.3).

As noted above, there are reasons to believe that the true risk from residential exposure to radon is somewhat higher than the central estimate from the pooled analysis of the European case–control studies of residential radon and lung cancer, on which our main economic analyses are based. This implies that both radon preventive and remedial measures will be somewhat more cost-effective than the figures given here. However, this cannot as yet be quantified.

The above calculations assume that radon policies are equally likely to reduce radon concentrations for current smokers, ex-smokers and lifelong non-smokers. For basic preventive measures in new homes this is likely to be true, but recent evidence suggests that remediation rates among lifelong non-smokers may be higher than rates among smokers. If this is true, then the cost-effectiveness of remediation in existing homes would, in practice, be less favourable than indicated by our analyses, possibly by a substantial amount.

Finally, we consider the overall costs of the policies suggested by our analyses in 2007 prices and without discounting. We estimate that the approximate annual cost of installing basic radon preventive measures in all new homes throughout the entire country would be £20 million (200,000 homes x £100). This cost would be borne by house purchasers and it might well decrease substantially if installation of such

TABLE 7.3 Homes already measured by the HPA: numbers of lung cancers averted [(i)] if radon remediation is carried out in all homes

Range of measured radon concentrations (Bq m^{-3})	Number of observations	Mean of measured radon concentrations	Estimated mean true radon	Number of lung cancers averted each year if remediation carried out on all homes with radon measurements in this range
<50	223,806	26	30	12.1
50–99	89,822	71	71	11.6
100–199	53,244	138	125	12.0
200–399	25,011	275	224	10.1
400+	10,541	702	490	9.4
Total	402,424	–	–	55.2

Note: (i) Reducing the radon concentrations in a home will, after a lag period, avert additional radon-induced lung cancers in every future year.

measures became routine, although if it were necessary to monitor the radon concentration in new homes, then this would incur an additional cost. If the full preventive measures policy described above were also applied in areas with mean long-term radon concentrations of at least 90 Bq m^{-3} and with the Action Level reduced to 100 Bq m^{-3}, we estimate the further annual costs would be approximately £136,000 for testing and installation, plus £8,000 annual running costs thereafter for the fans installed over that year for an Action Level of 100 Bq m^{-3}. For an Action Level of 50 Bq m^{-3} the corresponding testing and installation costs would be £190,000, plus £21,000 annual running costs.

For a policy that targeted existing homes in areas of the country with mean radon concentrations of 60 Bq m^{-3} or higher and with an Action Level of 100 Bq m^{-3}, we estimate that the invitation costs would be £790,000. If 30% of those invited to test accepted and 20% of those above the Action Level remediated, the testing costs would be £6.0 million, and the remediation would cost £5.7 million for the initial outlay with £191,000 each year for running costs. If 60% of those invited to test accepted the invitation and 50% of those above the Action Level remediated, the testing cost would be £12.0 million, and the remediation would cost £28.3 million for the initial outlay with £960,000 annual running costs.

While our conclusions are relevant to residential exposures we are aware that some of our findings and approaches may also have implications for other buildings, including offices and schools.

8 Recommendations

We are satisfied that the available evidence indicates a causal association between lung cancer and radon at the concentrations encountered indoors in ordinary homes and other buildings in many parts of the UK. About 1100 radon-induced lung cancer deaths occur each year in the UK, most of them as a result of exposure at concentrations well below the current Action Level of 200 Bq m^{-3}, and most of them in areas other than those currently designated as radon Affected Areas. This constitutes a material public health issue. Current policy focuses on individuals at highest risk and does not address the great majority of radon-induced lung cancers. We believe that the time is now appropriate to give emphasis to a population-based approach that aims to reduce the collective dose from radon exposure progressively until it is as low as reasonably achievable, thereby reducing progressively the number of radon-induced lung cancers that occur in the UK each year.

1 We therefore recommend that the Health Protection Agency should review its advice for control of indoor exposure to radon in the UK and that government departments should revise their policies to take account of the new information on the risks of exposure to indoor radon.

2 We also recommend that much greater emphasis should be given to reducing the mean indoor radon concentration throughout the UK, thereby gradually reducing the number of radon-induced lung cancers that occur each year.

3 We recommend that the HPA should review the concept of a radon Affected Area as it has tended to lead to the belief that other areas are not affected by radon.

In reviewing advice and policy we recommend that the health economic techniques that we have illustrated for evaluating the cost-effectiveness of alternative policies for control of indoor exposure to radon in terms of costs per quality adjusted life-year (QALY) gained and also the estimates of the numbers of lung cancers eventually avoided by the various policies should be taken into account in determining practical measures of control. The parameters used in these analyses depend on the details of the programmes proposed and will change as knowledge increases. For example, the incremental costs per QALY gained given in these recommendations are based on the radon risk estimate from recent European studies of residential radon and lung cancer, and there is some evidence that the true risk may be somewhat higher than this estimate. If this were true then the costs per QALY given in this report would decrease and the prevention of radon-induced lung cancer would be more cost-effective. In addition, our recommendations are based on the assumption that current smokers are as likely to act on a recommendation to remediate as lifelong non-smokers, and there is some evidence that current smokers may in fact be less likely to remediate than non-smokers. Now that a model has been developed, further cost-effectiveness analyses can be performed to take account of new information and also to investigate the potential consequences of specific aspects of the implementation of radon control policies. In the light of the above, we make the following recommendations.

4 We recommend that the HPA considers moving towards a population-based approach in which radon exposures are considered specifically in terms of mean long-term average radon concentration in an area, recognising that this is the quantity that is directly related to the typical risk from indoor radon to an individual living in that area. The suggested approach should also take into account the year-to-year variation in measured concentrations in individual homes that has been documented in several studies in the UK and which means that in any set of radon measurements the highest values will usually be substantial overestimates of the long-term average concentration in the homes concerned.

5 A ceiling cost of £20,000–£30,000 per QALY gained (discounted and at 2007 prices) is commonly used in decision-making for health care interventions paid for by the National Health Service and across other government departments. In new-build dwellings the installation of effective radon barriers throughout the UK would be highly cost-effective compared with this range, and would avert more than eight times as many radon-induced lung cancers than the present policy. We recommend that the HPA considers advising that effective radon barriers be required in new-build dwellings throughout the UK.

6 We recommend that the HPA considers a policy for new homes in high radon areas that requires the installation of a means of under-floor ventilation, such as a sump and pipework in addition to basic preventive measures, together with measurement of the radon concentration after occupation and the installation of a fan where the measurement exceeded a specific Action Level. Our calculations indicate that such a policy would be cost-effective within the maximum level of £30,000 per QALY if it were introduced in areas that had mean radon concentrations of at least 90 Bq m^{-3} in the absence of any radon preventive measures and with an Action Level of 100 Bq m^{-3} provided that there was 100% compliance with the policy. If remediation of homes with radon concentrations in the range 50–99 Bq m^{-3} can be shown to be as effective as it is at higher radon concentrations, then an Action Level of 50 Bq m^{-3} might be practicable. If so, then the number of radon-induced lung cancers averted with an Action Level of 50 Bq m^{-3} would be slightly higher than with an Action Level of 100 Bq m^{-3} and the cost-effectiveness would remain below the £30,000 maximum provided that compliance was 100%.

7 For existing homes, we recommend that in developing policy, the main objective should be averting as many lung cancers as possible whilst keeping cost-effectiveness below the maximum per QALY gained recommended by the National Institute for Health and Clinical Excellence (NICE). We note that reducing the Action Level from its current value of 200 Bq m^{-3} is likely to increase both the cost-effectiveness and the number of lung cancers averted by policies for existing homes. For example, a policy that targeted areas with mean long-term radon concentrations of 60 Bq m^{-3} or higher, offered free radon measurements, and then encouraged householders to remediate at their own expense if the measurement is above an Action Level of 100 Bq m^{-3}, would have cost-effectiveness below the maximum and would also avert more than twice as many lung cancers as the current government policy for England, which targets areas with mean radon concentrations of 64 Bq m^{-3} or above and has an Action Level of 200 Bq m^{-3}.

8 We note that both the cost-effectiveness and the number of lung cancers averted by remediating existing buildings are highly dependent on the proportion of householders who accept the offer of a free measurement and also the proportion who undertake remedial action after a measurement has indicated that the radon concentration in their home is above the Action Level. For example, if the proportion of householders accepting a free measurement could be increased from its current value of around 30% to around 60%, and if the proportion of householders undertaking remedial measures could be increased from its current value of around 20% to about 50%, then the number of lung cancers averted by the policy suggested above would increase by a factor of four. If this could be achieved at low cost, then the cost-effectiveness of remediating existing dwellings would improve, and it would become cost-effective to extend the policy to a wider area of the country, thus further increasing the number of lung cancers averted. We recommend that measures to improve uptake of testing and remediation continue to be explored.

9 Measurement of the radon gas concentration in existing homes can be viewed as a medical screening procedure. We have reviewed its potential efficacy in relation to current screening criteria and it meets all requirements. We recommend that the programme of radon measurements in existing homes be considered on the same terms as other medical screening procedures.

10 We note that dwellings with exceptionally high radon concentrations are occasionally encountered. Less than a dozen homes with measured radon concentrations above 10,000 Bq m^{-3} have so far been found in the UK. Since the risk to the occupants of these dwellings is high, especially if the occupants are smokers, and in most cases no action has yet been taken, we recommend that the HPA gives advice on practical measures that could be put in place to deal with the problem of exceptionally high radon concentrations, either by remediation or by condemnation.

11 As the great majority of radon-induced lung cancers occur in smokers, we recommend that measures to reduce smoking, which have been shown to be highly cost-effective and have the potential to avoid a large number of lung cancers, should continue to be given a high priority.

12 Where the HPA has already measured the radon concentration in a home, it may be cost-effective to carry out radon remediation if the measurement is 100 Bq m^{-3} or higher. Householders with measurements in the range 100–199 Bq m^{-3} have not previously been advised to remediate, and those with measurements above 200 Bq m^{-3} and who have not already remediated might do so given further encouragement, thereby averting additional lung cancers, especially if they are smokers. We recommend that a programme to encourage remediation in homes where a previous measurement was 100 Bq m^{-3} or above be considered.

13 In the present report, detailed consideration has been given only to homes, but it may well be that basic radon prevention would be cost-effective for other types of buildings as well. We recommend that a policy of basic radon prevention in other buildings, including schools, be considered.

In addition to the above recommendations relating to policy, we also make the following research recommendations.

14 We recommend that current efforts to carry out a pooled analysis of data from studies of residential exposure to radon and lung cancer be continued and completed, and that additional effort is expended to attempt to clarify the relationship between age at exposure to radon and the subsequent risk of developing radon-induced lung cancer.

15 The cost of effective radon barriers, their efficiency in reducing radon concentrations and their durability are all critical factors in determining their cost-effectiveness in new-build dwellings. We therefore recommend that the effectiveness of radon barriers and other radon preventive measures be explored to lower their cost and to improve their efficiency and durability, together with measures to ensure their proper installation.

16 Efforts to date have focused on remediation of existing dwellings where the measured radon concentration is above 200 Bq m^{-3}. The recommendations above place greater emphasis on remediation at lower radon concentrations, but detailed knowledge about remediation rates, remediation costs and remediation effectiveness at lower concentrations is sparse at present. We recommend that further work be carried out on remediation rates, remediation costs and remediation effectiveness as applied to dwellings with radon levels below 200 Bq m^{-3}.

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Appendix A
Radiation Quantities and Units

Quantities and units relevant to radon are summarised in Table A1. Different units have been used for these quantities historically: for example, the earlier unit for absorbed dose is the rad, while the SI unit is the gray.

TABLE A1 Radiation quantities and units

Quantity	Description	SI unit	Symbol	Relation to other units
Activity	Decays per second	becquerel	Bq	1 curie (Ci) = 3.7 10^{10} Bq
Activity concentration	Activity per unit volume	becquerel per cubic metre	Bq m^{-3}	1 picoCi per litre (pCi L^{-1}) = 37 Bq m^{-3}
Potential alpha energy concentration (PAEC)	Alpha energy from radon decay products per unit volume	joules per cubic metre	J m^{-3}	1 Working Level (WL) = 2.08 10^{-5} J m^{-3}
Exposure (traditional)	Charge per unit mass of air	coulombs per kilogram	C kg^{-1}	1 roentgen (R) = 2.58 10^{-4} C kg^{-1}
Exposure (radon)	–	Time integral of potential alpha energy concentration	J m^{-3} h	1 Working Level Month (WLM) = 3.5 10^{-3} J m^{-3} h 1 WLM = 6.37 10^{5} Bq m^{-3} h equilibrium equivalent radon exposure
Absorbed dose	Energy absorbed per unit mass	gray	Gy	1 rad = 0.01 Gy
Equivalent dose	Absorbed dose to an organ multiplied by radiation weighting factor	sievert	Sv	1 rem = 0.01 Sv
Effective dose	Sum of equivalent doses to organs multiplied by their organ weighting factors	sievert	Sv	1 rem = 0.01 Sv

A1 Quantities and units relating to radon exposure

A fundamental quantity is the *activity*, defined as the number of radioactive decays per second. The SI unit of activity is the becquerel (Bq), defined as one decay per second.

In the context of inhaled radon and its short-lived decay products it is usual to use *activity concentration* – the number of decays per second per cubic metre of air (Bq m^{-3}). Thus air with 200 Bq m^{-3} has, on average, 200 decays of radon nuclei per second in each cubic metre of air. Spending 8000 hours in an atmosphere containing an activity concentration of 100 Bq m^{-3} of radon would give a time integrated radon exposure of 8 10^5 Bq m^{-3} h or about 90 Bq m^{-3} y. (It should be noted that this is a special use of the term 'exposure'. Elsewhere in radiological protection the term 'exposure' is used to refer to the electric charge generated by radiation per unit mass of air.)

Radon decays through its chain of short-lived decay products before reaching the long-lived lead-210 (see Figure 1.1 of the main text). Most of the ionising radiation received as a result of exposure to radon is due to the short-lived decay products rather than radon gas itself. A reasonable indication of the risk associated with breathing any particular mixture of short-lived radon decay products is obtained by considering the *potential alpha energy concentration* (PAEC). This is the sum of the alpha energies per unit volume of air that the decay products will emit in decaying to the long-lived lead-210.

If none of the short-lived decay products is lost (for example, by ventilation or plating out on walls) there will be equal activities of radon and each of its short-lived decay products in air. This is described as a state of radioactive equilibrium. In practice, there will almost always be fewer decay products than the equilibrium activity. The *equilibrium equivalent radon concentration* (EER) of an atmosphere is the concentration of radon gas, in equilibrium with its short-lived decay products, which would have the same PAEC as the actual radon decay products in the atmosphere in question.

The *equilibrium factor*, *F*, is the ratio of the EER to radon concentration. The value of *F* is normally found to be in the range 0.3–0.5 in homes and in above-ground workplaces.

The historical unit of PAEC, the *Working Level* (WL), was traditionally used for describing the exposure of miners to radon decay products. The WL is so called because there was a period during which this was the level to which exposures in mines in the USA were controlled. One WL is equivalent to 3700 Bq m^{-3} of radon gas in equilibrium with its decay products. A *Working Level Month* (WLM) is defined as exposure to one WL for a working month of 170 hours. One WL is approximately equal to 7500 Bq m^{-3} of radon with an equilibrium factor of 0.5. One WLM is equal to a radon exposure (equilibrium factor of 0.5) of 1.26 10^6 Bq m^{-3} h, or about 144 Bq m^{-3} y.

A2 General dosimetric quantities

Absorbed dose is defined as the energy (from ionising radiation) absorbed per unit mass of the irradiated tissue. The SI unit of absorbed dose is the *gray* (Gy). Absorbed dose can be calculated for the body as a whole or, where energy deposition varies across the body, for each organ or tissue separately. Further quantities, more closely related to estimates of radiation risk, are derived from absorbed dose.

Some external sources of radiation give short bursts of radiation (for example, a medical X-ray set). In these instances it is natural to give the dose per examination. Other types of external exposure are protracted, such as those from cosmic rays. In these circumstances the dose per week or year can be given. A slightly different situation applies with radionuclides which enter the body by inhalation or ingestion, as is the case with radon decay products. Here the dose from any intake will generally fall off with time, but will continue after the intake ceases until the radioactivity (including that from decay products) has decayed away, or the material has been lost from the body by biological clearance mechanisms. The short-lived radon decay products have half-lives of less than an hour, but the exposures are protracted, so exposures and doses per year are normally quoted.

As noted above, the fundamental physical quantity used in radiological protection is the *absorbed dose*. However, it is found that different types of radiation differ in their ability to induce biological damage (particularly to induce cancers). This is reflected in *radiation weighting factors*, w_R, which the International Commission on Radiological Protection (ICRP) has estimated, using a number of sources of information, with the aim of generating a measure, for use in radiological protection, of the adverse health effects likely to be caused by different types of radiation.

The product of absorbed dose and w_R is called the *equivalent dose*. It is measured in *sievert* (Sv). For X-rays and gamma rays and for electrons, the value of w_R is 1.0, so the absorbed dose and the equivalent dose to an organ have the same numerical value but different units (Gy and Sv, respectively). For alpha particles, w_R is specified as 20. This is particularly relevant to radon because most of the risk is due to alpha particles.

Not only do different kinds of radiation differ in their ability to induce damage, the various tissues of the body differ in their sensitivity to radiation. Generally speaking, tissues where cells are actively dividing are more sensitive than those where they are not. This varying sensitivity makes it difficult to compare the effects of different types of partial body irradiation. For example, inhalation of radon decay products results, predominantly, in doses to the respiratory system, while inhalation of iodine-131 leads mainly to dose to the thyroid. The ICRP has specified *tissue weighting factors*, w_T, which are designed broadly to reflect the differing radiosensitivities of different tissues. Values of w_T are given in Table A2.

The *effective dose* is defined as the sum of the equivalent dose, H_T, to each tissue of the body multiplied by the corresponding w_T.

$$E = \Sigma\, w_T H_T$$

The unit of effective dose is also the sievert. This quantity is designed to be approximately proportional to the total risk of detriment to the subject. The effective dose corresponding to a pattern of partial body irradiation is the uniform whole body radiation dose which is estimated to give the same total risk. Effective doses thus allow different kinds of irradiation to be compared.

It can be seen that the accuracy of the calculation of the absorbed dose to organs (in Gy) depends upon how well the distribution of radon and its decay products within the body is known. The corresponding equivalent and effective doses (given in Sv) are derived from absorbed dose using radiation and tissue weighting factors which are specified by the ICRP on the basis of an assessment of the available evidence as it relates to radiological protection in general. It should be noted that the accuracy with which these

TABLE A2 Tissue weighting factors

Organ or tissue (T)	Tissue weighting factor, w_T
Gonads	0.20
Red bone marrow	0.12
Colon	0.12
Lungs	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05

After ICRP (1996). Age-dependent doses to members of the public from intakes of radionuclides: Part 4. Inhalation dose coefficients. ICRP Publication 71. *Ann ICRP*, **25**(3–4).

quantities reflect risk depends upon the validity of the assumptions made in defining the radiation and tissue weighting factors and on the extent to which they apply to the particular circumstances considered. Both radiation and tissue weighting factors are, of course, very simple summaries of complex biological responses. Nevertheless, they are useful tools which allow rough and ready comparisons of different kinds of radiation exposure.

It should be noted that epidemiological studies of the effects of exposure to radon, whether occupational or residential, do not use the effective dose concept. Analyses are carried out using measures of exposure.

Appendix B
Measurement of Radon Concentrations

B1 Rationale for measuring radon gas rather than decay products

Alpha particles from short-lived radon decay products deliver much larger doses to the body than radon gas does, so inhalation of radon decay products is of greatest concern for health. In practice, it is very difficult to measure long-term exposure to radon decay products. Measurement of exposure to radon gas, however, is relatively easy.

Radon decay product exposures may be estimated from a knowledge of radon gas exposures and the equilibrium factor, *F* (see Appendix A). In homes in the UK, *F* does not vary widely, and has an average value of about 0.4. Hence a measurement of radon gas exposure in each home is sufficient for estimating radon decay product exposures. Alternatively, estimates of risks from radon in dwellings may be expressed in terms of risk per unit radon gas exposure because of the small variation in *F*.

B2 Time period of measurement

The possible adverse effects of radon exposure depend on total exposure over many years. Although it would be convenient if this exposure could be estimated from a single short-term measurement, spot measurements of radon in the home may be misleading, despite the fact that they can give quite accurate measurements of the concentration at that time. This is because radon concentrations may vary, sometimes quite dramatically, from hour to hour, day to day, month to month, and also year to year (Lomas and Green, 1994; Miles, 2001; Darby et al, 2006).

Because of these variations, an estimate of the long-term average radon concentration over many years is needed. Ideally measurements made in any single year would take place over the full year, to cover variations with the seasons. However, detectors may be lost if left in a dwelling for so long, and many householders do not want to wait this long for results. For these reasons, routine measurements are usually made over a period of three months, and the annual average estimated using correction factors based on typical seasonal variations.

In interpreting measurements made within a single year, account also needs to be made of the substantial year-to-year variation that has been observed (see Appendix C).

There are several distinct circumstances in which estimates of radon concentrations in a dwelling might be needed. In case–control studies of the risk of disease associated with radon exposure, a measure of the true long-term average exposure during a time window or reference period over many years in the past is required. Alternatively, to guide a possible decision to undertake remedial work, the long-term average over many years going forward into the future is relevant. In virtually all circumstances, it is the long-term average radon concentration over many years that is of interest.

When time is short because a dwelling is being sold, screening measurements over a shorter period than three months may be adequate in many cases to determine whether or not a particular dwelling is above a particular threshold. Although the uncertainty on such short-term measurements is much greater than that for long-term measurements, in most cases the concentration measured will be found to be well below or well above the threshold. Under these circumstances, a confident verdict on whether the true radon concentration in the dwelling is above or below the threshold may be given, despite the uncertainty. If the results of the screening measurement are closer to the threshold, no conclusion can be drawn about whether the long-term average radon concentration is above or below the threshold.

B3 Radon and radon decay product measurement methods

B3.1 Etched-track detectors

Etched-track detectors have been the most commonly used instruments, both in case–control studies of long-term exposure and in the remediation setting. These instruments consist of a plastic detector in a small container. Radon gas diffuses into the container and decays, emitting alpha particles which leave invisible damage tracks in the plastic. When the device is returned to the laboratory, the plastic is etched in a caustic solution, producing pits where it has been damaged. The pits can be counted automatically, for example, under a microscope. Etched-track detectors are relatively cheap, and suitable for long-term measurements, often being deployed for three months. They are also known as passive alpha track detectors. The great majority of measurements of radon concentrations are made with etched-track detectors.

There are variations in results from similar detectors measuring in the same place over the same period. They result from differences between notionally similar detectors and between batches of detectors. The quality assurance standard applied by the HPA specifies that, for three-month measurements of radon concentrations in the range of about 50–1000 Bq m^{-3}, 95% of measurements should be within 20% of the true value.

B3.2 Electret ionisation chambers

An electret in an ionisation chamber holds an electrostatic charge which is gradually neutralised by the ionisation of the air by alpha particles emitted by radon and its decay products. Measuring the charge on the electret at the beginning and end of an exposure allows the radon concentration to be calculated. In making this calculation, an allowance must be made for ionisation caused by natural background radiation. Different types of electret and different sizes of ionisation chamber are available, suitable for measurements over periods of a few days to a few months. Care must be taken with electrets, as dropping them can cause a partial discharge, and an overestimation of the radon concentration.

B3.3 Charcoal detectors

These consist of a small container of activated charcoal, which absorbs radon out of the air. After exposure, the detector is sealed and returned to the laboratory, which can measure how much radon is still present. Radon has a 3.82-day half-life, and the radon absorbed at the beginning of the exposure decays away after a few days, so the measurement duration is limited and the device does not measure the true average exposure. Charcoal detectors are not suitable for long-term measurements, but may be used for screening purposes.

B3.4 Scintillation cell

A scintillation cell is a container with a coating on the inner wall of a material which emits a flash of light when struck by an alpha particle. These flashes can be counted using a photomultiplier. Scintillation cells can be used in the field, but it is necessary to wait for three hours before starting the measurement and they have now largely been superseded. Because they measure radon concentration at one point in time, they are not suitable for measuring radon exposures.

B3.5 Active monitors

Various types of electronic monitor are available. These sample the air continuously and measure either radon or its decay products, typically averaging over one hour. The measurement is repeated over successive time periods. This allows the variation in radon concentrations from hour to hour to be determined.

B3.6 Glass based measurements

Some of the short-lived decay products of radon in room air are lost by plating out on surfaces. These surfaces will include the glass protecting pictures or mirrors. When short-lived alpha emitting decay products on such surfaces decay, in a proportion of cases the recoil energy will embed the decay product, lead-210, in the glass. Because lead-210 has a long half-life (20 years) and because it is protected by being embedded in the glass, it can be detected many years later. In practice, this is done by using etched-track detectors to measure polonium-210, the alpha emitting decay product of lead-210. From this measurement, an estimate can be made of the cumulative radon exposure in the rooms where the glass object has been kept.

Since people will often carry pictures with them as they move from house to house, this technique allows an estimate of an individual's cumulative exposure. At one time it was thought that this technique might be an appropriate method for use in epidemiological studies, as it appeared to have the potential to derive a measure of the long-term average radon exposure experienced by an individual over many previous years, including periods in previous homes where measurement would be either costly or, if the home had been altered or demolished, impossible.

However, the method has a number of disadvantages. One of these is that the measure of exposure it provides is a weighted average of the exposure received in different years, with lower weights for more distant years, as determined by the 20-year half-life of lead-210. Another disadvantage is that aerosol conditions in rooms, including the effects of smoking, can affect the plate-out of decay products on surfaces and so affect the activity measured. For these reasons, this method has not been extensively used in recent epidemiological studies.

B4 Measurement of radon in water

Various methods are used to measure radon in water. One approach is to extract the radon by bubbling nitrogen through the water and condensing out the radon using liquid nitrogen; it can then be assessed using, for example, a scintillation cell. Liquid scintillation counting can also be used. In this method the water is shaken up with a liquid which emits flashes of light when an alpha particle passes through it. These flashes can be counted. Methods which use an etched-track device or electret to measure radon degassed from the water have also been used.

Von Philipsborn (1997) has described a simple method for assessing radon decay products in water. This makes use of the strong absorption of the decay products on commercially available glass-fibre filters.

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Von Philipsborn H (1997). Efficient adsorption of waterborne short-lived radon decay products by glass fibres. *Health Phys*, **72,** 277–281.

Appendix C
Variability and Distribution of Residential Radon Concentrations

S Darby, P McGale and S Read

C1 Variability between measured radon concentrations in different homes in an area

Surveys have shown that the distribution of measured radon concentrations in homes within a given area is usually very close to log-normal (see, for example, Figure 2.1 of the main text). The sample mean and sample variance of the natural logarithms of the measured values can be used to estimate the mean and also the median and other quantiles of the underlying distribution. The precision with which such quantities can be estimated will depend on the number of measurements. Also, for a given distribution and sample size, quantities in the centre of the distribution can be estimated more precisely than quantities in the upper tail of the distribution. To illustrate this, 1000 samples, each with 30 observations, were generated from a log-normal distribution similar to that arising in an area with 5% of radon measurements greater than 200 Bq m^{-3}. For each of the 1000 samples, the mean, median and the upper 30, 10, 5, 3 and 1 percentiles of the log-normal distribution have been estimated and the results summarised in Table C1 and Figure C1. The quantity that is estimated most precisely is the median (standard deviation of 8 Bq m^{-3} and coefficient of variation of 16%). The mean and upper 30 percentile are also estimated reasonably precisely. In contrast, the upper percentiles can be estimated only with considerable uncertainty and the estimated value often differs substantially from the true value.

C2 Year-to-year variability in measured residential radon concentrations

For the detectors and the measurement protocols that have been used for the measurements made by the HPA and also for the measurements in the case–control studies of residential radon concentration, the variability between multiple radon measurements made simultaneously in the same room is low (Miles et al, 2004). This indicates that the instrument measurement error in the radon detectors used is small. Numerous studies have, however, shown that the random variability between repeated measurements of radon concentration made in the same home but in different years is substantial. This is the case even when each measurement is made over a whole year, or made over several months with an appropriate seasonal adjustment, and the coefficient of variation for such repeated measurements is of the order of 50% (Darby et al, 2006). This variation indicates that the radon concentration in homes varies randomly from year to year, probably according to variations in the weather and in the lifestyle of the residents (eg degree of ventilation or central heating). It has been shown that the variance of repeated measurements made in the same home in different years is larger

TABLE C1 Summary statistics from 1000 samples, each with 30 observations, drawn from an underlying distribution of the form $4 + X$, where X has a log-normal distribution with parameters $\mu = 3.77$ and $\sigma^2 = 0.84$. This value of σ^2 corresponds to a geometric standard deviation of 2.50. The distribution is similar to that for radon measurements in a 1- or 5-km^2 area in the UK with 5% of observations above 200 Bq m^{-3}

		Properties of 1000 estimates				
Quantity	True value	Mean	Standard deviation	Smallest estimate	Largest estimate	Coefficient of variation (%)
Median	47	48	8	30	80	16
Mean	70	72	14	43	131	19
Upper percentiles:						
30	74	75	13	46	132	17
10	144	147	33	79	291	22
5	200	205	53	98	441	26
3	247	255	72	114	577	28
1	370	386	128	148	961	33

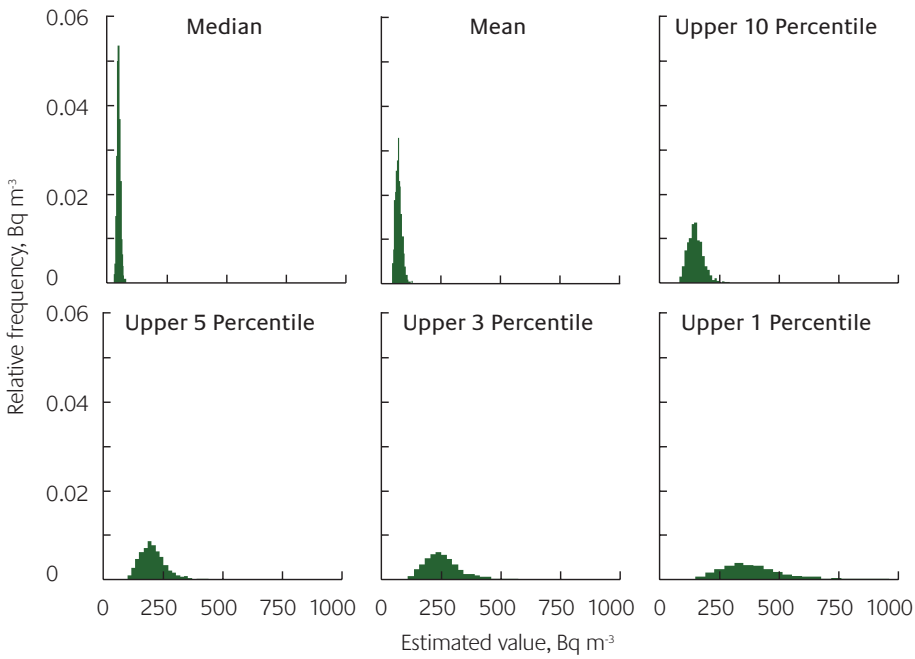

FIGURE C1 Histograms of estimated values (Bq m^{-3}) of the mean, median and upper 10, 5, 3 and 1 percentiles from 1000 samples, each of size 30, drawn from a log-normal distribution similar to that seen in an area where 5% of radon measurements are above 200 Bq m^{-3}. All areas are equal to one. (See legend to Table C1 for description of underlying log-normal distribution.)

in homes with larger long-term average radon concentrations, but that the coefficient of variation remains approximately constant over different values of the long-term average (Reeves et al, 1998). This suggests that the distribution of long-term radon mean concentrations in different homes in a given area is likely to be approximately log-normal.

An example illustrating the distribution of radon measurements that might be obtained if it were possible to measure the concentration in a home many times, each one in a different year, is shown in Figure C2. The long-term average concentration in this example is 100 Bq m^{-3}, and the coefficient of variation between repeated measurements is taken to be 50%. There are more measurements below the long-term average than above it but the mean absolute difference between the long-term average and the measurements that lie above it is larger than the mean absolute difference between the long-term average and the measurements that lie below it, a feature that occurs when a random variable has a skewed distribution such as the log-normal distribution.

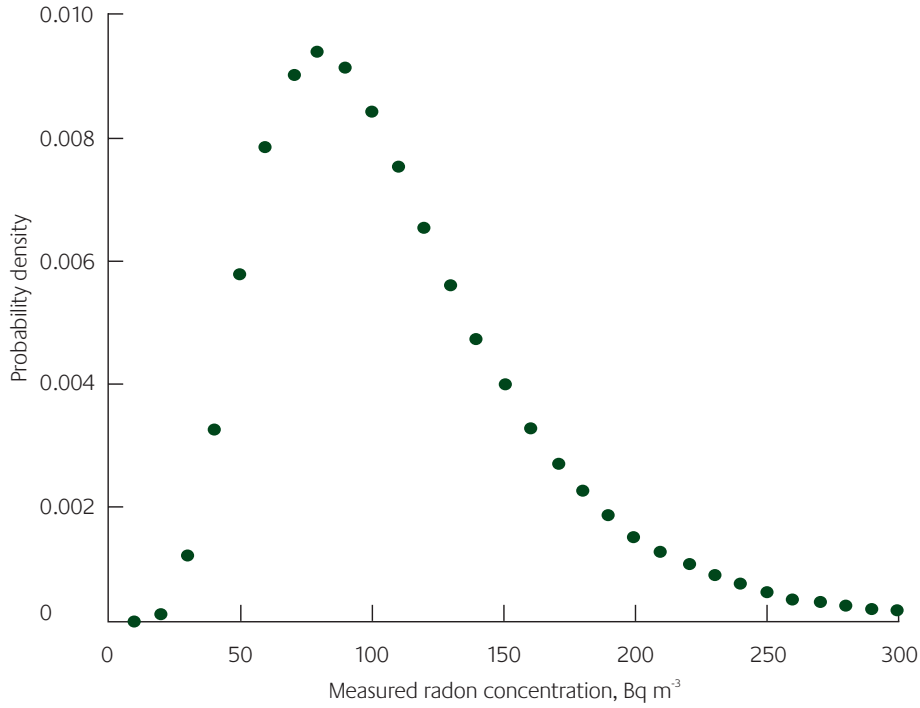

FIGURE C2 Distribution of measured radon gas concentrations that would arise from repeated measurements made in different years in a home in which there was a long-term average value of 100 Bq m^{-3} and a coefficient of variation for repeated measurements of 50%

C3 Relationship between measured residential radon concentration and the long-term average for the UK

Where information on the variability between repeat measurements, made in the same home but in different years, is available for a sample of homes in an area, it can be used in conjunction with a measurement made in a particular home to obtain a better estimate of the long-term average radon concentration in that home than is given simply by the measured value. When both the long-term average radon concentration and the distribution of measured values for a given long-term value are log-normally distributed, this relationship can be derived theoretically. If B is the background outdoor radon, $B + Y$ the estimated long-term average value corresponding to measured value $B + X$ made during the course of a single year (or over a few months and adjusted for seasonal variation to obtain an estimate for a full year), and $X' = \log_e X$, then

$$Y = \exp\{[\,(X'/\sigma_\varepsilon^2 + \mu/\sigma_x^2)/(\,1/\sigma_\varepsilon^2 + 1/\sigma_x^2)] + 0.5/(\,1/\sigma_\varepsilon^2 + 1/\sigma_x^2)\}$$

where σ_ε^2 is the variance of the logarithms of measurements made in the same home but in different years, σ_x^2 is the variance between different homes of the long-term average logarithm of radon concentration, and μ is the mean of the long-term average logarithm of radon concentration. It is clear that there is no single estimated long-term average value corresponding to a given measured value, as the relationship between the two involves not only the variance of repeated measurements but also both the mean and the variance of the population of the long-term average logarithm of radon concentrations from which the measured value is regarded as having arisen. For the UK as a whole a typical value for B is 4 Bq m^{-3}, and studies have shown that σ_ε^2 is around 0.2 (Hunter et al, 2004; Darby et al, 2006), while the results of the national survey (Wrixon et al, 1988) suggest values of 2.27 and 1.12 for μ and σ_x^2, respectively. For small areas of, say 1 or 5 km^2, measurements from the HPA database suggest that σ_x^2 is usually around 0.64, while μ has a wide variety of values, depending on the location of the area. The relationship between a given measured value and the corresponding best estimate of the long-term average value is illustrated in Table C2(a) under three different circumstances.

As illustrated in Figure C1, the percentiles in the upper tail of the log-normal distribution can be estimated only with considerable uncertainty from a limited number of measurements, while quantities in the centre of the distribution can be estimated much more precisely. It should be noted that, if a very large number of measurements were available, then quantities such as the upper percentiles of the distribution could be estimated accurately. However, as described in the following section, it would still be necessary to take account of measurement error. The theoretical relationship between the percentages of measurements above 200 Bq m^{-3} and the median and mean measured and long-term average radon concentrations in a small area when an infinite number of measurements are available, are given in Table C2(b), and the mean long-term average radon concentrations corresponding to particular mean measured values are tabulated in Table C2(c).

TABLE C2 Relationship between measured radon concentration and estimated long-term values. Calculations performed assuming outdoor radon concentration of 4 Bq m^{-3} and σ_ε^2 (ie the variance of the logarithms of measurements made in the same home in different years) = 0.2. In part (a) σ_x^2 (ie the variance of the long-term average logarithm of radon concentrations) = 1.12 if the measured value is considered as part of the UK as a whole. Elsewhere in part (a) and in parts (b) and (c), where the measured value is considered as part of a small area of 1 or 5 km^2, σ_x^2 = 0.64. In parts (b) and (c) it is assumed that a very large number of measurements is available, so that sampling error is eliminated

(a) Measured value and some examples of possible corresponding estimated long-term average values

Measured value (Bq m^{-3})	10	15	21	25	50	100	200	400	1000	2000
Estimated long-term average value (Bq m^{-3}) if the measured value is considered as part of the UK as a whole	11	16	21	24	44	78	139	250	541	973
Estimated long-term average value (Bq m^{-3}) if the measured value is considered as part of a small area where the average measured value is 10 Bq m^{-3}	10	13	17	19	32	52	87	147	292	493
Estimated long-term average value (Bq m^{-3}) if the measured value is considered as part of a small area where the average measured value is 100 Bq m^{-3}	15	22	29	33	58	98	165	280	561	950

(b) Relationship between percentage of measurements > 200 Bq m^{-3}, and median and mean of measured and long-term average concentrations for a small area

% of measurements > 200 Bq m^{-3}	1	3	5	10	30
Median of both measured and long-term average radon (Bq m^{-3})	27	39	47	65	125
Mean measured radon (Bq m^{-3})	39	57	70	96	188
Mean long-term average radon (Bq m^{-3})	36	52	64	87	171

(c) Relationship between mean long-term average and mean measured concentrations for a small area

Mean long-term average radon (Bq m^{-3})	5	10	15	20	25	30	35	40	45	50
Median of both measured and long-term average radon (Bq m^{-3})	4.7	8.4	12	16	19	23	27	30	34	37
Mean measured radon (Bq m^{-3})	5.1	11	16	22	27	33	38	44	49	55

C4 Effect of year-to-year variability in measured residential radon concentrations in the European pooling study

The aim of epidemiological studies of residential radon and lung cancer is to relate the risk of the disease to the long-term average radon concentration to which an individual is exposed. The data arising from such studies are analysed by regression, the name given to a general class of statistical methods that aim to identify the underlying systematic relationship between a dependent variable and one or more independent variables whilst providing an appropriate structure for the various sources of random variation involved. Here the dependent variable is the risk of lung cancer applicable to each individual in the study, and the independent variable of interest is the putative risk factor, residential radon. In addition, appropriate allowance needs to be made for the effect of other independent variables that are also risk factors for lung cancer, especially cigarette smoking.

It is crucially important to take the year-to-year random variation in radon measurements into account when estimating the relationship between residential radon concentration and the risk of lung cancer. Otherwise the underlying relationship between radon and the risk of lung cancer will be underestimated substantially (Frost and Thompson, 2000). The ideal situation would be to have the radon concentration in the homes of each individual under study averaged over the whole of the time window relevant to the risk of lung cancer. However, this is not practical as the time window is thought to extend over a period of at least 30 years prior to diagnosis. In the case–control studies, for most of the homes in which the study subjects have lived during the previous few decades, only a single radon measurement is available, taken over a period of a few months or, at most, over a year. Thus, for each home of interest, the measured concentration available in the study may be considerably higher or considerably lower than its long-term average.

When the data in the European study were analysed without taking into account the random year-to-year variability in the measured radon concentration in a home, the risk of lung cancer was estimated to increase by 8% (95% confidence interval, CI, 3–16%) per 100 Bq m^{-3} increase in the measured radon concentration (Darby et al, 2005, 2006). However, the data concerning the random year-to-year variability suggested that, for most individuals with measured concentrations above 800 Bq m^{-3}, the measured value was substantially higher than the long-term average value. Hence, although in the group with measured radon concentrations above 800 Bq m^{-3} (n =181) the mean of the measured concentrations was 1204 Bq m^{-3}, the mean of the estimated long-term average radon concentrations corresponding to them was only 678 Bq m^{-3} (Table C3, second and third columns). At the other end of the scale, in the group with measured radon concentrations below 25 Bq m^{-3} (n = 2040), the mean of the measured concentrations was 17 Bq m^{-3}, whereas the mean of the corresponding estimated long-term average radon concentrations was slightly higher, at 21 Bq m^{-3}. The overall mean measured radon concentration in the case–control studies was around 100 Bq m^{-3}. Figure C3 illustrates the general relationship between the estimated long-term average radon corresponding to a measured radon concentration when the overall mean measured radon is close to this.

The relationship between the risk of lung cancer and measured radon concentration, ignoring the random year-to-year variability in the measurements, is illustrated in Figure C4(a) for the data from the

TABLE C3 Means of measured and estimated long-term average radon concentrations, and relative risk of lung cancer in the pooled European studies of residential radon and lung cancer

	Radon concentration (Bq m^{-3})			
Range of measured values	Mean of measured values	Mean of estimated long-term average values	Numbers of lung cancer cases / controls	Relative risk (with 95% floated CI)
<25	17	21	566 / 1,474	1.00 (0.87–1.15)
25–49	39	42	1,999 / 3,905	1.06 (0.98–1.15)
50–99	71	69	2,618 / 5,033	1.03 (0.96–1.10)
100–199	136	119	1,296 / 2,247	1.20 (1.08–1.32)
200–399	273	236	434 / 936	1.18 (0.99–1.42)
400–799	542	433	169 / 498	1.43 (1.06–1.92)
800+	1,204	678	66 / 115	2.02 (1.24–3.31)
Total	104 / 97 (i)	90 / 86 (i)	7,148 / 14,208	

Note: (i) Weighted average for controls, with weights proportional to study-specific numbers of cases.

European study. As shown in Table C3, the effect of taking the random year-to-year variability into account is to increase the concentrations by a small amount at low values, and to decrease them substantially at higher values. The relationship between the risk of lung cancer and the long-term average radon concentration is shown in Figure C4(b). The slope is considerably steeper than in part (a), but in both the relationship is consistent with a linear model. When the random year-to-year variability in the radon measurements was taken into consideration, the risk of lung cancer was estimated to increase by 16% (95% CI 5–31%) per 100 Bq m^{-3} increase in the long-term average radon concentration (Darby et al, 2005, 2006).

Several methodological studies have been carried out considering the effect of year-to-year random variability in measured radon concentrations in the context of the case–control studies of lung cancer (Reeves et al, 1998; Fearn et al, 2008). These studies have investigated the effect of the random variability both theoretically and empirically. In particular, an artificial dataset with features similar to the data in the European study of residential radon was generated on a computer. In this artificial dataset, the true slope of the relationship between radon concentration and lung cancer risk was known, as were the 'long-term average' and the 'measured' radon concentrations for each individual. When the artificial data were analysed using the known long-term radon concentrations, the estimated slope was close to the correct value, as would be expected; when the data were analysed using the measured radon concentrations but ignoring the random year-to-year variability, the estimated slope was only about half the correct value. However, when the artificial data were analysed using the measured radon concentrations, but taking the random year-to-year variability into account with the methods that were employed for the European study, then the estimated slope was once again close to its correct value (Fearn et al, 2008).

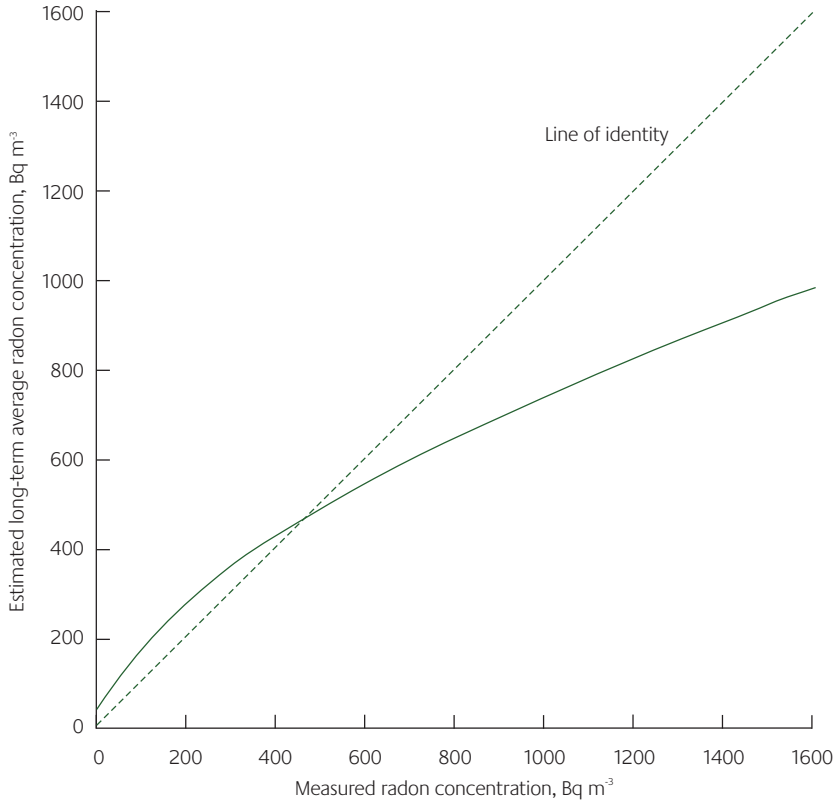

FIGURE C3 Estimated long-term average radon concentration versus measured radon. The values of σ_ε^2, σ_x^2 and μ are 0.2, 0.3 and 6.0, respectively. These are typical values for the European residential radon study. The straight line is $y = x$, ie the relationship that would be found if the true long-term average radon concentration were the same as the measured radon

C5 Distribution of residential radon concentrations in the UK

In Tables 4.1–4.3 of the main text the numbers of radon-induced deaths occurring in the UK each year have been estimated by assuming that the mean measured radon concentration in the UK is equal to that observed in the national representative survey (Wrixon et al, 1988) and assuming, as in Section C3 above, that the variance of the logarithms of measurements made in the same home but in different years is 0.2 (ie $\sigma_\varepsilon^2 = 0.2$).

In Table 4.4 of the main text it was necessary also to assume a specific form for the distribution of measured indoor radon concentrations in the UK. Outdoor radon concentration does not vary widely in the UK and, as mentioned above, it is around 4 Bq m^{-3}. When a background concentration of 4 Bq m^{-3} was subtracted from the observations in the national survey, the distribution of measured values was

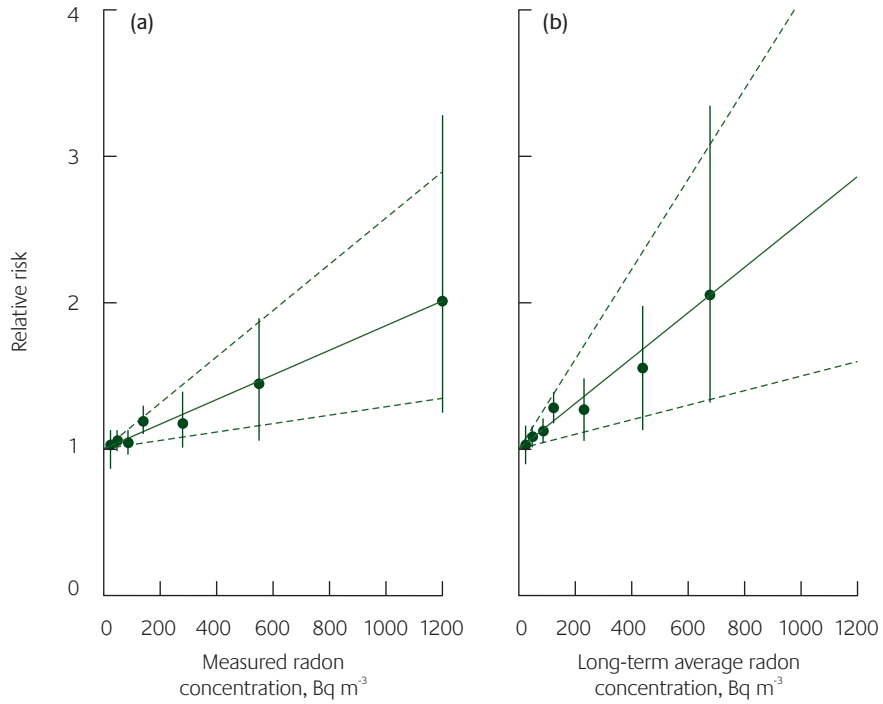

FIGURE C4 Relative risk of lung cancer according to (a) measured residential radon concentration and (b) long-term average residential radon concentration, with best-fitting straight lines and 95% confidence intervals. (Risks are relative to that for subjects with zero radon concentration.)

remarkably close to log-normal (see Figure C5). After subtracting 4 Bq m^{-3} and taking natural logarithms, the distribution of measured radon concentrations in the national survey had an estimated mean of 2.27 and an estimated variance of 1.32 (ie $\mu_{UK} = 2.27$ and $\sigma_{UK}^2 = 1.32$).

In Table 4.5, and also in Tables 7.1 and 7.2, it was necessary to make a further assumption about the distribution of the mean radon concentrations in small areas of 1 or 5 km^2. Measurements from the HPA database suggest that for small areas the variance of measured radon concentrations, on the logarithmic scale and after subtraction of outdoor radon concentration, is usually around 0.84, so that after subtraction of $\sigma_\varepsilon^2 = 0.2$ to allow for year-to-year variation, the variance of long-term radon concentrations within a small area, A, will be 0.64 (ie $\sigma_A^2 = 0.64$). Therefore, from the properties of the normal distribution, if M is the arithmetic mean of the long-term radon concentrations in dwellings in a small area, and if $M' = \log_e M$, then M' will have mean ($\mu_{UK} + 0.5\sigma_A^2$) of 2.59 and variance ($\sigma_{UK}^2 - \sigma_A^2 - \sigma_\varepsilon^2$) of 0.48, and a normal distribution has been assumed.

Finally, it should be noted that the measurements in the HPA database have a log-normal distribution after subtraction of outdoor radon (see Figure C6).

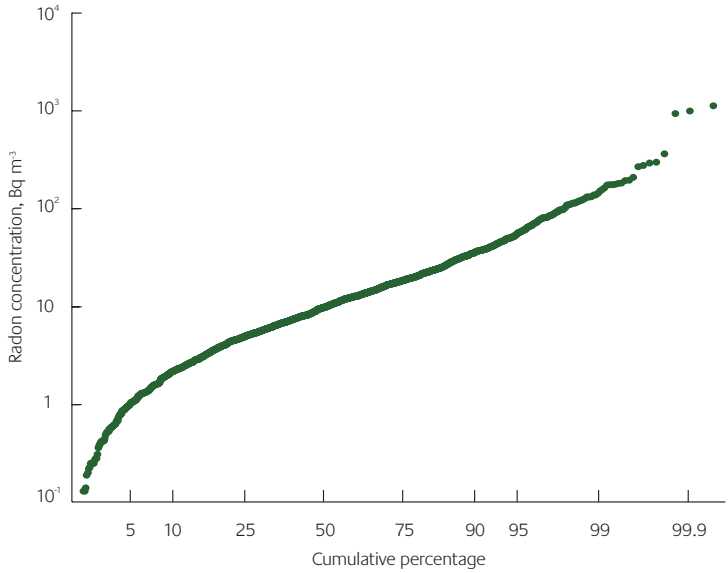

FIGURE C5 Log-normal probability plot of 2093 measurements of residential radon concentration from the UK national survey (Wrixon et al, 1988), after allowing for the outside air concentration by setting all measurements below 4.1 Bq m^{-3} to 4.1 Bq m^{-3}, and then subtracting 4 Bq m^{-3} from all measurements

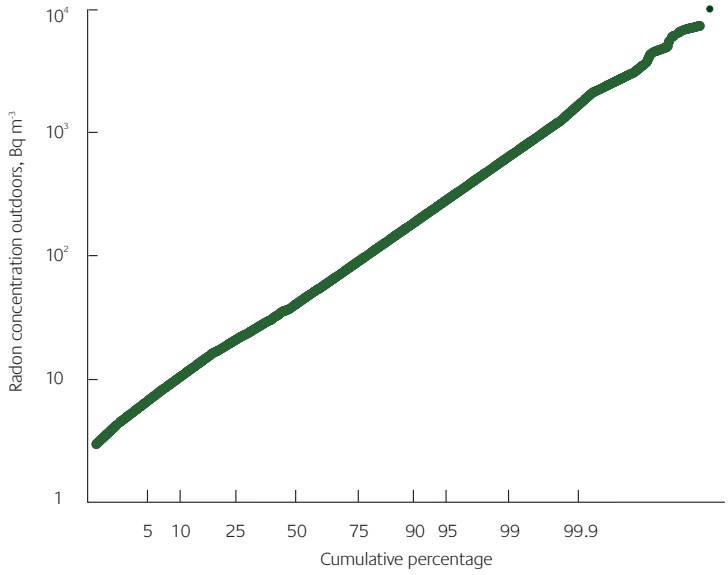

FIGURE C6 Log-normal probability plot of over 400,000 measurements of residential radon concentration from the HPA database, after allowing for the outside air concentration by setting all measurements below 4.1 Bq m^{-3} to 4.1 Bq m^{-3}, and then subtracting 4 Bq m^{-3} from all measurements

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Appendix D
Radon Policy and Programmes in the UK

A summary of radon policy in the UK is given in Table D1. The first step towards a policy occurred in 1987, when the (then) National Radiological Protection Board, NRPB, issued formal advice to government on radon in homes (NRPB, 1987). This covered both remedial measures in existing dwellings with high radon concentrations and, in outline, preventive measures in new buildings. The NRPB recommended that action should be taken to reduce long-term radon concentrations exceeding 400 Bq m^{-3} in existing dwellings and that precautions should be taken in the construction of new dwellings so that 100 Bq m^{-3} was not exceeded. The government accepted this advice and further asked the NRPB to undertake an extensive programme of measurements in areas where the highest radon concentrations were expected.

In 1990 the NRPB published revised advice on the control of radon in homes (NRPB, 1990a). It was recommended that steps should be taken to reduce radon concentrations which were above 200 Bq m^{-3}, this now being formally called an 'Action Level'. Where radon reduction was undertaken, emphasis was placed on reducing radon concentrations as far as possible, not just in edging below the Action Level. In 1990, the same Action Level of 200 Bq m^{-3} was recommended as a design limit for new dwellings, and the concept of a 'radon Affected Area' was introduced. This was a part of the country where measured radon concentrations in 1% or more of existing dwellings were expected to exceed the Action Level. It was expected at that time that anti-radon programmes would be largely concentrated within radon Affected Areas. Later that year, Devon and Cornwall became the first officially designated radon Affected Areas (NRPB, 1990b).

The NRPB developed maps of radon Affected Areas to show where radon problems were thought to be concentrated. Steps could then be taken to identify dwellings most at risk and to introduce preventive measures into new buildings in selected parts of the country. These programmes were conducted separately in England, Scotland, Wales and Northern Ireland.

In 1991, following the publication of the radon map for England (NRPB, 1990a), leaflets including an invitation to have a free radon measurement were issued via the Post Office® to all 650,000 households in Devon and Cornwall. About 12% of householders took up the offer. In cases where the measurement was above the Action Level, the householder was advised to undertake remedial work; in the great majority of cases the cost of this fell on the owner of the property. A series of other programmes followed, both in other high radon areas and also revisiting areas where programmes had already been conducted. The later programmes followed the same principle as the initial exercise of offering a free measurement, but with the cost of any remediation still falling on the householder.

It is straightforward to determine the proportion of householders who take up the offer of a free measurement. However, it is much less easy to determine the proportion of those who, when advised that the measured radon concentration in their home exceeded the Action Level, undertook remedial action.

TABLE D1 Summary of radon policy in the UK as at October 2007

Existing homes

HPA policy for the UK

It is recommended that anyone living in an area where 1% or more of homes are estimated to be above 200 Bq m^{-3} should be tested. Radon concentrations above this Action Level should be reduced below it.

Government policy for England

There is a series of targeted programmes offering free measurements on a rolling basis in areas where 5% or more of homes are estimated to be above 200 Bq m^{-3}. A free confirmatory retest after remedial works is offered to householders of dwellings identified with high radon concentrations.

Government policy for Wales

A pilot programme has offered free measurements in an area where 10% or more of homes are estimated to be above 200 Bq m^{-3}. A free confirmatory retest after remedial works is offered to householders of dwellings identified with high radon concentrations.

Government policy for Scotland

During the 1990s free measurements were offered to householders in the highest risk areas and anyone requesting a measurement in an area where more than 1% of homes were estimated to be over 200 Bq m^{-3}. A free confirmatory retest after remedial works is offered to householders of dwellings identified with high radon concentrations.

Government policy for Northern Ireland

A series of initiatives have offered free tests to householders in radon Affected Areas and tests continue to be available to householders on request unless they live in an area where less than 1% of homes are estimated to be above 200 Bq m^{-3}. A free confirmatory retest after remedial works is offered to householders of dwellings identified with high radon concentrations.

New homes

HPA recommendation for the UK

HPA advice is that precautions against radon should be considered for future homes within Affected Areas. Localities could be delineated in which all new homes are built in an approved manner likely to ensure that radon concentrations are as low as reasonably achievable, and at least below 200 Bq m^{-3}.

Government policy

Areas where it is estimated that <1% of homes are above 200 Bq m^{-3}: no requirements.

Areas where it is estimated that 1–2.9% of homes are above 200 Bq m^{-3}: no requirements in England and Wales, basic preventive measures required in Scotland and Northern Ireland.

Areas where it is estimated that 3–9.9% of homes are above 200 Bq m^{-3}: basic preventive measures required throughout the UK.

Areas where it is estimated that ≥10% of homes are above 200 Bq m^{-3}: full preventive measures required throughout the UK.

Note: The calculations in this report are based on the UK radon policy in effect as at October 2007. Since then, there have been changes in policy which would affect the results of some calculations. The HPA and the British Geological Survey have issued a new radon map for England and Wales (Miles et al, 2007), defining radon Affected Areas in terms of the estimated mean long-term radon concentrations in homes, rather than the measured concentrations in homes. This map is used for defining areas where basic or full radon protective measures are required in new buildings in England and Wales. Also, the HPA has issued advice to government (HPA, 2008) that all new buildings in the UK should include basic radon protective measures, rather than only new buildings in the areas defined above.

A special exercise was necessary to approach those concerned and to determine whether action had been taken. In later surveys, a second free measurement was offered to those with measurements above the Action Level if they undertook remedial work. This provided information on levels of remediation and its effectiveness, and also provided some incentive for remediation.

As time went on, surveys became more successful in persuading householders to take up the offer of a radon measurement. However, it was determined that still only 10–20% of householders who were informed that the measurement in their home was above the Action Level actually carried out remedial action (Bradley, 1996; Bradley and Thomas, 1996). Therefore, in 1999 the government changed the emphasis of its approach in order to encourage remediation. In particular, it was felt that the programme would be more successful if carried out in partnership with local authorities, as householders might identify more readily with a locally based campaign. Pilot studies of this approach were carried out in three local authority districts with the local authorities providing the public face of the initiative, but with resources to support the programme provided by central government. These pilot studies succeeded both in increasing interest in radon and in increasing the numbers of homes in which remedial works have been carried out.

A new programme was launched in July 2000, in partnership with a further 30 or so local authorities, to 'roll-out' the approach used in the pilot studies (POST, 2001). The radon campaign in each area was tailored to local conditions and requirements and the approaches taken were refined as experience was gathered. In 2005, the administration of the radon programme in England was transferred to the Health Protection Agency, which is continuing the practice of involving local authorities and has initiated the involvement of local health professionals and local community organisations.

The proportion of householders who accepted the offer of a measurement under the government programme (from 2000 to 2005) varied between 15 and 50%, depending on the amount of publicity and the percentage of homes already measured in the target area. It might be expected that the proportion of householders accepting the offer of a measurement would drop when areas are revisited, as the most interested householders have already taken up the offer. In fact, however, both the proportion taking up the offer of a measurement and the proportion of high radon concentrations remediated tended to increase with time as expertise in running the programmes increased. Important factors in this include the level of local media interest and whether or not the local authority and other local organisations provided encouragement and publicity. Broadly compatible measurement campaigns were also conducted in the other parts of the UK, in parallel with those in England. A summary of these radon remedial programmes has been published by Kendall et al (2005).

To date the NRPB/HPA measurement campaigns have involved measurements in approaching half a million UK homes. Results for England, Scotland, Wales and Northern Ireland are summarised in Chapter 2. The surveys have identified some 50,000 homes with measured values above 200 Bq m^{-3} (about half of the total estimated by the HPA to exist in the whole country), while measuring only about 2% of the housing stock.

In addition to programmes for identifying and remediating existing homes with high radon concentrations, some attention has also been given to reducing radon concentrations in those new homes at potentially greatest risk. Radon preventive measures have been developed to isolate the

building from the ground by means of a sealed membrane extending over the whole footprint of the house (BRE, 2005). If properly sealed, such a membrane can typically halve the radon concentration in the new building (Scivyer, 2001). If the membrane fails, or if a further reduction in radon concentration is required, provision can be made for sub-floor ventilation or depressurisation by means of a ventilated sub-floor void or a radon sump. A sump does not have any effect unless fitted with an electric fan, and the approach to date has been that a fan would generally not be fitted until testing had demonstrated a high radon concentration. Radon preventive measures are discussed further in Appendix L.

Interim guidance on radon preventive measures in new buildings in England and Wales was first issued by the (then) Department of the Environment in June 1988 (DOE, 1988). Changes were made to the building regulations in 1991 to require the adoption of anti-radon measures in new buildings in some areas of England and Wales (Parliament, 1991). The nature of the protective measures and the areas where they are required in new buildings are specified in Report BR211 of the Building Research Establishment (BRE, 1992). The provisions of BR211 have been updated as more information has become available (BRE, 2007). In England and Wales, in areas where more than 10% of measurements exceed the Action Level, full radon preventive measures are currently required. These comprise both a radon-proof barrier and a radon sump or ventilated sub-floor void. In areas where 3–10% of measurements exceed the Action Level, only 'basic preventive measures' (ie a radon-proof barrier) are required.

In Scotland, guidance on protective measures for new buildings in mapped areas was published in 1999 (BRE, 1999). Basic protection is required in areas where more than 1% of homes are expected to exceed the Action Level. This is a lower threshold than in England where basic protection is not required until the 3% level. Full protection is required above 10%.

Radon protective measures for Northern Ireland were explicitly incorporated in the Building Regulations (Northern Ireland) 2000 that came into force on 1 April 2001 (Parliament, 2000). The statutory instrument requires, as in Scotland, basic protection in areas where more than 1% of homes are expected to exceed the Action Level and improved protection above 10%. A report from the Building Research Establishment provides further details (BRE, 2001).

Further information on the development of the UK radon programme has been given by Kendall et al (2005).

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Appendix E
Evidence from Radiobiology on the Induction of Lung Cancer by Radon

Radon and its short-lived decay products emit alpha particles and also some beta particles and gamma rays. The latter carry about 4 and 8%, respectively, of the total decay energy (IARC, 2001) and deposit their dose, as low LET electrons, mostly well dispersed from the sites of decay. Because of the known high relative biological effectiveness of alpha particles for most cellular and tissue effects, the alpha particles (carrying about 88% of the decay energy) are expected to dominate the carcinogenic action. At low and moderate doses of radiation from radon and its short-lived decay products, the most relevant type of radiation is thus high LET alpha particle emissions that are isolated in space and time and with each particle able to traverse only a few cells adjacent to the point of the radionuclide decay.

There is strong evidence that a single alpha particle can cause complex clustered damage to the DNA of a cell and induce major genomic changes, including mutation and neoplastic transformation. Even allowing for cellular repair, the passage of a single alpha particle through a cell nucleus is quite likely to kill the cell or otherwise to cause permanent genomic changes to the surviving cell and its progeny. In addition, there is convincing evidence that most cancers are of monoclonal origin and derive from a damaged cell through a multistage process of genetic changes.

These considerations provide a mechanistic basis for a linear relationship, with no threshold and no dose-rate dependence, between alpha particle dose to the tissue and cancer risk at exposure levels at which the probability of a cell nucleus being traversed by more than one alpha particle is small (ie at exposure levels at which most of the nuclei are never traversed by even one particle). As exposure increases from zero, the insult to those cell nuclei that are traversed by an alpha particle remains the same, but the number of traversed nuclei increases in proportion to the exposure (ie the number of particles). On this basis, the carcinogenic risk is determined essentially by the product of the number of traversed cell nuclei and the effectiveness of a single alpha particle traversal to induce a critical cellular alteration, be it an immediate mutation, an induced genomic instability that leads to subsequent mutation(s) or a change in cell proliferation that contributes to the multistage carcinogenic process.

The above condition of single, isolated particles pertains to almost all levels of radon exposure in homes, and also to the lower and intermediate levels of exposure to radon-exposed miner cohorts in which excess lung cancer mortality has been observed. Using dosimetric models of exposure, it can be estimated that even at the UK Action Level for radon in homes (200 Bq m^{-3}), only a small minority of cell nuclei in the exposed lung tissue (around 1–3%, depending on cell type) will be traversed by an alpha particle in a year and many such locations will have no traversals throughout the person's lifetime (BEIR VI Committee, 1999). In the lowest-exposure miner cohort considered in the BEIR VI study (Radium Hill, see Appendix I, Table I1), on average only about 6–18% of the cell nuclei were traversed during the miners' entire working lives, while in the highest-exposure cohort (Colorado) each cell nucleus location

received on average 7–19 traversals during the working lives and this corresponds to 1–4 traversals per nucleus per year.

The BEIR VI analysis of lung cancer mortality in the miner cohorts concluded that the data were consistent with these mechanistic expectations of linear non-threshold dependence on dose at low and moderate exposures (BEIR VI Committee, 1999). It was only at high exposures and exposure rates (eg Colorado) that non-linearity was apparent and this had the effect of reducing the risk per unit exposure compared to that at lower exposures and/or exposure rates; this has been called an inverse dose-rate (or exposure-rate) effect. A variety of possible mechanisms were listed that could contribute to non-linearity at high exposures, due to the effects of multiple alpha particle traversals of cells or regions of tissue (BEIR VI Committee, 1999). Most of these mechanisms were, however, not expected to be of material importance at low exposures, during which only a minority of cell nuclei would be traversed and these essentially only by single alpha particles.

It was recognised by the BEIR VI Committee that alpha particles that miss the cell nucleus but pass through the cell cytoplasm might also be able to contribute to biological effect, that non-hit 'bystander' cells might respond to traversals through nearby hit cells, and also that local tissue effects, including cell killing and increased cell proliferation, might influence the expression of genomic damage.

Each of these phenomena could increase the effective target volume for multiple-track effects and extend expectations of non-linearity to lower doses and dose rates. We agree with the general assessment of the BEIR VI Committee and note that subsequent publications have added more substance to each of them (see, for example, Wu et al, 1999, Mothersill and Seymour, 2001, Morgan, 2003a,b, Little et al, 2002, Prise et al, 2003, Barcellos-Hoff and Brooks, 2001, and Belyakov et al, 2005).

However, there is at present little evidence to suggest that these effects are sufficiently strong to violate significantly the expectation of linearity for exposures in homes and the lower-exposure mines. To bring about significant alterations, these multi-track mechanisms would need to be very long ranged and/or long lived to link the actions of the separate alpha particles. Detailed modelling of the human lung, however, has highlighted the non-uniformity of deposition of radon decay products in the airways. As a consequence, in some locations multi-track exposure of cells should be much more likely than would be inferred from uniform exposure of the airways (Fakir et al, 2005).

Brenner and Sachs (2002, 2003) have suggested that the observed inverse dose-rate effect for lung cancers in the miner cohorts is due to bystander mechanisms being influential at low doses but tending to saturate at high doses. Little (2004), however, showed that equally good fits to the miner data could be obtained without including bystander effects if latency period, age at exposure and attained age are included in the model. The apparent inverse dose-rate effect then appears to be a result of the age dependencies.

The two-stage clonal expansion (TSCE) model applied to miner cohorts by Heidenreich et al (2004) differs fundamentally in that it allows for action of radiation not only at the initiation but also at the promotion stage of carcinogenesis. Their fits to several of the radon-exposed miner cohorts showed highly significant action of radon on promotion in all cases. Similar trends were found for lung cancer in radon-exposed rats (Kaiser et al, 2004). It has been suggested that the promotional action may be due to the

high probability of cell killing by a single alpha particle and the consequent opportunity for enhanced growth of nearby pre-existing spontaneous or radiation-induced initiated cells (Heidenreich and Paretzke, 2004).

E1 Conclusions

Alpha particle emissions dominate the carcinogenic action of radon and its decay products. The passage of a single alpha particle through the nucleus causes complex clustered damage to the DNA which is quite likely to kill the cell. Most cancers are of monoclonal origin and derive from a single damaged cell through a multistage process of genetic changes. These considerations suggest that, at the exposure levels relevant to most residential exposures (and all but the highest exposures of miners), the dose–response relationship for induction of cancer is likely to follow a linear non-threshold form with no dose-rate dependence. It is possible that alpha particles which do not pass through the cell nucleus may affect rates of cancer induction, but the evidence so far available suggests that any such effects would be small. These conclusions are consistent with those of the BEIR VI Committee (1999).

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Appendix F
Modelling Radon Doses to the Lung

Most of the radiation dose to the body from exposure to radon results from inhalation of its short-lived decay products. A discussion of the fate of inhaled materials is to be found in the human respiratory tract model of the International Commission on Radiological Protection (ICRP, 1994). This describes the deposition of radioactive material in the respiratory tract, its subsequent clearance and the doses to different parts of the respiratory system.

The ICRP recommends that the assessment and control of radon exposures should be based on direct epidemiological observations rather than on dosimetric modelling and that the ICRP human respiratory tract model should not be used for the assessment and control of radon exposures. However, lung modelling of radon doses can be used in a different way, to compare radon decay product doses received under different conditions.

The uncertainties in using lung modelling for this purpose are likely to be much smaller than those obtaining when using it to estimate absolute doses. This use of lung modelling allows, for example, the epidemiology of radon exposure in mines and in dwellings to be compared, despite the differences in exposure conditions, by calculating the absorbed dose to the lungs per unit radon exposure in both situations. It also provides a link to the calculation of doses from radon and its decay products to organs and tissues outside the lung, and where there is only limited direct epidemiological evidence on the magnitude of any risk (see Appendix K for further details).

F1 Estimation of absorbed dose to the lung from inhalation of radon

Several stages are involved in the calculation of absorbed radiation dose to the lung as a result of radon inhalation:

a determination of the physical nature of radon and its decay products in the atmosphere,

b characterisation of the morphology of the human airway and of the breathing habits of the population under investigation,

c modelling of the pattern of aerosol deposition within the lung and any subsequent clearance,

d determination of the energy deposited in the cells lining the lung airway.

Each of the stages in the assessment of dose to the respiratory tract is now discussed in more detail.

F1.1 Physical nature of radon and its decay products in the atmosphere

Radon-222 exists as a gas in the atmosphere. Its immediate decay products are themselves also radioactive and emit a mixture of alpha, beta and gamma rays (see Figure 1.1 of the main text). The decay products are atoms of solid elements, which become attached to aerosol particles in the atmosphere. This process passes through several stages. Initially the radioactive atoms rapidly coalesce with atmospheric gases and vapours to form clusters of molecules, which are particles in the size range 0.5–2 nm. These then attach themselves to existing aerosol particles in the atmosphere, which have a wide range of particle sizes between about 10 nm and 10 μm.

Measurements have suggested that decay product activity is distributed in several size-bands depending on the conditions and the measurement technique. These might typically be (Marsh and Birchall, 2000; Porstendorfer, 2001):

a the molecular clusters mentioned above, size range 0.5–2 nm,

b the nucleation mode covering sizes between about 2 and 100 nm,

c the accumulation mode from 0.1–1.0 μm,

d the coarse mode from 1–10 μm.

The usual convention is to refer to the amount of activity in molecular clusters as the 'unattached fraction' and the sum of the other modes as the 'attached fraction'. However, there is some controversy over this, as activity on particles in the nucleation size-band are found in atmospheres with no ambient aerosol, and therefore may not be strictly attached. The BEIR VI Report noted the problem of interpreting the term 'unattached fraction' and suggested there was a need for a more precise definition or for its use to be dropped (BEIR VI Committee, 1999). In this report the term is used in its conventional sense.

The distribution of activity between particles of different size depends on the environment. Important influencing factors are the concentration and size distribution of the ambient aerosol, the humidity and the process of attachment or plate-out of particles on to surfaces such as walls. In most cases nearly all the attached fraction is in the accumulation mode. Activity in the coarse mode is only found in outdoor aerosols, as plate-out on to surfaces removes particles of this size indoors. The unattached fraction is significantly affected by the ambient aerosol concentration (Porstendorfer, 2001). In homes, values of around 0.1 are typical, with lower values obtained if there is a smoker in the house, due to the high particle concentration from the smoke. In mines with heavy machinery, particle concentration is even higher and values of unattached fraction are around 0.01. Typical particle size distributions in the different environments are shown in Figure F1. Size distribution has a very important influence on the fate of the inhaled radioactive aerosol, as particles of different size have significantly different distribution patterns in the airway tree.

Most of the dose due to radon-222 inhalation is delivered by the radon decay products, with nearly all the absorbed energy coming from the alpha particle emissions of polonium-218 and polonium-214. Therefore in dose calculations it is more important to know the concentration of radon decay products

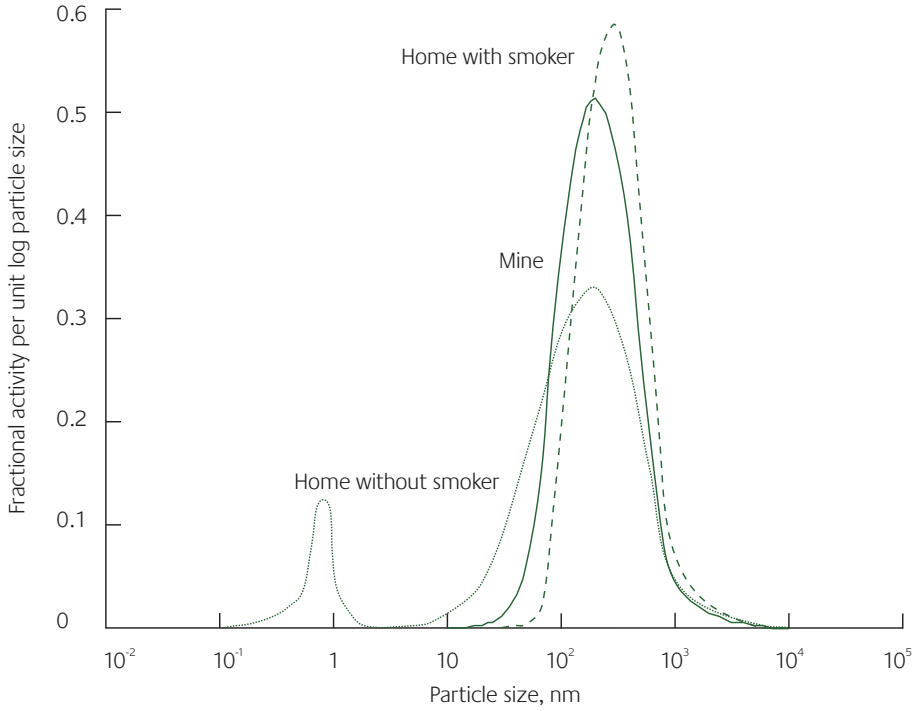

FIGURE F1 Typical relative activity size distribution of the potential alpha energy concentration of radon decay products in different environments (Porstendorfer and Reineking, 1999)

in the atmosphere than the concentration of radon itself. If radon and its decay products were in radioactive equilibrium, the activity of each of the decay products is equal to that of radon. However, in practice their concentrations are lower, due to air movement and attachment of particles to surfaces. The fraction of energy from decay products actually available for contributing to radiation dose is described by the equilibrium factor, *F* (see Appendix A). This varies considerably between different environments. Radon concentrations outdoors are generally low due to dilution caused by air movements, although measurements of the equilibrium factor suggest that it is relatively high at about 0.8 (Wrixon et al, 1988). Extensive measurements of *F* have been made in homes. Median values in different parts of the world are fairly consistent at around 0.4, although there is considerable variation between individual dwellings (BEIR VI Committee, 1999). Ventilation generally reduces the concentration of radon and its decay products by dilution, and also affects the equilibrium factor. Measurements in mines using only natural ventilation typically showed values of around 0.7–0.8 (Lubin et al, 1994), whereas in modern mines with forced ventilation, values are around 0.2 (Cavallo, 2000).

F1.2 Lung morphology and breathing parameters

The fate of the inhaled radon aerosol in the body depends on the morphology of the airway tree and the breathing pattern of the population being studied. The human airway can be considered to consist of two parts – the lungs and the airways leading to them, principally the nose and the mouth, which are referred to as the extrathoracic airways. The airway within the thorax consists of a branching structure in which each airway branch splits into two smaller airways. Starting from the trachea, which is the main windpipe feeding both lungs, there are around 23 generations of successive airway branches (Weibel, 1963). The branches become progressively smaller and more numerous as generation number increases. Figure F2 shows a cast of the human airway tree illustrating the branching structure. For simplicity the thoracic airway is often thought of as consisting of three major regions – the bronchial region, consisting of generations 0–8, the bronchiolar region, generations 9–15, and the alveolar–interstitial region, generations 16 onwards (ICRP, 1994). Measurements made on casts have allowed the development of systematic descriptions of lung morphology (Horsfield and Cumming, 1968; Weibel, 1991), which have helped in the assessment of particle deposition (ICRP, 1994).

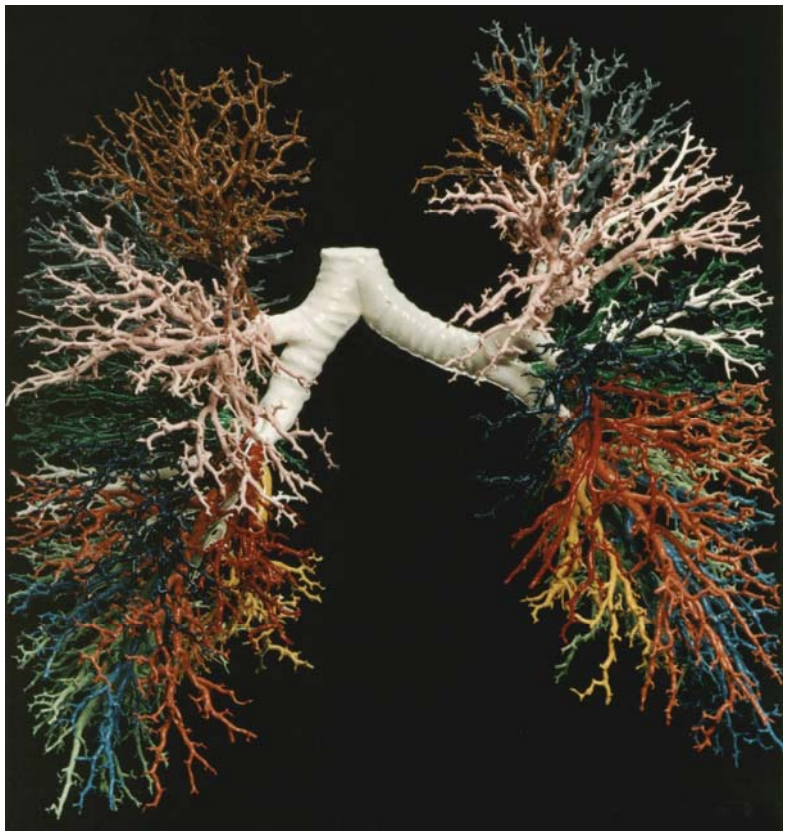

FIGURE F2 Cast of the human lung illustrating the branching structure of the airway tree

Particle deposition also depends on the breathing pattern and therefore information on this is required when modelling deposition. Values of breathing parameters are assigned for each activity in which the subjects in the study population normally participate, and an estimate made of the average fraction of time spent in each activity. This allows the average breathing rate to be calculated. In the BEIR VI calculations, for example, the average breathing rate used in calculating domestic dose for a male adult was 0.78 $m^3 h^{-1}$, whereas for miners it was 1.25 $m^3 h^{-1}$ (BEIR VI Committee, 1999).

F1.3 Modelling the pattern of aerosol deposition and clearance within the lung

Radon itself is a noble gas and therefore chemically inert. Following inhalation, most is exhaled before it has the opportunity to decay. It therefore generally gives a small component of the total dose to the body. By contrast, when the aerosol particles containing the radon decay products are inhaled, a significant fraction are deposited on the surface of the airway. Although a good proportion of the particles are ultimately cleared from the airway, their short half-lives mean that they have ample opportunity to release most of their energy before any appreciable clearance has occurred.

The ICRP human respiratory tract model (ICRP, 1994) can be used to predict the deposition of particles of a given size within the different regions of the airway tree: extrathoracic, bronchial, bronchiolar and alveolar–interstitial. The dependence of the deposition distribution on breathing pattern has also been modelled. For particles smaller than a micrometre such as those carrying radon decay products, the main mechanism of deposition is diffusion. The heavier particles of the attached fraction have a relatively low deposition probability of about 0.2, with over half of this penetrating deep into the alveolar region. The unattached particles of around 1 nm in diameter move more freely by diffusion and have a higher probability of being deposited. The total deposition fraction is around 0.8, with most deposition occurring in the extrathoracic airways (Hofmann, 1999). The variation of deposition efficiency with particle size for a male adult with the average breathing rate is shown in Figure F3. Both the total deposition and that in the bronchial region, where most lung cancers originate, are illustrated. Deposition in the bronchial region is significantly higher for unattached particles in the 0.5–2 nm range compared to that for typical attached particles in the 100–500 nm range. The model enables a detailed description of particle deposition using knowledge of the particle size distribution and breathing pattern of the population being studied.

The initial deposition patterns are modified by mechanisms which clear particles from the airways. Particles deposited in the bronchial and bronchiolar regions are subject to mucociliary clearance. The cells lining the airway have a surface of minute hair-like structures or cilia. These are continually moving and cause particles to be forced back up the airway tree towards the trachea. However, there is evidence that this process is relatively slow, possibly taking place over a time period of several hours, which is considerably longer than the half-lives of the radon decay products. This suggests that most of the dose due to these radionuclides will be deposited in the cells of the airway wall before they have the opportunity to be cleared. Although clearance does influence the dose distribution to some extent, it is not a major modifying factor. Particles may also be cleared from the bronchial and bronchiolar regions by absorption into the blood and from the alveolar–interstitial region by both clearance and absorption. These processes are generally considered to be very slow compared to the half-lives of the radon decay

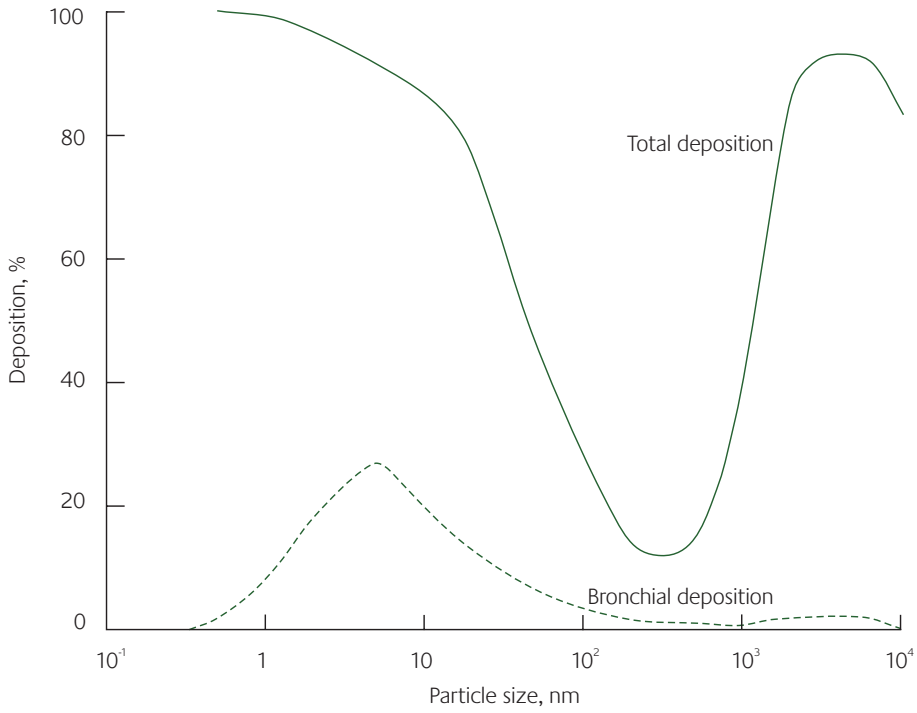

FIGURE F3 Variation of percentage deposition of inhaled particles with particle size for the whole respiratory tract and for the bronchial region alone. The data are estimated for a male adult at home. (After BEIR VI, using the ICRP 1994 model)

products (Marsh and Birchall, 1999) and therefore to have a minimal effect on dosimetry calculations. If more rapid absorption were to occur, then the dose to the lung would be reduced, and the dose to other organs increased.

F1.4 Calculation of absorbed dose to the cells lining the airway

The absorbed dose is defined as the energy deposited per unit mass in the bronchial epithelium, the cells lining the airway. The key cells in which lung cancer develops are thought to be the basal and secretory cells of the first five to eight generations of the airway (NRC, 1991). Before the alpha particles reach these cells, some of their energy is absorbed in the mucus and sol layers, which line the airway wall. By making certain assumptions regarding the thickness of these layers and of the cells themselves, the fraction of alpha particle energy deposited in the basal and secretory cells, and hence the absorbed dose, can be calculated. However, there is some variation in the values of these parameters assumed by different workers, and this has a significant effect on the dose obtained (Birchall and James, 1994; Porstendorfer and Reineking, 1999). In calculating the overall equivalent lung dose the relative weighting of the

bronchial, bronchiolar and alveolar–interstitial regions also needs to be defined. Either equal weighting is given to each of the regions (ICRP, 1994) or a heavier weighting is given to the bronchial region, to reflect the higher incidence of lung cancer originating in this location (Porstendorfer, 2001).

F1.5 Calculation of overall effective lung dose from inhaled radon decay products

The absorbed dose to the various parts of the lung is converted to an equivalent dose by multiplying by 20, the radiation weighting factor specified for alpha radiation. The ICRP has recommended that the equivalent dose to the lung as a whole should be taken as the equally weighted sum of equivalent doses to the bronchial, bronchiolar and alveolar–interstitial regions with a small contribution from dose to the lymphatic system. The dose to the lung as a whole is quite sensitive to the relative weights given to the various components.

The contribution of the lung dose to effective dose is then calculated by multiplying the equivalent dose to the lung as a whole by the appropriate tissue weighting factor, which is 0.12.

F2 Estimation of effective dose from inhaled radon decay products

Estimates of the effective dose due to radon exposure are needed if the total effective dose (and thus risk) from different sources are to be compared. Estimates of effective dose from radon decay products can be obtained from two sources:

a by converting measures of risk derived from epidemiological studies to effective dose by using standard estimates of risk per unit dose derived from other sources (for example, studies of the survivors of the atomic bombings in Japan),

b by calculating effective dose from absorbed doses as described above.

These two methods are discussed below.

F2.1 Estimation of dose conversion coefficients from epidemiology

In its 1990 recommendations, the ICRP estimated that the probability of detriment to health per unit effective dose was 5.6% per Sv for workers and 7.3% per Sv for the general public (ICRP, 1991). The ICRP compared these general radiation risk estimates with estimates of radon risks derived from studies of miners. As outlined in Section 2.2 of ICRP Publication 65, this comparison of risks was used to derive 'dose conversion coefficients' (DCCs), usually given as mSv per Working Level Month (mSv WLM^{-1}). These were estimated at 4 mSv WLM^{-1} at home and 5 mSv WLM^{-1} at work (ICRP, 1993). When detailed consideration is given to how estimates of radon risk should be transferred from studies of miners to the domestic environment, a number of problems arise, which are discussed in Section F2.2. However, the overall effect of allowing for these factors is small.

It should also be noted that when deriving effective dose from risks assessed by epidemiology, only the risk of lung cancer is considered. The total effective dose is a little higher than this, as other organs in the body also receive a dose. However, in normal circumstances, even the sum of these contributions is small compared to the lung dose.

The US Environmental Protection Agency has revised the estimates of radon risk from studies of miners using BEIR VI models and recent updates on smoking and lung cancer (EPA, 2003). This resulted in a DCC of 9 mSv WLM^{-1}. The European pooling of case–control studies of residential exposure to radon (Darby et al, 2005) gives an estimate of 4 mSv WLM^{-1}.

F2.2 Comparative dosimetry in homes and mines

Before completion of the large pooling studies described in Chapter 3, most authorities estimated risk factors for residential exposure from occupational studies of miners. The dose to sensitive tissue per unit exposure may differ in homes from that in mines and this and other factors complicate the comparison of risk factors from miner studies with those from the domestic environment. These are outlined below.

The approach adopted by the National Research Council (NRC, 1991) and the BEIR VI Committee (1999) uses the principle of comparative dosimetry. The absorbed dose per unit exposure in the domestic environment is calculated and compared to that for the conditions pertaining in mines. The comparison uses the *K*-factor, which is defined as:

$$K = (\text{Dose}_{\text{home}}/\text{Exposure}_{\text{home}})/(\text{Dose}_{\text{mine}}/\text{Exposure}_{\text{mine}})$$

The comparative dosimetry approach has the advantage over absolute dose calculation of depending on fewer assumptions, as the values of some parameters will be the same for both homes and mines.

The NRC (1991) calculated the *K*-factor between homes and mines, obtaining values of 0.7 for adults and 0.8 for children. Values of *K*-factor can also be inferred from the work of other groups which have compared dose calculations in both environments. Birchall and James (1994) performed dose calculations for conditions in mines and Marsh and Birchall (2000) for homes. Comparing these results, a value for *K* of 1.1 can be derived. A similar comparison of the work of Porstendorfer and Reineking (1999) and Portstendorfer (2001) suggests a ratio of 1.3.

The similar values for doses in mines and homes (0.7–1.3) are the result of two balancing factors. The high ambient particle concentration in mines results in a very low unattached fraction. Since this component of the aerosol is more efficiently deposited in the bronchial airways than the attached fraction (Figure F3), the doses per unit exposure in homes tend to be higher. This factor is balanced by the higher breathing rate of the miners, which tends to increase their dose.

It should be noted that Cavallo (2000) criticised the discussion of the *K*-factor in the BEIR VI Report (BEIR VI Committee, 1999). However, replies from Krewski et al (2002) and from James et al (2004) clarified the situation. These authors agreed that the statement in the original BEIR VI Report, that the *K*-factor expressed in terms of radon gas concentration, K_r, was unity, was incorrect. In fact their calculations led to the conclusion that the *K*-factor expressed in terms of radon decay product

concentration, K_d, was unity. This now agrees within the limits of the precision of the calculations with the other estimates of K_d. Given the concurrence of the restated BEIR VI results with other estimates of K_d, it seems reasonable to accept the conclusion that DCCs due to radon decay products in homes and mines are similar.

F2.3 Estimation of dose conversion coefficients from dosimetry

There have been a number of calculations of doses from inhalation of radon decay products, using the ICRP model of the human respiratory tract or variants on it. Birchall and James (1994) produced an estimate of DCC for miners of 13 mSv WLM^{-1}. This was based on typical aerosols found in mines and the miners' breathing parameters. Later work by the same group gave the equivalent value for domestic exposure as 15 mSv WLM^{-1} (Marsh and Birchall, 2000).

Marsh et al (2002) have investigated the effect on calculated doses of varying some of the parameters assumed in the ICRP human respiratory tract model (ICRP, 1994). They concluded that none of the parameters they varied could account for the difference in DCCs between this dosimetric approach and the ICRP approach based on epidemiology. However, the authors kept several of the calculational parameters fixed, and therefore one or more of these could still explain the discrepancy.

Porstendorfer and Reineking (1999) and Porstendorfer (2001) also suggested similar DCCs for mines and homes, although the absolute values they obtained were systematically lower (5.7 and 7.3 mSv WLM^{-1}, respectively). The reduced values obtained in these studies, result, in part, from different estimates of the depth of the sensitive cells in the bronchial epithelium.

Effective dose in the domestic environment has also been estimated by Hopke et al (1995). The authors calculated a value of 3.8 nSv per Bq m^{-3} h of radon gas concentration. Using the median value of equilibrium factor quoted in the paper of 0.408, this converts to 6 mSv WLM^{-1}.

Work by Kendall and Smith (2002), which includes analysis of doses to other organs as well as the lungs, derives values of between 5 and 20 mSv WLM^{-1}. The value depends primarily on the rates assumed for absorption of decay products from the lungs into the bloodstream. However, these calculations use standard parameters in the ICRP model of the human respiratory tract which the ICRP does not recommend for radon decay products.

F2.4 Choice between epidemiology and dosimetry

Dose conversion coefficients from dosimetry have tended to be higher than those derived from epidemiology. Calculations of effective dose from radon decay products are dependent on a large number of factors which are imperfectly known:

a the biological behaviour of the radioactive species involved,

b the location and nature of the sensitive cells,

c the relative contribution to risk from different parts of the lung,

d the risk–dose relationship, which is mainly based on exposure to low linear energy transfer (LET) radiation such as X-rays rather than the alpha particles emitted by radon decay products. It has been suggested that the radiation weighting factor for alpha particles, which is taken as 20 under the ICRP dosimetry framework, is too high.

Both methods for estimating effective dose involve considerable uncertainties. In Publication 65, the ICRP recommended the use of the epidemiological approach to effective dose estimation for radon exposures, as it is more direct (ICRP, 1993). The recent publication of the pooled residential case–control studies has strengthened this conclusion (Darby et al, 2005).

The various estimates of effective dose per unit exposure to radon decay products allow evaluation of the average dose to individuals exposed in UK homes. The average concentration of radon in the UK is 20 Bq m^{-3}, with an equilibrium factor of about 0.4 (NRPB, 2000). Table F1 gives estimates of the dose conversion coefficients and the resulting annual average doses as published by a number of authors. We regard the European pooling study (Darby et al, 2005) as the most reliable current source of information on the risks of domestic exposure to radon. However, as noted in Chapter 3, various factors mean that the true risk factor may be somewhat higher than that reported by Darby et al (2005).

F2.5 Relating the risk of radon to that of other ionising radiation sources

Exposure to background radiation arises from a number of sources and it is sometimes desirable to be able to quantify the relative risks they pose to the general public. This can be achieved by estimating the effective dose resulting from each source, as this is intended to be roughly proportional to risk.

The different estimates of average annual effective dose due to radon presented in Table F1 show that more recent values vary both above and below the 1.0 mSv figure defined by the NRPB in 1988 (Wrixon et al, 1988). However, the most reliable estimate comes from the European pooling study, which gives a range of 0.1 to 0.8 mSv, with a central value of 0.4 mSv, although there are reasons to believe that the true figure may be higher.

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TABLE F1 Estimated annual effective dose from radon in the UK based on the different estimates of the dose conversion coefficient for radon decay products in the home

Study	Dose conversion coefficient (mSv WLM^{-1})	Estimated annual effective dose at the UK mean radon concentration of 20 Bq m^{-3} (mSv)
Values based on dosimetry		
Hopke et al (1995)	6	0.7
Porstendorfer (1999)	7	0.8
Marsh and Birchall (2000)	15	1.7
Kendall and Smith (2002) (i) Type F	5	0.5
Kendall and Smith (2002) Type M	20	2.0
Values based on epidemiology		
Wrixon et al (1988)	10	1.0
ICRP (1993) (ii) Domestic	4	0.4
ICRP (1993) Occupational	5	0.5
EPA (2003) (ii) Domestic	7	0.7
EPA (2003) Occupational	9	0.9
Darby et al (2005)	4 (1–8)	0.4 (0.1–0.8)

Notes

(i) The two values quoted from Kendall and Smith (2002) were obtained by assuming different absorption rates from lung to blood, Type F and Type M clearance.

(ii) The two values quoted for the epidemiological studies are the different estimates of effective dose applying at home and work, respectively.

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Appendix G
Animal Evidence on Radon and Lung Cancer

A number of studies of the health effects of exposure of animals to radon have been undertaken, mostly in the 1960s when the main focus of attention was on radon in mines. For this reason, and also because of practical considerations involving sample sizes, many of the animal experiments were carried out at relatively high radon concentrations. The results also tended to be reported in terms of Working Levels and Working Level Months, the units used historically in radiological protection in mines.

Three main groups have been working on studies in which animals have been exposed to radon and its decay products alone or with other pollutants:

a CEA – Commissariat à l'Energie Atomique, France,

b PNNL – Pacific Northwest National Laboratory, USA,

c Harwell, UK.

Studies were conducted on Sprague-Dawley rats by the CEA (13,000 animals) and Harwell (2,000) and on hamsters (800), Wistar rats (6,000) and dogs (800) by PNNL. These studies represent the bulk of the evidence for the effects of exposure to radon and its decay products in animals. The findings have been reviewed by Monchaux et al (1994), Cross and Monchaux (1999) and Collier et al (2005).

Differences in the sensitivity of different species and strains of animal to lung tumour induction have been observed. For example, the Syrian hamster was found to be very resistant to lung tumour induction by radon and its decay products when compared with Wistar rats (Khan et al, 1995) and Groch et al (1997) reported differences in susceptibility between two strains of mice in the PNNL studies.

At exposure levels below 1000 WLM (800 MBq m^{-3} h radon gas exposure, at an equilibrium factor of approximately 0.8), a significant excess in the incidence of respiratory tract tumours is the principal and most consistent finding of the animal studies.

As noted above, the majority of the experiments have been conducted in rats and these studies provide the highest statistical power of any species studied. This is a consequence of the very low incidence of malignant lung tumours in control animals (CEA 0.6% in males, 0% in females; Harwell 0.4% in males) and the high sensitivity of the rat to lung tumour induction by radon and its decay products. However, the sites and types of tumours developed following exposure are somewhat different to those observed in humans. These differences have been explained by differences in the dosimetry between the species, with radon decay products depositing in different areas of the lung. In rats, the tumours originate at the periphery and bronchiolar–alveolar junction, but in humans they occur in the bronchial region. The choice of the rat to study effects of exposure to radon and its decay products has been defended by cytogenetic and molecular genetic studies of the CEA tumours, which have indicated similarities with human lung tumours, suggesting common underlying mechanisms for their development in both species (Dano et al, 2000).

The incidence of all primary (both benign and malignant) lung tumours is increased with cumulative doses up to 3000 WLM (2400 MBq m^{-3} h) in CEA experiments, to peak incidences of 47% in males and 28% in females. A similar dose–response relationship has been observed in male rats at Harwell for all primary lung tumours with the peak occurring at a similar cumulative exposure but being somewhat lower in magnitude (25% in males). Even with exposures as low as 25 WLM (20 MBq m^{-3} h), significant excess lung tumours have been observed (Monchaux and Morlier, 2000). In the Harwell studies, exposure to radon and its decay products produced the major effects on benign bronchial adenoma and malignant bronchial and bronchoalveolar adenocarcinomas. Significant elevations of these tumour types were seen at doses as low as 100 WLM (80 MBq m^{-3} h). Responses of other tumour types in the lung were not as sensitive and only became significant at higher doses such that for exposures at 1000 WLM (800 MBq m^{-3} h) all benign and malignant lung tumours were showing significantly elevated incidences.

No apparent threshold for lung cancer induction has been observed at PNNL, but studies at the CEA at low cumulative exposures and exposure rates have shown no evidence of lung tumour induction. For example, rats exposed at 25 WLM over 18 months (9.25 kBq m^{-3}) showed no excess of lung tumours (incidence 0.6%), but those exposed to the same cumulative exposure using a higher concentration (0.46 MBq m^{-3}) over a shorter period (4–6 months) showed a significantly higher tumour incidence.

A series of experiments at the CEA conducted at relatively low cumulative exposures of about 100 WLM (80 MBq m^{-3} h), comparable to lifetime exposures in dwellings with high radon concentrations or current underground mining exposures, indicates that the risk of lung cancer in rats decreases with exposure concentration (Monchaux and Morlier, 2002), confirming the results obtained at lower exposures. All these data suggest that the induction of lung cancer results from a complex interplay between cumulative dose and dose rate, with an optimal combination of these two parameters, ie a combination of cumulative dose and exposure concentration that results in a maximum risk of lung tumour induction. They support the hypothesis that, at low doses, the risk of lung cancer is determined by the rate at which the dose is delivered, and not by the total cumulative dose alone. These data suggest that there is a 'watershed' at cumulative exposures of about 50 WLM (40 MBq m^{-3} h). Below this exposure, decreasing the exposure concentration or protracting the time over which the dose is delivered results in a reduction in lung tumour risk. Above this level the converse is true with decreasing exposure concentrations or protracting the exposure time resulting in an increased lung cancer risk (Monchaux, 2005).

These data are consistent with those of underground miners showing an inverse dose-rate effect at high cumulative exposures, but a diminution of this effect at cumulative exposures lower than 50 WLM (40 MBq m^{-3} h) (Lubin et al, 1994). They support both an inverse dose-rate effect at high cumulative exposures and its diminution or disappearance at low cumulative exposures. The data from Harwell and PNNL confirm the CEA results for exposures at levels well above 50 WLM (40 MBq m^{-3} h), but only CEA data are available for exposures below this level. Studies at Harwell at 100 WLM (80 MBq m^{-3} h) cumulative exposure have demonstrated positive dose-rate effects for bronchoalveolar adenocarcinomas, indicating that the watershed may be slightly higher for this one tumour type. In the Harwell studies all other tumour types showed no effect of dose rate, although the exposure did result in an elevated incidence of benign adenoma. It has been postulated that the reasons for the existence of an apparent watershed can be related to the number of hits per cell. When cumulative exposures are

sufficiently low, and concentrations low or protraction high, few cells receive multiple hits. At these low doses, the inverse dose-rate effect seen at high cumulative exposures is diminished or does not occur (Morlier et al, 1992; Monchaux and Morlier, 2000).

Recently the data from the CEA, Harwell and PNNL studies have been combined and analysed by staff at GSF Neuherberg. Their analysis confirms that for high cumulative exposures, the lifetime excess absolute risk (LEAR) increases with increasing exposure durations and for low cumulative exposures the opposite trend occurs (Kaiser et al, 2004).

Both PNNL and the CEA have observed that, in rats, latency periods are decreased for older animals or that lung tumours generally occur between 800 and 900 days of age regardless of age at exposure. Combined analysis of the CEA, Harwell and PNNL data have shown that animals exposed later in life have substantially lower LEAR than animals exposed in early life (before 150 days) (Heidenreich et al, 2004).

Radon decay products are isotopes of solid elements and will quickly attract to themselves molecules of water and other atmospheric gases. These, in turn, attach to natural aerosol particles. The fraction of decay products which remains unattached will depend on the circumstances, but in dwellings might typically be about 10%. Unattached fractions are typically five- to ten-fold higher in homes than in mines. Increases of this order in the unattached fraction resulted in a doubling in the lung cancer risk per WLM in the PNNL data. However, at doses of 100 WLM (80 MBq m^{-3} h) there was no apparent difference between high and low unattached fractions in the Harwell studies in terms of lung tumour induction.

Rats were exposed to tobacco smoke and radon (1000 WLM or 800 MBq m^{-3} h) at the CEA. When cigarette smoke exposure occurred prior to radon exposure, animals showed a slight decrease in lung carcinoma incidence compared with rats exposed to radon only. But when the cigarette smoke exposure was after the radon exposure there was a highly significant four-fold increase in lung carcinomas. This resulted from an increase in squamous type tumours. Cigarette smoke promotion was also seen in levels of adenomatosis in the PNNL studies. It appears that pre-neoplastic lesions, induced by radon, are promoted by cigarette smoke.

Exposure to other pollutants (such as chrysotile asbestos, diesel or ozone) could only show co-carcinogenic effects at levels of the pollutant considerably higher than would be found in the domestic situation (Park et al, 1970; Monchaux et al, 1994).

At very high exposure levels (above 1000 WLM or 800 MBq m^{-3} h), which do not mimic human domestic exposure, excesses in non-malignant lung diseases (lung fibrosis and emphysema) have been observed in studies of rats at the CEA and PNNL. These result in significant life shortening and, as lung tumours occur relatively late in the life of the rat, excesses in lung malignancies decline at high exposure levels.

The efficiency with which radon decay products induce their effects in comparison with other forms of irradiation has been considered in a number of studies using animals. Generally comparisons have been drawn against cobalt-60 gamma irradiation or fission neutrons. When gross lung tumour incidence in rats is considered, the total dose affects the equivalence such that at low doses the ratio is 0.66 mGy WLM^{-1} (0.84 mGy MBq^{-1} m^3 h^{-1}). At higher total doses this ratio increases (Chemelevsky et al, 1984; Lafuma et al, 1989). Other studies have considered different endpoints. Micronucleus induction in alveolar macrophages (Johnson and Newton, 1994) showed a ratio of 9.8 mGy WLM^{-1} (12.5 mGy MBq^{-1} m^3 h^{-1})

for *in vitro* exposure of macrophages to plutonium-238 compared with exposure to radon and its decay products *in vivo*. Experiments with rat lung fibroblasts and Chinese hamster ovary cells irradiated *in vitro* compared with the same cells from animals exposed *in vivo* to radon and its decay products showed that radon exposure was 10.9 to 12.5 times more effective than cobalt-60 gamma irradiation at inducing micronucleus production (Brooks et al, 1994).

G1 Conclusions

Experiments to investigate the effects of exposure to radon were conducted in mice, rats, hamsters and dogs. The main focus of these experiments was to cast light on risks in mines and many of the experiments were designed with this aim in mind. The most consistent finding was of excess cancers of the respiratory tract. Rates of induced tumours increased with exposure until high doses. At high doses the rates of tumour induction depended on dose rate as well as on dose. There is some evidence that rates of lung tumours were higher in animals exposed at younger ages. At very high exposure levels there was evidence of increases in non-malignant lung disease.

However, there are some difficulties in applying these data to estimate quantitative risks of lung cancer from residential exposure:

a most experiments were conducted at radon concentrations higher than those to which humans are normally exposed,

b both background and induced levels of disease and the nature of induced tumours differ between humans and animals and also between animal species.

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Appendix H
International Reviews of Radon Risks

The association between exposure to radon and its decay products and lung cancer has been known for many years. There have been a number of authoritative reviews including those by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1982, 2000). In 1999, the BEIR VI Committee produced a report on the health effects of radon based on the evidence then available (BEIR VI Committee, 1999). The effects of radon have also been reviewed twice by the International Agency for Research on Cancer (IARC, 1988, 2001), and sufficient evidence was found to conclude that radon is carcinogenic to humans. Other reviews have also been published (see, for example, Samet and Eradze, 2000).

Here, we draw on these reports and also on more recent publications and summarise the quantitative evidence relating to lung cancer in humans with particular focus on public health issues. To do this we consider mainly the human evidence, and particularly the recent epidemiological studies of residential exposure to radon and lung cancer.

The first clear human evidence that radon can be a cause of lung cancer came from studies of underground miners of uranium and some other igneous rocks. These occupational studies have, in the past, been used to provide quantitative estimates of the risks to miners. There is, however, substantial uncertainty in extrapolating from the studies on miners to obtain a quantitative assessment of the risk of lung cancer from radon in the home. In particular, the exposures in mines were usually much higher than those in homes and they took place over a much shorter time period, as typically each miner was employed underground only for a few years. There are also major differences in the characteristics of the aerosols in the two environments, and these affect the way that the radon decay products are deposited in the airways. Many of the miners were also exposed to arsenic, which is itself an established lung carcinogen.

An additional difficulty with using data from the studies on miners to estimate the risks of residential radon exposure is that most of the miners' studies did not have access to information on smoking, the major risk factor for lung cancer, and therefore were not able to take it into account in the analysis. Where information was collected on smoking in the miners' studies it was usually very limited. Also none of the studies of underground miners included a substantial number of women or any young children.

H1 Lung cancer in radon-exposed miners

Some of the highest concentrations of radon in air 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. 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. However, 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 concentrations 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 small mining communities.

It was only in the 1950s and 1960s, when studies of igneous rock miners in areas other than the Erz mountains where there was exposure to high radon concentrations 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 concentrations 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 such mines in the past.

Studies have been carried out of the mortality patterns of several groups of radon-exposed miners and these form a major body of published evidence concerning the consequences of exposure to radon and its decay products. The review published as the BEIR VI Report (BEIR VI Committee, 1999, see Section H2) is particularly important. It should be noted that further work is in progress to collect and analyse data on three cohorts of miners with low concentrations and long durations of exposure to radon, in France, Czech Republic and Germany. Together, these three cohorts include more than 27,000 miners, with a detailed follow-up of annual individual exposures. Four case–control studies are also being developed within these cohorts, including a total of more than 1600 cases and 3600 controls. Reconstruction of cumulative exposure to radon and past consumption of tobacco is being completed (Tirmarche et al, 2003).

H2 BEIR VI Report

A major review of the data on miners was undertaken by the US National Academy of Sciences Committee on Health Risks of Exposure to Radon. Its report, published in 1999, is known as BEIR VI Report on the health effects of radon (BEIR VI Committee, 1999). The BEIR VI Committee considered 11 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, such as 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.

The BEIR VI Committee noted that the exposures of the miners had to be estimated on the basis of incomplete information and that ad-hoc procedures were used to complete gaps in the measurement data. Information on exposures was most often incomplete in the early years when exposures were highest.

In each of the 11 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 (see Table H1). Although the size of the radon-related increase varied by more than an order of

TABLE H1 Lung cancer mortality in cohort studies of underground miners occupationally exposed to radon (based on BEIR VI Committee, 1999)

Study	Source	Type of mine	Number of exposed miners	Mean total WLM	Mean exposure duration (years)	Number of lung cancer deaths	% increase in age-specific risk of lung cancer per WLM [(i)] (with 95% CI)
Yunnan, China	Xuan et al, 1993	Tin	13,649	286.0	12.9	936	0.16 (0.1–0.2)
West Bohemia, Czech Republic	Tomasek and Placek, 1999	Uranium	4,320	196.8	6.7	701	0.34 (0.2–0.6)
Colorado, USA [(ii)]	Roscoe, 1997	Uranium	3,347	578.6	3.9	334	0.42 (0.3–0.7)
Ontario, Canada [(iii)]	Kusiak et al, 1993	Uranium	21,346	31.0	3.0	285	0.89 (0.5–1.5)
Newfoundland, Canada	Morrison et al, 1988	Fluorspar	1,751	388.4	4.8	112	0.76 (0.4–1.3)
Malmberget, Sweden	Radford and St Clair Renard, 1984	Iron	1,294	80.6	18.2	79	0.95 (0.1–4.1)
New Mexico, USA	Samet et al, 1994	Uranium	3,457	110.9	5.6	68	1.72 (0.6–6.7)
Beaverlodge, Canada	Howe and Stager, 1996	Uranium	6,895	21.2	1.7	56	2.21 (0.9–5.6)
France	Tirmarche et al, 1993	Uranium	1,769	59.4	7.2	45	0.36 (0.0–1.2)
Port Radium, Canada	Howe et al, 1987	Uranium	1,420	243.0	1.2	39	0.19 (0.1–0.6)
Radium Hill, Australia	Woodward et al, 1991	Uranium	1,457	7.6	1.1	31	5.06 (1.0–12.2)
Total [(iv)]			60,606	164.4	5.7	2,674	

Notes

(i) See Appendix A for definitions of Working Level (WL) and Working Level Month (WLM).

(ii) Totals given exclude data above 3200 WLM.

(iii) Values given include all uranium miners, including those with previous gold mining experience.

(iv) Totals adjusted for miners and lung cancers included in both Colorado and New Mexico studies.

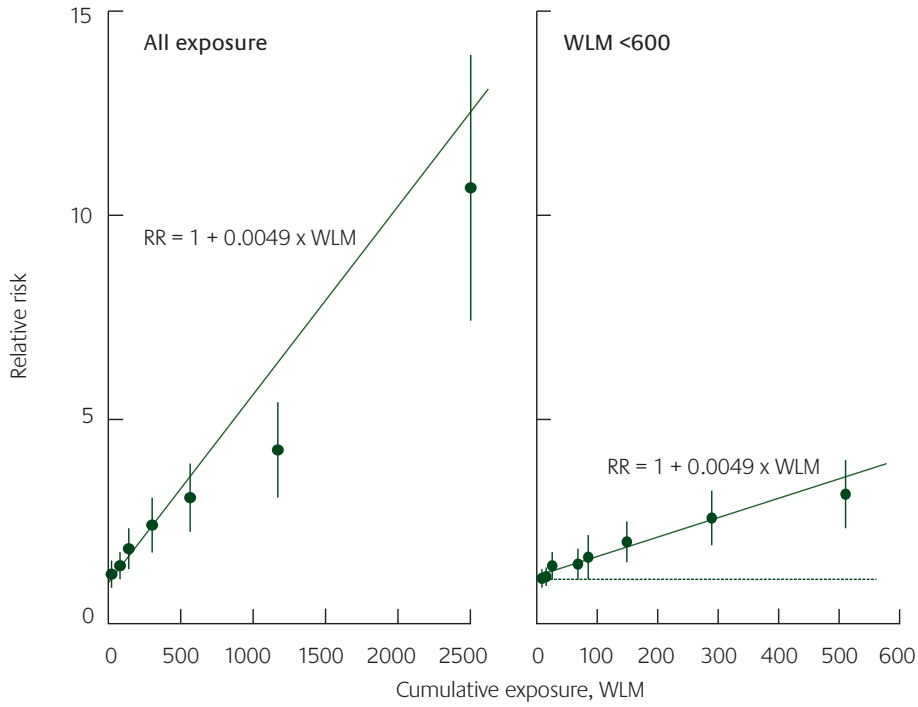

FIGURE H1 Relative risk (RR) of lung cancer with cumulative radon exposure in the cohort studies of underground miners occupationally exposed to radon (based on BEIR VI Committee, 1999)

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 (ie the proportional increase in the age-specific risk of lung cancer) rose linearly with increasing cumulative exposure, both overall and in the region below 600 WLM (Working Level Months, see Appendix A), which is of greatest interest when considering the effects of residential exposures (see Figure H1). The percentage increase in lung cancer 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 risk per unit increase in exposure was also greater in individuals who were aged around 50 years than in individuals who were aged around 60 or 70 years. Finally, mines where the radon concentrations were relatively low had a larger 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, 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 11 studies were collated centrally, and a combined analysis was carried out by the BEIR VI Committee.

After extensive technical investigations the preferred form of the model relating radon exposure to risk of death from lung cancer was:

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

where R is the proportional increase in the death rate 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, $w^* = (w_{5\text{-}14} + \theta_{15\text{-}24} w_{15\text{-}24} + \theta_{25+} w_{25+})$, with $w_{5\text{-}14}$, $w_{15\text{-}24}$ 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 $w_{5\text{-}14}$ is equal to one, while $\theta_{15\text{-}24}$ and θ_{25+} represent the contributions from exposures received 15–24 years and 25+ years previously, compared to exposures received in the period 5–14 years previously. Exposures occurring less than 5 years previously were assumed not to incur any risk. The parameter φ_{age} represents the modifying effect of age, while the parameter γ_z represents the modifying effect either of radon concentration or of exposure duration. Estimates of the parameters β, $\theta_{15\text{-}24}$, 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 H2.

H2.1 Joint effect of smoking and radon

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 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 studies on miners. 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 derive detailed models, as has been done for the data as a whole, but the relationship between the relative risk of lung cancer and cumulative radon exposure could be compared between lifelong non-smokers and others. In both lifelong non-smokers and others the relationship between the relative risk of lung cancer and cumulative radon exposure was approximately linear, but the increase in relative risk per unit increase in exposure was considerably greater among the lifelong non-smokers than among the current and ex-smokers (see Figure H2). To allow for this, the BEIR VI Committee recommended a higher value of β 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 footnote to Table H2 for parameter values). The large increase in relative risk of lung cancer per unit increase in exposure to radon for non-smokers was confirmed by a subsequent study of lifelong non-smoking uranium miners in the Colorado Plateau and adjacent areas of the USA (Gilliland et al, 2000).

It should be noted that, although the miner data suggested that the increase in *relative risk* per unit increase in radon exposure might be higher for never-smokers than for smokers, the suggested increase in the *absolute value* of the risk of lung cancer per unit increase in radon exposure would still be much higher for smokers than for never-smokers, as smokers have much higher rates of lung cancer than never-smokers in the absence of radon exposure.

TABLE H2 Parameter estimates from BEIR VI preferred models in combined analysis of eleven studies of underground miners occupationally exposed to radon (based on BEIR VI Committee, 1999). See text for description of models

Exposure-age-duration model [i]		Exposure-age-concentration model	
β x 100 [ii,iii]	0.55 [iv]	β x 100 [ii,iii]	7.68 [iv]
Time-since-exposure windows		***Time-since-exposure windows***	
$\theta_{5\text{-}14}$	1.00	$\theta_{5\text{-}14}$	1.00
$\theta_{15\text{-}24}$	0.72	$\theta_{15\text{-}24}$	0.78
θ_{25+}	0.44	θ_{25+}	0.51
Attained age (years)		***Attained age (years)***	
$\phi_{<55}$	1.00	$\phi_{<55}$	1.00
$\phi_{55\text{-}64}$	0.52	$\phi_{55\text{-}64}$	0.57
$\phi_{65\text{-}74}$	0.28	$\phi_{65\text{-}74}$	0.29
ϕ_{75+}	0.13	ϕ_{75+}	0.09
Duration of exposure (years)		***Concentration of exposure (WL[iii])***	
$\gamma_{<5}$	1.00	$\gamma_{<0.5}$	1.00
$\gamma_{5\text{-}14}$	2.78	$\gamma_{0.5\text{-}1.0}$	0.49
$\gamma_{15\text{-}24}$	4.42	$\gamma_{1.0\text{-}3.0}$	0.37
$\gamma_{25\text{-}34}$	6.62	$\gamma_{3.0\text{-}5.0}$	0.32
γ_{35+}	10.2	$\gamma_{5.0\text{-}15.0}$	0.17
		$\gamma_{15.0+}$	0.11

Notes

(i) Risk projections for the USA were carried out by the BEIR VI Committee for both models and shown to give very similar results. In the present report projections based on the exposure-age-concentration model are given.

(ii) Units are WLM^{-1}.

(iii) See Appendix A for definitions of WLM and WL.

(iv) The BEIR VI Committee recommended that, when separate estimates of β x 100 are required for smokers and non-smokers, these should be 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.

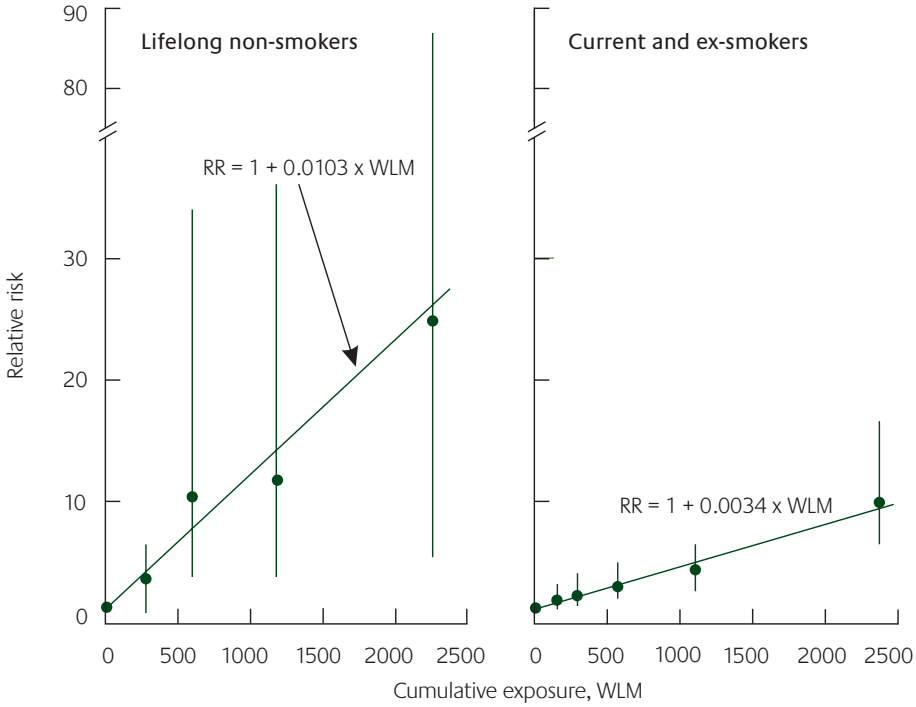

FIGURE H2 Relative risk (RR) of lung cancer with cumulative radon exposure among lifelong non-smokers and others in the six cohort studies of underground miners for which smoking information was available (based on BEIR VI Committee, 1999)

H2.2 Other studies of occupational exposure to radon

A study of tin miners in the UK has also been undertaken (Hodgson and Jones, 1990). This found that lung cancer mortality rates in the miners were higher than in the population in general and that the increased risk increased with duration of work underground. However, there were no direct measurements of historical radon concentrations so the cohort has not been used for quantitative risk assessment.

Since publication of the BEIR VI Report, several papers have been published giving further analyses of the North American and the Chinese cohorts of radon-exposed miners (Langholz et al, 1999; Stram et al, 1999; Hauptmann et al, 2001; Hazelton et al, 2001; Hornung, 2001; Duport, 2002; Archer et al, 2004; Heidenreich et al, 2004). But further follow-up has been conducted only for the Czech cohort (Tomásek, 2002; Tomásek and Zarska, 2004) and the French cohort (Rogel et al, 2002; Laurier et al, 2004). In addition, a large cohort of uranium miners in the former German Democratic Republic has been established (Kreuzer et al, 2002), as have cohorts of radon-exposed coal miners in Poland (Skowronek and Zemla, 2003) and Brazil (Veiga et al, 2004). A recent analysis of the German cohort, comprising 59,000 uranium miners, indicated that lung cancer risks were raised more than 35 years following

exposure (Grosche et al, 2006). In contrast, a case–control study that was conducted within this area of Germany, and that also took account of smoking history, reported that the relative risk of lung cancer among uranium miners decreased with time since exposure, except perhaps for exposures received more than 45 years previously (Brüske-Hohlfeld et al, 2006). In both studies, however, estimates of radon exposures during the early years of mining are particularly uncertain.

Despite these additional publications, the analyses of the BEIR VI Committee remain the most complete published summary to date of the risk of lung cancer from occupational exposure to radon.

H3 IARC evaluation of radon risks

In 1969 IARC initiated a programme to evaluate the carcinogenic risk of chemicals on humans and to produce monographs on individual chemicals or groups of chemicals. IARC first designated radon and its decay products as a human carcinogen in 1988. In 2001, IARC published a new monograph on the carcinogenic risks associated with the ingestion or inhalation of a variety of radioactive chemicals (IARC, 2001). This included radon. The monograph considered 12 cohort studies of underground miners exposed to high concentrations of radon-222 and its short-lived decay products, and additionally an analysis of the pooled data from 11 of these studies. All of these studies showed clear evidence of an increased risk of lung cancer associated with exposure to radon. In none of the studies was convincing evidence found for an increase in the risk of death from cancer other than lung cancer.

The monograph included an evaluation of 13 case–control studies that had investigated the possible association between lung cancer and residential exposure to radon. A meta-analysis, based on published data, of eight of these studies had been carried out in 1997. This and the five studies published after this date found a positive association between exposure to radon and lung cancer. The risk estimates from these studies of residential exposure were consistent with those predicted from the studies of underground miners.

IARC also reported that lymphocyte chromosomes showed increased levels of damage associated with radon concentrations above 200 Bq m^{-3} in cohorts from Sweden and Germany.

H4 EPA evaluation of radon risks

In 2003 the US Environmental Protection Agency (EPA) published its assessment of health risks from indoor radon, which it noted was the second leading cause of lung cancer after cigarette smoking (EPA, 2003). The EPA noted that its assessment was essentially based on the BEIR VI models but with some technical adjustments. First of all, the EPA modified and extended the approach used in the BEIR VI Report, constructing a single model giving numerical results midway between those obtained using the two BEIR VI preferred models. The EPA noted that the BEIR VI definition of excess risk effectively omitted premature deaths caused by radon in people who would otherwise have eventually died of lung cancer and the EPA modified its calculations to include all radon-induced lung cancer deaths. The EPA also used more detailed smoking prevalence and more recent mortality data for its calculations than those used by

the BEIR VI Committee. Furthermore the EPA provided numerical estimates of the risk per unit exposure, in terms of the number of lung cancer deaths per WLM, whereas the BEIR VI Committee estimated the fractional increase in lung cancer incidence due to radon.

Based on its analysis the EPA estimated that out of a total of 146,000 lung cancer deaths in the USA in 1995, 21,100 were radon related. This is 14.4% of the lung cancer deaths in the USA for that year, compared to the central estimate of risk of 11% (2–21%) quoted by the BEIR VI Committee. Among non-smokers an estimated 26% were radon related. Estimates of risks are given in the EPA report for both smokers and non-smokers. A Monte-Carlo uncertainty analysis (ie based on methods that rely upon repeated random sampling to compute their results) – which accounted for only those factors that can be quantified – indicated that estimates for the American population generally and for smokers in particular may be accurate to within factors of about two or three.

Finally the EPA report concluded that the effects of radon and cigarette smoking are more than additive, so that smokers are at a higher absolute risk from exposure to radon. The EPA anticipated that indoor radon will remain an important public health issue, contributing to thousands of lung cancer deaths annually.

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Appendix I
Epidemiology of Lung Cancer from Residential Exposure to Radon

The majority of human exposure to radon occurs environmentally rather than occupationally and, for the most part, occurs indoors, especially in small buildings such as houses. As radon concentrations in houses and other buildings can be reduced at low cost during the construction phase, and at moderate cost in existing buildings (see Appendix L), it is of potential public health importance to determine whether exposure to radon decay products in buildings, in particular in the home, increases the risk of lung cancer and, if it does, the magnitude of the risk.

The first human evidence that exposure to radon, chiefly from short-lived radon decay products, could cause lung cancer came from groups of underground miners of uranium or other igneous rocks who were occupationally exposed to substantial concentrations of radon (Appendix H). It was shown that radon caused many hundreds of deaths from lung cancer among those studied and that radon can cause lung cancer in both smokers and non-smokers. There is substantial uncertainty in extrapolating from the studies on miners to obtain a quantitative assessment of the risk of lung cancer from radon in the home. This source of uncertainty can be avoided by studying the risk of residential exposure to radon directly. However, even the largest of the epidemiological studies of residential radon and lung cancer that has been carried out to date has only limited statistical power. The recent publication of pooled analyses, in which the individual data from several individual studies have been collated centrally and then a single analysis of the combined data carried out, has, for the first time, provided clear, direct estimates of the risk of lung cancer from residential radon. We are now able to draw on evidence from several such analyses in reaching conclusions on the quantitative risks of radon exposures, and we here focus on these analyses.

Several research groups have undertaken epidemiological studies to investigate directly the risks of lung cancer from radon in the home. The main characteristics of the published studies are summarised in Table I1, whilst the results of the major studies are given in Table I2 and in Figure I1. Most of these studies used a case–control design in which a predetermined number of individuals who had developed lung cancer were identified, as well as a predetermined number of control individuals who had not developed the disease, but who were otherwise representative of the population from which the cases of lung cancer were drawn. The radon exposure history of each individual in the study was obtained, and the exposure histories of the two groups were then compared. Early studies estimated residential radon concentrations from building characteristics (Axelson et al, 1979, 1981; Edling et al, 1984a,b; Damber and Larssen, 1987). It was soon appreciated, however, that individual measurements of the radon concentration in each home were necessary. In later studies the radon concentration for each home of interest was usually determined using passive alpha etched-track detectors, often with one detector in a living room and one in a bedroom, and left in place for a period of between three months and a year, depending on the study. In order to obtain information about the long-term exposure history, many of

the studies measured the radon concentrations of homes in the past as well as the present homes of the individuals taking part in the study, while some studies specifically selected individuals who had lived for a very long time in the same home.

In the case–control studies of residential radon and lung cancer, the measurements of radon concentrations were usually made some time after the exposure of interest has occurred. In many cases the family of the study participant was still living in the home when the measurement was made, and no major alterations to the home had been carried out. Such measurements are likely to be representative of the exposures of interest. In other cases, there may have been alterations to the home, or the study participant may no longer have been living in it at the time the measurement was made. Such measurements are likely to be less closely related to the exposure of interest. Finally, for some homes it may not have been possible to obtain a measurement and it was necessary to construct an estimate of the likely radon concentration, usually based on other homes in the same area.

For all the studies summarised in Table I1, estimates of radon concentration in the home and smoking histories were obtained for each individual in the study. In addition to these studies, a number of geographical correlation studies (sometimes known as 'ecological studies') have also been published. In these ecological studies, attempts were made to look for associations between average radon concentrations and average lung cancer rates in geographical areas of various sizes. The best known of these is the study of Cohen (1995, 2001) in the USA. Such studies face severe methodological difficulties (Greenland, 2001; Lubin, 2002) and are liable to give biased and misleading estimates of the risk. This has been demonstrated in two of the case–control studies of residential radon, where parallel individual and ecological analyses of identical datasets have been carried out and have had discrepant results (Lagarde and Pershagen, 1999; Darby et al, 2001). The explanation for this is that in the ecological analyses it was not possible to control adequately for determinants of lung cancer risk such as cigarette smoking.

Using an ecological approach similar to that of Cohen, Puskin (2003) examined correlations between radon concentrations and a variety of cancers other than lung cancer, including some that are related to smoking. For these other smoking-related cancers, Puskin also found inverse correlations with radon concentrations. In contrast, he found no association between radon and those cancers that are not linked to smoking. Puskin concluded that the negative association observed for lung cancer can be explained in terms of confounding by smoking without invoking any kind of beneficial or hormetic effect of low level radiation exposure.

Cigarette smoking is the dominant cause of lung cancer. Urban areas tend to have lower radon concentrations than rural areas, partly because they tend to be located on alluvial deposits (see Chapter 2) and partly because more people tend to live upstairs in apartments in urban areas than in rural areas. People living in urban areas have also tended in the past to be more likely to smoke cigarettes. Hence, in the studies of the effects of radon in the home, smoking and radon concentration are negatively correlated (see Table I3).

(text continues on page 135)

TABLE I1 Epidemiological studies of residential radon and lung cancer in which data on the radon and smoking histories of each individual in the study have been obtained

	Setting	Source	Study design	Subjects	Exposure assessment	In pooled analyses?	Reason not in pooled analyses
1	Sweden, Southern	Axelson et al, 1979	Case–control	37 cases, 178 controls	Type of house construction	N	Fewer than 150 cases of lung cancer; crude exposure assessment
2	Sweden, Southern	Axelson et al, 1981	Case–control	27 cases, 183 controls	Type of house construction	N	Fewer than 150 cases of lung cancer; crude exposure assessment
3	USA, Washington County	Simpson and Comstock, 1983	Cohort	298 cases	Type of house construction	N	Crude exposure assessment
4	Sweden, Southern	Edling et al, 1984a	Case–control	23 cases, 202 controls	Type of house construction	N	Fewer than 150 cases of lung cancer; crude exposure assessment
5	Sweden, Southern	Edling et al, 1984b	Case–control	23 cases, 202 controls	1 and 3 month measurements	N	Fewer than 150 cases of lung cancer
6	Sweden, North	Pershagen et al, 1984	Case–control	30 cases, 30 controls	Type of house construction	N	Fewer than 150 cases of lung cancer; crude exposure assessment
7	Sweden	Pershagen et al, 1984	Case–control	23 cases, 23 controls	Type of house construction	N	Fewer than 150 cases of lung cancer; crude exposure assessment
8	Sweden, Northern	Damber and Larsson, 1987	Case–control	589 cases, 582 deceased controls, 453 living controls	Type of house construction	N	Crude exposure assessment

TABLE I1 ***continued***

	Setting	Source	Study design	Subjects	Exposure assessment	In pooled analyses?	Reason not in pooled analyses
9	Sweden, Stockholm	Svensson et al, 1987	Case–control	292 cases, 584 controls	Geology and living near ground level; some grab sample results	N	Crude exposure assessment
10	Sweden, Southern	Axelson et al, 1988	Case–control	177 cases, 677 controls	Type of house construction; some 2 month measurements	N	Crude exposure assessment
11	Belgium	Poffijn et al, 1989	Case–control			N	Fewer than 150 cases of lung cancer
12	Sweden, Stockholm County	Svensson et al, 1989	Case–control	187 cases, 160 hospital controls, 177 population based controls	Geology and living near ground level; some 2 week measurements	N	Crude exposure assessment
13	China, Shenyang	Blot et al, 1990	Case–control	308 cases, 356 controls	Alpha track measurements over at least several months	Y	
14	Hungary, Eastern	Deri et al, 1992	Case–control	33 cases, 66 controls	Measurements over at least several months	N	Fewer than 150 cases of lung cancer
15	Sweden, Stockholm	Pershagen et al, 1992	Case–control	201 cases, 378 controls	Alpha track measurements over at least several months	Y	
16	USA, New Jersey	Schoenberg et al, 1992	Case–control	480 cases, 442 controls	Alpha track measurements over at least several months	Y	
17	USA, Missouri (MO-I)	Alavanja et al, 1994	Case–control	538 cases, 1183 controls	Alpha track measurements over at least several months	Y	
18	Canada, Winnipeg	Létourneau et al, 1994	Case–control	738 cases, 738 controls	Alpha track measurements over at least several months	Y	
19	Sweden (nationwide)	Pershagen et al, 1994; Lagarde et al, 1997	Case–control	1281 cases, 2576 controls	Alpha track measurements over at least several months	Y	

TABLE I1 ***continued***

	Setting	Source	Study design	Subjects	Exposure assessment	In pooled analyses?	Reason not in pooled analyses
20	Russia, Moscow	Zaridze et al, 1995	Case–control	79 cases, 135 controls	Short-term measurements, lasting 30 minutes	N	Fewer than 150 cases of lung cancer. Also short-term radon measurements only
21	Finland (nationwide)	Auvinen et al, 1996	Case–control	517 cases, 517 controls	Alpha track measurements over at least several months	Y	
22	Finland, South	Ruosteenoja et al, 1996	Case–control	291 cases, 495 controls	Alpha track measurements over at least several months	Y	
23	UK, South West England	Darby et al, 1998	Case–control	982 cases, 3185 controls	Alpha track measurements over at least several months	Y	
24	USA, Missouri (MO-II)	Alavanja et al, 1999	Case–control	512 cases, 553 controls	Alpha track measurements over at least several months	Y	
25	Bulgaria, Town of Rakovski	Pressyanov et al, 1999	Standardised incidence ratio		Measurements in a sample of homes for 2 months	N	Crude exposure assessment
26	USA, Connecticut	Sandler et al, 1999	Case–control	963 cases, 949 controls	Alpha track measurements over at least several months	Y	
27	USA, Utah–South Idaho	Sandler et al, 1999	Case–control	511 cases, 862 controls	Alpha track measurements over at least several months	Y	
28	Germany, Schneeberg area	Conrady et al, 2000	Case–control	72 cases, 288 controls	Measurements over at least several months	N	Fewer than 150 cases of lung cancer
29	USA, Iowa	Field et al, 2000	Case–control	413 cases, 614 controls	Alpha track measurements over at least several months	Y	
30	Sweden (never-smokers)	Lagarde et al, 2001	Case–control	258 cases, 487 controls	Alpha track measurements over at least several months	Y	

TABLE I1 ***continued***

	Setting	Source	Study design	Subjects	Exposure assessment	In pooled analyses?	Reason not in pooled analyses
31	Italy, Alpine area	Pisa et al, 2001	Case–control	138 cases, 291 controls	Measurements over at least several months	N	Fewer than 150 cases of lung cancer
32	Czech Republic	Tomásek et al, 2001	Cohort	210 cases	Alpha track measurements over at least several months	Y	
33	Spain, North West	Barros-Dios et al, 2002	Case–control	163 cases, 241 controls	Alpha track measurements over at least several months	Y	
34	Austria, Tyrol	Oberaigner et al, 2002	Case–control	183 cases, 188 controls?	Alpha track measurements over at least several months	Y	
35	China, Gansu	Wang et al, 2002	Case–control	768 cases, 1659 controls	Alpha track measurements over at least several months	Y	
36	France	Baysson et al, 2004	Case–control	486 cases, 984 controls	Alpha track measurements over at least several months	Y	
37	Italy, Latzio	Bochicchio et al, 2005	Case–control	384 cases, 404 controls	Alpha track measurements over at least several months	Y	
38	Germany, Eastern	Wichmann et al, 1999, 2005	Case–control	1312 cases, 1717 controls	Alpha track measurements over at least several months	Y	
39	Germany, Western	Wichmann et al, 1998, 2005	Case–control	1651 cases, 2515 controls	Alpha track measurements over at least several months	Y	
40	Russia, Middle Urals	Kirdin et al, 2001; Lezhnin et al, 2001	Case–control	323 cases, 444 controls	Alpha track measurements over 3 months	N	Investigators unaware of study
41	USA, Connecticut and Utah	Sandler et al, 2006	Case–control	1474 cases, 1811 controls	Alpha track measurements over 12 months	N	Too recent
42	USA, Worcester County, Massachusetts	Thompson et al, 2008	Case–control	200 cases, 397 controls	Alpha track measurements over 12 months	N	Too recent

TABLE I2 Results of the major epidemiological studies of residential radon and lung cancer

Setting	Source	Average measured radon concentration (Bq m^{-3})	% increase in risk per 100 Bq m^{-3} measured radon (with 95% CI)	% increase in risk per 100 Bq m^{-3} long-term average radon (with 95% CI)
Europe				
Austria	Oberaigner et al, 2002	198	46 (<0–>5)	162 (<0–>500)
Czech Republic	Tomásek et al, 2001	500	19 (0–207)	>500 (6–>500)
Finland, nationwide	Auvinen et al, 1996	103	3 (<0–17)	–2 (–38–124)
Finland, south	Ruosteenoja et al, 1996	215	6 (–8–158)	35 (<0–>500)
France	Baysson et al, 2004	133	11 (–1–41)	17 (–1–62)
Germany, Eastern	Wichmann et al, 1999, 2005; Kreuzer et al, 2003	76	18 (0–56)	38 (–2–139)
Germany, Western	Wichmann et al, 1998, 2005; Kreienbrock et al, 2001	50	<0 (<0–39)	15 (–36–161)
Italy	Bochicchio et al, 2005	108	10 (–18–140)	10 (–20–168)
Spain	Barros-Dios et al, 2002	131	<0 (<0–59)	–14 (<0–87)
Sweden, nationwide	Pershagen et al, 1994; Lagarde et al, 1997	96	11 (–4–46)	17 (–5–67)
Sweden, never-smokers	Lagarde et al, 2001	74	24 (–8–95)	28 (–12–126)
Sweden, Stockholm	Pershagen et al, 1992	134	12 (–14–141)	17 (–21–387)
UK	Darby et al, 1998	55	4 (–5–22)	5 (–7–28)
Overall: Europe		105	8 (3–16)	16 (5–30)

TABLE I2 ***continued***

Setting	Source	Average measured radon concentration (Bq m^{-3})	% increase in risk per 100 Bq m^{-3} measured radon (with 95% CI)	% increase in risk per 100 Bq m^{-3} long-term average radon (with 95% CI)
North America				
Connecticut	Sandler et al, 1999	33	2 (–21–51)	–
Iowa	Field et al, 2000	127	44 (5–16)	–
Missouri-I	Alavanja et al, 1994	63	1 (<0–42)	–
Missouri-II	Alavanja et al, 1999	56	27 (–12–153)	–
New Jersey	Schoenberg et al, 1992	26	56 (–22–297)	–
Utah–South Idaho	Sandler et al, 1999	57	3 (–20–55)	–
Winnipeg	Letourneau et al, 1994	120	2 (–5–25)	–
Overall: North America		67	11 (0–28)	–
China				
Gansu	Wang et al, 2002	223	19 (5–47)	–
Shenyang	Blot et al, 1990	85	–5 (<0–8)	–
Overall: China		201	13 (1–36)	–

	Number of lung cancers / controls	% increase in risk per 100 Bq m^{-3} measured radon (with 95% CI)
Study (p for heterogeneity: 0.94)		
Austria	183 / 188	46
Czech Republic	171 / 713	19
Finland: nationwide	881 / 1,435	3
Finland: south	160 / 328	6
France	571 / 1,209	11
Germany: eastern	945 / 1,516	18
Germany: western	1,323 / 2,146	−2
Italy	384 / 405	10
Spain	156 / 235	−11
Sweden: nationwide	960 / 2,045	11
Sweden: never-smokers	258 / 487	24
Sweden: Stockholm	196 / 375	12
UK	960 / 3,126	4
Age (p for trend: 0.93)		
<55 years	1,100 / 2,582	−7
55–64 years	2,506 / 4,818	14
65+ years	3,542 / 6,808	7
Sex (p for heterogeneity: 0.19)		
Male	5,521 / 10,388	11
Female	1,627 / 3,820	3
Smoking (p for heterogeneity: 0.98)		
Current cigarette smoker	3,575 / 3,322	7
Ex-smoker	2,465 / 4,930	8
Lifelong non-smoker	884 / 5,418	11
Other	224 / 538	8
Histological type (p for heterogeneity: 0.07)		
Squamous cell	2,479 / 14,208	−1
Adeno	1,698 / 14,208	6
Small cell	1,379 / 14,208	31
Other type	754 / 14,208	3
Unknown	838 / 14,208	8
Overall, using measured radon	7,148 / 14,208	8
Overall, using long-term average radon	7,148 / 14,208	16

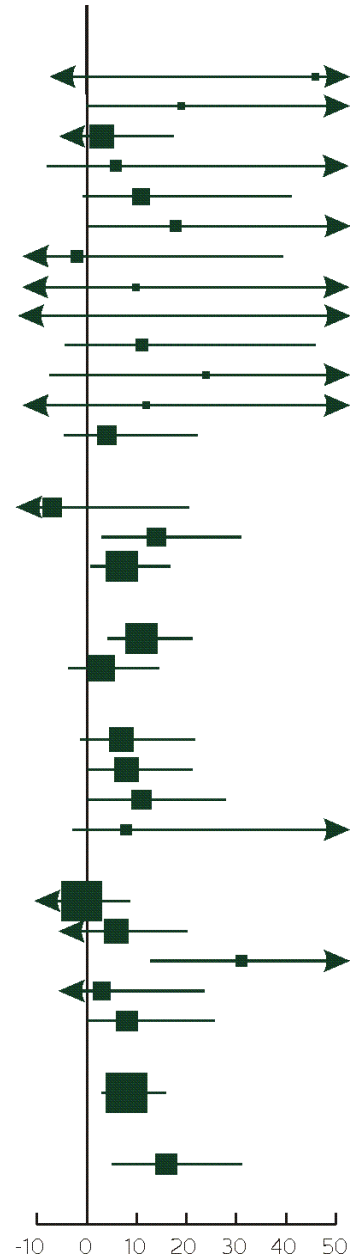

FIGURE I1 Summary of results of the major European studies of residential radon and lung cancer, as used in the European pooling study (from Darby et al, 2005). Smoking status 'other' corresponds to occasional smokers and current smokers of cigars, cigarillos or a pipe only. Areas of plotted squares are inversely proportional to the standard error of the percentage increase in risk per 100 Bq m^{-3}

TABLE I3 Percentage of control individuals without lung cancer who were found to be current or past smokers of cigarettes by category of measured radon concentration in European studies of residential radon and lung cancer (Darby et al, 2006)

Measured radon concentration (Bq m^{-3})	Percentage who have ever smoked cigarettes
<100	61
100–199	60
200–399	59
400–799	54
800+	52

p for trend = 0.001.

Because of this negative correlation between radon concentration and smoking, it is essential to make very careful allowance for the effect of smoking in analyses estimating the lung cancer risk associated with radon in the home and, to do this reliably, data on the smoking habits of each individual taking part in the study are needed. In contrast to the studies of miners, the studies of radon in the home almost all included detailed information on individual smoking habits obtained by interviewing the person concerned, or a close relative. Some of the studies were restricted to never-smokers. Other studies were restricted to women, partly because the occupational studies of miners provide no data on women and partly because women have, in the past, tended to smoke less and to spend more time in the home than men.

A number of papers have been published in which the authors have combined the information from several studies of residential radon. Some of these papers have been based on the publications of individual studies and combined the published results (Lubin and Boice, 1997; Lubin, 1999; Pavia et al, 2003). Such papers have all concluded that the radon-related risk of lung cancer varies significantly from one study to another. However, it is likely that this variation arises because the methodology used to analyse the various studies differs considerably from study to study, notably in the quantification of the radon exposure histories of each individual and the methods used to allow for differing smoking histories between individuals. Such differences cannot be eliminated without access to basic data for each individual involved in the studies. Therefore, although such meta-analyses (ie critical review and combination of the results of multiple studies based on published information about them, with a quantitative summary of all the results collectively) are easier to carry out than pooled analyses of the original data of the individual studies, they are of limited value in the present example, where control of confounding by smoking is such a key issue.

A more appropriate method of combining the information from several studies of the risk of lung cancer from residential radon is to assemble and collate the component data on the radon concentration, smoking history and other relevant factors for each individual in the original study, and undertake a

pooled analysis of these data. Based on this individual information a uniform definition and categorisation of radon exposures is possible, as is a uniform and detailed adjustment for the effect of smoking. For these reasons, we focus on the evidence from pooled analyses of individual data in assessing the risks of lung cancer associated with residential radon exposure.

Four pooled analyses based on individual data have been undertaken: of thirteen European studies (Darby et al, 2005, 2006), of seven North American studies (Krewski et al, 2005, 2006), of two Chinese studies (Lubin et al, 2004) and of two German studies (Wichmann et al, 2005). The two studies involved in the pooling of German studies were both included in the European pooling study and therefore will not be discussed further in the present report. Table I4 summarises the three major pooling studies. Results were reported in terms of the percentage increase in the risk of lung cancer per 100 Bq m^{-3} increase in residential radon exposure.

TABLE I4 Summary of estimates of the risk of lung cancer associated with residential radon in pooling studies that have combined individual data from a number of case–control studies

					Percentage increase in risk of lung cancer per 100 Bq m^{-3} increase in radon concentration (with 95% CI)		
					Based on measured radon		Based on long-term average radon [ii]
Study	Number of studies included	Number of cases of lung cancer	Number of controls	Exposure window (years)	Including all subjects	Limited [i]	
European	13	7,148	14,208	5–34	8 (3–16)	9 (3–18)	16 (5–31)
North American	7	3,662	4,966	5–30	11 (0–28)	18 (2–43)	–
Chinese	2	1,050	1,995	5–30	13 (1–36)	32 (7–91) [iii]	–
Weighted average of above studies	22	11,860	21,169		10 (8–12)	–	~20 [iv]
Miners exposed at <0.5 WL [vi]	11	2,787	n/a		19 30 [v]	–	–

Notes

(i) Considering only individuals with one or two homes during the period of interest and who had at least 20 years' coverage.

(ii) Allowing for year-to-year random variation in measured radon.

(iii) Considering only individuals who had a single home during the period of interest.

(iv) Informal estimate, indicating the likely effect of removing the bias induced by random year-to-year variation in radon concentrations (see text).

(v) Considering only miners with cumulative exposures <50 WLM.

(vi) See Appendix A for definitions of WL and WLM.

The exposure quantity of interest in the epidemiological studies is the *long-term average radon concentration* that the individual was exposed to in homes occupied during the reference period of 25 or 30 years ending 5 years prior to the diagnosis of lung cancer (or prior to a comparable reference date for the individuals who had not developed lung cancer). This quantity is not directly available. Instead the quantity that is available for each individual is the *measured radon concentration*, usually calculated by taking a weighted average of the measurements made in each home, with weights proportional to the length of time that the individual lived in the home.

A number of studies have shown that, when the radon concentration is measured in a home in several different years, the measurements vary considerably, reflecting principally the year-to-year variation in the radon concentration in that home. The coefficient of variation between measurements made in different years in the same home is typically around 50%, even when each measurement is either made over a whole year, or over several months with an appropriate seasonal correction (Darby et al, 2006). The distribution of measured radon concentrations is usually highly skewed and in all the component case–control studies was found to be approximately log-normal. In addition, studies of magnitude of the year-to-year variability have shown that it is proportional to the radon concentration (Reeves et al, 1998). This means that the year-to-year variability is much larger at high radon concentrations than at low radon concentrations. Analyses of data from the radon case–control studies that are based simply on measured radon concentrations ignoring the year-to-year variability will underestimate the risk of radon-induced lung cancer, and special methods need to be employed to take the year-to-year variation into account (see Appendix C for further discussion).

A number of case–control studies are too recent to have been included in either the European or the North American pooling studies (see Table I1). Two of these (Kirdin et al, 2001; Sandler et al, 2006) reported risks that are in line with the results reported by the pooling studies, but one of them (Thompson et al, 2008) reported a significant negative association between the risk of lung cancer and residential radon exposure. This study included 200 cases compared with several thousand cases in the North American pooling study. Therefore, if it had been possible to include it in the pooling study it would have made little difference to the overall result. Also, the precision of adjustment for smoking was not as great as that in the European pooling.

I1 European pooling study

The largest of the pooling studies to date is that based on the European studies (Darby et al, 2005, 2006). It analysed data from all 13 European studies of residential radon and lung cancer that included over 150 people with lung cancer and 150 control individuals without lung cancer, incorporated detailed smoking histories and sought radon measurements in homes where these individuals had lived during the past 15 years or more. In total, more than 7,000 lung cancer cases and over 14,000 controls were included. The study examined the effect on lung cancer risk of exposures to radon during the 30-year period ending 5 years prior to the diagnosis of lung cancer (or prior to a comparable reference date for control individuals). Two features of the analysis were the very fine stratification used to allow for the effect of smoking and the allowance for year-to-year random variability that is present when the radon

concentration in a home is measured in different years. The rationale for allowing for random year-to-year variability is explained in Appendix C.

The European pooling study showed a clear association between increasing exposure to radon and lung cancer. There was no significant variation between the proportional increases in risk estimated for the component studies, and the results were not dominated by any single study. The risk appeared to be approximately linear with no evidence for a threshold below which there was no risk (Figure I2). In particular, the results were incompatible with a threshold above 150 Bq m^{-3} (ie 150 Bq m^{-3} was the 95% upper confidence limit for any threshold).

Furthermore, the investigators found a statistically significant association between radon concentration and lung cancer even when analysis was restricted to people in homes with measured radon concentrations below 200 Bq m^{-3} ($p = 0.04$). When individuals with measured radon in the range 100–199 Bq m^{-3} (mean 136 Bq m^{-3}) were compared with those with measured radon below 100 Bq m^{-3} (mean 52 Bq m^{-3}), their risk of lung cancer was increased by 20% (95% CI 3–30%, $p = 0.01$). The

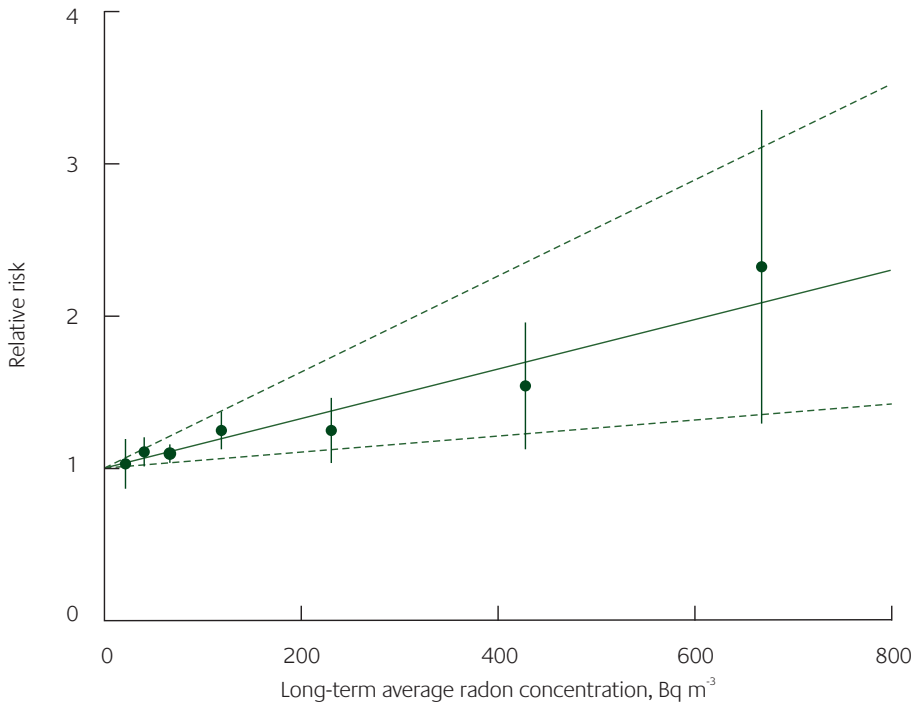

FIGURE I2 Relative risk of lung cancer versus long-term average residential radon concentration in the European pooling study (based on Darby et al, 2005). Relative risks and 95% confidence intervals are shown for categorical analyses and also the best-fitting straight line. Risks are relative to that at 0 Bq m^{-3}

proportional increase in risk per 100 Bq m^{-3} did not vary significantly with age, sex or smoking status. However, it was somewhat larger for small cell lung cancer (31.2%, 95% CI 12.8–60.6%) than for other histological types (2.6%, 95% CI <0–10.2%).

When the analysis was conducted in terms of measured radon concentrations, it was estimated that the risk of lung cancer increased by 8% per 100 Bq m^{-3} increase in mean measured radon concentration (95% CI 3–16%). When the analysis was repeated in terms of long-term average radon concentrations (ie taking into account the random year-to-year variability in measured radon concentration), the final estimated risk coefficient was higher, at 16% per 100 Bq m^{-3} (95% CI 5–31%). As there was some uncertainty in the magnitude of the random year-to-year variability in measured radon concentrations, the investigators performed a sensitivity analysis to examine whether the final estimated risk depended strongly on the magnitude of the year-to-year variation used (Darby et al, 2006). This sensitivity analysis confirmed that the value of the final risk estimate was stable for a range of values for the year-to-year variability.

Radon measurements were available for an average of 23 years of the full 30-year period of interest. For the remaining dwellings it was not possible to obtain a radon measurement, eg because a house had been demolished. Where measurements had not been possible, estimated values were used in the construction of the mean measured radon concentration for an individual, based on the mean measured value in all the dwellings for individuals in the control group in the local area. Darby et al examined the way that risk estimates varied with the number of homes that the individuals taking part in the study had occupied during the 30-year period of interest, and also with the proportion of the 30-year period for which measurements were available. There was no significant difference between the risk estimates for those with complete and incomplete radon exposure histories.

I2 North American pooling study

Krewski and co-workers (2005, 2006) published a pooled analysis which involved 3662 cases and 4966 controls from seven studies in the USA and Canada. The reference period considered was the 25-year period ending 5 years before the diagnosis of lung cancer, slightly shorter than in the European pooling study.

In this analysis there was also detailed allowance for smoking, but it was less finely stratified than that used in the European pooling study.

When compared with individuals whose mean measured radon concentrations were below 25 Bq m^{-3}, individuals in various categories of higher mean measured radon concentration had increased risks of lung cancer, but for no individual category was the increase statistically significant (see Figure I3). The estimated increase in risk per 100 Bq m^{-3} measured radon concentration was 11% (95% CI 0–28%). There was no significant heterogeneity between the levels of risk estimated for the component studies, and the risk estimate did not change substantially when any of the studies was excluded from the analysis.

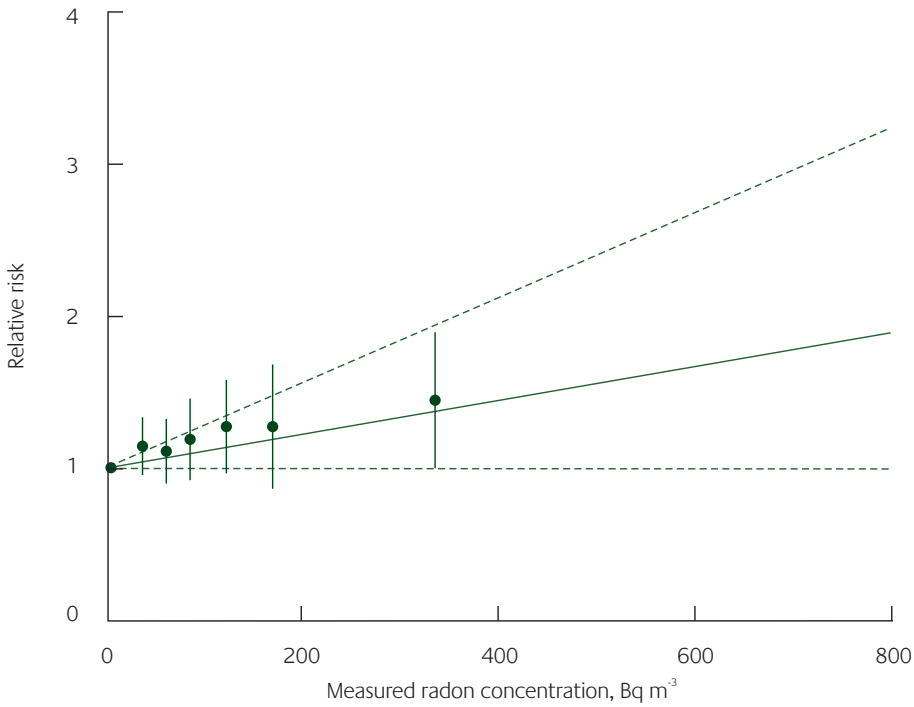

FIGURE I3 Relative risk of lung cancer versus measured residential radon concentration in the North American pooling study (based on Krewski et al, 2005). Relative risks and 95% confidence intervals are shown for categorical analyses and also the best-fitting straight line. Risks are relative to those in the category <25 Bq m^{-3}

Krewski et al also considered analyses restricted to various subgroups. When the analysis included only individuals for whom radon exposure histories were more complete, in that monitoring data were available for at least 20 years out of the 25-year exposure time window, the increase in risk per 100 Bq m^{-3} measured radon was 14% (95% CI 1–35%). A similar result was obtained by restricting attention to those who had lived in only one or two homes. As discussed above, this differs from the finding in the European pooling study, where the risk estimate for a subgroup analysis including only individuals with at least 20 years' coverage was similar to the overall estimate.

As with the European pooling study, the relative risk for small cell lung cancer [increase in risk per 100 Bq m^{-3} of 23% (95% CI 8–88%)] was larger than that for other types of lung cancer (9% for adenocarcinoma and squamous cell cancer).

There was no significant variation in the estimated proportional increase in risk per 100 Bq m^{-3} with smoking status, sex, age or educational level.

I3 Chinese pooling study

Lubin and co-workers (2004) published a study which involved 1050 cases and 1996 controls from two studies in two areas, Gansu and Shenyang; the first of these studies was much the larger. As with the North American pooling study, the reference period considered was 5–30 years before diagnosis. Four smoking categories were considered in the analysis.

For the pooled data, the increase in risk per 100 Bq m^{-3} was 13.3% (95% CI 1–36%). This positive slope was chiefly due to the Gansu data, although the results of the two component studies were compatible with each other. For subjects resident in only one home for the whole of the 25-year reference period, the increase in risk per 100 Bq m^{-3} measured radon was 32% (95% CI 7–91%) based on the pooled data.

The investigators did not report a separate result for small cell lung cancer.

As with the European and North American poolings, there was no significant difference between the estimated proportional increase in risk per 100 Bq m^{-3} calculated for the different smoking categories.

The risk estimate was higher in analyses restricted to those study subjects who had provided information on confounders themselves compared to those where it had been obtained from a surrogate.

I4 Overall appraisal of risk of residential exposure to radon based on the three pooling studies

The best evidence of the risks of residential radon exposure would come from a pooled analysis of individual data from all the residential studies, including all those in the three pooling studies described above. Such a world pooling is currently under way, but its results will not be available for some time. Nevertheless, the three pooling studies present very similar broad pictures of the risks of lung cancer from residential exposure to radon. There is overwhelming evidence that radon is acting as a cause of lung cancer in the general population and at concentrations found in ordinary homes. In particular, there is substantial evidence that there is a risk even below 200 Bq m^{-3}, the concentration at which action is currently advocated in many countries. In the European pooling study the dose–response relationship appears linear, with no evidence of a threshold dose (Figure I2). The results of the North American pooling are also consistent with a linear dose–response relationship with no threshold, although greater uncertainty is involved (Figure I3).

The three major pooling studies reported increased risks of lung cancer based on measured radon concentrations of 8% (95% CI 3–16%), 11% (0–28%), and 13% (1–36%) per 100 Bq m^{-3} (Table I4). As these three estimates are statistically compatible with each other, a weighted average of them, with weights proportional to their variances, can be calculated. This gives a joint estimate from the three pooling studies, based on measured radon concentrations, of 10% per 100 Bq m^{-3}.

In each of the three major pooling studies the investigators also reported alternative analyses. All the studies considered subsets of the data for individuals for whom measurements were more complete in the exposure time window or who had lived in only one or two homes. In the case of the North American

and Chinese poolings, these restricted analyses resulted in higher risk estimates than did the basic analysis; in the case of the European pooling the results of the restricted analysis were very similar to those of the analysis in which all subjects were included. The authors of the North American and Chinese poolings argued that these restricted analyses gave more reliable risk estimates. This was on the basis that exposure estimates based on direct measurements in the homes in question are better than those that include imputed values and that it is easier to make reliable estimates of exposure for individuals who have lived in few dwellings. However, it should be noted that those who respond to requests for measurements in their homes may differ in, for example, socioeconomic status from those who do not. It is therefore possible that exclusion of subjects for whom the radon information is incomplete could bias the observed relationship between radon exposure and lung cancer (Vach and Blettner, 1991; Greenland and Finkle, 1995; Demissie et al, 2003; Lyles and Allen, 2003).

As described above, estimates based on measured radon concentration will underestimate the true risks associated with residential radon, due to the year-to-year random variation in radon concentrations in a home. The only pooling study that has to date carried out a detailed analysis of the risks of residential radon based on long-term average, as opposed to measured, radon concentrations, is the European pooling. In this study, the estimate based on long-term average concentrations was double the estimate based on measured radon concentrations. Data from repeated radon measurements made in separate years in the same home in China show a similar amount of variation to that seen in the European studies (Lubin et al, 2004; Darby et al, 2005), suggesting that an estimate from the Chinese study based on long-term average radon concentration would also be approximately double the estimate based on measured radon. No data from North America on repeated measurements made on different occasions in the same home are available at present, but it seems likely that the association with measured radon there also underestimates the relationship between lung cancer and long-term average radon concentration.

Darby et al (2005, 2006) noted a number of factors that could not be included in the analysis of the pooling studies. In particular, there would have been errors in the assignment of individuals to smoking categories, variation in the radon concentration between the different rooms in a home and, in some countries, there may have been systematic changes in the radon concentrations over the last few decades, due to increased energy efficiency. There may also be some element of risk resulting from exposure to radon concentration outside the 25- or 30-year exposure time window considered. Additionally, there may be some inaccuracy in the reported smoking habits of the individuals in the studies (Heid et al, 2006). The overall effect of these factors may mean that the true effect of radon might be somewhat higher than the estimated risk in the European pooled analysis after correction for year-to-year random variation in measured radon concentrations, and higher than the risks reported in the other two pooling studies.

On the basis of the pooling studies it appears that the relative risk of radon exposure (characterised as the proportional increase in risk per 100 Bq m^{-3} increase in residential radon concentration) is similar between sexes, across age groups and between current smokers, ex-smokers and lifelong non-smokers.

I5 Absolute risk of radon-induced lung cancer in the UK based on estimates of risk from the European pooling study

If the proportional increase in risk per unit radon concentration is approximately independent of smoking history then, as lung cancer is much commoner in cigarette smokers than in lifelong non-smokers, radon poses a much greater absolute hazard to cigarette smokers, and to recent ex-smokers, than to lifelong non-smokers or to long-term ex-smokers. Absolute risks of lung cancer are not available from the epidemiological studies of residential radon and lung cancer, as the number of individuals with lung cancer and also the number of unaffected control individuals was determined by the study design. However, appropriate estimates of the absolute death rate from lung cancer in lifelong non-smokers at different ages are available from a large prospective study carried out by the American Cancer Society (Peto et al, 1992). These estimates are consistent with estimates derived from data in the UK based on the follow-up of British doctors (Doll et al, 2004; Darby et al, 2006). Based on the absolute risks of lung cancer among lifelong non-smokers in the American Cancer Society study, the cumulative risk to age 75 years of mortality from lung cancer among current smokers in the UK study, in the absence of other causes of death, was 15.9% (Peto et al, 2000). For those giving up smoking at 50 and 30 years the cumulative risks to 75 years were much lower, at 6.0 and 1.7%, respectively, while for lifelong non-smokers, the cumulative risk to 75 years was 0.4%. These estimates of the effect of smoking are based on data for males only. However, women's smoking habits have changed in recent years to become more like those of men. Therefore, it is likely to be more relevant to model future risks among women who currently smoke cigarettes in the UK than on an estimate based on the smoking habits of the female cases of lung cancer and controls in the UK study.

If the risk of lung cancer increases by about 16% per 100 Bq m^{-3} increase in long-term mean radon concentration, regardless of smoking status, then at long-term mean radon concentrations of 0, 100, 200, 400 and 800 Bq m^{-3}, respectively, the cumulative absolute risks of lung cancer by age 75 years would be 0.4, 0.5, 0.5, 0.7 and 0.9% in lifelong non-smokers, and 15, 17, 19, 23 and 30% in current cigarette smokers (see Figure I4). For those who gave up smoking cigarettes at age 50 years, the corresponding risks would be considerably lower than those for continuing smokers, at 5.5, 6.4, 7.2, 8.9 and 12.2%, while for those who gave up smoking at 30 years the risks would be much lower, at 1.6, 1.8, 2.1, 2.6 and 3.5%, respectively. These absolute risk estimates are somewhat higher than those estimated in the European pooling study, as the relative risks of lung cancer in cigarette smokers compared with lifelong non-smokers are somewhat higher in the UK than in other European countries (Crispo et al, 2005).

I6 Summary of all evidence on radon risks

There is overwhelming evidence from epidemiology and from animal experiments (reviewed in Appendix G) that exposure to radon and its decay products leads to an increased risk of lung cancer. This is consistent with the evidence for the induction of lung cancer by external sources of ionising radiation (UNSCEAR, 2000). In this section we consider how quantitative estimates of these lung cancer risks may be derived in order to help determine policies for control of radon as a public health hazard.

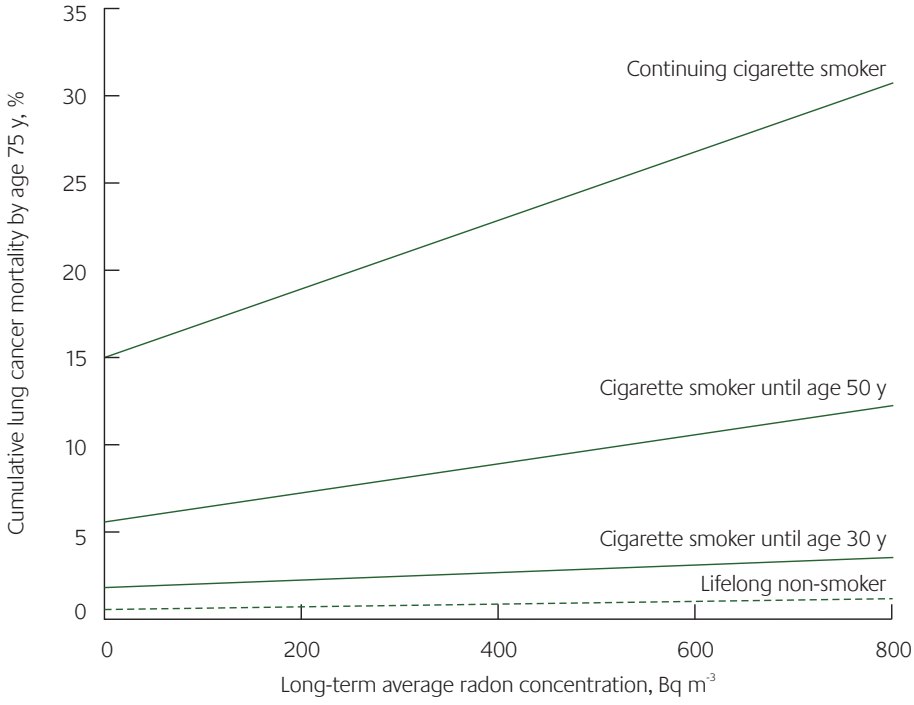

FIGURE I4 Cumulative absolute risk of death from lung cancer by age 75 years versus long-term average radon concentration at home for continuing cigarette smokers, ex-smokers and lifelong non-smokers in the UK (see Table 4.3 for sources of data and method of calculation)

All the sources of information have their strengths. It is also true that each paints a broadly similar picture. Nevertheless, in deciding upon a quantitative estimate of the risks of residential exposure to radon, we have depended rather little on the data from animal experiments. It is true that these involved carefully measured exposures and allowed investigation of the possible effects of potential co-carcinogens, in particular tobacco smoke. Nevertheless, many of these studies were conducted at radon concentrations higher than those of interest in a domestic context. Moreover, the differences observed between different animal species and strains emphasise the difficulties in extrapolating these data to humans (see Appendix G).

There is no doubt that the best and most direct quantitative risk estimates are based on epidemiological studies of humans, rather than on animal studies. Nevertheless, it is inevitable that there will be a low dose region, where the statistical uncertainties are too large for epidemiology to provide direct evidence. As we have noted above, direct evidence now extends down to radon concentrations below 200 Bq m^{-3}, and it is important to form a view on risks at lower concentrations, because the vast majority of the population is exposed at these levels (see Chapter 4). The usual assumption in radiological protection is

that radiation risks are proportional to the dose, decreasing as doses decrease, but without any threshold below which risks are zero. We are convinced by the evidence from radiobiology (see Appendix D) that this assumption is appropriate in the case of radon exposures and, based on it, calculations indicate that this is where the majority of the radon-induced deaths will occur.

In the past, radon risk estimates for residential exposures have been based on evidence from epidemiological studies of miners (reviewed in Appendix H). With the publication of the pooled analyses of individual data from a number of residential case–control studies, it is time to consider the relative weight to be given to the two types of study in deciding radon risk estimates for purposes of controlling residential exposures.

Epidemiological studies of residential radon exposure have been discussed above. Individually, the component studies had limited statistical power, and meta-analysis of the published results suggested that the results of the studies were inconsistent. However, recent pooled analyses have combined the data on all the individuals in a number of residential studies. These pooled analyses have greater power than the individual component studies and have also demonstrated that the latter were mutually consistent, despite the initial indications to the contrary from the meta-analyses of summarised results from the individual studies.

The analyses of pooled data from the residential studies have certain advantages over the studies on miners:

a Exposures were received under similar aerosol conditions to those of interest.

b Exposures were received at similar concentrations to those of interest (thus avoiding problems from high dose-rate effects and also from extrapolating from high dose rate to low dose rate).

c There is little confounding from possible exposure to occupational carcinogens such as arsenic.

d Detailed individual smoking histories are available for all study participants, thus enabling the risks of cigarette smoking to be accounted for in the analysis.

e Detailed individual exposure data are available, generally based on individual measurements in the homes where the individual has lived, although the measurements were usually made some time after the period of interest.

f A substantial quantity of data is available regarding the uncertainties in the assessment of residential radon exposures, and analyses have been carried out taking these uncertainties into account.

g Data are available for both men and women for all ages at which lung cancers occur.

The miners' studies also have certain strengths:

a There were large exposures leading to clear excesses of lung cancer, allowing detailed examination of the shape of the dose–response relationship, at least at high exposures.

b The ability to examine time-since-exposure effects – many individuals in the residential studies did not move house very often, so their measured exposure in one year is similar to that in the next. In

contrast, the miners were usually employed underground for only a few years. Furthermore they often had huge changes in their exposures from year to year.

c The data on miners potentially provide information on risks over a lifetime, not just a window of 30 years or so – continued follow up will be needed to realise this potential to the full.

d Some of the miners' studies have exposure estimates based on contemporary measurements (albeit incomplete and for areas rather than individuals).

Some further points are relevant to the studies on miners.

a For the majority of the studies of radon-exposed miners there is no information on the smoking habits of the individuals concerned and in the studies where there is such information it is usually very limited. This means that it is not possible to take the effect of smoking history into account in studying the risk of radon-induced lung cancer in the miners and the resulting risk estimates may well be subject to confounding by smoking.

b The uncertainties in these measurements and their effect on the resulting risk estimates have yet to be studied in detail.

c There is substantial heterogeneity between the risk estimates in the miner studies; the risk estimates from individual studies vary by more than an order of magnitude.

d The data on miners suggest a large age effect. The relative risk of radon-induced lung cancer among miners decreases with increasing attained age so that miners aged over 75 years have about 10% of the proportional increase in risk per unit exposure of the risk among those aged less than 55 years. This age dependence is not replicated in the residential studies; however, the uncertainties in the data are not small and the possibility exists of confounding by age- or time-related changes in smoking habits. Further work is desirable to clarify this matter.

e The risk estimates seen in the miners' studies are often affected by high dose-rate effects.

f The conditions of exposure in mines, in terms of aerosol conditions, attached fraction etc, differ substantially from those occurring in homes.

The residential studies give more direct evidence on the public health hazard presented by radon than do the miners' studies. They also have much more detailed information on smoking, and it is now recognised that it is important to take account of the year-to-year variability in the radon measurements in evaluating these studies. We therefore conclude that the association with long-term average concentration observed in the pooled analysis of European studies is the best current basis for risk estimation. The 95% confidence interval on the proportional increase in the risk of lung cancer per 100 Bq m^{-3} increase in long-term average radon concentration in the home covers the range 5–31%. The central estimate is 16% per 100 Bq m^{-3}. However, there are reasons to believe that the true risk might be somewhat higher than this. Uncertainty around this estimate has been taken into account in the sensitivity analysis referred to in Chapter 6.

The risk estimate from the European pooling is compatible with those from the other two pooling studies, and is broadly consistent with those based on the data on miners. Darby et al (2005, 2006) reported that for miners exposed at less than 0.5 WL risks were in the range 19–30% per 100 Bq m^{-3} (see Table I4).

There is thus very substantial congruence between the evidence from miners' studies and from residential studies. We note, however, that there are the following two areas of discrepancy.

a The miners' studies suggest that the relative risks in non-smokers are substantially larger than in smokers, although the difference does not reach statistical significance; the residential studies suggest that they are similar.

b The miners' studies suggest that the risks vary with age at expression; again, the residential studies do not.

In both cases, we regard the case for variation as not proven and we have based our risk appraisal primarily on the evidence from the residential studies. However, the question of possible variation in risk with age is important, particularly so far as exposures of children are concerned.

Lung cancer is predominantly a disease of middle and old age. Children are generally thought to be somewhat more radiosensitive than adults (ICRP, 1991). However, epidemiological studies of lung cancer in miners suggest an excess risk which peaks within 20 years after exposure and then decreases. If this applies to radon, then the natural lung cancer incidence in those exposed to radon in childhood will be very low during the period in which the greatest proportional increase in risk is operative. The deleterious consequences of radon exposure in absolute terms would then also be very low.

Some direct evidence comes from the European pooling study which included 222 cases of lung cancer and 588 controls who were aged less than 45 years, so that the period of exposure that was considered in the analysis started at less than 10 years of age for them. For these individuals there was no evidence of any radon-related risk of lung cancer. In fact, their risk of lung cancer tended to decrease with increasing radon exposure, by 11% per 100 Bq m^{-3} (95% CI <–16–35%; Darby et al, 2006). Some further direct evidence comes from one of the miners' studies (Xuan et al, 1993). This was a cohort study of about 17,000 tin miners of whom 80% worked underground. About 40% of the latter were first exposed before 15 years of age. The excess relative risk per WLM tended to be somewhat higher for first exposure before age 20 years, but the pattern was not clear and weakened when correction was made for exposure to arsenic. Some analyses of the atomic bomb survivors, who were, of course, predominantly exposed to external gamma radiation, had previously suggested that the lung cancer risk is somewhat lower in those exposed before age 20 years than in older people. However, a more recent analysis (Pierce et al, 2003) suggested that this finding was an artefact due to variation in lung cancer rates between birth cohorts.

Direct evidence is thus sparse and other sources of information are ambiguous. It would be incautious to conclude that the radiological consequences of radon exposure in childhood were negligible. However, there is no direct evidence that children are much more sensitive than adults and it is clear that

lung cancer is very rare before age 35 years in all populations. This is an area where further research would be useful.

In summary, we judge that the magnitude of the risk from radon is such that exposure to 100 Bq m^{-3} for 30 years increases the risk of lung cancer by 5–31% with a central risk estimate of around 16%. The latter is likely to be an underestimate rather than an overestimate. The residential studies do not suggest that this relative risk varies with age, sex or smoking status. Despite some evidence from the miners' studies for a higher relative risk in non-smokers and for a relative risk which declines after the age of 50 years, on balance we prefer to follow the residential studies.

For a given residential radon concentration, the risk of lung cancer is much higher in smokers than in non-smokers so the absolute magnitude of the additional risk caused by radon is much larger in smokers than in non-smokers. The evidence from epidemiological studies of miners exposed to radon suggests that the main part of the risk expresses itself within the 30 or 40 years following exposure.

There is some evidence from the case–control studies that small cell lung cancer may be particularly associated with radon exposure. However, this is less clear in the miners' studies.

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Appendix J
Radon Doses to Organs Other than the Lung

Radiation doses may be received from external sources such as an X-ray set or from radionuclides within the body, also called internal emitters. It is often possible to make an accurate and direct measurement of the former. However, it is generally not possible to measure the doses from radionuclides to organs and tissues: instead these doses must be calculated. This is done in two stages. In the first, the number of radioactive decays in each body organ is estimated using the physical decay data and biokinetic models which describe where in the body the material goes and for how long it is retained. In the second stage of the calculation, each organ or tissue in which radioactive decays take place is considered to be a source of radiation. The energy absorbed in all the organs or tissues of interest, considered as 'targets', from decays in this source tissue is calculated. Summing the contributions from all source tissues gives the total dose to the target tissues.

The contribution to the dose in a target tissue per radioactive decay in the source is called the specific effective energy (SEE). The calculation of SEEs for alpha particles is simple if the radionuclide concerned is distributed throughout a tissue which is large compared to the range of the alpha particles (typically several tens of micrometres). It can then be assumed that all the energy is absorbed in the source tissue and none in any other. However, where the target tissue is small and/or the radionuclide is on its surface the situation is more complex. The calculation of doses then depends on the exact position of the sensitive target cells in relation to the radionuclide. This more complex situation applies to most of the calculations considered here.

The ICRP recommends that the assessment and control of radon exposures should be based on direct epidemiological observations rather than dosimetric modelling (see Appendix F). However, assessment of the risk from radon and its decay products to tissues outside the lung requires the calculation of doses for which ICRP models are used.

J1 Doses to stomach

Radon is soluble in water and ingestion can be an important route of exposure if high concentrations are found in drinking water (Hursh et al, 1965). If such water is ingested, the radon remains in the stomach for perhaps an hour before being passed to the small intestine where it is transferred to blood and is rapidly lost from the body (BEIR VI Committee, 1999). Because all the radionuclides concerned have short physical and/or biological half-lives, most of them decay in the stomach and dose to stomach dominates that to other organs, both for radon itself and for its decay products.

In contrast to the situation for inhalation, doses from ingestion of radon gas dominate those from ingestion of the decay products even if it is conservatively assumed that all the decay products are in equilibrium. This is because radon, with its much greater half-life, irradiates tissues for longer than do its

decay products. Once it leaves the stomach, ingested radon gas enters the bloodstream, passes through the liver and is lost from the lungs. The stomach receives much the highest dose, with minor contributions from liver and lung. Fatty tissues (eg red bone marrow and breast) also receive higher doses than other body organs because of radon's preferential solubility in fat. Other body organs receive doses which are typically two orders of magnitude lower than the committed effective dose.

A number of estimates of doses to different body organs resulting from ingestion have been made. The more recent calculations tend to use pharmacokinetic models in which data on blood flow through the various organs are combined with estimates of partition coefficients, ie the ratios of concentration of radon in the organ to that in the blood leaving the organ. The BEIR VI Committee (1999) and Khursheed (2000) have published the results of two such sets of calculations. The results are broadly compatible, although the BEIR VI dose to stomach, while still the highest to any organ, is lower than that calculated by Khursheed. This is because the BEIR VI calculation models diffusion of radon into the stomach wall, while that of Khursheed assumes that radon is held in the stomach until it is absorbed from the small intestine. The doses listed in Table J1 have been calculated using the method of Khursheed, whose results were more comprehensive. Full details of the calculation are in Section J4.

Table J1 lists ingestion doses for a notional individual consuming water containing 1000 Bq L^{-1}. This is the current UK Action Level for radon in private water supplies. Most radon concentrations are very much lower. Over a year, this individual would receive a stomach dose of about 50 mSv with a couple of millisievert to the small intestine. Doses to liver, red bone marrow and breast are about 1% of the stomach dose with other organ doses about a factor of ten smaller again.

J2 Doses to skin

Radioactive radon decay products in the atmosphere may deposit on surfaces in the environment including human skin. The alpha particles emitted will deliver a radiation dose to the outer layers of the skin in areas exposed to the atmosphere such as the hands and face. Radon decay products depositing on clothing will in general give rise to minimal skin dose as the alpha particle energy will be absorbed in the material.

The radiation effects in the skin are assumed to originate from target cells located in the basal layer, which lies within and at the base of the epidermis. It is possible that, where the skin is thin, these sensitive cells lie within the range of alpha particles from radon decay products with consequent risk of skin cancer. The main potential risk of low doses of ionising radiation on skin is the development of non-melanoma skin cancer. However, the risk per unit radiation dose is very much lower than for other organs, as the skin is much less sensitive to radiation. Moreover this type of skin cancer has a high survival rate, although this does not imply that the effects are negligible.

Radon skin dosimetry is based on experimental measurements of the amount of radon plate-out on to surfaces, and calculation of the dose to the basal layer of skin, making various assumptions about skin anatomy. Most experimental measurements on radon plate-out have been carried out *in vitro* by measuring deposition of radon decay products on the walls of large chambers containing known radon

concentrations (George et al, 1983; Miles, 1986). More recently the dose to the skin surface has been measured *in vivo* using personal 'wristwatch' dosemeters attached to the skin surface (Eatough et al, 1999). The aim of the calculations is to estimate a dose equivalent to the skin corresponding to a given ambient concentration of radon in the atmosphere (Eatough and Henshaw, 1992). There is a large range of measured values of plate-out and also large variation in the parameters used in the dose calculation. Therefore in addition to the average dose per unit concentration, it is also usual to quote a range corresponding to the extreme values of the parameters used in the calculation.

There are several factors which have an important influence on the dose received by the skin.

a Particle velocity Particles with higher velocity have a higher probability of depositing on surfaces. The unattached fraction of radon decay products diffuses much more rapidly than the attached fraction and therefore has a much higher proportion of plate-out on the skin.

b Air movement This has two effects on deposition. First, it increases particle velocity thus increasing plate-out, as described above. Second, if radon decay products are deposited on the skin from still air, a boundary layer will be set up close to the skin in which the decay products have been depleted by plate-out. If there is air movement, this depleted layer will constantly be replaced with air containing a fresh supply of decay products. This means that estimates of skin doses that are based on measurements or calculations of deposition under still conditions are likely to be lower than those incurred in reality. Both these effects tend to increase the outdoor radon skin dose compared to that received indoors for the same radon decay product concentration.

c Rainfall It has been suggested that doses to skin from radon decay products may be enhanced by rainfall (Paatero, 2000; Henshaw et al, 2001). This is probably due to the rapid diffusion of unattached decay products becoming quickly incorporated into water droplets (Blaauboer and Smetsers, 1997). Measurements using plastic alpha etched-track detectors suggest that the radon decay products contained in raindrops depositing on skin might give rise to high dose rate (Henshaw et al, 2001). The practical implications of these observations on skin dose are not easy to assess, as most people are averse to rain and try to minimise the amount of wetted skin.

d Proximity to electrical power lines Fews et al (1999) have studied the effect of high voltage power lines on deposition of radon decay products, combining experimental measurements and a theoretical analysis. The authors observed enhanced deposition within the vicinity of power lines and suggested a possible mechanism. The 50 Hz oscillating electric field from the power lines causes charged decay products to oscillate, particularly when perturbed by a conducting human body. In doing so the decay products may come into contact with skin in a way that they would not have done in the absence of the field. This process interacts synergistically with the effect of air movement discussed above. Fews et al (1999) argue that the mechanism is important both to the small unattached fraction and to the attached fraction, and therefore that skin doses may be increased by a factor of between 1.2 and 2.0 as a result of living in the vicinity of power lines. These conclusions have been questioned by Swanson and Jeffers (2000), who argued that the effect has been overestimated because the deposition of the attached fraction of decay products is little affected by AC fields, buildings and trees screen the electric fields in most situations and in practice people spend very little of their time outdoors under power lines.

e **Location of deposition with respect to sensitive cells** The location of deposition on the skin surface also affects the dose received. In many parts of the body the alpha particle energy is harmlessly absorbed in the dead outer layers of the skin. However, where the skin is thin – for example, on the face – the alpha particles may be able to reach the sensitive basal cells where it is thought that the cancers originate. The depth of the sensitive cells is a crucial factor in assessing the dose from radon decay products. Charles (2004) has argued that the sensitive cells may lie too deep to receive any significant dose.

The BEIR VI Report quoted an absorbed dose rate to exposed areas of skin of 125 μGy y^{-1} as a result of domestic exposure to the average UK radon concentration of 20 Bq m^{-3} (BEIR VI Committee, 1999). This is based on the work of Eatough and Henshaw (1992) who expressed their results in terms of the dose equivalent to skin as 2.5 mSv y^{-1} (range 1.7–17). Some recent studies using measurements from personal dosemeters suggest that considerably higher skin doses, of 23 mSv y^{-1} (range 8.8–31.5) (Eatough et al, 1999) and 18 mSv y^{-1} (Fews et al, 1999), are received as a result of the average UK exposure. The main reason for the large increase in the more recent dose estimates is that the earlier work of Eatough and Henshaw (1992) considered only indoor exposure, whereas the later studies considered both indoor and outdoor exposure. Skin deposition is much higher outdoors due to the greater air movement and also the possibility of wet deposition from rainfall, and this can significantly increase the overall dose, even allowing for the relatively small proportion of time spent outdoors by most people. For people who spend a greater amount of time outdoors, the implication is that doses would be even higher. However, as we have noted above there are significant uncertainties in the position of the sensitive cells and quantification of any risk is difficult.

J3 Doses to haematopoietic tissue

As noted above, radon is more soluble in tissues with a higher fat content (Nussbaum and Hursh, 1957). Whether the primary route of intake is inhalation through the lungs or ingestion, radon gas will enter the bloodstream and irradiate body organs. Fat itself is not thought to be radiosensitive. However, Richardson et al (1991) pointed out that red bone marrow has a reasonably high fat content and this also receives a relatively high dose.

Richardson et al (1991) calculated doses to haematopoietic tissues from inhalation of radon and its decay products. They modelled red bone marrow as a mixture of spherical fat cells and haematopoietic tissue. The distribution of fat cells was taken to vary with age and across the body. We note that, in contrast to the ICRP model in which retention of short-lived decay products in lung is assumed to be sufficiently long that only a very small fraction escapes, Richardson et al considered that short-lived radon decay products might be absorbed from the lung and contribute to dose to red bone marrow. However, even under the assumptions of Richardson et al, the majority of the dose to haematopoietic tissue would be from radon gas rather than from its short-lived decay products or polonium-210. Blood will also receive a dose from decay products which are absorbed from the lung and from radon gas; the doses recorded for muscle in Table J1 give an indication of their magnitude.

Khursheed (2000) calculated doses to red bone marrow from inhalation of radon and its decay products. These calculations assumed a homogeneous mixture of fatty source tissue and haematopoietic target tissue. The assumption of homogeneous mixing lends an element of conservatism because it does not allow for self-absorption of alpha energy within the source fat cell. However, the results of Khursheed are not dissimilar to those of Richardson et al. Khursheed's calculations are the basis for the calculations given in Table J1. As described above, this table includes results for two alternative standard assumptions about the solubility of radon decay products, 'Type F' and 'Type M', the names coming from Fast or Moderate solubility. For inhalation of radon with its decay products, the dose to red bone marrow from radon is perhaps a factor of two higher than those from the radon decay products if Type F behaviour applies and an order of magnitude higher than for Type M behaviour. Dose to red bone marrow remains, however, much less than the dose to lung. For an individual breathing air containing 200 Bq m^{-3} of radon the dose to red bone marrow is about 0.7 mSv per year. This is comparable with the annual dose from cosmic and natural gamma rays. It is much less than the dose to lung at this radon concentration.

J4 Comparison of doses to different organs

The radiation dose received by an organ gives an indication of the risk caused by an exposure. It is therefore interesting to compare doses calculated for different organs and for different modes of exposure. Kendall and Smith (2002) have presented doses to various organs and tissues (and to the fetus) from inhalation and from ingestion of radon gas and its short-lived decay products. Doses to the skin from external radiation by radon decay products were also considered. The results presented here are based on that publication. The calculations are based on a number of assumptions which are outlined below. However, the original publication should be consulted for full details.

Table J1 presents doses from ingestion of radon decay products and from radon itself. These data are from the ICRP compendium of dose coefficients (ICRP, 1998) and from Khursheed (2000), respectively. The table gives estimates of annual doses by inhalation to an individual exposed to an atmosphere containing 200 Bq m^{-3} of radon gas and annual doses by ingestion to an individual who drank water containing 1000 Bq L^{-1}. These are the respective current UK Action Levels for radon in the air in dwellings and for radon in private water supplies. Estimates of annual dose to the skin from radon decay products are also given in Table J1. Doses from different concentrations of radon may be obtained by linear scaling. Typical UK doses would be lower than those given in Table J1 by about a factor of ten for radon in air and perhaps a factor of 1000 for radon in water. If water is boiled before being consumed, most of the radon is lost, and this must be considered in dose assessments.

Kendall and Smith (2005) did not give estimates of doses to the ET airways from inhaled or ingested radon gas, nor from ingested radon decay products. There are two components to these doses, from activity absorbed into body tissues and, for radon gas, dose from radon within the airways. Systemic activity will give a dose to the ET airways very similar to that from muscle. Results presented by Marsh et al (2005) and by Bailey et al (1996) allow estimates of the doses from radon gas in the airways to be made: these are larger than those from systemic activity.

TABLE J1 Annual doses (mSv) to organs and tissues from inhalation and ingestion of radon and its decay products. The doses shown are committed equivalent doses to organs and committed effective doses. The annual intake of air for an adult is taken to be 7300 m^3 and the annual water intake to be 600 litres (NRPB, 2000). The dose to the fetus from radon gas is taken to be that to muscle over nine months

	Exposure to air containing radon at 200 Bq m^{-3} with radon decay products (F = 0.41)[(i)]			Ingestion of water containing 1000 Bq L^{-1} of radon with decay products[(ii)]	
Organ/tissue	Decay products Type F	Decay products Type M	Radon gas	Decay products	Radon gas
Lung	35.8	159	1.2	0.01	1.26
ET airways[(iii)]	44.5	70.9	0.42	0.02	0.04
Stomach	0.19	0.08	0.06	1.15	50.4
Small intestine	0.17	0.05	0.06	0.51	2.6
Colon	0.16	0.02	0.05	0.13	0.1
Red bone marrow[(ii)]	0.28	0.03	0.65	0.03	0.66
Bone surfaces	1.48	0.17	0.03	0.12	0.03
Liver	0.43	0.05	0.09	0.04	0.57
Breast	0.15	0.02	0.42	0.01	0.44
Kidney	5.20	0.54	0.05	0.25	0.05
Gonads	0.15	0.02	0.05	0.02	0.05
Brain	0.15	0.02	0.06	0.01	0.06
Bladder	0.21	0.02	0.05	0.02	0.05
Muscle	0.15	0.02	0.05	0.02	0.05
Effective dose	**5.30**	**19.7**	**0.28**	**0.17**	**6.00**
Fetus	0.06	0.01	0.04	0.01	0.06
Skin[(iv)]	25	25	–	–	–

Notes

(i) Based on Kendall and Smith (2002).

(ii) Based on ICRP (1998) and Khursheed (2000).

(iii) ET airways are the extrathoracic airways, ie nose, mouth and throat.

(iv) Dose by deposition of decay products, based on Eatough and Henshaw (1992), quoted by the BEIR VI Committee (1999). The skin dose is not affected by lung absorption Type.

The ICRP has recommended that the usual model for the human respiratory tract should not be used for calculating doses from radon decay products (ICRP, 1993). Nevertheless, lung doses are needed to put doses to other tissues into context. For want of a generally accepted model, Table J1 presents calculations of doses from inhalation of short-lived decay products under two alternative standard assumptions about their solubility, known as Type F and Type M behaviour. The range of doses for Type F and Type M may be taken as an indicative spectrum of doses from radon decay products.

It is assumed that 10% of the activity of radon decay products is associated with the unattached fraction. It is further assumed that the activities of the radionuclides

Radon-222 : Polonium-218 : Lead-214 : Bismuth-214

are in the ratio of

1.0 : 0.9 : 0.45 : 0.225

This corresponds to an equilibrium factor, *F*, of 0.41, typical of domestic, and many occupational, conditions. The annual committed effective dose is then about 5 mSv if Type F behaviour is assumed or about 20 mSv for Type M.

The doses from radon decay products to organs outside the respiratory tract depend on the rate at which material is cleared to blood. This clearance is much faster for Type F material than for Type M, and the former give larger doses to organs outside the respiratory tract. Nevertheless, short-lived radon decay products which are cleared from lung to blood will usually decay in blood before entering organs and this tends to decrease their radiological significance. It is informative to consider doses to the kidney, the most exposed such organ. For Type M material the dose to the kidney is always at least an order of magnitude lower than the committed effective dose and two orders of magnitude lower than the lung dose. For Type F material, the kidney dose is relatively larger, but still always substantially lower than that to lung. It may be noted that the doses to the extrathoracic part of the respiratory tract are also higher than those to the kidney and may be higher than those to the lung as a whole.

These doses from radon decay products may be compared with those which would result from breathing radon gas, in the absence of its decay products. The calculated committed effective dose from radon gas is about 0.3 mSv, an order of magnitude lower than that from decay products if they display Type F behaviour and two orders of magnitude lower than for Type M.

J5 Comparison of doses received from inhalation

It is interesting to consider the relative magnitude of doses to different organs and tissues from inhalation of radon gas. As with the decay products, the lung is the organ receiving one of the highest doses, in this case largely because of the contribution from decays in the air within the lung. However, radon is more soluble in tissues with a higher fat content. Fat (which is not thought to be radiosensitive) receives the highest dose of all. Red bone marrow has a reasonably high fat content and this also receives a relatively high dose, as does the female breast. The doses to these two tissues are perhaps a factor of

two higher than those from the radon decay products if Type F behaviour applies and an order of magnitude higher than for Type M. They remain, however, much less than the doses to lung.

A fetus can take up material from the mother's bloodstream via the placenta. Elements vary in their ability to cross this placental barrier. It has been recommended by the ICRP (2001) that the concentration of lead in the fetus is taken to be equal to the concentration in the mother regardless of when the intake occurred; for polonium and bismuth the concentration ratio (fetal : maternal) is taken to be 0.1, indicating modest discrimination against these elements by the placenta. However, for all radionuclides considered here the dose to the fetus is very similar to that to the maternal muscle, and this assumption is held to be true for intakes of radon too.

J6 Comparison of doses received from ingestion

In contrast to the situation for inhalation, doses from ingestion of radon gas dominate those from ingestion of the decay products even if it is conservatively assumed that all the decay products are in equilibrium. This is because radon, with its much greater half-life, irradiates tissues for longer than do the decay products. Once it leaves the stomach, ingested radon gas enters the bloodstream, passes through the liver and is lost from the lungs. The stomach receives much the highest dose, with minor contributions from liver and lung. Fatty tissues (eg red bone marrow and breast) also receive higher doses than other body organs because of radon's preferential solubility in fat. Other body organs receive doses which are typically two orders of magnitude lower than the committed effective dose.

As discussed in relation to inhalation, the fetus does not preferentially take up radon from the bloodstream and would receive a dose similar to that to muscle.

J7 Doses received by children

Kendall and Smith (2005) have also discussed how doses to ten-year-old children and one-year-old infants would differ from those to adults. For inhalation and ingestion, the general pattern of doses to organs is broadly similar to that in adults. Much the largest doses are received by the organ of intake (respiratory tract and stomach, respectively). Otherwise, tissues with higher fat content tend to receive somewhat higher doses from radon gas than other tissues. Dose coefficients (dose per unit intake factors) for children are generally larger than those for adults. However, total annual doses are more similar across the age groups because of smaller intakes of air and water by children. Radon decay products deposited on skin may be able to induce skin cancer. However, the location of the sensitive cells is not known with certainty and they may lie too deep to receive significant dose. If they are irradiated, it is likely that doses to children would be larger than those for adults.

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Appendix K
Cancers Other than Lung and Other Possible Health Effects

In this appendix the epidemiological evidence relating to conditions other than lung cancer, including haematological malignancies, childhood cancers, gastric cancer, skin cancer and other conditions, are considered. The appendix draws on epidemiological and related evidence relating to risks from exposure to radon gas, for a wide range of conditions. In general the available evidence is sparse. For this reason a more systematic approach has been adopted than in other appendices, to enable the reader to find the information more easily.

Physical and biological processes mean that small concentrations of radon and its decay products will be established in all organs and tissues. This means that, when the radionuclides decay, all organs and tissues will receive some dose. These doses can be calculated using mathematical modelling and the results of such calculations have been given in Appendix J of this report.

However, in most cases the calculated doses to organs and tissues other than the lung are too small for there to be any expectation that any effects, such as an increased risk of cancer, could be detected. This is because the probability of an effect of radiation exposure decreases as the dose decreases. A large and well-designed epidemiological study of an appreciable radiation risk – for example, lung cancer induced by radon decay products – may find a statistically significant association overall between exposure and risk (see Appendices H and I). However, if attention is concentrated on successively lower dose subsets of the data it is inevitable that a point will be reached at which no statistically significant association can be seen because of random variations in effects. Indeed, such are the difficulties in establishing really large studies that even the entire data of a well-conducted epidemiological study, or a pooled analysis of several studies, may not establish a statistically significant association even though there is a real underlying causal relationship. Therefore, for cancers other than lung cancer, it is necessary to consider all sources of evidence when making judgements on the magnitude of the possible risks from radon.

Ever since the aetiological role of radon as a cause of lung cancer in highly exposed mining populations was elucidated in the 1960s, there has been speculation that exposure to radon and its decay products might increase the risk of certain malignancies at other sites (see, for example, Axelson and Forastiere, 1993). In this appendix we consider the evidence for associations between exposure to radon and its decay products and a number of types of cancer other than lung cancer and some non-malignant conditions.

Animal and other experimental evidence supporting a biological effect on tissues other than the lung is weak and contradictory (Bridges et al, 1991; Albering et al, 1992; Lloyd et al, 1993; Cole et al, 1996; Ruttenber et al, 2001; Smerhovsky et al, 2001).

As described in Appendix G, animal studies on the effects of radon exposure have been carried out, notably at three main laboratories: Harwell, UK (Collier et al, 2005), Commissariat à l'Energy Atomique, France (CEA) (Monchaux et al, 1994) and the Pacific Northwest National Laboratory, USA (PNNL) (Cross and Monchaux, 1997). The principal finding of all the animal studies has been that of lung tumour

induction (see Appendix G). In addition, at high exposure levels non-malignant lung damage reduced lifespan at both the CEA and PNNL. In most animal studies all tumours and other health effects, whether occurring in the lung or not, are investigated and reported. The incidence of tumours other than those of the lung in control animals is generally considerably higher than that of lung tumours and this reduces the power of the studies. Kidney tumours have also shown increased incidence, significantly so for some studies from two of the three laboratories. Increases in leukaemia incidence following exposure to radon and its decay products have not been observed in experimental animals by any of the laboratories. Various other tumours have been observed at slightly elevated levels, but not consistently across the three laboratories. However, combined analyses of the CEA, Harwell and PNNL data has indicated that exposure to radon and its decay products results in significant lethal effects in addition to lung tumours (Kaiser et al, 2004).

Cytogenetic studies of human peripheral blood lymphocytes suggest that structural chromosomal aberrations in radon-exposed miners are much higher than in an unexposed population (Bilban and Jakopin, 2005) and that chromosomal aberrations persist over many years once radon exposure ceased (Meszaros et al, 2004). These observations support the possibility that conditions other than lung cancer might be a long-term consequence of radon exposure.

K1 All-cause mortality other than lung diseases

As noted above, some studies exist of the effects of radon on human lymphocytes. One such study has shown an elevated frequency of various cytogenetic aberrations amongst radon-exposed miners in the Czech Republic compared with that found in the general population (Smerhovsky et al, 2001). This might suggest that various malignancies could be implicated as risks to those heavily exposed to radon.

K1.1 Ecological studies

An ecological study from the Japanese spa of Misasa with high radon concentrations in its water supply (Mifune et al, 1992) showed lower overall standardised cancer mortality ratios (SMR) than expected, albeit based on less than 100 deaths. An ecological study from a uranium mining and milling area of Texas (Boice et al, 2003) showed no overall excess of cancer when contrasted with deaths in four 'control' counties, with the relative risk of death being 1.0 (95% CI 0.9–1.1) based on 1223 deaths. No measures of exposure to any type of radiation accompanied these studies.

K1.2 Cohort studies

Several cohort studies have given results of 'overall' rates of death and 'all-cancer' deaths as part of their analyses. Hodgson and Jones (1990) studied a cohort of UK tin miners from 1941–1986 and found that all causes of death other than lung cancer, silicotubercolosis and silicosis were elevated with an SMR of 139.8 for those miners with exposures of over 30 years. This was a non-significant excess and there was no dose-related linear trend (in contrast to the other named conditions).

A cohort of Misasa residents showed no overall cancer excesses (Ye et al, 1998).

However, a study of North Swedish iron-ore miners (Darby et al, 1995a) showed an elevated mortality from all cancers other than lung cancer of 1.21 (95% CI 1.03–1.41) based 162 deaths. This excess was not correlated with radon exposure. In the combined analysis of 11 studies of underground miners who were occupationally exposed to high concentrations of radon, all of which had statistically significant excesses of lung cancer that were related to cumulative radon exposure (Darby et al, 1995b), the mortality for all cancers other than lung cancer was not significantly elevated in exposed miners for any time period (SMR 1.01, 95% CI 0.95–1.07) nor in those exposed for over 10 years, based on 1179 and 1063 deaths, respectively.

K1.3 Conclusions

Overall there is no available evidence to suggest that radon exposures contribute directly to excess mortality other than that due to lung cancer.

K2 Childhood cancer

Estimates of bone marrow dose suggest that there may be a small proportion of childhood leukaemia as a result of exposure to radon and related sources, but an extremely powerful study (ie with very large case numbers) would be needed to detect it.

K2.1 Ecological studies

The association between childhood cancer and indoor radon exposure has been investigated in several ecological studies. Some of the studies were from the UK and involved partially overlapping datasets.

In England and Wales, Alexander et al (1990) considered the correlation between acute lymphoblastic leukaemia (ALL) in children aged up to 14 years and exposures to radon in households for 22 administrative counties during the period 1984–1988. The correlation coefficient between county standardised morbidity ratios and the geometric means of the radon concentration was 0.65 ($p < 0.005$). The data were based on location at diagnosis, but previous dwellings were not taken into account. Potential confounding by socioeconomic variables has been discussed, but its effect is unclear (Lucie, 1990; Wolff, 1991).

Muirhead et al (1991, 1992) considered data on leukaemia and non-Hodgkin's lymphoma in children during the period 1969–1983 in 459 county districts in England and Wales and regional districts in Scotland. Average indoor radon concentrations and gamma dose rates were used. With data aggregated into counties, the regression coefficient for indoor radon concentration was positive and that for indoor gamma dose negative, both being of borderline statistical significance. However, when analysis was made by district within each country, the coefficient for radon was negative and that for indoor gamma dose was positive. The differences in the trend with indoor radon and indoor gamma dose between the

analysis based on counties and that based on the smaller districts within them suggest that the between-county analysis was affected by geographical confounding factors.

In Devon and Cornwall, Thorne et al (1996) found that the incidence of childhood malignancies of all types was lower in postcode sectors with radon exposures of 100 Bq m^{-3} or more, compared to sectors with lower exposure, but this difference was not statistically significant. When specific types of childhood cancer were considered, the incidence of neuroblastoma was statistically significantly higher in the sectors of higher exposure than in those with lower exposure. This may be a chance finding in view of the multiple significance tests performed. Furthermore, the incidence rate of neuroblastoma in the sectors of higher exposure was similar to that for the rest of the UK (Parker and Craft, 1996).

Gilman and Knox (1998) studied the geographical distribution of the place of birth of children who died from cancer under the age of 16 years in Great Britain during the period 1953–1981 in relation to measurements of indoor radiation. The rate ratio of cumulative mortality due to leukaemia and lymphoma for twice the mean value of radon exposure compared with the mean value (27 Bq m^{-3}) was 1.06 (95% CI 0.99–1.12). The corresponding rate ratio for solid cancers was 1.08 (95% CI 1.02–1.15). This analysis was adjusted for socioeconomic status, birth density and birth year. There was no confounding by measured indoor and outdoor gamma radiation levels or by a different measure of socioeconomic status.

Henshaw et al (1990a,b) found a correlation between the incidence of total childhood cancer in 13 countries and the population averaged arithmetic mean radon concentrations, weighted according to the number of measurements, of 0.78 ($p < 0.01$). The correlation coefficient was not reduced, but became less significant, when attention was restricted to the six countries for which the data were more reliable (Butland et al, 1990).

Collman et al (1991) examined cancer death rates among children aged under 15 years during the period 1950–1979 in counties within North Carolina ranked according to estimates of average groundwater radon concentration. Counties were grouped into low, medium and high thirds. For total childhood cancer the relative risks compared with the lowest third were 1.16 (95% CI 1.05–1.28) for the middle third and 1.23 (95% CI 1.11–1.37) for the highest. The corresponding relative risks for childhood leukaemia were 1.26 and 1.33. The authors noted that some confounding factor might be responsible for this positive trend, but they were unable to identify one.

Hoffmann et al (1993) studied the incidence of childhood malignancies during the period 1970–1989 around a uranium processing plant in the Rheinland-Pfalz state of Germany. There appeared to be an excess of childhood leukaemias within 5 km of the plant (7 cases diagnosed at ages under 20 years observed versus 2.3 expected), but not of solid tumours of childhood. The subsoil of the region within 20 km of the plant contained high concentrations of natural uranium ore, and there were high levels of external gamma radiation and high concentrations of indoor radon. However, the geographical distributions of these exposures did not appear to account for the distribution of leukaemia cases.

Kohli et al (2000) examined the relationship between ground radon concentrations and childhood cancer diagnosed up to 1995 in children born in Östergötland (Sweden) in 1979–1992. Ground radon exposure was considered in two different ways: either by level of exposure in the area in which the child was born

or by level of exposure on the basis of each area of residence during the study period. No association was found between total childhood cancer and either of these measures of exposure. However, there appeared to be a positive association between ALL and each of these measures.

Evrard et al (2005) conducted an ecological study of associations between indoor radon concentrations and childhood leukaemia incidence in France. Over 4000 cases were registered during the study period (1990–1998). Analysis was in terms of 348 national geographical units (*zones d'emploi*). The investigators found a significant association between childhood acute myeloid leukaemia (AML) and mean indoor radon concentration but no association with ALL.

K2.2 Case–control studies

In a study of childhood cancer diagnosed in Great Britain during the period 1991–1996, radon measurements were obtained from the home at diagnosis of 2226 cases (50.5% of those eligible) and 3773 controls (31.46% of those eligible) (UKCCS Investigators, 2002). The controls were matched with cases on age, sex and region of residence. Radon measurements were made (over a period of six months) using etched-track radon detectors, in virtually all homes both in the bedroom of the index child and in the living room. The arithmetic mean radon concentration in homes measured was 24.0 Bq m^{-3}, with the mean in cases (21.1, standard deviation, SD, 31.0) being slightly lower than that in controls (25.5, SD 42.4). There was an inverse association between childhood cancer risk and radon concentration. This was apparent both overall and separately for each diagnostic group (ALL, other leukaemias, non-Hodgkin's lymphomas, Hodgkin's disease, central nervous system tumours and other solid tumours). Analysis of the relationship between housing features and radon concentrations suggested that differences in housing characteristics between cases and controls could account for differences in radon concentrations between their homes. However, adjustment for these housing characteristics had little effect on the relationship between childhood cancer and radon concentration.

As part of a large study of ALL in children in the USA, radon measurements in homes occupied by the families in the five-year period prior to diagnosis were obtained for 505 cases and 443 controls (Lubin et al, 1998). For children aged under five years at diagnosis, efforts were made to measure all homes in which the subjects had resided for at least six months, while for older children, homes in which the subjects had resided for a year or longer within the five-year reference period were eligible for measurement. Subjects were included provided that the measured homes covered at least 70% of the child's life during the reference period. Time weighted average radon concentration within the exposure assessment period for each subject was calculated, weighted by length of residence. The mean time weighted average radon concentration was 68.7 Bq m^{-3} for cases and 75.7 Bq m^{-3} for controls. Analysis was made both for the 281 individually matched case–control pairs, with adjustment for sex, and also for all 505 cases and 443 controls for whom radon measurements were obtained, with adjustment for age and sex. Both matched and unmatched analyses showed no association between ALL and radon exposure. Subgroup analysis was also made by age. In the subgroup aged under two years at diagnosis, there was a decrease in risk with increasing radon concentration that was not statistically significant. This may be a chance finding, but a similar pattern was observed in a case–control study of childhood AML drawn from the same target population (Steinbuch et al, 1999) (see below).

In a parallel study of AML, radon measurements were obtained for 173 cases with AML or myelodysplastic syndrome diagnosed under the age of 18 years, and 254 controls matched with cases on age, ethnic group and geographical area recruited by random digit dialling (Steinbuch et al, 1999). In contrast to the study of ALL, measurements were only made in the residence occupied by the child at the time of diagnosis or the reference date. Only unmatched analyses were made. The mean time weighted average radon concentration was 49.8 Bq m^{-3} for cases and 56.0 Bq m^{-3} for controls. No association between childhood AML and time weighted average radon concentration was observed. Analysis stratified by age (less than two years, or two or more years) was made because it was considered that childhood environmental exposures are less likely than pregnancy and parental factors to affect the risk for infant myeloid leukaemia. Among children aged under two years at diagnosis, there was an inverse association between radon concentration and the risk of AML, whereas among those aged two or more years, the estimated relative risk was increased among those with a higher radon exposure.

In Lower Saxony (Germany), radon measurements from the room in which the child stayed most of the time were obtained for 82 cases of leukaemia, 209 matched controls for the leukaemia cases and 82 cases with CNS tumours, retinoblastoma, neuroblastoma or rhabdomyosarcoma (Kaletsch et al, 1999). The 90th percentile of all measurements was set as a cut point to distinguish between higher and lower exposures: this was 70 Bq m^{-3}. There was no association with leukaemia (odds ratio 1.3, 95% CI 0.3–5.3). The odds ratio for solid tumours associated with high levels of radon exposure was 2.6 (95% CI 1.0–7.1), adjusted for degree of urbanisation, socioeconomic status, age and gender. The elevation in odds ratio was mainly accounted for by CNS tumours (6 cases in the higher category of exposure and 35 in the lower).

Maged et al (2000) identified 240 potentially eligible children aged 2–14 years with ALL diagnosed in the period 1996–1998 in a single hospital in Cairo (Egypt). Exclusion criteria included prenatal maternal and postnatal exposure to radiation, and postnatal exposure to chemicals of known carcinogenic potential. Two healthy controls were 'selected randomly by private communication' and matched with cases on age and sex. Cases and controls had resided in Cairo since birth. Radon detectors were placed in the bedroom of the index child and the living room for three months. There was a positive association between ALL and radon concentration. Given the small case referent numbers and the unmatched design, some doubt must be cast on this finding.

In Östergötland (Sweden), concern among parents of children with cancer regarding high radiation levels in their homes led Stjernfeldt et al (1987) to study the gamma and alpha radiation levels in the homes of a group of children. Cases comprised 28 children with all types of childhood cancer who had been regularly seen at the university paediatric unit between 1980 and 1984. Measurements could be made in all of the dwellings in which the child with cancer had lived from the time of conception to diagnosis for 15 cases. For each of these, a playmate matched on age and sex was chosen as a control. There was no appreciable difference between the groups in gamma radiation or radon decay product exposure. For the other 13 children with cancer, access to all dwellings in which they had lived was not possible, and measurements were only done at their current residence. On the basis of current residence, the estimated median cumulative radon decay product exposure was similar to that of the other groups.

Wakefield and Kohler (1991) reported a study in which indoor radon concentrations over the same three-month period were assessed in the bedroom and living room of dwellings for a group of children in the Wessex Health Authority Region. The cases comprised 42 children with cancer diagnosed in a three-year period, 45% of whom had ALL. There were 39 controls, matched with cases on age and area of residence. Cases who had moved house during the year prior to diagnosis and controls who had moved house in the year prior to the diagnosis of the matched case were not recruited. The mean indoor radon concentrations were similar between the groups.

Pobel and Viel (1997), in a case–control study of childhood leukaemia in the vicinity of the nuclear reprocessing plant at La Hague, France, found an increased risk of leukaemia associated with a surrogate for radon exposure. The relative risk per year of residence in homes reported to be made of granite material or built on granite ground was 1.18 (95% CI 1.03–1.42). The presence of granite in the building materials could be verified for the current home but not previous homes. No association was observed with the length of residence in homes with double-glazing, which might have been expected to lead to higher concentrations of radon gas within homes.

A small case–control study of osteosarcoma in people aged under 25 years has been carried out to identify risk factors in an area of Cornwall where a high incidence had been noted (Wright and Pheby, 2006). An association with radon concentration was reported. However, the study included only six cases, and they were identified via word of mouth and the media. Furthermore, the controls were self-selected volunteers and only short-term radon measurements over a period of 24 hours were made. These methodological limitations preclude any conclusions being drawn from this study.

Raaschou-Nielsen et al (2008) conducted a study of associations between inferred radon levels and childhood leukaemia (1153 cases with 2 controls for each case), CNS tumours (922 cases with 3 controls per case), and malignant lymphoma (325 cases with 5 controls per case) diagnosed in Denmark between 1968 and 1994. Cases and controls were selected from registries and the study was free of the selection biases that may be introduced by incomplete participation. Radon exposures of cases and controls were estimated using a model based on geographical region, soil type and house characteristics rather than by direct measurements in the homes in question. The authors acknowledged that this was a weakness, because, although their model gave good general predictions of radon levels, the actual radon concentrations in adjoining and apparently similar dwellings can differ substantially. Inferred cumulative radon exposure was associated with risk for ALL (860 cases), with rate ratios of 1.21 (95% CI 0.98–1.49) for levels of 260–890 Bq m^{-3} y and 1.63 (95% CI 1.05–2.53) for exposure to more than 890 Bq m^{-3} y, when compared with the lowest category, less than 260 Bq m^{-3} y. There was no association with CNS tumours or lymphoma, either overall or for specific subtypes.

K2.3 Conclusions

Large case–control studies in Great Britain and the USA and a somewhat smaller study in Germany do not suggest a relationship between childhood leukaemia overall or ALL and indoor radon concentrations. By contrast, a positive association between ALL and radon concentration has been reported in a much smaller study in Egypt; it is difficult to exclude bias as a possible explanation of this finding as radon

measurements could only be made for about one-fifth of cases eligible for inclusion in the study, and details of control selection are unclear. A large case–control study in Great Britain does not suggest any relationship between radon and other forms of childhood cancer, and in particular did not find an increased risk for CNS tumours that had been observed in an earlier small German study. We did not give weight to the ecological studies in drawing conclusions for the following reasons. First, the characteristics of individual dwellings have a substantial effect on radon concentrations in homes, so concentrations in an area may be poor indicators of individual exposure. Second, there is evidence that geographical confounding factors can influence the direction of the relation between childhood cancer and areal measures of radon concentration (Muirhead et al, 1991,1992).

K3 Adult haematological malignancies

Epidemiological evidence supporting the suggestion that radon may play an aetiological role in certain haematological malignancies diagnosed in adults comes largely from geographical correlation studies (Henshaw et al, 1990a,b; Collman et al, 1991; Mifune et al, 1992; Eatough and Henshaw, 1993; Miller et al, 1993; Viel, 1993).

Detailed epidemiological studies relating individual radon exposure to disease onset have failed to confirm the hypothesis that naturally occurring radon gas is a material cause either of haematological malignancies in general, or of leukaemia in particular (Hodgson and Jones, 1990; Darby et al, 1995a,b; Forastiere et al, 1998; Law et al, 2000; Auvinen et al, 2002; Toti et al, 2005).

K3.1 Ecological studies

Henshaw et al (1990a,b) using international cancer registry data and national estimates of mean household radon concentration concluded that for all myeloid leukaemias, 6–12% of the incident cases in the UK could be attributable to radon exposure to the bone marrow. A similar analysis by Eatough and Henshaw (1993) using English county data for mean radon concentration and monocytic leukaemia data (ICD 206) also found a significant correlation.

However, a study from a high radon area in Japan found no correlation with adult leukaemias (Mifune et al, 1992).

Miller et al (1993) using data from 18 Canadian cities found no correlation with AML data for all ages. Viel (1993) reported on an analysis of 41 French administration areas. The analysis of adult ALL showed no correlation with mean radon concentrations. However, a positive correlation was noted for adult AML deaths with an odds ratio of 1.41 (95% 1.23–1.62). This was based on over 1000 AML deaths.

In a study from North Carolina (Collman et al, 1991), no correlation was observed between leukaemia mortality by case residences and radon-222 concentrations in drinking water.

K3.2 Cohort studies

In the pooled analysis of over 60,000 underground miners, each of whom was followed for an average of almost 17 years, no significant excesses in the numbers of deaths from any of the haematological malignancies examined (ICD-9 200–208) was found, when all of the available data were analysed (Darby et al, 1995b). Mortality from leukaemia (ICD-9 204–208) was, however, increased ($p \leq 0.01$) when the analysis was restricted to the 10 years following first employment. The excess was not due to any particular subtype and no discernable heterogeneity between studies was detected. No evidence of an association with cumulative radon exposure in any time period was found, and the authors concluded that the excess was unlikely to be due to radon (Darby et al, 1995b).

However, in a case–cohort study of 23,043 Czech uranium miners there was a significant association between cumulative radon exposure and both all leukaemias and chronic lymphocytic leukaemia, although not with other leukaemias, lymphomas or multiple myeloma (Rericha et al, 2006).

An excess of leukaemia deaths was observed in a study of tin miners from the UK (Hodgson and Jones, 1990) but based on only seven persons and with no trend with exposure.

K3.3 Case–control studies

Two case–control studies have examined the relation between residential radon concentration and leukaemia in adults: one from Italy compared radon concentrations in the homes of 44 cases with AML to that of 211 controls (Forastiere et al, 1998; Toti et al, 2005) and one from the UK compared radon concentrations in the homes of 578 cases with acute leukaemia to that of 983 controls (Law et al, 2000). No associations between radon and leukaemia were found in either study.

In a nested case–control study from Finland in an area of high natural uranium and other radionuclides in drinking water, 35 cases of leukaemia were contrasted to a random sample from the rest of the cohort of 274 persons (Auvinen et al, 2002). Radon concentration from the well water was 80 Bq L^{-1} from cases and 130 Bq L^{-1} from controls, giving no indication of risks from this source.

K3.4 Conclusions

The evidence base for leukaemia risks is much more limited than that for lung cancer and so our conclusions have to be more cautious. The authors of a review of epidemiological studies published over the 14-year period 1988–2001 which, as well as the miners' cohorts included 19 ecological studies and 8 case–control studies, concluded that the available data did 'not provide evidence for an association between radon exposure and leukaemia' (Laurier et al, 2001). While we generally agree with this view, the evidence for an association with some leukaemias cannot be entirely discounted.

K4 Gastric cancers

No excess of stomach cancer has been observed in animal experiments.

Calculations suggest that if water containing high concentrations of radon is ingested, the dose to the lining of the stomach can be significant and this implies some risk of stomach cancer. Elevated levels of stomach cancer have been seen in survivors of the atomic bombings (UNSCEAR, 2000).

K4.1 Ecological studies

Kjellberg and Wiseman (1995) reported a correlation between stomach cancer and radon concentrations in an ecological study set in Pennsylvania, USA. No such correlations have been reported from Japan (Mifune et al, 1992). However, Wilkinson (1985) reported high stomach cancer mortality rates in areas of New Mexico with high deposits of uranium. In a study on radon concentrations in groundwater and the risk of gastric cancer, no correlation was found from North Carolina, USA (Collman et al, 1991).

K4.2 Cohort studies

In the combined cohort of miners exposed to radon, Darby et al (1995b) reported a significant excess mortality from stomach cancer (SMR 1.33, 95% CI 1.16–1.52, based on 217 observed deaths). However, no trend in mortality with cumulative exposure was found. Doses to the stomach from radon in air are relatively low. It is also possible that, in mines, other radioactive materials cleared from the lung may be swallowed and irradiate the stomach. The authors concluded that the excess was unlikely to be caused by exposure to radon.

An excess of stomach cancer deaths was also seen in the study of UK tin miners from 1941–1986 by Hodgson and Jones (1990) but no trend with exposure was detected. No excess gastric cancer was observed in a cohort from the Japanese spa town of Misasa (Ye et al, 1998).

A study of Swedish iron miners (Darby et al, 1995a) showed an excess of gastric cancer (1.45, 95% CI 1.64–1.98) based on 40 cases. A dose–response relationship was lacking. This study was included in the combined cohort of miners (Darby et al, 1995b) mentioned above.

A nested case–control study for areas with high radon levels in drinking water in Finland failed to find any association between radon, radium-226 and uranium concentrations and stomach cancer risk (Auvinen et al, 2005).

K4.3 Conclusions

Despite some case excesses seen in several studies, it appears unlikely that radon gas contributes directly to the risk of gastric cancer.

K5 Skin cancer

Calculations suggest (see Appendix J) that the dose to the basal layers of the skin from radon decay products is not negligible and that, if these are the cells at risk, there is some risk of skin cancer. On the basis of such calculations, Eatough and Henshaw (1995) have estimated that perhaps 2% of non-melanoma skin cancers in the UK may be caused by exposure to radon decay products. This estimate depends critically on the depth of the sensitive cells.

In the Harwell animal studies, small but significant excesses in hyperkeratosis (in animals exposed at 1000 WLM, 800 MBq m^{-3} h radon gas exposure, at an equilibrium factor of approximately 0.8) and malignant trichoepithelioma (animals exposed at 100 WLM, 80 MBq m^{-3} h with high unattached fractions) were found.

Non-melanoma skin cancers have been observed in populations exposed to low LET (lightly ionising) radiation (UNSCEAR, 2000). Reporting of non-melanoma skin cancers is often patchy and they are difficult to study by conventional epidemiology using cancer registries.

K5.1 Ecological studies

Etherington et al (1996) reported a correlation between radon concentrations and non-melanoma skin cancer in an ecological study in Devon and Cornwall.

K5.2 Cohort studies

Non-melanoma skin cancers have been observed in a cohort of miners exposed to radon (Sevcova et al, 1978). In the pooled analysis of 11 miners' cohorts, although the overall study was large (some 1100 deaths were observed from cancers other than lung cancer), there were only 27 deaths from all types of skin cancer combined. For malignant melanoma (ICD-9 172), 18 deaths were observed with 19.6 expected (SMR 92, 95% CI 54–145) and for other types of skin cancer, 9 deaths were observed with 5.6 expected (SMR 160, 95% CI 73–303) (Darby et al, 1995b).

K5.3 Conclusions

Mortality is not an appropriate measure to detect an excess of non-melanoma skin cancer due to the rarity of directly attributable deaths from the condition and no conclusions can be drawn from the studies available.

Malignant melanoma does not appear to be associated with radon exposure.

K6 Other cancers

Several animal studies have found excesses of cancers other than those discussed above (see Appendix G).

In the animal studies conducted by the CEA on Sprague-Dawley female rats, an excess of mammary tumours was observed over controls at 1600 WLM (1250 MBq m^{-3} h) with an incidence of 78% compared with 30% observed normally in controls. With the Wistar rat at PNNL, exposures at 640 WLM (500 MBq m^{-3} h) resulted in a doubling of benign mammary tumour incidence. In the studies at Harwell, all groups showed mammary tumour incidences that were not significantly different from those in controls, with the exception of animals exposed at 100 WLM (80 MBq m^{-3} h) with high unattached fractions. In these animals there appeared to be a four-fold increase in the incidence of mammary fibroadenoma over the control incidence of 1%.

PNNL studies showed slight but significant increases in kidney carcinomas, but the carrier aerosol for the exposures was uranium ore dust that may account for this finding. A slight, but non-significant excess of kidney tumours was reported by the CEA in animals exposed up to 1000 WLM (800 MBq m^{-3} h) (G Monchaux, CEA: personal communication). In one of the three studies conducted at Harwell a significantly elevated incidence of renal adenocarcinomas was observed in animals exposed at 500 WL, 1000 WLM (800 MBq m^{-3} h) (3 cases observed out of 50 animals where 1 was expected). Co-exposure to uranium ore dust did not occur at the CEA or Harwell, so there is an indication of a slight increase in kidney tumours associated with exposure to radon and its decay products from all three laboratories.

In the Harwell studies, small but significant excesses in fibroma were observed in animals exposed at 1000 WLM (800 MBq m^{-3} h). This finding has not been reported by the other two laboratories.

In studies at the CEA, the only other findings were slight and non-significant excesses in osteosarcoma and liver tumours.

A markedly higher deposition of radon decay products is found in the nasal passages (Harwell studies), this results in lesions being found in the nose of animals exposed at high unattached fractions (PNNL studies). The higher deposition may also account for the increased skin (trichoepithelioma) incidences observed in the PNNL studies.

None of these animal observations has been followed by further human evidence either from experimental observation of cellular changes or from epidemiology.

K6.1 Cohort studies

The various cohort studies reported in this appendix examine a wide range of other diseases, largely other cancers.

The vast majority of other reported conditions are statistically within 'expected' boundaries. The few exceptions include a deficit of tongue, mouth, pharyngeal and colon cancer in the combined underground miners cohort (Darby et al, 1995b) and an excess of primary liver cancers based on

50 cases (overall ratio of observed to expected cases, O/E, of 1.73, 95% CI 1.29–2.28). No link, however, was found with cumulative exposure.

In contrast, the Swedish iron miners study (Darby et al, 1995a) found an excess of rectal cancer (O/E 1.94, 95% CI 1.03–3.31) based on 13 cases.

The tin miners cohort study from the UK (Hodgson and Jones, 1990) found a significant excess of mortality as a result of accidents, poisoning and violence (based on 51 deaths, SMR 140.6) as well as showing that silicosis and silicotubercolosis were in excess and demonstrated a dose response.

K6.2 Conclusions

There is little support for association with other cancers in human populations and little to contrast between animal experiments and epidemiological results.

K7 Other conditions

K7.1 Multiple sclerosis

The unusual geographical distribution of cases of multiple sclerosis has engendered numerous hypotheses correlating case onset with geomorphological features. One such correlation study from Norway (Bølviken et al, 1997) suggested residential links between multiple sclerosis cases and areas of granite and hence radon exposure. This was also the subject of a hypothesis-generating exercise from Ireland along similar lines (Gilmore and Grennan, 2003). A further and more detailed study from Norway (Bølviken et al, 2003) gives more ecological support based on residence area within the country.

No directly measured studies have been undertaken. No conclusions can be drawn from these studies.

K7.2 Teratological and reproductive effects

No teratological or reproductive effects were found when PNNL exposed pregnant Sprague-Dawley rats to 1600 WLM (1250 MBq m^{-3} h) (Cross and Monchaux, 1999). Maintenance in an area of high radon concentrations in France was found to adversely affect fertility in both male and female mice and male rabbits. These findings were associated with increased incidences of chromosomal aberrations found in somatic cells. However, repetition of the studies in the laboratory with controlled exposure to radon and its decay products demonstrated that these effects were probably due to the high gamma dose rate in the area (100 mGy h^{-1}) rather than the exposure to radon and its decay products (Leonard et al, 1981, 1985). In separate studies a Romanian group has also reported a delayed loss of fertility 10 months after exposure to 20 WLM (16 MBq m^{-3} h) of radon decay products over 52 days. This was associated with a loss of weight of the ovaries of the exposed animals compared with controls and histological changes including a lack of mature graffian follicles and a disturbed corpus luteum (Ramboiu et al, 1999).

No epidemiological evidence exists to support any human effects.

K7.3 Effect on immune system

Exposure of C57BL/6 mice to 1000 WLM or 2500 WLM (800 or 2000 MBq m^{-3} h) resulted in decreases in the total number of cells in the thymus, lung-associated lymph nodes and peripheral lymph nodes (Nagarkatti et al, 1996). The percentage of T cells in these tissues increased, apparently as a result of increased numbers of immature T cells. The authors proposed that cell killing of mature T cells in the lungs as a direct result of exposure resulted in a redistribution of the mature and immature cells throughout the immune system.

K7.4 Non-malignant lung disease

As reported in Appendix G, at high exposure levels (>>1000 WLM, 8000 MBq m^{-3} h) significant excesses in non-malignant lung disease (lung fibrosis and emphysema) have been observed by PNNL and the CEA. At lower exposure levels these diseases were not found unless there was concurrent exposure to other pollutants.

K8 Overall conclusions

On the basis of radiobiological considerations of the distribution of radon and its decay products, there is a small theoretical risk of malignant and other damage to a variety of organs other than the lung.

Some animal experiments tend to support this view. However, this is only on the basis of high radon exposure levels being used. In addition, the effects of other sources of radiation cannot be ruled out in some studies.

The epidemiological evidence is mixed, in that most positive associations are found by ecological (or geographical) studies. However, it is now recognised that indoor radon concentrations vary widely within any area so that average concentration in an area is a poor surrogate for the concentration in a particular house. Results based on such studies are thus very unreliable.

Most case–control studies in which individual radon measurements were available found little or no risk of any conditions, including childhood cancers, adult haematological malignant diseases, gastric cancer and skin cancer.

In general, the cohort studies discussed in this appendix show no excess risk of any malignancy with a few exceptions – for example, some leukaemias. In the cases of gastric cancer and primary liver cancer, significant excesses were found, but without a significant dose response.

There is no published epidemiological evidence to support observations regarding effects on the immune system or teratological or reproductive effects.

In overview, if radon and its decay products do have effects on organs other than the lung, the effect is so weak as to be generally undetectable.

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Appendix L
Reducing Radon Concentrations

This appendix describes the practical measures by which high radon concentrations can be avoided in new buildings, or reduced in existing buildings.

L1 Practical control of radon concentrations in new buildings

Changes were made to the building regulations in 1991 to require the adoption of anti-radon measures in new buildings in parts of England and Wales and these have been updated in later building regulations. The nature of the preventive measures and the areas in which they are required were specified in Report BR211 of the Building Research Establishment (BRE, 1999) and updated in 2007 (BRE, 2007). The guidance specifies two levels: 'basic protective measures' and 'full protective measures'. Maps show where the basic or full measures are required.

For basic protection against radon, an effective seal against radon entry from the ground can be incorporated at little cost when the floor of a new building is being constructed. The standard measures required for protection against ingress of moisture can be adapted to provide protection against radon ingress. Radon enters buildings principally with a flow of air from the ground, diffusion through barriers being very slow in comparison. The aim of basic radon protective measures is therefore to provide a continuous barrier across the whole area of the building.

The radon barrier comprises a cavity tray through the wall, sealed to a membrane, typically a 300 μm (1200 gauge) polyethylene membrane laid across the concrete floor slab. In order to be an effective radon barrier, it is necessary that the joints in the membrane and gaps around service penetrations are well sealed, the membrane is not punctured and good standards of design and workmanship are applied. Where there is a risk of the membrane being punctured, or it is difficult to seal joints in the membrane, it may be advisable to use a more robust membrane. Some membranes are offered for this specific purpose.

It has been found that installation of a radon barrier approximately halves indoor radon concentrations (Woolliscroft, 1992). The cost of adapting standard moisture protection measures to provide a radon barrier for a typical three bedroom semi-detached house, consisting of fitting cavity trays, sealing all penetrations of the membrane, and upgrading the membrane itself if necessary, is estimated to be about £100 (C Scivyer, BRE: personal communication, 2008). The price might be somewhat higher if a more robust radon barrier material with prefabricated 'top hat' sleeves (collars) to seal around pipe penetrations and pre-formed corner pieces were specified.

In areas where radon concentrations are particularly high, further preventive measures, described as 'full radon protection', are required in new buildings. In addition to a radon barrier in the floor, means of

ventilation under the floor must be installed. This can take the form of a suspended concrete floor with a ventilated space underneath, or a radon sump, described in more detail in Section L2.1.

The use of a suspended concrete floor with ventilation underneath instead of a solid concrete floor halves radon concentrations independently of the reduction achieved by installation of a radon barrier (Woolliscroft, 1992). However, a radon sump installed at the time of building a house is not normally connected to piping above ground level, and so has no effect on indoor radon concentrations by itself. If a measurement of radon after the house is occupied demonstrates that there is a high radon concentration in the building, piping and an electric fan can be fitted to extract air from the sump and reduce radon entry into the building. Measurement of indoor radon concentrations after construction is not currently compulsory. The cost of installing a sump and associated pipework during construction is estimated at £100 in addition to the basic measures described above. The capital and installation costs of fitting and connecting an electric fan when a sump/pipe are already fitted are estimated to be £300. The replacement cost of a fan will be £200 with a life of 10 years and a running cost of approximately £60 per year.

L2 Practical control of radon concentrations in existing buildings

Incorporating radon remedial measures into an existing building is less easy than including preventive measures during construction. Nevertheless, radon remedial measures are not technically difficult and are not expensive compared with other building works (see Table L1 and BRE, 1993).

The choice of method for reducing radon concentrations depends on the reduction factor required and the type of floor. In general, the best way to lower radon concentrations is to reduce the pressure difference that draws radon into a building. Table L1 compares typical costs and the measured effectiveness of various radon remedial measures (Naismith et al, 1998). These are discussed in more detail below.

L2.1 Sump under solid floor

A radon sump is a void of around 10–25 litres created beneath the floor slab and linked by pipework to the outside. The sump void can sometimes be produced by boring through an external wall below floor level. An electric fan draws air from the sump, reversing the pressure difference that draws radon into the room, and discharges the radon-laden air outside. It is much the most effective radon remedial measure. In some cases a sump is used without an electric fan, and is known as a passive sump. These are less effective in reducing radon concentrations than powered sumps.

L2.2 Powered ventilation under suspended floor

Radon concentrations under a suspended floor can be diluted with a fan mounted in the wall to blow air into or draw it from the under-floor space. The effect on radon concentration, however, depends in a complicated way on how air moves into and around the space, and the design or operation of a system can sometimes require careful adjustments. The fan can be set up so as to blow air into or extract air

TABLE L1 Typical costs and effectiveness of various radon remedial measures in existing homes (after Naismith et al, 1998)

Location	Remedial method	Method of operation	Typical reduction	Capital cost	Running costs
Below solid floor	Sump with fan	Soil gas drawn into low pressure cavity and exhausted outside	90%	£1,000	£60 per year plus £200 every 10 years
Below suspended floor	Mechanical ventilation	Increased flow of fresh air reduces under-floor radon concentration	60%	£300	£60 per year plus £200 every 10 years
Below suspended floor	Increased natural ventilation	Increased flow of fresh air reduces under-floor radon concentration	50%	£300	–
Mounted in roof space	Powered input ventilation	Reduced indoor underpressure, increased ventilation	60%	£500	£10 per year
Above floor	Increased natural ventilation with crack sealing	Dilution of radon concentration	50%, reducing with time	£1,300	–

from the under-floor space. The choice between these alternatives depends on the local characteristics of the ground and property construction and may require experimentation. It is essential, however, to avoid drawing damp air under the floor or otherwise creating conditions that will cause timber to rot.

L2.3 Increased natural ventilation under suspended floor

Improvement of under-floor airflow can sometimes be achieved with additional air bricks or by replacing existing ones. Plastic air bricks with a large open area can significantly increase ventilation under the floor providing there are no obstructions such as foundation or stub walls. To be effective, air bricks are required on at least two opposite walls with no obstruction between them, and should provide at least 1500 mm^2 of open area for each metre run of wall.

L2.4 Powered input ventilation to house

Permanent extra ventilation can be introduced into a building using commercial systems that blow filtered air from the roof space into the house. These units are principally designed for reducing condensation, but they also reduce the underpressure in the house. This reduces the ingress rate of radon-laden air from the ground. They are most effective in dwellings that are well sealed, and more effective in bungalows than dwellings with more than one storey.

L2.5 Increased natural ventilation with crack sealing

Sealing cracks in the floor is a simple and appealing way of combating high radon concentrations. However, it is likely to be ineffective unless a significant opening is closed up. It may well be necessary to ensure that almost all cracks are sealed. Over time, the effectiveness of this measure decreases, presumably as cracks open up again.

L3 References

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Appendix M
Cost-effectiveness Analysis of Radon Preventive and Remedial Programmes in UK Homes

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Radon prevention and remediation have been the subject of several previous economic evaluations, amongst which are studies in the UK (Field et al, 1996; Kennedy et al, 1999), Spain (Colgan and Gutiérrez, 1996), the USA (Marcinowski and Napolitano, 1993; Ford et al, 1999) and Norway (Stigum et al, 2003). However, none of these previous economic analyses has made use of the most recent estimates of the risk of lung cancer following exposure to radon (BEIR VI Committee, 1999; Lubin et al, 2004; Darby et al, 2005, 2006; Krewski et al, 2006). In addition, current UK policy concerning existing homes, which has been based on offering free radon measurements to selected households, with the decision to remediate and the costs of remediation normally falling on the householder, is under review, partly because the proportion of householders in affected homes who decided to take action has typically been very low (in the range 10–20%). Policy interest has therefore moved towards finding more cost-effective strategies, either through increasing testing and remediation uptake, or through consideration of more comprehensive rules concerning new house building.

The various policy choices all involve different combinations of costs and benefits, and this appendix reports on the methods and results of a new economic evaluation, undertaken with the specific intention of informing discussion about current and future UK policy in this area using the most up-to-date information available, while acknowledging that cost-effectiveness is only one of the inputs to the decision-making process.

We evaluate two main types of programme. First, we consider the cost-effectiveness of radon prevention in new homes, using a range of values to define areas in which such remediation takes place. At present, 'basic preventive measures' are required in new homes in England and Wales in areas where at least 3% of homes have measured radon concentrations above the current Action Level of 200 Bq m^{-3} (ie areas with mean long-term radon concentrations of 52 Bq m^{-3} or higher), and 'full preventive measures' (consisting of basic preventive measures plus fitting a means of under-floor ventilation such as a radon sump and pipe) are required in areas where more than 10% of homes have measured radon concentrations above 200 Bq m^{-3} (ie areas with mean long-term radon concentrations of 87 Bq m^{-3} or higher) (BRE, 2007). In Scotland and Northern Ireland basic preventive measures are required where at least 1% of homes have measured radon concentrations above 200 Bq m^{-3}.

Second, we consider the cost-effectiveness of policy concerning existing homes. Much of the radon measuring is done by the HPA which takes the measurements over a period of three months and then

makes an adjustment for seasonal variation. At present, if the radon measurements in 1% or more of homes in an area, usually an area of 1 or 5 km^2, are estimated to be above 200 Bq m^{-3} (ie the mean long-term radon concentration is 36 Bq m^{-3} or higher), the area is designated as a 'radon Affected Area' and current HPA policy is to recommend that occupants of homes in these areas have their homes tested and carry out remediation if the radon measurement (again taken over a period of three months and seasonally adjusted) is above 200 Bq m^{-3}. Government policy in England is to focus on areas where measurements in 5% or more of existing homes are likely to exceed 200 Bq m^{-3} (ie areas with mean long-term radon concentrations of 64 Bq m^{-3} or higher), and to invite householders in these areas to have their homes tested, and to remediate if they are found to be over 200 Bq m^{-3}. Our baseline analyses consider an area which just qualifies under this policy, but we provide results across a range of alternatives.

In our main analyses, we have adhered to the methodological framework now considered appropriate in the economic evaluation of health interventions, using a societal perspective in which all direct costs are included (Gold et al, 1996; DH, 2004; Drummond et al, 2005).

M1 Methods

A model was constructed to estimate the expected number of lung cancer deaths in a particular population in the presence and absence of radon remediation. These estimates were then combined with information on the costs of radon measurement and remediation and of lung cancer treatment to calculate the cost-effectiveness of various remediation programmes compared to no programme. Cost-effectiveness is calculated as the ratio of net change in cost to net change in outcome, with outcome (lung cancer deaths averted) expressed in terms of life-years and quality adjusted life-years (QALYs) gained; this facilitates comparison of the cost-effectiveness of radon remediation with that of other public health and health care interventions.

M1.1 Risk

It is assumed that the main objective of household remediation programmes is a reduction in radon concentrations in homes in order to decrease the number of deaths from radon-induced lung cancer. We estimate the number of deaths from radon-induced lung cancer that would arise following exposure to a certain indoor radon concentration using two methods. First, we use data from a recent analysis which brought together the individual data from a total of 13 European epidemiological studies that investigated directly the risk of lung cancer from residential radon in the home (Darby et al, 2005). Based on this analysis, the risk of lung cancer is estimated to increase by 16% per 100 Bq m^{-3} increase in the long-term average radon concentration in the home. We refer to this as our *direct* estimate of risk. Our second method estimates the relationship between the risk of lung cancer and indoor radon concentration indirectly, and we follow the preferred risk models proposed by the BEIR VI Committee (1999), based on radon-exposed miners (see Appendix H), in which the death rate from lung cancer was assumed to vary linearly with cumulative radon exposure, subject to modification by time since exposure,

attained age and either radon concentration or duration of exposure. Here the approach based on radon concentration is used. We refer to this as our *indirect* estimate of risk.

Using both the direct and indirect estimates of the relationship between radon concentration and lung cancer, we estimate the lifetime risk of lung cancer in the UK for the whole population, and separately for current cigarette smokers and lifelong non-smokers at various radon concentrations, in each case allowing for the competing risk of death from other causes. We then estimate the difference between the risk at pre-remediation and post-remediation concentrations and the resulting number of lung cancers averted per household remediating. We make the simplifying assumption that all lung cancer cases result in death from lung cancer (5-year survival from lung cancer in England in 1998–2001 was only 6% in men and 7% in women), and our estimate of the number of life-years lost from each lung cancer death is based on the estimated remaining life expectancy at the time of death from radon-induced lung cancer; separate calculations are performed for current cigarette smokers and for never-smokers. Any estimated benefits of radon prevention or remediation, in the form of life-years gained, are then spread over a 100-year period of analysis, discounted to present values. In the baseline case these benefits are spread evenly, with no assumed latency period or lag between reduction of radon exposure and alteration of lung cancer risk. Sensitivity analyses are also reported showing the effects on the results of introducing latency periods of up to 20 years. Evidence from, for example, UK case–control studies of smoking and lung cancer indicate that the risk of lung cancer begins to fall within five years after cessation of smoking (Peto et al, 2000). Similarly, in the BEIR VI Report, most weight is given to the period 5–14 years after the radon exposure window, with progressively smaller weights to periods 15–24 and 25+ years after exposure (BEIR VI Committee, 1999).

Although the majority of our calculations are presented for both direct and indirect estimates of risk, our conclusions are based on the direct estimates, as we judged them to be more relevant to the risk from indoor radon at home (see Chapter 3).

M1.2 Perspective

We include direct costs incurred or saved by households, by the NHS, and by the HPA. The net cost of radon remediation was calculated by obtaining information on the cost of identifying homes with measured radon concentrations over 200 Bq m^{-3}, the capital, maintenance, running and replacement costs of remedial work, the treatment costs for lung cancer cases, and the health care use of individuals during any period of extended life expectancy. We assumed that initial programme costs, including all measurement and initial remediation costs, were incurred in year 1. All costs are expressed in 2007 UK pounds sterling.

M1.3 Time horizon and discounting

The costs and benefits of radon remediation are modelled over a period of 100 years, and are discounted to present values. The discount rate, or social time preference rate, is used to reflect the value society attaches to present, as opposed to future, consumption, and has two elements:

a time preference, reflecting the extent to which individuals prefer present consumption over future consumption, assuming no change in *per capita* consumption, and also reflecting the risk that unforeseen external events or technological changes may seriously affect the likely returns on the investment,

b growth in real income over time, which implies that future consumption will be more plentiful relative to the present and so will have lower marginal utility.

UK estimates suggest that the first element has a value of approximately 1.5% per year, and the second – equivalent to the trend rate of growth of real *per capita* income – is approximately 2% per year, giving a discount rate of 3.5% per year, and this is the normal discount rate recommended by the Treasury when appraising possible investments (HM Treasury, 2003). This is the discount rate that the National Institute for Health and Clinical Excellence recommends be applied to future costs and health outcomes (NICE, 2004). However, it has been suggested that the marginal utility of health benefits does not fall as incomes rise, and therefore that only the time preference element of the discount rate should be applied to these health benefits (Gravelle and Smith, 2001); following this argument, the Department of Health has recommended that costs be discounted at 3.5% per year and health benefits at 1.5% per year (DH, 2004) and this approach is also followed by the Health and Safety Executive (NERA, 2007). In this report, all costs are discounted at 3.5% per year and health benefits at 1.5% per year, with other rates used in sensitivity analyses.

M1.4 Willingness to pay for health benefit

Having estimated the cost-effectiveness of particular interventions, calculated as the ratio of net change in cost to net change in outcome, it will frequently be the case that the intervention produces additional health benefits but at added cost. It is then necessary to assess whether the intervention represents good value for money, ie whether the additional health benefits are worth the additional cost. One approach to this, adopted by NICE in England, is to make a judgement about the acceptability of using resources for some new intervention in comparison with other possible uses of these scarce resources: in effect, to assess the opportunity cost of using NHS resources in this way. Using this approach, NICE has indicated that, although it does not have a fixed threshold, interventions with a cost-effectiveness below a most plausible estimate of £20,000 per QALY gained are likely to be viewed favourably, whilst the case for supporting interventions with a cost per QALY gained of over £30,000 would have to rely quite heavily upon other factors such as an innovative technology, particular features of the health problem, or wider societal costs and benefits (NICE, 2004). Other researchers, however, have inferred from decisions reached by NICE that its cost-effectiveness ceiling may in fact be higher than this stated range (Dakin et al, 2006; Raftery, 2006).

The approach advocated by NICE would be one useful way of judging whether radon prevention and remediation interventions are likely to be good value for money. However, NICE is primarily concerned with value for money to the NHS, and its ceiling range of £20,000–£30,000 per QALY gained is at least implicitly a judgement about the opportunity cost to the NHS of using resources in different ways. Radon interventions have a wider range of costs and savings that fall not only on the NHS but also on other public agencies such as the HPA, and in particular on households themselves. An alternative is to look at

the results of studies which have tried to place direct valuations on a 'statistical life' or values of preventing 'statistical fatalities' – for example, to inform decisions about investments in transport safety. In the UK this figure, based on the gross output that people would have produced if they had not died prematurely as well as additional amounts for the pain and suffering involved, was approximately £1.4 million in 2004 (DOT, 2007). Given an average remaining life expectancy of those killed in road accidents of 40 years, this is approximately equivalent to £35,000 per year of life lost. Another approach was used in a study commissioned by the Department for Environment, Food and Rural Affairs, which asked a population sample how much they would be prepared to pay annually over an average lifetime for an improvement in air quality that resulted in improved life expectancy. The results of this survey indicated that the value implicitly placed on an additional year of life was £27,600 (Chilton et al, 2004), a figure also broadly in line with the ceiling range being used by NICE, and the adoption of this figure across different government departments has subsequently been recommended by an Interdepartmental Group on Costs and Benefits (IGCB, 2004).

In summary, although arrived at using different methods, the values placed on an additional year of life as implied by existing valuations of statistical lives and by stated preference surveys are not in practice radically different from the £20,000–£30,000 range used by NICE. In the report below, figures show the range £20,000–£30,000 per QALY gained as a broad indication of the likely upper limits of willingness to pay for health gain.

M1.5 Uncertainty

Uncertainty around the various assumptions and data inputs used to construct the model is dealt with in two ways. First, the results of one-way sensitivity analyses are reported, in which key input variables are varied across a plausible range to assess their impact on the results, holding all other variables constant.

Second, the model has been constructed to perform probabilistic sensitivity analysis, in which the input values of parameters can be simultaneously and independently varied repeatedly around the central estimates using random draws from specified distributions or ranges, with costs, effects and cost-effectiveness recorded on each run (Doubilet et al, 1985; Claxton et al, 2005). In the probabilistic analyses we included all variables also included in the one-way sensitivity analyses: for the percentage increase in the risk of lung cancer per 100 Bq m^{-3} increase in radon concentration we used the log-normal distribution; for the percentage reduction obtained by remediation measures and for the average home occupancy level we used beta distributions; for initial remediation cost per household and for the mean NHS/hospice treatment cost per lung cancer case we used gamma distributions; and for other variables (such as unit costs of testing) we used uniform distributions. The results are displayed on a two-dimensional cost-effectiveness plane, on which is plotted the incremental change in effectiveness (x-axis) and the incremental change in costs (y-axis) of radon remediation versus no remediation. The results are also displayed in the form of a cost-effectiveness acceptability curve, which plots the probability that the intervention is cost-effective at different levels of willingness to pay for health gain (Van Hout et al, 1994). The sensitivity analyses assess the potential effect of changed circumstances on the results. For example, there is evidence that new local-authority-based exercises are achieving a higher level of remediation than has historically been attained and that this is very dependent on the level of advice and support

offered to householders (Appendix D) (Kendall et al, 2005). However, not all such future conditions can be anticipated, and programme techniques may well change. The numerical results presented here, in particular the cost per quality adjusted life-year saved of generic remediation programmes, are indicative of the cost-effectiveness of current programmes and some possible alternatives in the UK. The same approach could be used to make specific estimates for programmes in other countries or for other proposed future programmes, each with its own attributes.

M2 Data

Data on the size of the total UK population, subdivided by age and sex, were obtained from UK national statistics for the year 2005 (ONS, 2006a), and the corresponding numbers of lung cancer deaths were obtained by combining published 2005 figures for England and Wales, Scotland and Northern Ireland (ONS, 2005). The lung cancer death rate in never-smokers was based on American Cancer Society data (Thun et al, 2006), adjusted from the mean indoor radon concentration in the USA to that in the UK; the lung cancer death rate in current cigarette smokers was derived from a recent study of cigarette smoking in the UK (Peto et al, 2000) and estimates of the age- and sex-specific lung cancer death rates for lifelong non-smokers (Thun et al, 2006), together with estimates of the age- and sex-specific proportion of the population who are lifelong non-smokers (ONS, 2006a). The proportion of the population who have never smoked regularly, and the proportion who are current cigarette smokers, by age and sex, were obtained from an analysis of General Household Survey data for 2004 (ONS, 2006a; Professor M Jarvis, University College, London: personal communication). Life expectancy at the time of death from lung cancer was calculated separately for male and female current cigarette smokers and never-smokers using data on all-cause mortality attributable or not to smoking (Doll et al, 2004; Peto et al, 2006).

M2.1 Distribution of indoor radon concentrations

Information on the distribution of measured radon concentrations in homes in the UK as a whole was obtained from national survey data on radon concentrations (Wrixon et al, 1988; Gunby et al, 1993; Green et al, 2002) and information on the variation between radon measurements made in the same home in different years was obtained from previous studies (Lomas and Green, 1994; Hunter et al, 2004; Darby et al, 2006). Information on measured radon concentrations in areas of 1 or 5 km^2 and on the estimated proportion of homes with measured values likely to be above 200 Bq m^{-3} in each area was provided by the HPA. Mean long-term average radon concentrations were derived using the methods described in Appendix C. We set the cost of inviting householders to have their homes tested at £1.65, including administration, postage and materials: this is based on the estimated average cost per invitation letter sent in a screening programme for colorectal cancer, updated to 2007 prices (Garvican, 1998). The proportion of householders invited to test who accept was taken to be 30%, in line with the proportions accepting the current Defra programmes and a Department of the Environment, Transport and the Regions pilot study in the Derbyshire, Cherwell and Mendip areas (DETR, 2000). The HPA offers a service for measuring radon concentrations, based on delivery, removal, reading and reporting from a pair of etched-track detectors in two rooms for three months, for £42. These and other unit costs are reported in Table M1.

TABLE M1 Unit costs used in analyses, in UK £s in 2007 values

	Baseline value	Source
Basic preventive measures such as sealed membranes in new homes	£100	HPA, DCLG and BRE: see Section M2.2
Full preventive measures, consisting of basic preventive measures plus fitting a means of under-floor ventilation such as a radon sump and pipe, in new homes	£200	HPA, DCLG and BRE: see Section M2.2
Electric fan: capital and installation cost when sump/pipe already fitted	£300	HPA, DCLG and BRE: see Section M2.2
Electric fan: replacement cost	£200	
Electric fan: running cost per year	£60	HPA, DCLG and BRE: see Section M2.2
Electric fan: capital and installation cost with retrofitting of sump/pipe	£1,000	HPA, DCLG and BRE: see Section M2.2
Invitation to test	£1.65	Cost per invitation letter in screening programme for colorectal cancer, updated to 2007 prices (Garvican, 1998)
Measurement of radon: delivery, removal, reading and reporting from two etched-track detectors in different rooms for three months	£42	HPA current price, as of July 2008
Average remediation cost per household (initial)	£762	Naismith et al, 1998, adjusted to 2007 prices using the Building Cost Index
Lifetime remediation cost per household (discounted over 100 years, 35% with active measures requiring replacement every 15 years plus running costs)	£1,987	Naismith et al, 1998, and model: see Section M2.2
NHS annual *per capita* expenditure on all other health care during added life expectancy	£7,817	DH, 2007
Mean NHS/hospice treatment cost per lung cancer case	£16,840	Wolstenholme and Whynes, 1999

M2.2 Radon remediation and prevention

The costs of radon preventive measures in new homes are influenced by building practices and regulations, the experience of builders, and the type of home and precise methods of construction. For the purposes of this analysis, we have arrived at a set of cost assumptions following discussions with Jon Miles (Health Protection Agency), Richard Shipman (Department for Communities and Local Government) and Chris Scivyer (Building Research Establishment). While a formal Regulatory Impact

Assessment would be desirable, the costs used in the present analyses are believed to represent a fair assessment of current regulation and practice. We assume that the installation of basic radon preventive measures, consisting essentially of radon-proofing existing damp-proof or vapour control membranes across a building's footprint during construction by creating gas-proof seals around pipe penetrations, installing cavity trays and upgrading the membrane itself if necessary, would typically cost approximately £100 per building. Upgrading these basic measures to provide full preventive measures, consisting of fitting a means of under-floor ventilation, such as a radon sump and extract pipe, would cost an additional £100. Similar figures have been quoted for installation of radon-resistant features in new homes in North America (FPTRPC, 2006).

For the new homes intervention, we assume average radon concentrations in new buildings are the same as the mean concentration for the area as a whole. It is further assumed that these basic measures, when installed in a new home, will reduce radon by approximately 50% (Naismith, 1997). The full preventive measures do not affect radon levels by themselves, but make it easier to fit at a later date active measures such as an electric fan, which we estimate will cost £300 including fan and installation costs, will have to be replaced every 15 years at a cost of £200, and will have annual running costs of £60. Active measures are taken to reduce radon levels in a new home by approximately 90%, similar to that found in existing homes. Retrofitting a sump and pipework and installing an electric fan in a new home where no provision was made for them at the time of construction is estimated to cost £1,000.

In our evaluation of existing homes, once the measured radon concentration in a tested home was found to be above 200 Bq m^{-3} and the householder advised to remediate, the proportion of householders who actually do remediate was set at 20%, in line with previous surveys (Bradley and Thomas, 1996). This figure includes households being surveyed for the first time and some households that may have been surveyed previously. Clearly, this could change as the proportion of households previously surveyed increases, but there is little evidence on this. The pre-remediation measured radon concentration in households carrying out remediation was based on the expected value given the mean measured value in the area of interest and assuming a log-normal distribution (Gunby et al, 1993) with left-truncation at the recommended Action Level. There is conflicting evidence on whether the householders who choose to remediate have radon levels typical of all homes over 200 Bq m^{-3}: in the pilot study in the Derbyshire, Cherwell and Mendip areas the mean measured radon concentration in all homes found to be above the current Action Level was 396 Bq m^{-3}, but was 18% higher, at 467 Bq m^{-3}, in those households that took remedial action (DETR, 2000); however, a 2007 HPA evaluation and equity audit of the radon programme in England (Dr Y Chow, HPA: personal communication) found no evidence that those who remediate had higher average radon levels than those who did not. Here we assume that the former have radon levels typical of all homes over the current Action Level. The reduction in radon concentrations following remediation depends on the type and hence on the cost of the actions performed; Naismith et al (1998) reported an average reduction in measured radon concentrations of approximately 85% in existing homes and an average remediation cost of £630 in a study of almost 1000 homes undertaking a range of remediation measures, equivalent to £762 when adjusted to 2007 prices using the Building Cost Index. Almost identical results were obtained by Kennedy et al (1999) for a sample of 62 homes in Northamptonshire. Assuming approximately 35% of homes remediating had installed active measures requiring replacement of fans and running costs (Naismith et al, 1998), this is

equivalent to a discounted cost of £1987 per household over 100 years. The average household size was set at 2.4 people in line with national data for 2006 (ONS, 2007a); this figure has not changed appreciably in recent years and so we hold it constant into the future. The average home occupancy level has been reported to be approximately 80% in some studies (Kendall et al, 1994), but may since have fallen: we used a value of 70%, or around 17 hours per day, in line with the Time Use Survey 2005 (ONS, 2007b).

M2.3 Health care costs

Detailed estimates of the four-year hospital costs of lung cancer diagnosis, treatment and follow-up were reported in 1999 (Wolstenholme and Whynes, 1999): these indicated an average cost per case for non-small cell lung cancer of £6150 at 1993 prices. We doubled the estimated in-patient palliative care costs (£3962 per patient receiving palliation) to make approximate allowance for hospice care following discharge from hospital, and inflated the resulting figure by 56.4% using the Hospital and Community Health Services Pay and Prices Index to give a figure of £16,840 per case at 2007 prices. This estimate will not reflect changes in medical practice in recent years, such as shorter lengths of stay or the introduction of new treatment regimens.

There is currently a lack of consensus on how to treat additional health care costs incurred during any period of extended life expectancy in economic evaluations of interventions to improve health: current NICE guidelines suggest that only costs for diseases related to the intervention should be included, but in practice these may be hard to define. Here all health care costs are included in the baseline analyses, and they were estimated using data from the Department of Health on hospital and community health services expenditure per person by age group (DH, 2007). Assuming all other NHS expenditure was similarly distributed, uprating to 2007 prices gave a figure of £7817 per year per person aged 75 years or over (average age at death from lung cancer was 72.2 years in 2004). These costs are varied in various sensitivity analyses.

M3 Results for new homes

M3.1 Basic preventive measures in new homes

Table M2 reports the central estimates of cost-effectiveness for the installation of basic preventive measures in all new homes during construction in areas where approximately 3% of homes have radon measurements above the current Action Level of 200 Bq m^{-3}, ie areas where the mean long-term radon concentration is 52 Bq m^{-3}. Practical policies target areas above a threshold, rather than areas at a particular value as in this example. However, it is necessary to calculate cost-effectiveness at particular values in order to determine where to set a threshold.

TABLE M2 New homes: main estimates of effects, costs and cost-effectiveness for basic preventive action in all new homes in an area where 3% of homes have measured radon concentrations above 200 Bq m^{-3}, based on the HPA methodology of considering one radon measurement of three months' duration with seasonal adjustment. This corresponds to an area with a mean long-term concentration of 52 Bq m^{-3}

Current government policy in England and Wales requires preventive measures in new homes in areas with mean long-term radon concentrations at or above this level

	Radon risk estimate	
	Direct [(i)]	**Indirect [(ii)]**
Initial		
Lifetime cumulative risk of death from lung cancer (% per person)	6.38	6.62
Post-preventive action		
Lifetime cumulative risk of death from lung cancer (% per person)	6.14	6.18
Health gain per household		
Lung cancer cases averted	0.006	0.010
Total life-years gained	0.08	0.14
Total life-years gained – discounted	0.04	0.07
Total QALYs gained	0.06	0.11
Total QALYs gained – discounted	0.03	0.06
Resource use and costs per household		
Radon prevention cost – discounted	£100	£100
NHS lung cancer treatment costs averted – discounted	£29	£52
Other NHS costs incurred by added life expectancy – discounted	£177	£322
Net cost – discounted – societal	£248	£369
Net cost – discounted – NHS	*£148*	*£269*
Net cost – discounted – households	*£100*	*£100*
Cost-effectiveness		
Cost per life-year gained – discounted – societal	£6,226	£5,101
Cost per QALY gained – discounted – societal	**£7,953**	**£6,516 [(iii)]**
Cost per QALY gained – discounted – NHS	*£4,752*	*£4,752*
Cost per QALY gained – discounted – households	*£3,201*	*£1,764*

Notes
(i) From studies of residential radon and lung cancer in Europe (Darby et al, 2005, 2006).
(ii) From studies of radon-exposed miners (BEIR VI Committee, 1999).
(iii) Shaded value corresponds to current policy in England and Wales.

Using the direct estimate of the risk of lung cancer, based on the European studies of residential radon (Darby et al, 2005, 2006), the cost-effectiveness model predicts that the cumulative lifetime risk of lung cancer for a member of the general population at pre-remediation radon concentrations is 6.38%. Post-remediation, the lifetime risk falls to 6.14%. This is equivalent to a reduction of 0.006 lung cancer cases in a household of average size, which in turn is equivalent to 0.08 life-years gained, or 0.04 discounted life-years gained. Using the indirect estimate of the risk of lung cancer, based on the BEIR VI analysis of the experience of radon-exposed miners, the lifetime risks of radon-induced lung cancer are higher, and the total undiscounted life-years gained per household from remediation are 0.14.

Under a policy of installing basic radon preventive measures in new homes, the only costs are those of the preventive measures, ie £100 per home. Using the direct estimate of lung cancer risk, savings from lung cancer treatment costs averted, when discounted, reduce costs by £29 per household, while additional costs to the health service from added life expectancy are £177, leading to a net societal cost of £248 per household. Using the indirect estimate of lung cancer risk, the net societal cost is higher, at £369, mainly due to a larger estimate of additional life expectancy.

Combining the outcomes and costs reported above, the cost per QALY gained of radon prevention in areas with mean long-term radon concentrations of 52 Bq m^{-3} (ie 3% of homes have measurements above 200 Bq m^{-3}) is £7,953 using the direct risk estimate. Using the indirect risk estimate, with higher risks of radon-induced lung cancer and therefore higher benefits from radon prevention, the cost per QALY gained falls to around £6,516. Both these figures are well below the maximum levels (£20,000–£30,000 per QALY gained) that might typically be considered cost-effective when assessing alternative ways of improving health.

M3.2 Basic measures in new homes under different assumptions

Table M3 and Figure M1 show the cost-effectiveness of a policy of installing basic radon preventive measures in new homes in areas at different mean long-term radon concentrations. As the mean radon concentration decreases, the cost per life-year gained increases. However, even in areas at 10 Bq m^{-3}, the cost per QALY gained is still only just over £21,000 for calculations based on the direct estimate of risk and below £14,000 for calculations based on the indirect estimate of risk. When the whole country is considered, the cost per QALY gained is £11,400 using the direct risk estimate and £8,400 using the indirect risk estimate. This suggests that a basic preventive measures policy of fitting membranes in all new homes throughout the entire UK would be highly cost-effective.

Figures M2(a) and (b) summarise the results of one-way sensitivity analyses of the effect of installing basic radon preventive measures in all new homes throughout the UK, using direct and indirect estimates of the radon-related lung cancer risk, respectively. In these analyses, single parameters are varied while all other parameters are held constant. Under the direct estimate of the lung cancer risk, the cost per QALY gained would increase from the baseline of £11,400 to approximately £21,400 if the cost of installing basic measures was £250 instead of the £100 assumed; alternatively, if the cost of basic measures was only £50, the cost per QALY gained would fall to £8,100. These results are also sensitive to the percentage reduction in radon obtained from fitting a membrane, and to the risk of radon-related

TABLE M3 New homes: effect on cost per quality adjusted life-year (QALY) gained of varying the definition of areas where basic preventive measures are fitted in all new homes

	Cost per QALY gained [(i)]		
Mean long-term radon concentration in area	**Direct risk estimate**	**Indirect risk estimate**	**% of national housing stock in areas with mean above this value**
10	£21,406	£13,909	87.5
15	£15,858	£10,860	60.9
20	£13,084	£9,335	39.6
25	£11,419	£8,421	25.6
30	£10,310	£7,811	16.7
35	£9,518	£7,376	11.2
36 [(ii)]	£9,383	£7,301	10.3
40	£8,923	£7,049	7.6
45	£8,461	£6,795	5.2
50	£8,091	£6,592	3.7
52 [(iii)]	**£7,953**	**£6,516** [(vi)]	3.2
55	£7,788	£6,425	2.6
60	£7,536	£6,287	1.9
64 [(iv)]	£7,372	£6,196	1.5
65	£7,323	£6,170	1.4
70	£7,140	£6,069	1.0
75	£6,982	£5,982	0.8
80	£6,843	£5,906	0.6
85	£6,720	£5,838	0.5
87 [(v)]	£6,667	£5,809	0.4
90	£6,612	£5,779	0.4
Whole country			
21	£11,427	£8,425	100

Notes
(i) Discounted – societal.
(ii) That is, 1% of measurements >200 Bq m^{-3}.
(iii) That is, 3% >200 Bq m^{-3}.
(iv) That is, 5% >200 Bq m^{-3}.
(v) That is, 10% >200 Bq m^{-3}.
(vi) Shaded value corresponds to current policy in England and Wales.

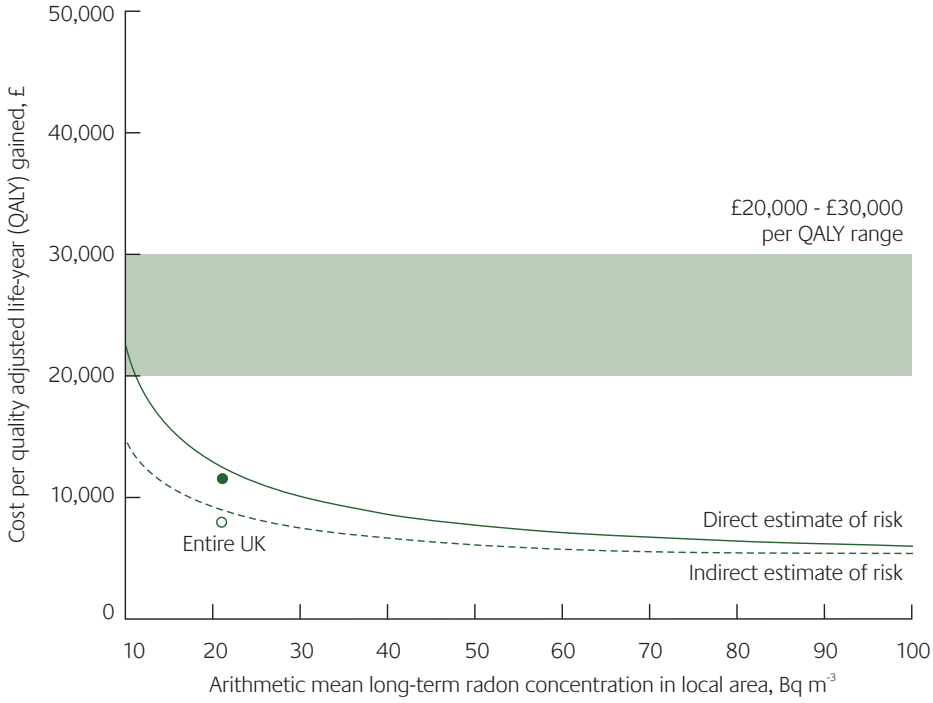

FIGURE M1 New homes: relation between long-term mean radon concentration (Bq m^{-3}) in a small area of 1 or 5 km^2 and cost per quality adjusted life-year gained (discounted – societal) for the installation of basic preventive measures in all new homes. Results shown for direct estimate (ie based on European pooling study) of radon risk and indirect estimate (ie based on BEIR VI) of radon risk

lung cancer: at the upper 95% confidence limit for the direct estimate, the increase in lung cancer risk would rise to 31% per 100 Bq m^{-3} and the cost per QALY gained would fall to £8,300, while at the lower 95% limit the increased risk would be only 5% per 100 Bq m^{-3} and the cost per QALY gained would rise to £25,600. Finally, the results are sensitive to alternative assumptions concerning the discount rate: at 0% for outcomes and 3.5% for costs, cost-effectiveness falls to £6,000 per QALY gained under the direct estimate of lung cancer risk, while at a 6% discount rate for both costs and outcomes, the cost-effectiveness ratio increases to £28,200.

Figures M3(a) and (b) show the results of probabilistic sensitivity analyses for the direct and indirect estimates of lung cancer risk, respectively, in which all the main model parameters are varied independently and simultaneously 10,000 times using the specified distributions, and the cost-effectiveness results are recorded on each occasion.

Figure M4 extends this by showing a cost-effectiveness acceptability curve, which plots the probability that the cost per QALY gained of radon prevention in new homes is cost-effective, as the decision-maker's willingness to pay for health gain is varied from 0 to £100,000 per QALY. In this instance, the

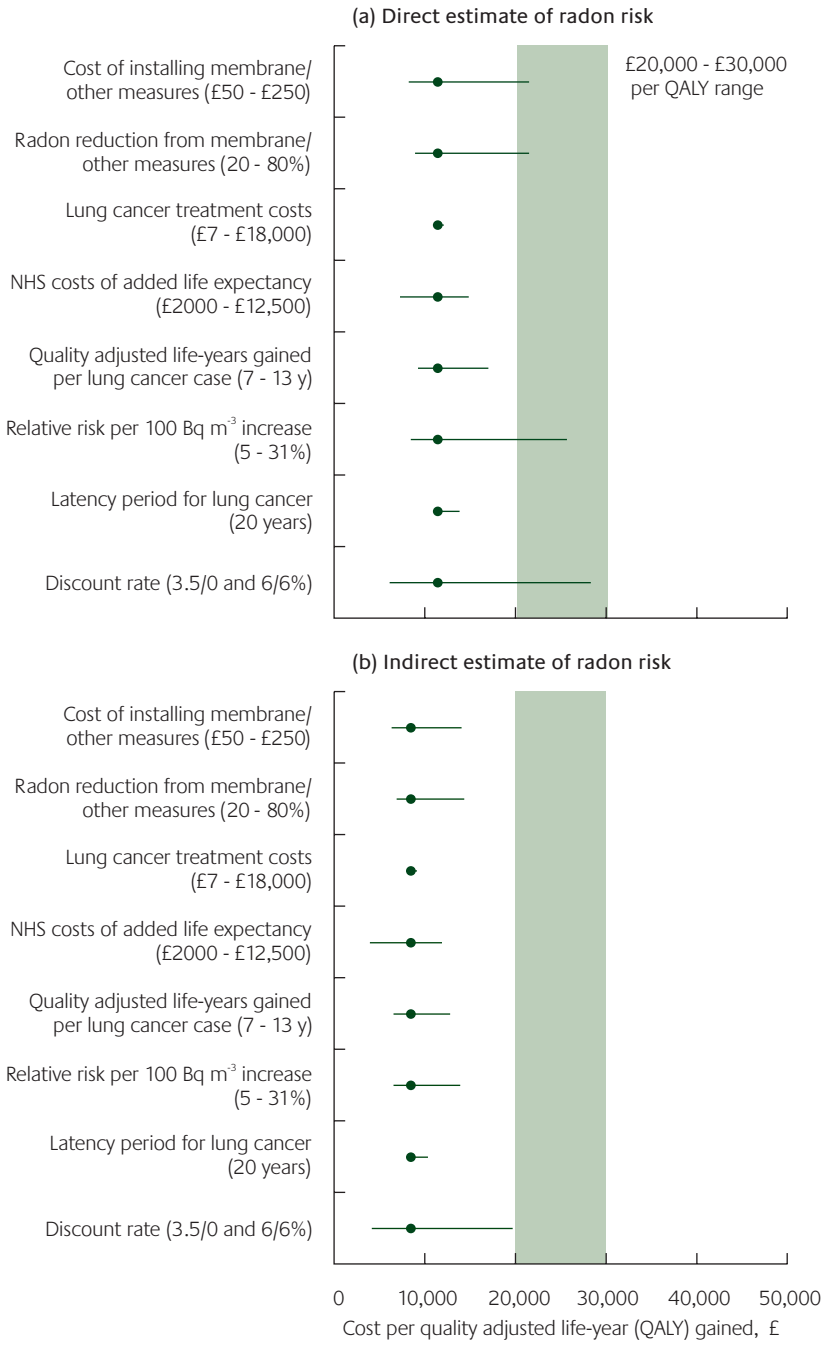

FIGURE M2 New homes: sensitivity to changes in main parameter values of installing basic preventive measures in all new homes: (a) direct estimate of radon risk and (b) indirect estimate of radon risk

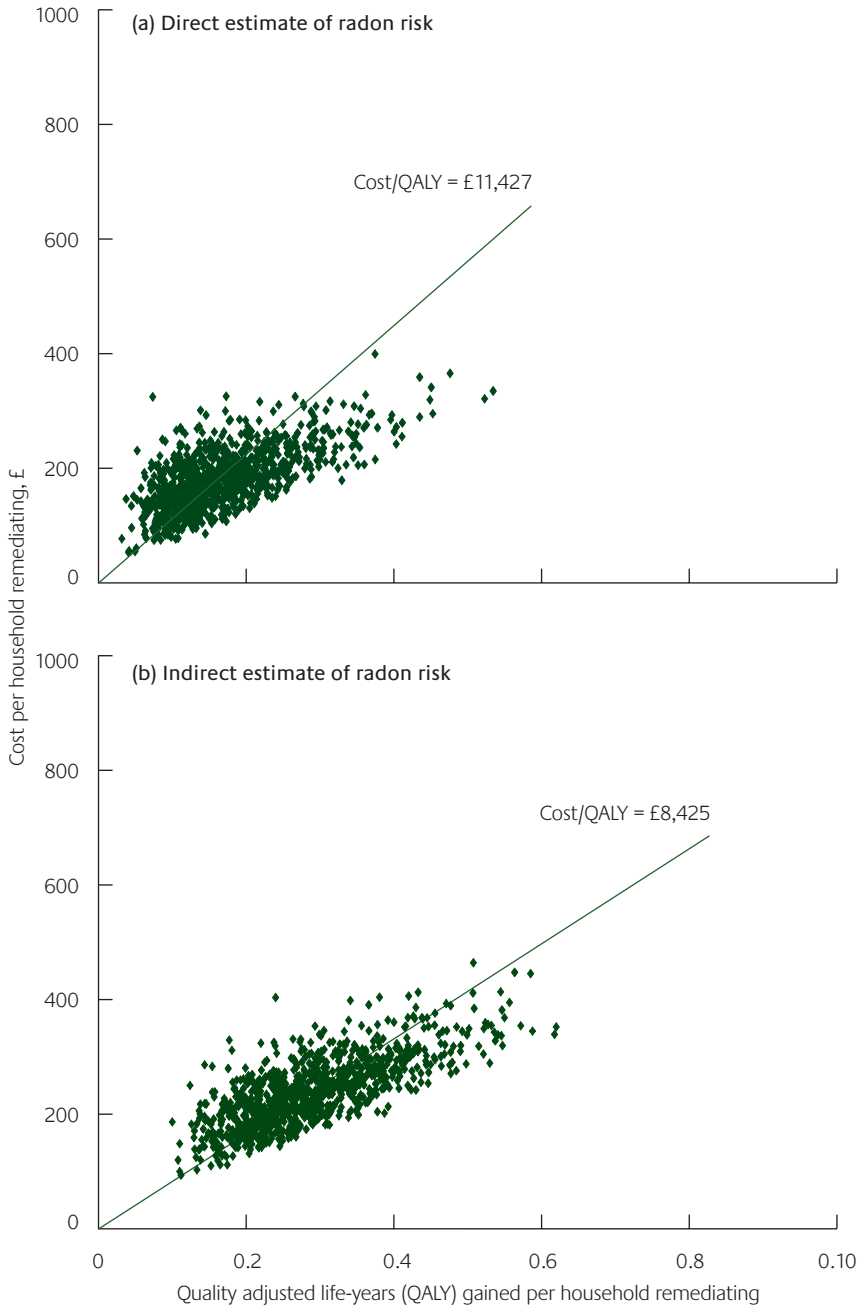

FIGURE M3 Cost-effectiveness plane showing uncertainty around cost and QALYs gained from installing basic preventive measures in all new homes in the UK: 10,000 simulations allowing all key variables to vary simultaneously and independently around baseline values: (a) direct estimate of radon risk and (b) indirect estimate of radon risk

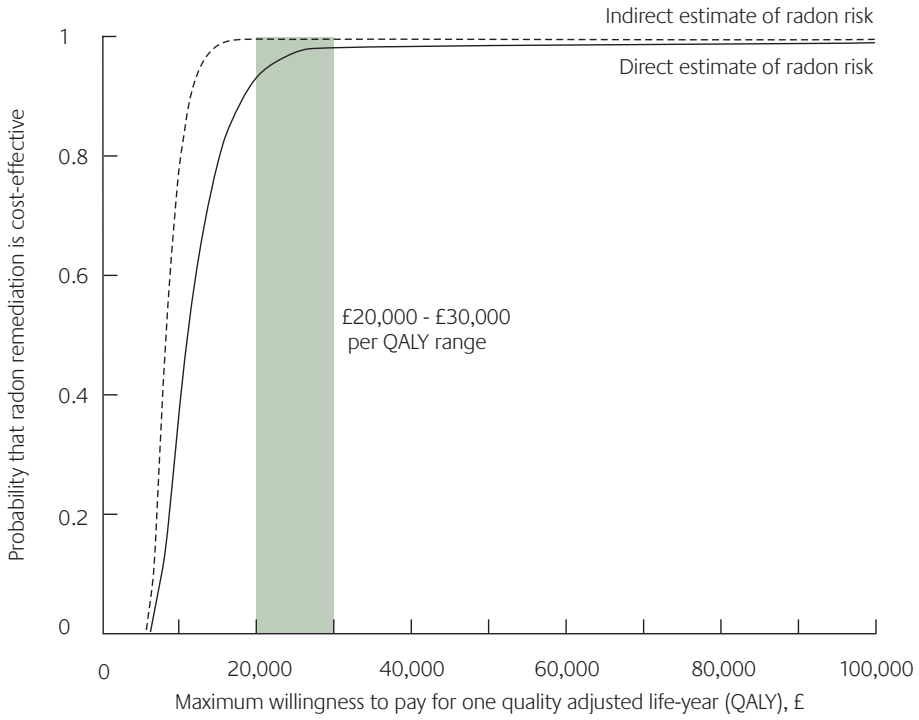

FIGURE M4 New homes: cost-effectiveness acceptability curve for the data in Figure M3, showing probability that the cost per QALY gained of installing basic preventive measures in new homes in all areas is cost-effective, as a function of a decision-maker's willingness to pay for health gain

probability that the intervention is cost-effective (using the direct risk estimate) when decision-makers are prepared to spend up to £20,000 per QALY gained is 94%; when they are prepared to spend up to £30,000 per QALY gained this probability rises to almost 100%.

Finally, Figure M5 shows the cost-effectiveness of a variety of scenarios in which the basic measures installed during construction are enhanced at higher cost but with higher effectiveness – for example, by fitting more resistant membranes or initiating a tighter inspection regime. The figure shows the additional benefit in relation to the additional cost – for example, spending £150 instead of £100 on basic measures, in order to increase effectiveness from 50% to 55% or 60%, etc. The results indicate that enhanced basic measures can be cost-effective, but only if the additional radon reduction obtained is at least 10% – for example, spending £200 instead of £100 on enhancing basic measures in order to obtain a radon reduction of 55% instead of 50% would have a cost-effectiveness of almost £67,000 per QALY gained, but if the radon reduction obtained was larger – for example, 65% – spending £200 instead of £100 would be cost-effective, with a cost per QALY just below £30,000.

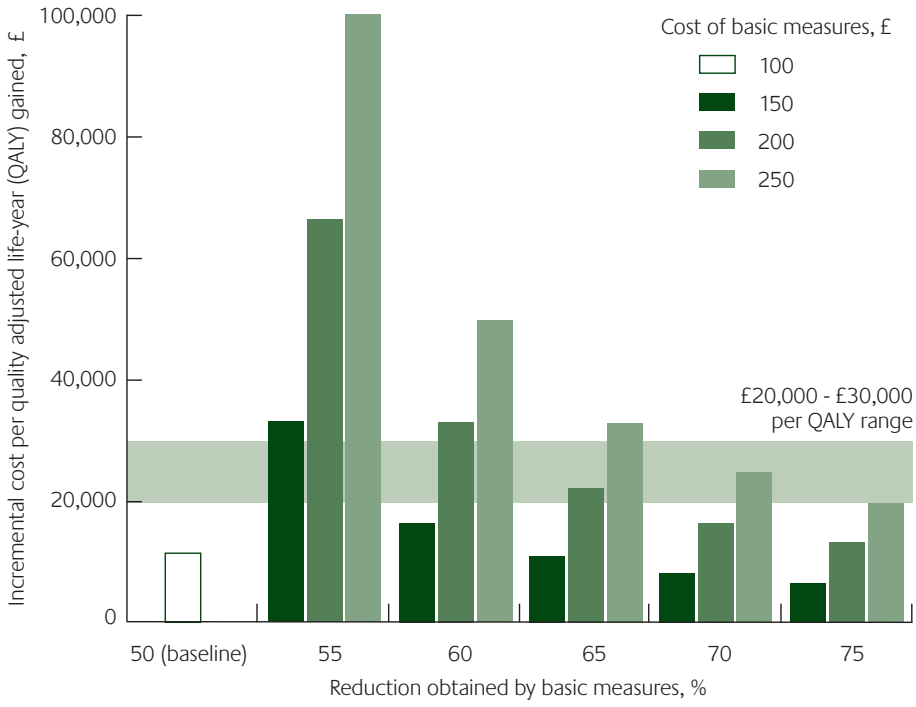

FIGURE M5 New homes: cost per QALY gained (discounted – societal) of installing basic preventive measures in new homes in all areas, as the cost of these measures and the reduction in radon obtained by them are varied

M3.3 Full preventive measures in new homes

Table M4 shows the outcomes, costs and cost-effectiveness of the existing full preventive measures policy in areas where, in the absence of any preventive measures, 10% of homes have measured radon concentrations above 200 Bq m^{-3}, ie where the mean long-term radon concentration would be 87 Bq m^{-3}. As this policy consists simply of installing basic preventive measures plus fitting a means of under-floor ventilation such as a sump and pipework, but without the installation of a fan to activate them, the effectiveness is similar to that of the basic measures policy while the cost is higher. In areas with these radon concentrations, the cost per QALY gained of basic measures alone would be £6700 using the direct estimate of lung cancer risk, when compared to no action (column 1), while the cost per QALY gained of installing full protective measures in such areas would be £8600 compared to no action (column 2). However, £100 of extra cost is being incurred for no extra benefit compared with the basic measures policy, and so the incremental cost-effectiveness of full preventive measures in such areas compared to the basic measures policy (column 3) is infinite.

TABLE M4 New homes: main estimates of effects, costs and cost-effectiveness for current full preventive measures policy in all new homes in an area where 10% of homes have measured radon concentrations above 200 Bq m^{-3}, based on the HPA methodology of considering one radon measurement of three months' duration with seasonal adjustment. This corresponds to an area with a mean long-term concentration of 87 Bq m^{-3}. Based on direct estimates of risk [(i)]

Current government policy in England and Wales requires full preventive measures – ie the installation of a membrane and also fitting a means of under-floor ventilation such as a sump but with no requirement to test the radon concentration or to activate the sump – in areas with mean long-term average radon concentrations at or above this level

	(1) Basic measures only versus no action	(2) Full preventive measures versus no action	(3) Incremental cost-effectiveness of full compared to basic measures
Initial			
Lifetime cumulative lung cancer risk (%)	6.71	6.71	0.00
Post-preventive action			
Lifetime cumulative lung cancer risk (%)	6.31	6.31	0.00
Health gain per household			
Lung cancer cases averted	0.010	0.010	0.000
Total life-years gained	0.13	0.13	0.00
Total life-years gained – discounted	0.07	0.07	0.00
Total QALYs gained	0.10	0.10	0.00
Total QALYs gained – discounted	0.05	0.05	0.00
Resource use and costs per household			
Radon prevention cost – discounted	£100	£200	£100
NHS lung cancer treatment costs averted – discounted	£48	£48	£0
Other NHS costs incurred by added life expectancy – discounted	£296	£296	£0
Net cost – discounted – societal	£348	£448	£100
Cost-effectiveness			
Cost per life-year gained – discounted	£5,219	£6,718	∞
Cost per QALY gained – discounted	£6,667	£8,581	∞

Note: (i) From studies of residential radon and lung cancer in Europe (Darby et al, 2005, 2006).

The idea underlying the full preventive measures policy is to facilitate the installation of active preventive measures at a later stage if the house proves to have a high radon concentration after the installation of basic preventive measures. A more appropriate policy would, therefore, include evaluation of the radon concentration after the installation of the basic measures and where necessary the activation of additional measures, and an evaluation of such a policy would consider the entire sequence of events. Table M5 reports the outcomes, costs and cost-effectiveness of a policy of installing full preventive measures in areas where, in the absence of basic preventive measures, 10% of homes would have measured radon concentrations above 200 Bq m^{-3}, then testing all homes after completion and occupation, to find those that still have measured radon levels above the current Action Level of 200 Bq m^{-3}, and finally installing electric fans in these homes. It should be noted that, although the mean long-term radon concentration would initially have been 87 Bq m^{-3} in the area, basic measures would have already been installed, reducing the radon levels by 50%. As a result, only 1.8% of homes in such areas would still be expected to have measured radon levels over 200 Bq m^{-3}. Using the direct risk estimate, the cost-effectiveness model predicts that the cumulative lifetime risk of lung cancer in homes still above 200 Bq m^{-3} would be 7.57%. After installation of electric fans, the lifetime risk falls to 6.07%. This is equivalent to a reduction of 0.04 lung cancer cases in a household of average size, which in turn is equivalent to 0.48 life-years gained, or 0.25 discounted life-years gained. Using the indirect estimate of risk, the lifetime risks of lung cancer are higher, and the total undiscounted life-years gained are 0.86.

As mentioned above, at the radon concentration considered, only 1.8% of homes are likely to have measured radon concentrations above 200 Bq m^{-3} after the installation of basic radon preventive measures. Therefore, 55 homes will have been fitted with sumps and pipework during construction for every home still above 200 Bq m^{-3}, at a total installation cost of £5486. In addition, 55 homes will have to be tested for every home found still to require radon remediation, at a further cost of £2304. Savings from lung cancer treatment costs averted, when discounted, reduce costs by £180, while additional costs to the health service from increased life expectancy add £1108, leaving a net cost of £10,450 per household remediating. The net cost is higher using the indirect risk estimate, mainly due to a higher estimate of additional life expectancy.

Combining the outcomes and costs reported above, the cost per QALY gained of this policy in areas where in the absence of basic measures approximately 10% of homes would have had measurements above 200 Bq m^{-3}, is £53,507 based on the direct estimate of risk. Using the indirect risk estimate, with higher lifetime lung cancer risks and therefore higher benefits from radon prevention, the cost per QALY gained falls to around £31,727. These figures are above the levels (£20,000–£30,000 per QALY gained) that might typically be considered the maximum willingness to pay when assessing alternative ways of improving health.

Tables M6(a) and (b) show the same analysis as in Table M5 for the direct and indirect estimates of risk, respectively, but varying both the Action Level and the targeted area in which the initial full preventive measures are installed during construction. It is evident that, using the direct estimate of risk, a policy targeted on areas with long-term mean radon concentration prior to installation of basic preventive measures would not be cost-effective at the current Action Level of 200 Bq m^{-3}, even in areas with mean radon concentrations as high as 100 Bq m^{-3}. Cost-effectiveness would, however, be improved if the

Action Level were reduced. If the Action Level were set at 100 or 50 Bq m^{-3} then the policy would become cost-effective in areas with mean radon concentrations of 90 Bq m^{-3} or above. Using the indirect estimate of risk, the existing policy is borderline cost-effective, but would be much more cost-effective if the Action Level were very much lower: assuming that active measures to reduce radon achieved the same proportional reduction in radon concentrations at 25 Bq m^{-3} as is known to be achievable above 200 Bq m^{-3}, then an Action Level of 25 Bq m^{-3} would be more cost-effective than the current value of 200 Bq m^{-3}, and it would also be cost-effective over a much wider proportion of the country. The above calculations assume that testing and installation and activation of fans is carried out with 100% compliance. If there was even moderate lack of compliance, then, based on the direct estimate of risk, the policy would not be cost-effective anywhere in the UK.

The need to focus on areas with high radon levels in order for the policy presented in Tables M5 and M6(a) and (b) to be cost-effective arises from the fact that basic preventive measures will already have halved radon levels, thereby reducing the numbers of homes likely still to have measurements above 200 Bq m^{-3} (from 10% to 1.8% in the example given in Table M5). This increases considerably the costs of detecting the remaining homes above the current Action Level, and also means that large numbers of homes (55 in this example) are having full preventive measures installed during construction for every one that will ultimately require further radon remediation. The high lifetime costs of the active measures, even when discounted to present values, are another factor making cost-effectiveness hard to achieve.

An alternative, therefore, would be to consider a policy in which only basic measures are installed during construction and then, in specified areas, all homes are tested once completed to find those that still have radon levels above 200 Bq m^{-3}, with subsequent installation of sumps, pipework and electric fans in those homes. The cost of retrofitting a sump will be higher than it would have been during construction, but the work would need to be done only in homes that remained over 200 Bq m^{-3}. Here it is assumed that a sump that costs £100 to install at the time of construction would cost more, around £700, to install at a later date. In addition, compliance may fall due to the added disruption involved: our calculations assume that it falls from 100% when sumps are pre-fitted to 60% when retrofitted. Table M6(c) shows the results for this policy, across a range of Action Levels and targeted areas. It is evident that the benefits of such a policy – ie avoiding the fitting during construction of sumps that will never be used – are more or less cancelled out by the higher costs of retrofitting sumps in those homes requiring them and by the likely reduction in compliance amongst householders due to the added disruption. If compliance fell to below our assumed value of 60%, the policy would be even less cost-effective. Thus the policy outlined in Table M6(c) provides no advantage over that described in Tables M6(a) and (b).

(text continues on page 207)

TABLE M5 New homes: main estimates of effects, costs and cost-effectiveness of a policy of installing full preventive measures (as currently defined) in areas where 10% of homes are likely to have measured radon concentrations above 200 Bq m^{-3} (ie areas with a mean long-term concentration of 87 Bq m^{-3}), then testing all new homes in such areas once completed to find those which still have radon levels above 200 Bq m^{-3}, and installing and activating electric fans in 100% of those homes.

Current government policy in England and Wales requires full preventive measures – ie the installation of a membrane and sump but with no requirement to test the radon concentration or to activate the sump – in areas with a mean long-term average radon concentration at or above this level

	Direct [(i)]	Indirect [(ii)]
Initial		
Lifetime cumulative lung cancer risk (%)	7.57	8.75
Post-remediation		
Lifetime cumulative lung cancer risk (%)	6.07	6.05
Health gain per household remediating		
Lung cancer cases averted	0.04	0.06
Total life-years gained	0.48	0.86
Total life-years gained – discounted	0.25	0.45
Total QALYs gained	0.37	0.67
Total QALYs gained – discounted	0.20	0.35
Resource use and costs per household remediating		
Number of sumps and pipework fitted during construction	55	55
Cost of fitting sumps and pipework during construction	£5,486	£5,486
Number of invitations to test	55	55
Invitation costs	£91	£91
Number of radon tests	55	55
Radon testing cost	£2,304	£2,304
Radon remediation cost – discounted	£1,642	£1,642
Sub-total: invitation, testing and remediation costs – discounted	£9,522	£9,522
NHS lung cancer treatment costs averted	£605	£1,093
NHS lung cancer treatment costs averted – discounted	£180	£325
Other NHS costs incurred by added life expectancy – discounted	£1,108	£2,003
Net cost – discounted – societal	£10,450	£11,199
Net cost – discounted – NHS	*£928*	*£1,677*
Net cost – discounted – households	*£9,522*	*£9,522*
Cost-effectiveness		
Cost per life-year gained – discounted	£41,891	£24,839
Cost per QALY gained – discounted – societal	**£53,507**	**£31,727 [(iii)]**
Cost per QALY gained – discounted – NHS	*£4,752*	*£4,752*
Cost per QALY gained – discounted – households	*£48,755*	*£26,974*

Notes
(i) From studies of residential radon and lung cancer in Europe (Darby et al, 2005, 2006).
(ii) From studies of radon-exposed miners (BEIR VI Committee, 1999).
(iii) Shaded value corresponds to current government policy in England and Wales.

TABLE M6

(a) New homes: effect on cost per quality adjusted life-year gained of varying both the targeted area and the Action Level within the targeted areas for the policy shown in Table M5. Results shown for direct estimate of radon risk

Targeted area (Bq m^{-3} long-term mean prior to installation of basic preventive measures)	Cost per quality adjusted life-year gained (discounted – societal)						
	Action Level (Bq m^{-3} measured value)[i,ii]						
	25 Bq m^{-3}	50 Bq m^{-3}	100 Bq m^{-3}	150 Bq m^{-3}	200 Bq m^{-3}	300 Bq m^{-3}	400 Bq m^{-3}
20 [iii]	**£185,400**	£697,600	£5,628,300	£24,606,300	£77,942,900	£460,322,100	£1,809,163,900
25	**£109,400**	£249,600	£1,393,000	£5,126,700	£14,447,500	£72,503,400	£253,918,600
30	**£82,700**	£134,700	£547,900	£1,757,500	£4,532,700	£20,158,200	£64,846,100
35	**£69,200**	£90,600	£280,100	£796,000	£1,906,800	£7,693,500	£23,133,700
36 [v]	**£67,200**	£85,000	£249,800	£693,500	£1,638,500	£6,493,300	£19,286,700
40	**£61,000**	£69,200	£169,400	£430,600	£966,200	£3,587,800	£10,197,500
45	**£55,300**	£57,000	£115,000	£263,500	£556,500	£1,919,500	£5,196,300
50	£51,000	**£49,300**	£84,900	£176,500	£352,100	£1,135,200	£2,942,600
52 [vi]	£49,500	**£46,800**	£76,000	£151,800	£295,600	£926,700	£2,360,400
55	£47,600	**£44,000**	£66,700	£126,800	£239,400	£724,200	£1,804,600
60	£44,900	**£40,200**	£54,900	£96,100	£172,200	£490,400	£1,177,900
64 [vii]	£43,000	**£38,000**	£48,500	£80,200	£138,400	£377,000	£882,000
65	£42,500	**£37,300**	£46,800	£76,100	£129,700	£348,400	£808,300
70	£40,500	**£35,000**	£41,000	£62,400	£101,400	£257,500	£578,000
75	£38,700	**£33,100**	£36,800	£52,700	£81,900	£196,700	£427,700
80	£37,100	**£31,500**	£33,500	£45,500	£67,900	£154,600	£325,800
85	£35,600	**£30,200**	£30,900	£40,100	£57,500	£124,500	£254,500
87 [viii] [iv]	£35,000	**£29,600**	£29,800	£38,000	**£53,500** [ix]	£113,000	£227,800
90	£34,300	£29,000	**£28,800**	£36,000	£49,700	£102,400	£203,200
95	£33,200	£28,000	**£27,100**	£32,700	£43,700	£85,800	£165,400
100	£32,100	£27,100	**£25,700**	£30,000	£39,000	£73,100	£136,900

TABLE M6 *continued*

(b) New homes: effect on cost per quality adjusted life-year gained of varying both the targeted area and the Action Level within the targeted areas for the policy shown in Table M5. Results shown for indirect estimate of radon risk

Targeted area (Bq m^{-3} long-term mean prior to installation of basic preventive measures)	Cost per quality adjusted life-year gained (discounted – societal)						
	Action Level (Bq m^{-3} measured value) [i,ii]						
	25 Bq m^{-3}	50 Bq m^{-3}	100 Bq m^{-3}	150 Bq m^{-3}	200 Bq m^{-3}	300 Bq m^{-3}	400 Bq m^{-3}
20 [iii]	**£104,100**	£386,100	£3,102,500	£13,567,300	£43,001,800	£254,285,200	£1,000,584,400
25	**£62,300**	£139,500	£769,700	£2,829,300	£7,975,600	£40,073,300	£140,521,100
30	**£47,600**	£76,300	£304,100	£971,600	£2,504,500	£11,147,700	£35,905,900
35	**£40,200**	£52,000	£156,500	£441,300	£1,055,100	£4,257,400	£12,816,200
36 [v]	**£39,100**	£49,000	£139,800	£384,800	£907,000	£3,593,800	£10,686,100
40	**£35,700**	£40,200	£95,500	£239,800	£535,800	£1,987,200	£5,652,800
45	**£32,600**	£33,500	£65,500	£147,600	£309,600	£1,064,500	£2,882,500
50	£30,200	**£29,300**	£49,000	£99,600	£196,700	£630,500	£1,633,700
52 [vi] [iv]	£29,400	**£27,900**	£44,000	£85,900	£165,500	£515,200	£1,311,100
55	£28,400	**£26,400**	£38,900	£72,100	£134,400	£403,100	£1,003,000
60	£26,800	**£24,300**	£32,400	£55,200	£97,300	£273,700	£655,600
64 [vii]	£25,800	**£23,000**	£28,900	£46,400	£78,700	£211,000	£491,500
65	£25,500	**£22,700**	£28,000	£44,100	£73,800	£195,100	£450,700
70	£24,400	**£21,400**	£24,800	£36,600	£58,200	£144,800	£322,900
75	£23,400	**£20,400**	£22,400	£31,200	£47,400	£111,100	£239,600
80	£22,500	**£19,500**	£20,600	£27,300	£39,700	£87,800	£183,100
85	£21,800	**£18,800**	£19,200	£24,300	£33,900	£71,100	£143,500
87 [viii]	£21,400	**£18,500**	£18,600	£23,100	**£31,700** [ix]	£64,800	£128,700
90	£21,100	£18,100	**£18,000**	£22,000	£29,600	£58,900	£115,000
95 [iv]	£20,400	£17,600	**£17,100**	£20,200	£26,300	£49,700	£94,000
100	£19,800	£17,100	**£16,300**	£18,700	£23,700	£42,600	£78,200

TABLE M6 *continued*

(c) New homes: cost per quality adjusted life-year gained across different targeted areas and Action Levels within the targeted areas for alternative 'full preventive measures' policy in which basic measures are installed in all new homes, which are then measured. Sumps, pipework and electric fans are then fitted only in those homes with measured radon levels above the Action Level, with 60% compliance. Results shown for direct estimate of radon risk

Targeted area (Bq m^{-3} long-term mean prior to installation of basic preventive measures)	Cost per quality adjusted life-year gained (discounted – societal)						
	Action Level (Bq m^{-3} measured value)[i,ii]						
	25 Bq m^{-3}	50 Bq m^{-3}	100 Bq m^{-3}	150 Bq m^{-3}	200 Bq m^{-3}	300 Bq m^{-3}	400 Bq m^{-3}
20 [iii]	**£151,000**	£390,400	£2,874,500	£12,480,300	£39,489,100	£233,137,600	£916,242,100
25	**£106,800**	£160,200	£727,500	£2,613,600	£7,331,300	£36,730,000	£128,604,200
30	**£89,000**	£99,600	£298,200	£906,300	£2,309,300	£10,219,800	£32,849,900
35	£78,800	**£75,400**	£161,400	£418,500	£978,800	£3,906,700	£11,724,700
36 [v]	£77,200	**£72,200**	£145,900	£366,500	£842,700	£3,298,800	£9,776,400
40	£71,900	**£63,000**	£104,500	£232,900	£501,900	£1,827,100	£5,173,000
45	£66,700	**£55,500**	£76,200	£147,700	£294,000	£981,900	£2,640,000
50	£62,500	**£50,500**	£60,300	£103,200	£190,100	£584,400	£1,498,400
52 [vi]	£60,900	**£48,800**	£55,600	£90,500	£161,400	£478,700	£1,203,500
55	£59,000	**£46,900**	£50,600	£77,600	£132,700	£376,000	£921,900
60	£56,000	£44,100	**£44,100**	£61,700	£98,400	£257,400	£604,400
64 [vii]	£54,000	£42,300	**£40,500**	£53,500	£81,100	£199,900	£454,400
65	£53,400	£41,800	**£39,600**	£51,300	£76,600	£185,300	£417,100
70	£51,000	£39,900	**£36,300**	£44,100	£62,100	£139,100	£300,300
75	£48,900	£38,300	**£33,700**	£38,900	£52,000	£108,200	£224,100
80	£47,000	£36,900	**£31,700**	£35,000	£44,700	£86,700	£172,400
85	£45,300	£35,700	**£30,100**	£32,100	£39,300	£71,400	£136,200
87 [viii]	£44,500	£35,100	**£29,400**	£30,900	£37,200	£65,500	£122,600
90	£43,700	£34,600	**£28,800**	£29,800	£35,200	£60,000	£110,100
95	£42,300	£33,600	**£27,700**	£27,900	£32,000	£51,500	£90,800
100 [iv]	£40,900	£32,700	£26,700	**£26,400**	£29,400	£45,000	£76,300

Notes to Table M6

(i) Based on the current HPA methodology of considering one three-month radon measurement with seasonal adjustment.

(ii) There is little information available at present on the percentage reduction in radon concentration that would result from installing a fan with pre-remediation concentrations of 25 or 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher pre-installation concentrations, ie 90%.

(iii) Entries in bold denote the most cost-effective Action Level for each targeted area.

(iv) Entries between the lines have cost per quality adjusted life-year gained between £20,000 and £30,000.

(v) That is, 1% of measured values >200 Bq m^{-3}.

(vi) That is, 3% >200 Bq m^{-3}.

(vii) That is, 5% >200 Bq m^{-3}.

(viii) That is, 10% >200 Bq m^{-3}.

(ix) Shaded area corresponds to the current UK policy for full preventive measures in new homes.

M4 Results for existing homes

M4.1 Remediation of existing homes

Table M7 reports central estimates of the model concerning existing homes, when invitations are targeted on areas in which approximately 5% of homes have measured radon levels above 200 Bq m^{-3}. Such areas will have a mean long-term radon concentration of 64 Bq m^{-3}. Using the direct risk estimate, the cost-effectiveness model predicts that the cumulative lifetime risk of lung cancer at pre-remediation radon concentrations in such areas is 7.82%. Post-remediation, the lifetime risk falls to 6.19%. This is equivalent to a reduction of 0.04 lung cancer cases in a household of average size, which in turn is equivalent to 0.52 life-years gained, or 0.27 discounted life-years gained. Using the indirect risk estimate, the lifetime risks of lung cancer are higher, and the total undiscounted life-years gained are also higher at 0.93.

The mean indoor radon concentration in an area determines the average number of homes that have to be tested to identify one over the Action Level. At a long-term average of 64 Bq m^{-3}, 333 invitations to test will result in 100 homes tested, five found to be above 200 Bq m^{-3}, and one remediating. The cost of the invitations is £550 and the cost of testing is £4200. These costs, together with remediation costs, come to a discounted total of £6801. Against this, around £195 is saved from the averted lung cancer treatment costs and £1203 is incurred in health care costs during added life expectancy using the direct estimate of lung cancer risk, and consequently the net cost is £7809 per household remediating. A slightly higher net cost of £8620 using the indirect risk estimate arises from the slightly higher predicted numbers of lung cancer cases averted and hence additional health care costs during increased life expectancy.

Combining the outcomes and costs reported above, the cost per quality adjusted life-year gained is £36,800 using the direct risk estimate. This figure is above the level (£20,000–£30,000 per QALY gained) that might typically be considered the maximum willingness to pay when assessing alternative ways of improving health. If the calculation is repeated using the indirect risk estimate, the cost per quality adjusted life-year gained is £22,500, ie within the NICE ceiling range of £20,000–£30,000.

M4.2 Remediation of existing homes under different assumptions

Figure M6 shows the main cost-effectiveness results for existing home remediation with the current Action Level of 200 Bq m^{-3}, but varying the mean radon concentration in the area of interest. As the mean radon concentration in an area decreases, the proportion of homes expected to be above 200 Bq m^{-3} falls, and so the cost per QALY increases, and vice versa.

Table M8(a) shows how the cost per QALY gained of remediating existing homes varies as both the mean radon concentration in the targeted area and the Action Level are varied. The results are shown using the direct risk estimate, and Table M8(b) shows the same information based on the indirect risk estimate. The current HPA recommendation is that people living in areas where at least 1% of homes have measured values above 200 Bq m^{-3} (ie corresponding to a mean long-term average of at least 36 Bq m^{-3}) have their homes measured. However, the cost-effectiveness ratios for areas with a long-term mean of 36 Bq m^{-3} are £154,700 per QALY based on the direct estimate of risk and £87,700 based on the indirect estimate of risk, both substantially above the current NICE ceiling of £20,000–£30,000 per QALY.

TABLE M7 Existing homes: main estimates of effects, costs and cost-effectiveness for a policy of inviting householders to test in areas where 5% of homes are likely to have measured radon concentrations above 200 Bq m^{-3}, and recommending they remediate if measurements are above the current Action Level of 200 Bq m^{-3} (assuming 30% of households accept invitation to test and 20% found to be over Action Level agree to remediate)

This reflects current government policy in England. Measured radon levels based on the current HPA methodology of one radon measurement of three months' duration with seasonal adjustment. Areas where 5% of homes have measurements over 200 Bq m^{-3} have a mean long-term radon concentration of 64 Bq m^{-3}

	Radon risk estimate	
	Direct [(i)]	Indirect [(ii)]
Initial		
Lifetime cumulative risk of lung cancer death (% per person)	7.82	9.20
Post-remediation		
Lifetime cumulative risk of lung cancer death (% per person)	6.19	6.27
Health gain per household remediating		
Lung cancer cases averted	0.04	0.07
Total life-years gained	0.52	0.93
Total life-years gained – discounted	0.27	0.49
Total QALYs gained	0.40	0.73
Total QALYs gained – discounted	0.21	0.38
Resource use and costs per household remediating		
Number of invitations to test	333	333
Invitation costs	£550	£550
Number of radon tests	100	100
Radon testing cost	£4,200	£4,200
Radon remediation cost – discounted	£2,051	£2,051
Sub-total: invitation, testing and remediation costs – discounted	£6,801	£6,801
NHS lung cancer treatment costs averted	£656	£1,185
NHS lung cancer treatment costs averted – discounted	£195	£353
Other NHS costs incurred by added life expectancy – discounted	£1,203	£2,171
Net cost – discounted – societal	£7,809	£8,620
Net cost – discounted – NHS	*£1,008*	*£1,819*
Net cost – discounted – HPA	*£4,750*	*£4,750*
Net cost – discounted – households	*£2,051*	*£2,051*
Cost-effectiveness		
Cost per life-year gained – discounted	£28,833	£17,633
Cost per QALY gained – discounted – societal	**£36,828**	**£22,523 [(iii)]**
Cost per QALY gained – discounted – NHS	*£4,752*	*£4,752*
Cost per QALY gained – discounted – HPA	*£22,404*	*£12,412*
Cost per QALY gained – discounted – households	*£9,673*	*£5,359*

Notes
(i) From the European study of residential radon and lung cancer (Darby et al, 2005, 2006).
(ii) From studies of radon-exposed miners (BEIR VI Committee, 1999).
(iii) Shaded value corresponds to current government policy in England.

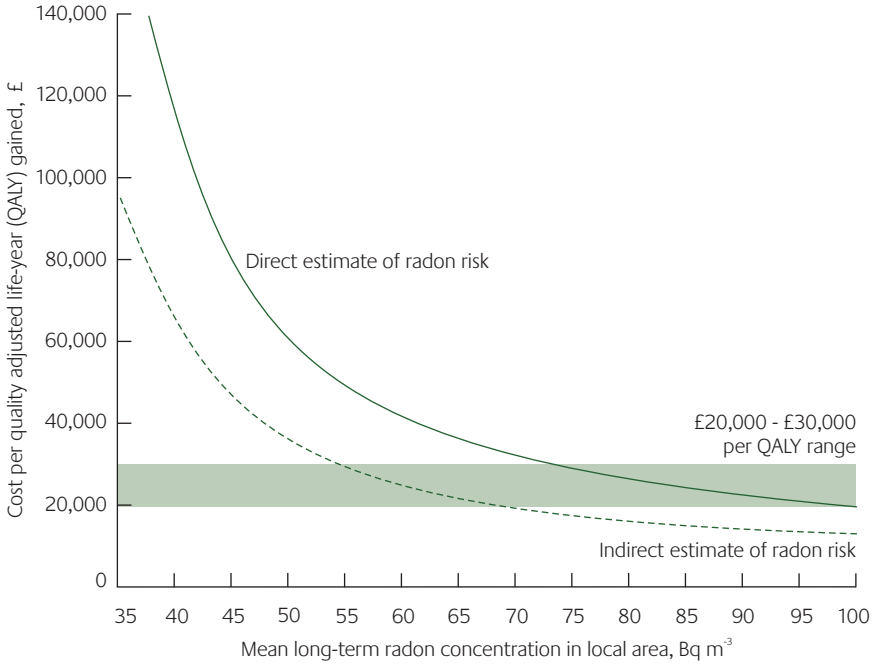

FIGURE M6 Existing homes: relation between long-term mean radon concentration in an area (Bq m^{-3}) and cost per quality adjusted life-year gained (discounted – societal), for invitations to test and a recommendation to remediate for measurements above the current Action Level of 200 Bq m^{-3}. Results shown for direct estimate (ie based on European pooling study) and indirect estimate (ie based on BEIR VI) of radon risk

Current government policy for England concerning existing homes targets areas with a long-term mean of at least 64 Bq m^{-3} (ie at least 5% of measurements are above 200 Bq m^{-3}). The cost per QALY based on the direct estimates of risk is £36,800, which is also above the current NICE ceiling. Policy for existing homes could be more cost-effective if the current Action Level were reduced. For example, if it were reduced to 100 Bq m^{-3}, it would then become cost-effective to apply the policy in areas with mean radon concentrations down to 60 Bq m^{-3}. Using indirect risk estimates, the policy also becomes more cost-effective if the Action Level is reduced from its current value of 200 Bq m^{-3}, and with a lower Action Level it would become cost-effective in areas with lower long-term mean radon concentrations.

Table M8(c) shows, for the direct risk estimate, that the current policy concerning existing homes could be cost-effective at much lower mean long-term radon concentrations, and therefore over a much larger geographical area, if the proportion of people accepting an initial invitation to test could be increased from 30% to 60%, and the proportion agreeing to remediate could be increased from 20% to 50%, with no change in overall costs. In practice this would require more efficient or more targeted methods of persuasion, or a transfer of at least some of the remediation costs currently borne by householders to another party. If these acceptance and remediation rates could be achieved, then based on the direct risk estimate, and with an Action Level of 100 Bq m^{-3}, the policy would be borderline cost-effective in areas with a long-term mean radon concentration of 45 Bq m^{-3}. *(text continues on page 215)*

TABLE M8

(a) Existing homes: effect on cost per quality adjusted life-year gained of varying both the targeted area and the Action Level within the targeted area. Results shown for direct estimate of radon risk

Targeted area (Bq m^{-3} long-term mean)		Cost per quality adjusted life-year gained (discounted – societal) Action Level (Bq m^{-3} measured value)[i,ii]						
		25 Bq m^{-3}	50 Bq m^{-3}	100 Bq m^{-3}	150 Bq m^{-3}	200 Bq m^{-3}	300 Bq m^{-3}	400 Bq m^{-3}
20	[iii]	**£85,200**	£105,600	£285,200	£744,300	£1,682,500	£6,271,900	£17,840,700
25		**£69,800**	£72,000	£138,100	£300,100	£608,500	£1,980,300	£5,145,100
30		£60,600	**£56,900**	£86,100	£159,700	£293,800	£851,800	£2,056,100
35		£54,100	**£48,500**	£62,200	£100,900	£170,100	£444,300	£1,005,900
36 [v]		£53,100	**£47,200**	£58,900	£93,400	£154,700 [ix]	£395,900	£885,400
40		£49,300	**£43,000**	£49,200	£71,600	£111,500	£264,300	£564,600
45		£45,400	**£39,100**	£41,400	£55,100	£79,900	£173,000	£350,000
50		£42,200	£36,200	**£36,200**	£44,900	£61,200	£121,700	£233,900
52 [vi]		£41,000	£35,100	**£34,400**	£41,600	£55,400	£106,500	£200,200
55		£39,500	£33,800	**£32,500**	£38,100	£49,300	£90,600	£165,800
60		£37,200	£31,900	**£29,800**	£33,400	£41,300	£70,600	£123,300
64 [vii]		£35,600	£30,700	**£28,200**	£30,700	**£36,800** [x]	£60,000	£101,100
65		£35,200	£30,300	**£27,700**	£30,000	£35,600	£57,100	£95,300
70		£33,400	£28,900	**£26,000**	£27,400	£31,500	£47,600	£76,100
75		£31,800	£27,700	**£24,600**	£25,300	£28,400	£40,700	£62,500
80		£30,400	£26,600	**£23,500**	£23,700	£25,900	£35,500	£52,500
85	[iv]	£29,100	£25,600	£22,500	**£22,400**	£24,000	£31,500	£45,000
87 [viii]		£28,500	£25,200	£22,100	**£21,800**	£23,200	£29,900	£42,000
90		£27,900	£24,700	£21,700	**£21,300**	£22,400	£28,400	£39,200
95		£26,800	£23,900	£20,900	**£20,300**	£21,100	£25,900	£34,700
100	[iv]	£25,900	£23,200	£20,300	**£19,500**	£20,100	£23,900	£31,200

TABLE M8 *continued*

(b) Existing homes: effect on cost per quality adjusted life-year gained of varying both the targeted area and the Action Level within the targeted area. Results shown for indirect estimate of radon risk.

Targeted area ($Bq\ m^{-3}$ long-term mean)	Cost per quality adjusted life-year gained (discounted – societal)						
	Action Level ($Bq\ m^{-3}$ measured value)[i,ii]						
	25 $Bq\ m^{-3}$	50 $Bq\ m^{-3}$	100 $Bq\ m^{-3}$	150 $Bq\ m^{-3}$	200 $Bq\ m^{-3}$	300 $Bq\ m^{-3}$	400 $Bq\ m^{-3}$
20 [iii]	**£49,000**	£60,300	£159,400	£412,900	£931,700	£3,473,300	£9,891,900
25	**£40,600**	£41,800	£78,300	£167,800	£338,400	£1,098,700	£2,856,100
30	£35,500	**£33,500**	£49,600	£90,300	£164,600	£474,100	£1,143,300
35	£31,900	**£28,800**	£36,400	£57,900	£96,200	£248,400	£560,700
36 [v]	£31,300	**£28,100**	£34,600	£53,700	£87,700 [ix]	£221,500	£493,800
40 [iv]	£29,300	**£25,800**	£29,300	£41,700	£63,800	£148,700	£315,800
45	£27,100	**£23,700**	£25,000	£32,600	£46,400	£98,100	£196,600
50	£25,400	£22,100	**£22,100**	£26,900	£36,000	£69,700	£132,200
52 [vi]	£24,700	£21,500	**£21,100**	£25,100	£32,800	£61,200	£113,500
55	£23,900	£20,800	**£20,100**	£23,200	£29,400	£52,400	£94,400
60	£22,600	£19,700	**£18,600**	£20,600	£25,000	£41,300	£70,700
64 [vii]	£21,800	£19,000	**£17,700**	£19,100	**£22,500** [x]	£35,400	£58,400
65	£21,500	£18,800	**£17,400**	£18,700	£21,900	£33,800	£55,200
70 [iv]	£20,500	£18,100	**£16,500**	£17,300	£19,600	£28,600	£44,500
75	£19,700	£17,400	**£15,700**	£16,100	£17,800	£24,700	£36,900
80	£18,900	£16,800	**£15,100**	£15,200	£16,500	£21,800	£31,300
85	£18,200	£16,300	£14,600	**£14,500**	£15,400	£19,600	£27,200
87 [viii]	£17,800	£16,000	£14,300	**£14,200**	£15,000	£18,800	£25,500
90	£17,500	£15,800	£14,100	**£13,900**	£14,600	£17,900	£24,000
95	£16,900	£15,300	£13,700	**£13,400**	£13,800	£16,500	£21,500
100	£16,400	£14,900	£13,300	**£12,900**	£13,300	£15,400	£19,500

TABLE M8 *continued*

(c) Existing homes: effect on cost per quality adjusted life-year gained of varying both the targeted area and the Action Level within the targeted area. Results shown for direct estimate of radon risk, with invitation acceptance rates increased from 30% to 60%, and remediation rates increased from 20% to 50%

Targeted area ($Bq\ m^{-3}$ long-term mean)	Cost per quality adjusted life-year gained (discounted – societal)						
	Action Level ($Bq\ m^{-3}$ measured value)[i,ii]						
	25 $Bq\ m^{-3}$	50 $Bq\ m^{-3}$	100 $Bq\ m^{-3}$	150 $Bq\ m^{-3}$	200 $Bq\ m^{-3}$	300 $Bq\ m^{-3}$	400 $Bq\ m^{-3}$
20 [iii]	£69,700	**£65,700**	£125,100	£294,600	£646,200	£2,373,400	£6,731,700
25	£60,000	**£50,900**	£68,400	£126,300	£240,700	£755,600	£1,947,100
30	£53,500	**£43,500**	£47,900	£72,700	£121,600	£330,000	£782,700
35	£48,600	£39,000	**£38,200**	£50,100	£74,600	£176,100	£386,700
36 [v]	£47,800	£38,300	**£36,800**	£47,100	£68,700 [ix]	£157,800	£341,200
40	£44,800	£35,800	**£32,700**	£38,600	£52,100	£108,000	£220,200
45	£41,600	£33,400	**£29,200**	£32,000	£39,900	£73,400	£139,200
50	£38,900	£31,500	**£26,700**	£27,800	£32,600	£53,900	£95,300
52 [vi]	£37,800	£30,700	**£25,900**	£26,400	£30,300	£48,100	£82,500
55	£36,500	£29,800	£25,000	**£25,000**	£27,900	£42,000	£69,500
60	£34,500	£28,400	£23,600	**£22,900**	£24,700	£34,300	£53,400
64 [vii]	£33,100	£27,500	£22,700	**£21,700**	£22,900	£30,200	£44,900
65	£32,700	£27,200	£22,400	**£21,400**	£22,400	£29,100	£42,700
70	£31,100	£26,100	£21,500	**£20,200**	£20,600	£25,400	£35,400
75 [iv]	£29,700	£25,200	£20,700	**£19,200**	£19,300	£22,700	£30,100
80	£28,400	£24,300	£20,000	£18,400	**£18,300**	£20,600	£26,300
85	£27,300	£23,500	£19,400	£17,800	**£17,400**	£19,000	£23,400
87 [viii]	£26,800	£23,100	£19,100	£17,500	**£17,000**	£18,400	£22,300
90	£26,200	£22,800	£18,900	£17,200	**£16,700**	£17,800	£21,200
95	£25,300	£22,100	£18,400	£16,700	**£16,100**	£16,800	£19,400
100 [iv]	£24,400	£21,500	£17,900	£16,300	**£15,600**	£15,900	£18,000

Notes to Table M8

(i) Based on the current HPA methodology of considering one three-month radon measurement with seasonal adjustment.

(ii) There is little information available at present on the percentage reduction in radon concentration that would result from installing a range of remediation measures with pre-remediation concentrations of 25 or 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher pre-installation concentrations, ie 85%.

(iii) Entries in bold denote the most cost-effective Action Level for each targeted area.

(iv) Entries between the lines have cost per quality adjusted life-year gained between £20,000 and £30,000.

(v) That is, 1% of measured values >200 Bq m^{-3}.

(vi) That is, 3% >200 Bq m^{-3}.

(vii) That is, 5% >200 Bq m^{-3}.

(viii) That is, 10% >200 Bq m^{-3}.

(ix) The current HPA recommendation for the UK is that people living in areas where 1% of more of homes have radon measurements above 200 Bq m^{-3} measure the radon in their home and carry out remediation if the measurement is above 200 Bq m^{-3}.

(x) Shaded area corresponds to the current policy in England for surveys of existing homes.

TABLE M9 Existing homes: effects of reducing Action Level from 200 Bq m^{-3} to 150, 100, 50 or 25 Bq m^{-3} or increasing it to 300 or 400 Bq m^{-3}
Results shown for a notional group of 1000 homes in an area where the long-term mean radon concentration is 60 Bq m^{-3}. Calculations based on direct estimate of radon risk

	Action Level (Bq m^{-3} measured value) [i,ii]						
	25 Bq m^{-3}	50 Bq m^{-3}	100 Bq m^{-3}	150 Bq m^{-3}	200 Bq m^{-3}	300 Bq m^{-3}	400 Bq m^{-3}
Number invited	1000	1000	1000	1000	1000	1000	1000
Number agree to test	300	300	300	300	300	300	300
Number found over Action Level	229	134	52	24	13	5	2
Number decide to remediate	46	27	10	5	3	1	<1
Invitation costs	£1,650	£1,650	£1,650	£1,650	£1,650	£1,650	£1,650
Radon testing costs	£12,600	£12,600	£12,600	£12,600	£12,600	£12,600	£12,600
Radon remediation cost – discounted	£94,096	£54,949	£21,448	£10,032	£5,298	£1,864	£800
NHS lung cancer treatment costs averted – discounted	£3,080	£2,348	£1,314	£781	£493	£225	£117
Other NHS costs during added life expectancy – discounted	£18,959	£14,455	£8,090	£4,808	£3,037	£1,388	£720
Net cost – discounted – societal	£124,225	£81,306	£42,473	£28,309	£22,091	£17,276	£15,653
Total life-years gained – discounted	4.27	3.25	1.82	1.08	0.68	0.31	0.16
Total quality adjusted life-years gained – discounted	3.34	2.55	1.43	0.85	0.54	0.24	0.13
Cost per life-year gained – discounted	£29,106	£24,985	£23,322	£26,154	£32,316	£55,302	£96,529
Cost per QALY gained – discounted – societal	£37,177	£31,914	£29,789	£33,407	£41,278	£70,638	£123,298

Incremental results: ie additional cost, and additional effects, and ratio of additional costs to additional effects when moving from one action level to lower level:

	25 v 50 Bq m^{-3}	50 v 100 Bq m^{-3}	100 v 150 Bq m^{-3}	150 v 200 Bq m^{-3}	200 v 300 Bq m^{-3}	300 v 400 Bq m^{-3}
Incremental cost compared to higher AL	£42,918	£38,833	£14,164	£6,218	£4,815	£1,623
Incremental QALYs compared to higher AL	0.79	1.12	0.58	0.31	0.29	0.12
Incremental cost per QALY gained compared to higher AL	£54,070	£34,614	£24,490	£19,915	£16,568	£13,801

Notes

(i) Based on the current HPA methodology of considering one three-month radon measurement with seasonal adjustment.

(ii) There is little information available at present on the percentage reduction in radon concentration that would result from installing a range of remediation measures with pre-remediation concentrations of 25 or 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher pre-installation concentrations, ie 85%.

Table M9 shows in more detail the effect of altering the Action Level from its current value of 200 Bq m^{-3} in an area with a long-term mean radon concentration of 60 Bq m^{-3}. It is assumed that other parameters do not change: for example, the proportion of households over each Action Level that decide to remediate is held constant, as is the assumed cost of remediating and the proportional reduction in radon concentration achieved by remediation. These assumptions may not hold in practice – for example, propensity to remediate could either decline or increase at lower Action Levels – but there is little evidence in this area. As the Action Level increases above 200 Bq m^{-3}, cost-effectiveness deteriorates. In contrast, as the Action Level falls from 200 Bq m^{-3}, the cost per QALY gained initially falls, as the health benefits obtained increase more rapidly than the costs. At 100 Bq m^{-3} the cost per QALY gained has fallen to £29,800, but thereafter it begins to rise as the Action Level is further reduced. Where the upper part of the table shows the costs and effects of each policy against a comparator of no action, the lower part recalculates the same figures to show the incremental costs and benefits of each Action Level against the next highest. The same data are plotted in Figure M7. Again it is clear that, starting from an Action Level of 400 Bq m^{-3}, it would be cost-effective to move to an Action Level lower than

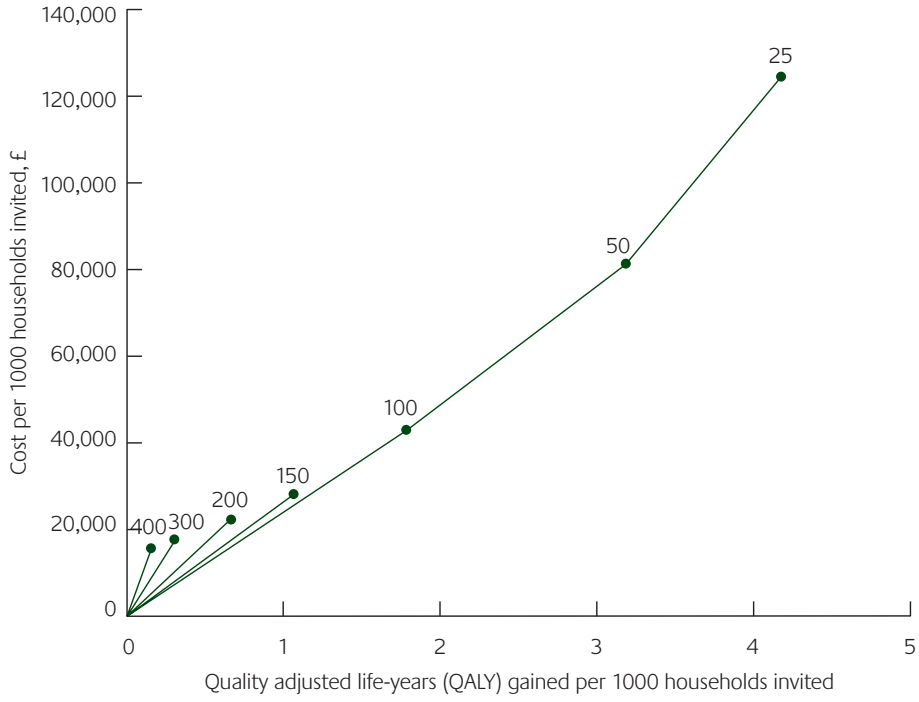

FIGURE M7 Cost-effectiveness plane showing net costs and QALYs gained from inviting 1000 existing households to test for radon, at seven different Action Levels with all other parameter values as in Table M9. Results shown for direct estimate of radon risk

For example, the incremental cost per incremental QALY gained for an Action Level of 100 Bq m^{-3} compared to 150 Bq m^{-3} is £24,490 [ie (£42,473 – £28,309)/(1.43 – 0.85) from Table M9]

200 Bq m^{-3} but, in an area with a long-term mean radon concentration of 60 Bq m^{-3}, the incremental cost-effectiveness of moving below an Action Level of 100 Bq m^{-3} is likely to be above the ceiling range of £20,000–£30,000 per QALY gained.

Figures M8(a) and (b) summarise the results from one-way sensitivity analyses of 11 parameters and assumptions using direct and indirect risk estimates, respectively, from a baseline of invitations to test and a recommendation to remediate for measurements above an Action Level of 100 Bq m^{-3} for an area with a long-term radon concentration of 60 Bq m^{-3}. It is evident that the results are particularly sensitive to the risk of radon-induced lung cancer: in comparison with the baseline estimate of £29,789 per QALY gained when the lung cancer risk increases by 16% per 100 Bq m^{-3}, an increase of only 5% – the lower 95% confidence limit from the European pooling study (Darby et al, 2005) – raises the cost per QALY gained to £82,800, while an increase of 31% – the upper 95% confidence limit – lowers the cost per QALY gained to £18,100.

Changes in the proportion of households over the current Action Level that decide to remediate also have a substantial impact on the results. From a baseline assumption that 20% of householders with homes found to be over the Action Level decide to remediate, increasing the remediation rate to 50% reduces the cost per QALY gained to £23,800. If this remediation rate were pushed higher, as might be achieved by, for example, transferring all or part of the cost of remediation from the householder to a public agency, the cost-effectiveness would be substantially enhanced: at an 80% remediation rate, for example, the cost per QALY gained would fall to £17,000 per QALY gained.

The results are also sensitive to alternative assumptions concerning the discount rate: at 3.5% for costs and 0% for outcomes, the cost-effectiveness falls to £20,200 per QALY gained; at a 6% discount rate for costs and outcomes, the cost-effectiveness ratio rises to £63,500. Assuming that the latency period before lung cancer risk falls in 20 years (ie spreading the benefit from radon reduction over years 21–100 instead of evenly over years 1–100) pushes the cost per QALY gained up from £29,800 to £35,700.

Changes in the baseline unit cost of invitations and the treatment costs of lung cancer have little effect on the results.

Figures M9(a) and (b) show the results of a probabilistic sensitivity analysis, in which all key variables are permitted to vary simultaneously across specified distributions, and the costs and QALYs gained from radon remediation in existing homes are repeatedly observed and recorded during 10,000 simulations: each plotted point on the figure therefore has a cost and effect coordinate and an associated cost-effectiveness ratio, equivalent to the slope of a line between that point and the origin. The baseline estimate of £29,789 per QALY gained in the direct risk estimate results is shown as a line on the figure, at which point approximately 50% of simulations are above and 50% below the line. Figure M10 extends this by showing a cost-effectiveness acceptability curve, which plots the probability that the cost per QALY gained of radon remediation in existing homes is cost-effective, as the decision-maker's willingness to pay for health gain is varied from £0 to £100,000 per QALY. In this instance, the probability that the intervention is cost-effective when decision-makers are prepared to spend up to £30,000 per QALY gained is approximately 54% using direct risk estimation and 97% using indirect risk estimation.

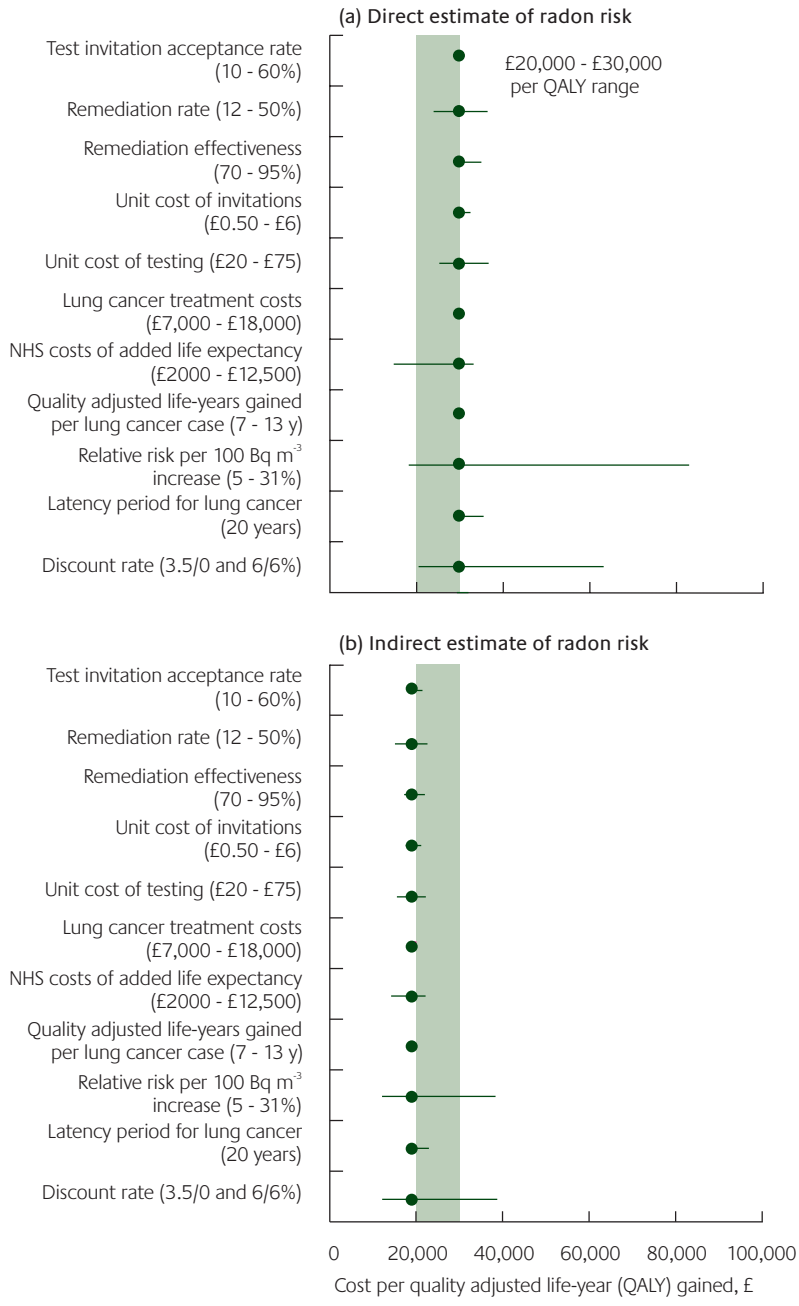

FIGURE M8 Existing homes: sensitivity of results to changes in main parameter values of results in Table M6, ie invitations to test and a recommendation to remediate for measurements above an Action Level of 100 Bq m^{-3} for an area with a long-term radon concentration of 60 Bq m^{-3}: (a) direct estimate of radon risk and (b) indirect estimate of radon risk

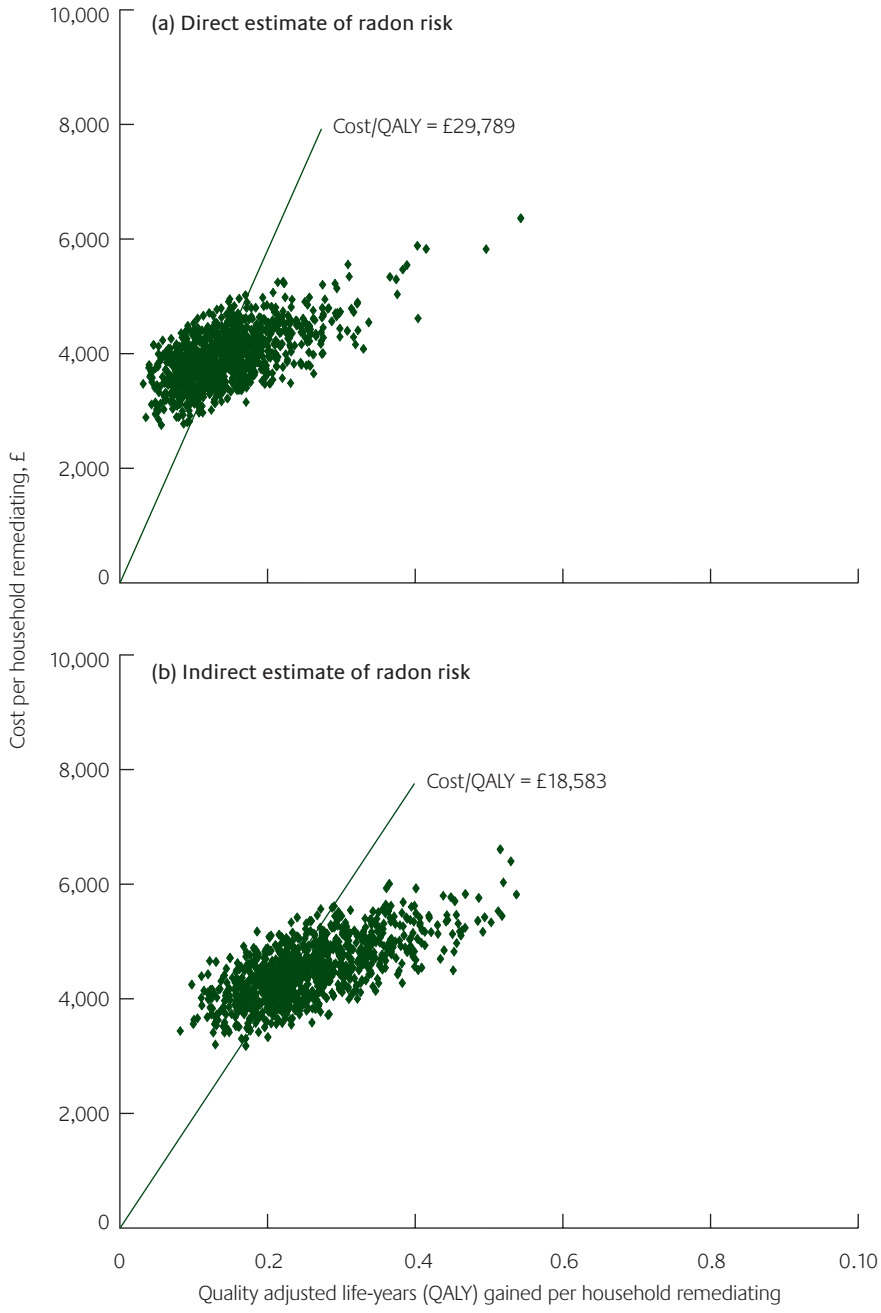

FIGURE M9 Cost-effectiveness plane showing uncertainty around cost and QALYs gained from radon remediation in existing homes: 10,000 simulations allowing all key variables to vary simultaneously and independently, with an Action Level of 100 Bq m^{-3} and in a targeted area with a long-term mean radon concentration of 60 Bq m^{-3}: (a) direct estimate of radon risk and (b) indirect estimate of radon risk

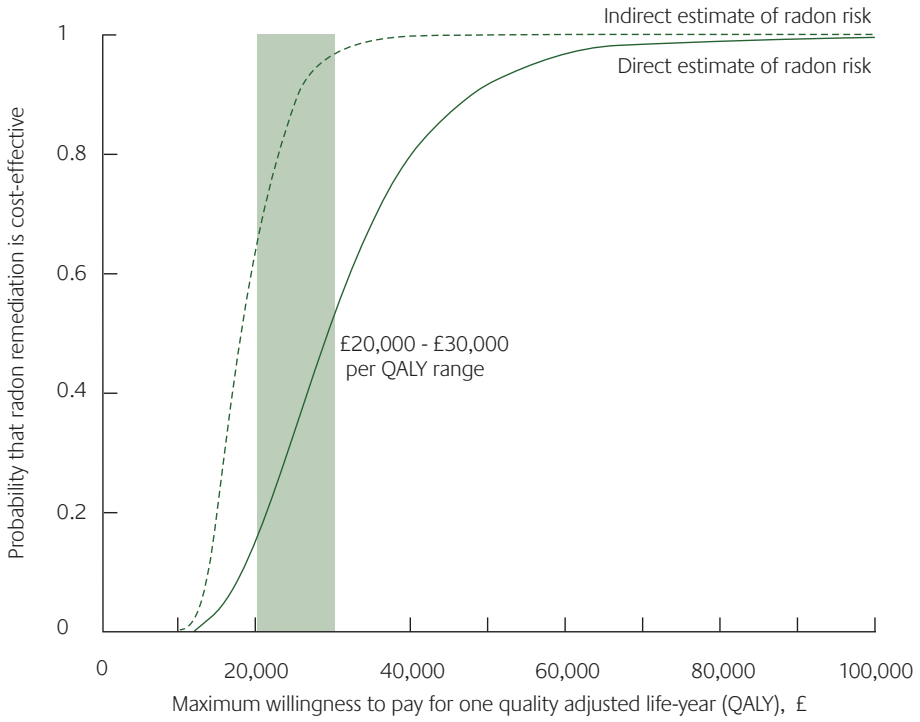

FIGURE M10 Existing homes: cost-effectiveness acceptability curve for the data in Figure M9, showing probability that the cost per QALY gained of radon remediation in existing homes is cost-effective, as a function of a decision-maker's willingness to pay for health gain

M4.3 Remediation of homes according to smoking status

Table M10 shows the cost-effectiveness of invitations to existing householders in areas with a long-term mean radon concentration of 60 Bq m^{-3} to test and remediate if their measured radon level is over an Action Level of 100 Bq m^{-3}, both for the entire population and also under the hypothetical assumption that the household consisted entirely of never-smokers (ie those with the lowest lung cancer risk for a given radon concentration) or entirely of current cigarette smokers (ie those with the highest lung cancer risk for a given radon concentration). It is evident that remediation of homes where measurements are above 100 Bq m^{-3} is likely to be cost-effective for households consisting of current cigarette smokers (£13,700 per QALY gained), but not for households consisting of never-smokers (£173,000 per QALY gained). It should be noted that these calculations assume that the smoking status in a home is held constant for the entire length of time considered in the calculation, ie 100 years. In practice, smoking status may be unchanged over long periods for a given household, but this is less likely to be the case for a given house which is likely to be occupied over a long period by several different households.

TABLE M10 Existing homes: lung cancer risk, remediation costs and cost per life-year and quality adjusted life-year gained for the total population, and for households consisting entirely of never-smokers or current cigarette smokers, based on invitations to existing households in areas with a long-term mean radon concentration of 60 Bq m^{-3} to test and remediate if their measured radon level is over an Action Level of 100 Bq m^{-3}, ie results shown for direct estimate of radon risk (assuming 30% of households accept invitation to test and 20% found to be over 100 Bq m^{-3} agree to remediate)

	Household of:		
	Population prevalence of smoking [(i)]	Never-smokers only [(ii)]	Current cigarette smokers only [(iii)]
Lifetime cumulative lung cancer risk (%)			
Initial	7.13	0.94	28.52
Post-remediation	6.09	0.80	24.81
Health gain per household remediating			
Lung cancer cases averted	0.03	<0.003	0.09
Total QALYs gained [(iv)]	0.14	0.02	0.35
Resource use and costs per household remediating			
Invitation, testing and remediation costs [(iv)]	£3,414	£3,414	£3,414
NHS lung cancer treatment costs averted [(iv)]	£126	£17	£447
Other NHS costs incurred by added life expectancy [(iv)]	£774	£110	£1,870
Net cost	£4,062	£3,506	£4,836
Cost-effectiveness			
Cost per quality adjusted life-year gained – societal	£29,789	£173,720	£13,727
Cost per QALY gained – discounted – NHS	*£4,752*	*£4,590*	*£4,037*
Cost per QALY gained – discounted – HPA	*£9,995*	*£67,514*	*£3,868*
Cost per QALY gained – discounted – households	*£15,043*	*£101,616*	*£5,822*

Notes

(i) Calculated using age- and sex-specific lung cancer mortality rates for entire population of the UK.

(ii) Calculated using age- and sex-specific lung cancer mortality rates for a population of lifelong non-smokers (Thun et al, 2006).

(iii) Calculated using age-and sex-specific lung cancer mortality rates for current cigarette smokers in the UK. These rates were derived from observed age- and sex-specific lung cancer death rates for 2005 for the total UK population, estimates of lung cancer death rates for lifelong non-smokers (Thun et al, 2006), estimates of the relative risks relative risks for UK men with differing smoking habits relative to continuing cigarette smokers (Peto et al, 2000), and estimates of the proportion of the population according to smoking status for categories of age and sex (ONS, 2006a, and Professor M Jarvis, University College, London: personal communication).

(iv) Discounted.

Table M11 shows the lowest mean radon level in a targeted area at which the current policy of remediation of existing homes – ie as in Table M8(a) – becomes cost-effective, for the entire population, for never-smokers and for current cigarette smokers. It also shows the Action Level that is most cost-effective for that targeted area. For the entire population and for current cigarette smokers, it would be possible to lower the current Action Level and widen the currently targeted areas and still be within acceptable cost-effectiveness limits. For never-smokers, cost-effective remediation would be possible only if the targeted areas had high radon levels compared to the existing policy and if, in addition, the Action Level were considerably higher than at present. It should be noted that the best policy may not correspond to the most-cost-effective Action Level, eg if a lower Action Level avoids more lung cancers and still remains below the maximum cost per QALY.

TABLE M11 Existing homes: lowest value of long-term average radon concentration in targeted area for which the current policy of remediation in existing homes – ie as in Table M8(a) – becomes cost-effective (ie discounted – societal cost per QALY <£30,000, or cost per QALY <£20,000) and most cost-effective Action Level for that area, for the entire population, never-smokers and current cigarette smokers (assuming 30% of householders accept invitation to test and 20% found to be over Action Level agree to remediate). Results shown for direct estimate of radon risk

	(1) Lowest value of long-term mean radon concentration in targeted area for which there is an Action Level with cost-effectiveness below maximum cost per QALY gained (Bq m^{-3} long-term mean)	(2) Most cost-effective Action Level for targeted area as in column (1) (Bq m^{-3} measured value) [i,ii]
	Maximum cost per per QALY gained £30,000:	
Entire population [iii]	60	100
Never-smokers	420	400
Current cigarette smokers	25	25 [iv]
	Maximum cost per per QALY gained £20,000:	
Entire population [iv]	100	150
Never-smokers	800	400
Current cigarette smokers	45	25 [iv]

Notes

(i) Based on the HPA methodology of considering one three-month radon measurement with seasonal adjustment.

(ii) The best policy may not correspond to the most-cost-effective Action Level, eg if a lower Action Level avoids more lung cancers and still remains below the maximum cost per QALY.

(iii) Taken from part (a) of Table M8.

(iv) There is little information available at present on the percentage reduction in radon concentration that would result from installing a range of remediation measures with pre-remediation concentrations of 25 or 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher pre-installation concentrations, ie 85%.

M4.4 Remediation of homes where a measurement has already been made

A large number of radon measurements have already been made in the UK, mainly by the HPA. Table M12 shows cost-effectiveness in circumstances where a radon measurement already exists for a particular home, and where the householder decides to remediate without any invitation or testing costs incurred. Results are shown for the entire population and also for hypothetical populations consisting entirely of never-smokers or of current cigarette smokers. Cost-effectiveness ratios are shown at different radon

TABLE M12 Existing homes: cost per quality adjusted life-year gained (discounted – societal) for the total population, and for households consisting entirely of never-smokers or current cigarette smokers, where radon measurements already exist. Results shown for direct estimate of radon risk

	Household consisting of:		
Radon level in home	Population prevalence of smoking [i]	Never-smokers [ii]	Current cigarette smokers [iii]
25 [iv]	£86,800	£561,400	£35,100
50 [iv]	£45,800	£283,100	£19,700
75	£32,200	£190,300	£14,500
100	£25,300	£143,900	£12,000
150	£18,500	£97,500	£9,400
200	£15,100	£74,300	£8,100
300	£11,700	£51,100	£6,800
400	£10,000	£39,500	£6,200
500	£8,900	£32,500	£5,800
600	£8,300	£27,900	£5,500

Notes

(i) Population results calculated using age- and sex-specific lung cancer mortality rates for the UK in 2005.

(ii) Never-smoker results calculated using age- and sex-specific lung cancer mortality rates for a population of lifelong non-smokers (Thun et al, 2006).

(iii) Current cigarette smoker results calculated using age-and sex-specific lung cancer mortality rates for current cigarette smokers in the UK. These rates were derived from observed age- and sex-specific lung cancer death rates for 2005 for the total UK population, estimates of lung cancer death rates for lifelong non-smokers (Thun et al, 2006), estimates of the relative risks relative risks for UK men with differing smoking habits relative to continuing cigarette smokers (Peto et al, 2000), and estimates of the proportion of the population according to smoking status for categories of age and sex (ONS, 2006a, and Professor M Jarvis, University College, London: personal communication).

(iv) There is little information available at present on the percentage reduction in radon concentration that would result from installing a range of remediation measures with pre-remediation concentrations of 25 or 50 Bq m^{-3}. The calculations presented assume that it would be similar to that achieved at higher pre-installation concentrations, ie 85%.

levels for such homes, ranging from 25 to 600 Bq m^{-3}. For a household of lifelong non-smokers, the societal cost per QALY gained decreases from £561,000 at 25 Bq m^{-3} to £27,900 per QALY gained at 600 Bq m^{-3}. In these circumstances it would only be cost-effective to remediate homes consisting entirely of never-smokers once the radon level in the home exceeds about 600 Bq m^{-3}, depending on the ceiling cost-effectiveness ratio used. For households involving current or ex-smokers, however, the cost-effectiveness ratio is much more favourable even at radon levels as low as 50 Bq m^{-3}.

M5 Discussion and conclusions

This analysis has used best current estimates of the relationship between radon exposure and lung cancer risk to estimate the cost-effectiveness of radon remediation programmes in existing homes and new homes. By evaluating these programmes in similar terms to other health interventions, comparisons can be made based on outcomes and costs per life-year gained.

Concerning new homes, the analysis indicates that the current government policy in England of installing basic preventive measures in all new homes where the long-term mean indoor radon concentration is at least 36 Bq m^{-3} (ie at least 3% of measurements are over the current Action Level of 200 Bq m^{-3}) is likely to be highly cost-effective (cost per QALY gained below £8000), and that this could be extended to all areas of the UK and remain within the generally accepted range of cost-effectiveness. There is some evidence from the south west of England, where installing preventive measures in new homes is seen as routine, that the material and labour costs have fallen over time, and this would further improve the cost-effectiveness of such a policy. In addition, there is some evidence that the installation of radon-proof membranes will have other benefits, such as better damp-proofing, and thus preventing the entry of other soil pollutants.

The current policy of full preventive measures, consisting of installing not only a radon-proof membrane but also a means of under-floor ventilation such as a sump and associated pipework, is only of value where it is likely that active measures such as an electric fan are fitted subsequently in homes where the radon concentration remains high when only the basic measures are fitted. Our analyses show that a policy that requires testing in all new homes in an area once they are complete and then the installation of fans in those that still have radon concentrations above 200 Bq m^{-3} has a cost-effectiveness ratio of around £53,500 per QALY gained based on the direct estimate of risk, ie substantially above the levels that might typically be considered the maximum willingness to pay when assessing alternative ways of improving health. Such a policy would be more cost-effective if the Action Level were reduced substantially. With an Action Level of 100 or 50 Bq m^{-3} such a policy could have a cost-effectiveness ratio below £30,000 per QALY gained, provided that the proportional reduction in radon concentration achieved by installing an active measure such as a fan could be maintained at around 90% and 100% compliance achieved. However, even with a substantially reduced Action Level and 100% compliance, such a policy would only have a cost-effectiveness ratio below £30,000 per QALY if it were targeted on areas where the long-term mean radon concentration before basic measures are installed is around 90 Bq m^{-3} or higher. For it to have a cost-effectiveness ratio below £20,000 per QALY, the long-term mean radon concentration in the targeted areas would need to be well over 100 Bq m^{-3}, making such a

policy of little practical relevance for the UK. The need to focus this policy on high radon areas arises from the costs of testing, which are substantial, and the high lifetime costs of the active measures.

Concerning existing homes, our model indicates that the current policy for England, in which remediation programmes are targeted in areas with long-term mean indoor radon concentrations down to 64 Bq m^{-3} (ie 5% of houses have measured radon concentrations over the 200 Bq m^{-3} Action Level), and which relies upon identifying radon-affected existing homes and inviting the householders to remediate at their own expense, has at its boundary a cost-effectiveness ratio of £36,800 per QALY gained, which falls well above the assumed NICE ceiling range of £20,000–£30,000 per QALY gained, while the current HPA recommendation that people living in homes where the radon concentration is 36 Bq m^{-3} (ie at least 1% of homes have measured radon concentrations above 200 Bq m^{-3}) has an even higher cost-effectiveness ratio at its boundary.

Our analyses have shown that increasing the Action Level from its current value of 200 Bq m^{-3} is likely to result in a deterioration in the cost-effectiveness ratio. In contrast, lowering the Action Level from 200 Bq m^{-3} is likely to result in a more favourable cost-effectiveness ratio, as the health benefits initially increase more rapidly than the costs. For very low Action Levels, however, the situation is reversed and the cost-effectiveness starts to increase. The value at which the most favourable cost-effectiveness ratio occurs depends on the long-term mean of the radon concentration in the area under consideration. For mean values between 50 and 80 Bq m^{-3}, the most favourable cost-effectiveness ratio is achieved at an Action Level of 100 Bq m^{-3}. It should be noted, however, that the minimum value of the cost-effectiveness ratio may not correspond to the optimum policy if, for example, a lower Action Level avoids more lung cancers while the cost-effectiveness ratio remains below the permitted maximum.

A policy of remediating existing homes in areas with a long-term mean radon concentration of 60 Bq m^{-3} or above and targeted with an Action Level of 100 Bq m^{-3} would be cost-effective given a maximum cost-effectiveness ratio of £30,000 per QALY. However, if the maximum cost-effectiveness ratio were only £20,000 per QALY, then a policy of remediation in existing homes would not be cost-effective at any Action Level unless the mean long-term radon concentration in the area were 100 Bq m^{-3} or higher.

The analyses presented here assume that those in whom lung cancers are averted by prevention or remediation have smoking habits, and therefore lung cancer risks, that are typical of the entire population. This is likely to be the case for prevention in new homes where the builder will usually install basic preventive measures at an early stage in the construction process. For existing homes, however, a recent study has shown that remediation rates among householders who are lifelong-non-smokers are about twice those of householders who are current smokers (Dr Y Chow, HPA: personal communication). The consequence of this is that the cost-effectiveness of remediation in existing homes would, in practice, be even less favourable than indicated by our analyses, possibly by a substantial amount.

The poor cost-effectiveness of the current policy for existing homes is mainly due to the low proportions of householders (historically around 20%) undertaking remediation following a measurement over the Action Level. Lowering the costs of identifying existing homes that are likely to have high measured radon and, particularly, increasing the proportion of identified homes in which householders undertake remediation, would substantially improve cost-effectiveness. For example, it has long been evident that an individual's likelihood of taking remedial action is affected by the way in which risk information on

radon is presented (Smith and Desvousges, 1990; Smith et al, 1995; Kendall et al, 2005). It has also been shown that the level of advice and support offered to householders is important in determining the proportion who remediate (Appendix D). The link between risk information provided to the householder and the propensity to take remedial action is at present poorly understood or researched. More research in this area is needed and may result in higher remediation rates and improved cost-effectiveness.

Our analyses of the effects of varying the targeted area and the Action Level assume that all other parameters remained unchanged: for example, that the assumed average cost of remediating remains unchanged, and also that the proportion of householders in homes over the Action Level who decide to remediate is the same whether that level is set at 200 or 100 Bq m^{-3}. There is some evidence in favour of this from a recent study evaluating the current radon programme which suggests that remediation is at least as likely at lower as at higher radon levels above the current Action Level of 200 Bq m^{-3} (Dr Y Chow, HPA: personal communication).

Our analyses also assume that the proportional reduction in radon concentration achieved by radon prevention or remediation is unchanged regardless of the radon concentration that would be present without any remediation. There is some evidence that this assumption is appropriate for basic radon preventive measures, such as the installation of a radon-proof membrane. For active remediation measures, however, almost all experience to date has been in buildings where the initial radon concentration is at least 200 Bq m^{-3}. Further research on the efficacy of radon remediation measures in buildings with lower pre-remediation radon concentrations is also needed.

The analysis presented here has been performed from a societal perspective, and includes all direct costs incurred by individuals, local and central government departments and the health service. In this important respect the perspective differs from that of agencies such as NICE, which is primarily concerned with health and social care costs. Typically, householders bear the cost of taking remedial action, and this is likely to be one factor influencing remediation rates. Transferring some or all of these costs to another party might result in increased remediation rates and, if it did this, it would improve substantially the overall cost-effectiveness ratio for remediation in existing homes. Such cost transfers do occur in other policy areas: health and local authorities, for instance, in certain circumstances pay towards the costs of adapting homes for people with disabilities. A difference is that radon remediation has benefits not only to the existing home occupier but to future occupiers, supporting an argument that householders should be re-imbursed for at least a share of the costs. Against this, however, is the fact that a documented low radon concentration might be favourably reflected in the house price, especially if it were in a high radon area.

The fact that both radon and smoking increase the risk of lung cancer has frequently resulted in the cost-effectiveness of radon remediation programmes and smoking cessation programmes being compared. Smoking cessation interventions typically have very favourable cost-effectiveness ratios: one UK analysis of nicotine replacement therapy in general practice reported a net cost per life-year gained of less than £1000 in 1999 (Stapleton et al, 1999). This suggests that providing such interventions should be a priority for the health service and that every effort should be made to reduce lung cancer risk by means of such interventions. However, assuming that such interventions are indeed being fully provided, there is no reason why such a low cost-effectiveness ratio should be the benchmark for radon remediation or

other interventions relevant to smoking-related diseases: instead, we have adhered to the broad approach followed by a number of government agencies, and discussed in Section M1.4 above, which suggests that the maximum willingness to pay for health benefit typically lies somewhere between £20,000 and £30,000 per life-year or per quality adjusted life-year gained.

The results presented here are sensitive to whichever discount rate is selected. The baseline analysis uses the Department of Health approved annual discount rates of 3.5% for costs and 1.5% for health benefits (DH, 2004); the long periods over which the health benefits from reduced radon-induced lung cancer deaths are realised mean that different discount rates on benefits will strongly affect the results.

In summary, the analysis reported here provides evidence to assist in forming policies concerning radon remediation in new and existing homes, and a framework to assess the value for money of different options.

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Appendix N
Radon Testing as a Health Screening Programme

Screening is a public health service in which members of a defined population, who do not necessarily perceive they are at risk, are offered a test, to identify those individuals who are more likely to be helped than harmed by further tests or treatment to reduce the risk of a disease or its complications.

Radon testing in homes has a number of similarities to a health screening programme. The main difference is that properties rather than individuals are tested, and that preventive measures are available for the properties and not individual people, although, of course, it is the individual people in the properties who will receive the benefit.

Routine health screening programmes in the NHS are overseen by the UK National Screening Committee (NSC). This group advises Ministers, the devolved national assemblies and the Scottish Parliament on all aspects of screening policy. The NSC assesses proposed new screening programmes against a set of internationally recognised criteria. Assessing programmes in this way is intended to ensure that they do more good than harm at a reasonable cost. In 1996, the NHS was instructed not to introduce any new screening programmes until the NSC had reviewed their effectiveness. Radon testing would not be within the remit of NHS screening programmes as these are focused on individuals. However, NHS premises could be (and have been) included in radon surveys. Moreover, in considering the public health implications of radon, it is helpful to consider radon testing in homes against the criteria used to evaluate health screening tests.

N1 Limitations of screening

Screening has important ethical differences from clinical practice, as it targets healthy people, offering to help individuals to make better informed choices about their health. However, there are usually risks involved and it is important that people have realistic expectations of what a screening programme can deliver. Whilst screening has the potential to save lives or improve quality of life, it is not a fool-proof process. Screening can reduce the risk of developing a condition or its complications but it cannot offer a guarantee of protection. In any screening programme, there is an irreducible minimum of false positive results (wrongly reported as having the condition) and false negative results (wrongly reported as not having the condition). The NSC is increasingly presenting screening as risk reduction to emphasise this point.

The NSC publishes criteria for appraising the viability, effectiveness and appropriateness of a screening programme*. Ideally all the criteria should be met before screening for a condition is adopted by the NHS. These criteria are listed below with annotation to show whether and how screening of homes for high radon concentrations meets the criteria.

* NSC (2003). Criteria for appraising the viability, effectiveness and appropriateness of a screening programme. UK National Screening Committee. Available at www.nsc.nhs.uk/pdfs/criteria.pdf.

N2 Criteria for adopting screening

Condition

1 The condition should be an important health problem.
Lung cancer is a major health problem. Radon exposure is an established cause of lung cancer.

2 The epidemiology and natural history of the condition, including development from latent to declared disease, should be adequately understood and there should be a detectable risk factor, disease marker, latent period or early symptomatic stage.
The epidemiology of radon and lung cancer is well known. The latent period is less well understood but is long (perhaps some 5–25 years after exposure).

3 All the cost-effective primary prevention interventions should have been implemented as far as practicable.
Considerable efforts have been made to implement primary prevention programmes targeting tobacco smoking. Additional primary prevention interventions are the installation of radon barriers in new homes. This is already a requirement in areas with higher radon concentrations, and the present report recommends expanding the area in which such measures are required. Such primary prevention is only possible in new homes. In high radon areas it can be supplemented – for example, by offering free radon testing in existing homes and recommending remediation work at the householder's expense. Such measures have been implemented in the UK, but with limited uptake. Measures to improve uptake might be considered on the basis of the results of cost-effectiveness analyses, as discussed in Chapter 6 (and in more detail in Appendix M).

4 If the carriers of a mutation are identified as a result of screening the natural history of people with this status should be understood, including the psychological implications.
Not applicable for radon exposure and lung cancer.

Test

5 There should be a simple, safe, precise and validated screening test.
Radon testing of homes fits all these criteria.

6 The distribution of test values in the target population should be known and a suitable cut-off level defined and agreed.
The distribution of radon concentrations in different part of the country is well known and there is a defined radon Action Level (currently 200 Bq m^{-3}). However, the majority of lung cancer deaths attributable to radon exposure are likely to occur at concentrations lower than this.

7 The test should be acceptable to the population
This criterion does not really apply to the radon test. The placement of two small plastic detectors in the house is clearly much more 'acceptable' than clinical screening tests such as mammography or cervical cytology. However, the uptake rates in population radon screening

programmes are low. This is usually attributed to lack of recognition of the hazard rather than unacceptability of the test.

8 There should be an agreed policy on the further diagnostic investigation of individuals with a positive test result and on the choices available to those individuals.
There are established procedures for giving advice to householders with high tests, including repeat tests if it is suspected that an erroneous result may have been obtained due to an error in placement of the detectors. Re-tests are offered after remediation work has been done.

9 If the test is for mutations the criteria used to select the subset of mutations to be covered by screening, if all possible mutations are not being tested, should be clearly set out.
Not applicable for radon.

'Treatment'

10 There should be an effective treatment or intervention for patients identified through early detection, with evidence of early treatment leading to better outcomes than late treatment.
The information in the present report provides a basis for evidence-based policies, and highlights gaps in evidence relevant to policies.

11 There should be agreed evidence-based policies covering which individuals should be offered treatment and the appropriate treatment to be offered.
This evidence is well known.

12 Clinical management of the condition and patient outcomes should be optimised in all health care providers prior to participation in a screening programme.
There are radon prevention programmes in new-build dwellings in high radon areas already. Further information regarding their long-term efficacy would be desirable.

Screening programme

13 There should be evidence from high quality randomised controlled trials that the screening programme is effective in reducing mortality or morbidity. Where screening is aimed solely at providing information to allow the person being screened to make an 'informed choice' (eg Down's syndrome or cystic fibrosis carrier screening), there must be evidence from high quality trials that the test accurately measures risk. The information that is provided about the test and its outcome must be of value and readily understood by the individual being screened.
Randomised trials of prevention versus no prevention measures would probably not be ethical for new-build dwellings and would certainly not be timely. Randomised trials evaluating the efficacy and durability of different methods of radon prevention would, however, be useful. For remediation in existing homes, there is scope to compare alternative approaches to maximise uptake of testing and implementation of remediation. There is good evidence regarding radon concentrations and lung cancer risk.

14 There should be evidence that the complete screening programme (test, diagnostic procedures and treatment/intervention) is clinically, socially and ethically acceptable to health professionals and the public.
The health economics work in this report provides information to support radon testing. Some householders are probably deterred from seeking radon testing because of an unwillingness to pay for remediation and a fear that it will be difficult to sell the home if a high radon concentration is found.

15 The benefit from the screening programme should outweigh the physical and psychological harm (caused by the test, diagnostic procedures and treatment).
There is evidence that publicity about early radon measurement campaigns caused some anxiety among householders about possible effects on house prices, but these worries have not been borne out in practice. There is no evidence about the effects of radon testing on resolve to quit smoking.

16 The opportunity cost of the screening programme (including testing, diagnosis and treatment, administration, training and quality assurance) should be economically balanced in relation to expenditure on medical care as a whole (ie value for money).
The health economics work in this report provides information on the circumstances under which radon testing might be cost-effective.

17 There should be a plan for managing and monitoring the screening programme and an agreed set of quality assurance standards.
Quality assurance mechanisms for radon testing are in place.

18 Adequate staffing and facilities for testing, diagnosis, treatment and programme management should be available prior to the commencement of the screening programme.
Not a problem for the current programme. Funding of remediation remains a barrier that could invalidate the programme in terms of these criteria.

19 All other options for managing the condition should have been considered (eg improving treatment or providing other services), to ensure that no more cost-effective intervention could be introduced or current interventions increased within the resources available.
Significant resources are already committed to smoking cessation activities. Increasing these is likely to have a highly cost-effective beneficial effect in reducing radon-related lung cancer among current smokers. Expanding the areas in which radon prevention is a requirement in new buildings is also likely to be cost-effective according to current NICE guidelines. For existing homes in high radon areas, more effective strategies have not been proposed for ex-smokers or for lifelong non-smokers.

20 Evidence-based information, explaining the consequences of testing, investigation and treatment, should be made available to potential participants to assist them in making an informed choice.
Such information is readily available, including telephone help.

21 Public pressure for widening the eligibility criteria for reducing the screening interval, and for increasing the sensitivity of the testing process, should be anticipated. Decisions about these parameters should be scientifically justifiable to the public.
At present radon does not engender the same level of public debate as traditional health screening programmes.

In summary, radon prevention in new homes would appear to meet all relevant criteria for a screening programme identified by the UK National Screening Committee. Radon testing and remediation may also be appropriate if it is cost-effective. To date, however, programmes have often failed, as homes are often not remediated following a test result indicating a high radon concentration.

Glossary

Absorbed dose The quantity of energy imparted by ionising radiation to a unit mass of matter such as tissue. Absorbed dose has the units J kg^{-1} (joules per kilogram) and the special name gray (Gy). 1 Gy = 1 J kg^{-1}. Absorbed doses can be estimated for the body as a whole or for organs or tissues.

Action Level That radon concentration above which it is recommended that remedial action is taken to reduce exposures.

ALL Acute lymphoblastic leukaemia.

Alpha emitter A radionuclide which decays through emission of alpha particles.

Alpha particle A charged particle emitted during the radioactive decay of many heavy radionuclides. It is the nucleus of a helium atom, consisting of two protons and two neutrons. An alpha particle has low penetrating power but high linear energy transfer (LET).

AML Acute myeloid leukaemia.

Becquerel (Bq) The international (SI) unit for the number of nuclear disintegrations occurring per unit time, in a quantity of radioactive material. 1 Bq = 1 nuclear disintegration per second.

BEIR US Committee on the Biological Effects of Ionizing Radiation.

Beta particle An electron emitted during radioactive decay.

Bias In the most general sense, bias is any effect which causes an investigation to give results which differ systematically from the true values, ie errors which are not random errors. In epidemiology, 'bias' may arise in the particular circumstances where a subset of individuals selected for study are not representative of the population being studied or where the information available for those at different degrees of risk is not comparable. For example, in a cohort study, the exposed group might be more (or less) likely to be lost to follow-up than the unexposed group. In a case–control study, the controls might be more likely than the cases to have forgotten that they were exposed to the agent in question. Bias is a very serious flaw in epidemiological studies and increasing the study population will not, of itself, reduce the errors (as would be the case if errors were random). Other things being equal, bias is more likely to be a problem in case–control than in cohort studies. In a cohort study, some kinds of bias can be eliminated by studying the whole population at risk rather than a sample, if this is practicable.

Bystander effect Genetic damage appearing in unirradiated cells close to irradiated cells.

Case–control study An investigation into the extent to which a group of persons with a specific disease (the cases) and comparable persons who do not have the disease (the controls) differ with respect to exposure to putative risk factors.

Cohort study Cohort studies compare a group with the exposure under consideration to a group without the exposure, or with a different level of exposure, or to the country as a whole. The groups

(cohorts) are followed over a period of time, and the disease occurrence is compared between the groups or between the cohort and rates expected from national statistics.

Confidence interval Indicates the (im)precision of an estimate derived from a finite number of observations as might be available from a study. In this way a confidence interval conveys the effects of sampling variation on the precision of, for example, an age-standardised rate calculated from a limited time period. Specifically, the true rate will be inside the 95% confidence interval on 95% of occasions, although the study rate remains the best estimate of the true value.

Confounding Confounding is a problem in epidemiological studies which arises when there is an exposure which is associated with both the factor that is being investigated and the disease under study. This would give rise to an apparent relationship between the factor being investigated and the disease, even if the factor did not cause the disease. For example, suppose lung cancer was being studied in workers exposed to a particular chemical. If those exposed to higher levels of the chemical smoked more than other workers, then the chemical would be associated with lung cancer even if it did not actually cause the disease. The problem can be addressed in the design and analysis of studies but requires that data on the confounder be collected.

Cost-effectiveness analysis A comparative analysis of two or more alternatives in terms of the costs and health consequences. Typically, the incremental cost per incremental unit of health gain is computed; decision-makers must then decide whether they are willing to pay for the health gain at this cost per unit. Health consequences can be measured in different ways such as cases detected, events, deaths, life-years or quality adjusted life-year (QALYs – see separate entry).

Decay The process of spontaneous transformation of a radionuclide. The decrease in the activity of a radioactive substance.

Decay chain Series of decay products resulting from a long-lived radioactive atom (nucleus). It includes the products which result from the fact that some atoms (nuclei) can decay in more than one way.

Decay product A nuclide or radionuclide produced by decay. A decay product may be formed directly from a radionuclide or as a result of a series of successive decays through several radionuclides.

DNA A chemical made up of a linear sequence of different molecules called bases (adenine, thymine, cytosine and guanine) constituting the genetic material of organisms. There are four bases and the permuted sequence of these is read as a code which determines the composition and properties of the organism. The simplest organisms such as bacteria have nearly five million bases in their genetic material; humans have more than three-hundred million bases.

Dose Sometimes used as an abbreviation for absorbed dose or, if the context is clear, other dosimetric quantities such as effective dose. 'Dosimetry' is the science of estimating doses.

Ecological study See geographical correlation study.

Effective dose Effective dose is the sum of the weighted equivalent doses in all the tissues and organs of the body. It takes account of the biological effectiveness of different types of radiation and variation in

the susceptibility of different organs and tissues to radiation damage. Thus it provides a common basis for comparing exposures from different sources. Unit sievert (Sv).

Epidemiology Epidemiology is the study of the distribution and putative causes of diseases in human populations. Descriptive epidemiology analyses the age, sex and whereabouts of those who have particular diseases and the methods allow changes in case rates with time or place to be studied and case clusters to be investigated. Analytical epidemiology investigates possible causes of particular diseases using case–control or cohort approaches.

Equilibrium factor The ratio of PAEC (potential alpha energy concentration) for the actual mixture of radon decay products to that which would apply at radioactive equilibrium. Usually abbreviated as *F*.

Equivalent dose The quantity obtained by multiplying the absorbed dose by a factor to allow for the different effectiveness of the various ionising radiations in causing harm to tissue. Unit sievert, symbol Sv. Usually the factor for gamma rays, X-rays and beta particles is 1 but for alpha particles it is 20.

Etched-track detector Passive detector that records the tracks of alpha particles.

Excess absolute risk (EAR) The absolute difference between the instantaneous disease rates in two groups of people, eg those exposed to radiation at a given level and those unexposed. An EAR of 0 corresponds to neither an increase nor a decrease in risk.

Excess relative risk (ERR) The relative risk minus 1. Thus, an ERR of 0 corresponds to a relative risk of 1 and signifies no raised risk. In instances where the trend in relative risk with dose has been estimated, the change in relative risk per unit dose is often denoted as ERR Gy^{-1} or ERR Sv^{-1}.

External and internal exposures External exposure arises from radioactive sources which remain outside the body. Internal exposure arises from radioactive materials which are taken inside the body, through inhalation or ingestion. An alpha particle has a very short range and hence very little penetrative power, so that if it were to come from an external source it would be unlikely to penetrate the surface of the skin, giving up most of its energy in the dead surface skin layers. If, however, an alpha particle were emitted from a source that had been inhaled into the lungs its closer proximity to living cells could result in damage to those cells. Internal exposures are generally received from sources that have been inhaled or ingested. Beta and gamma radiation sources can give rise to either internal or external exposures.

Gamma radiation A photon (quantum) of electromagnetic radiation that may be emitted from the nucleus of an atom during radioactive decay.

Gene A unit of genetic material consisting of a specific DNA sequence which usually contains the instructions to produce one type of protein. A gene may exist in more than one form (or allele) thus contributing to the differences between individuals.

Genetic material The genetic material of almost all organisms is DNA, a chemical comprising a linear sequence of bases constituting a code which determines the properties of the organism.

Geographical correlation study A study which is based on averaging of disease rates and measure(s) of exposure over geographical areas and attempts to correlate them. Also known as an ecological study. Such studies are susceptible to specific types of bias.

Gray (Gy) Unit of ionising radiation dose (J kg^{-1}), calculated without weighting of the particular radiation type by its relative biological effectiveness.

Half-life ($t_{½}$) The time taken for the activity of a radionuclide to lose half its value by decay. During each subsequent half-life its activity is halved again so its activity decays exponentially.

Health economics Application of the theories and tools of economics to the topic of health and health care.

Hormesis A theory that suggests that low doses of radiation have a beneficial effect.

Hypothesis A suggested explanation for an observed phenomenon, ideally one that can be tested experimentally. See also null hypothesis.

IARC International Agency for Research on Cancer.

ICD International Classification of Diseases.

ICRP International Commission on Radiological Protection. It consists of experts in radiology, genetics, physics, medicine and radiological protection from a number of countries. Established in 1928, it meets regularly to consider the results of research on the effects of radiation and publishes recommendations on all aspects of radiological protection, including dose limits for man.

Ion Atom or molecule that has lost or gained one or more electrons and is therefore electrically charged.

Ionisation The process by which a neutral atom or molecule acquires or loses an electron. The production of ions.

Ionise Remove or add one or more electrons from a neutral atom or molecule.

Ionising radiation Radiation which is sufficiently energetic to remove electrons from atoms in its path. In human or animal exposures, ionising radiation can result in the formation of highly reactive particles in the body which can cause damage to individual components of living cells and tissues.

Malignancy Cancerous growth, a mass of cells showing uncontrolled growth, a tendency to invade and damage surrounding tissues and an ability to seed daughter growths to sites remote from the primary growth.

Meta-analysis An analysis that combines data from a number of comparable studies, leading to a quantitative summary of the results. In the field of epidemiology the term is usually used to refer to an analysis based on previously published summary statistics, rather than on individual patient data. Such analyses are limited by the fact that it is not possible to evaluate or alter the treatment of confounding variables from that used in the original publication. See also pooling study.

Multiplicative (relative) risk model A model in which a unit of exposure induces an increase in the disease rate that is proportional to the underlying age-specific rate.

Nuclide A specific isotope characterised by the number of protons and the number of neutrons contained in the nucleus.

Null hypothesis The statistical hypothesis that one variable has no association with another variable or set of variables, or that two or more population distributions do not differ from one another.

PAEC Potential alpha energy concentration. The sum of the energy of all the alpha particles which the radon decay products will emit in decaying to lead-210.

Pooling study In radon epidemiology, the term 'pooling study' has been used to describe analyses that combine the individual data from a number of comparable studies, leading to a quantitative summary of the results. In contrast to meta-analyses of published data, the pooling studies have been able to adjust much more precisely for the confounding effect of smoking.

Power The power of a study is its potential ability to reject the null hypothesis in favour of a specified alternative hypothesis. In mathematical terms, power is specified as the probability that the null hypothesis can be rejected, based on a test with specified statistical significance. For example, the null hypothesis might be that a specific agent carries no increased risk of a particular disease; the alternative hypothesis, that it doubles risk. A planned study may then be estimated to have, say, a 90% probability of rejecting the null hypothesis of no increased risk, based on a test at the 5% level, if the alternative hypothesis were true.

QALY A quality adjusted life-year. If quality of life is impaired by pain, disability, anxiety or some other dimension of ill-health, a period of time in that state is likely to give an individual less utility than they would obtain from the same period of time in full health. The quality adjusted life-year attempts to place a numerical weight on the utility derived from different health states by trying to quantify preferences for these states. For example, if asked to choose between 12 months in a health state of moderate pain and anxiety, with some restrictions on mobility and usual functioning, or some lesser period of time in a state of full health, a respondent may indicate they would consider 12 months in the former health state to be equivalent to 9 months of full health – ie they would give up 3 months of life expectancy to be freed from these health problems. This would equate to a quality adjusted life-year of 0.75. The QALY is favoured by health economists as a measure of health outcome when conducting cost-effectiveness analysis because it permits comparison across many different interventions that could affect quality of life, survival or some combination of these.

Radioactivity The property of radionuclides of spontaneously emitting ionising radiation. The activity of radioactive decay is measured in becquerel (Bq).

Radionuclide A type of atomic nucleus which is unstable and which may undergo spontaneous decay to another atom by emission of ionising radiation (usually alpha, beta or gamma).

Radon (Rn) Radon-222 is a radioactive gas with a half-life of 3.82 days. It is a decay product of the long-lived uranium isotope uranium-238. (The numbers 222, 238, etc, denote the total number of protons and neutrons in the nucleus.)

Radon decay products The most important decay products of radon in relation to health are the radioactive polonium isotopes polonium-218 and polonium-214. Since radon gas is present in air at very low concentrations, traces of polonium isotopes are formed and deposited in the respiratory system. (The numbers 214, 218, etc, denote the total number of protons and neutrons in the nucleus.)

Relative risk (RR) A ratio of the risk of disease or death of those exposed to the risk to those not exposed to the risk. It may be necessary to make allowance for confounding factors, such as age.

Risk The probability that an event will occur, eg that an individual will become ill or die within a stated period of time or age. Also, a non-technical term encompassing a variety of measures of the probability of a (generally) unfavourable outcome. (See also relative risk.)

Sievert (Sv) The international (SI) unit of effective dose, obtained by weighting the equivalent dose in each tissue in the body with ICRP-recommended tissue weighting factors, and summing over all tissues. Because the sievert is a large unit, effective dose is commonly expressed in millisievert (mSv) – ie one-thousandth of one sievert – and microsievert (mSv) – ie one-thousandth of one millisievert. The average annual radiation dose received by members of the public in the UK is 2.6 mSv.

Significance level The probability of obtaining a result at least as extreme as that observed in the absence of a raised risk. A result that would arise less than 1 in 20 times in the absence of an underlying effect is often referred to as being 'statistically significant'.

Significance test A result that lies outside the range of values expected to occur on the majority of occasions, if some specified hypothesis is true, is said to be statistically significant. A probability (p-value) of 0.05 for such an occurrence is commonly used to separate 'significant' from 'non-significant' results. This boundary is arbitrary.

Standardised incidence ratio (SIR) As standardised mortality ratio, but referring to the incidence of disease rather than death.

Standardised mortality ratio (SMR) The ratio of the observed number of deaths from a given cause in a cohort to that expected in the general population, based on the same mixture of ages and sexes and interval of follow-up. SMRs are often (but not always) quoted as percentages. For example, an SMR of 100 indicates that the mortality rate in the cohort is the same as that in the general population.

Statistical power The probability that, with a specified degree of confidence, an underlying effect of a given magnitude will be detected in a study.

Statistical significance In an investigation of, for example, whether exposure to a particular agent is associated with a certain type of cancer, statistical tests will be carried out to assess the probability that a result at least as extreme as that observed could have arisen by chance if the null hypothesis were true. Researchers will commonly describe a result as statistically significant if this probability is 5% (1 in 20) or less. (See also p-value.)

Synergy A joint effect of two agents that is greater than the sum of each acting alone.

Thorium (Th) A radioactive metal which can exist in various isotopic forms. The most important isotope is thorium-232, which decays through a series of decay products which emit alpha, beta and gamma radiation to radon-220 (also known as thoron), which has a half-life of 56 seconds.

Tumour Mass of tissue formed by unregulated growth of cells; can be either benign or malignant.

UNSCEAR United Nations Scientific Committee on the Effects of Atomic Radiation.

Uranium (U) A radioactive metal which can exist in various isotopic forms. The most important isotopes are uranium-235 (the only naturally occurring readily fissile isotope) and uranium-238. Both isotopes decay through a series of decay products which emit alpha, beta and gamma radiation. Uranium-235 gives rise to radon-219, with a half-life of 4 seconds and uranium-238 gives rise to radon-222, with a half-life of 3.82 days.

Working Level (WL) 1 WL is any combination of short-lived radon decay products in one cubic metre of air that will result in the ultimate emission of 1.3 10^8 MeV of alpha energy.

Working Level Month (WLM) 1 WLM is the amount of radiation exposure accumulated during 170 hours at 1 WL, or 3.5 mJ m^{-3} h.

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