International Commission on Radiological Protection

Statement on Radon

Approved by the Commission in November 2009

(1) The Commission issued revised recommendations for a System of Radiological Protection in 2007 (ICRP, 2007) which formally replaced the Commission’s 1990 Recommendations (ICRP, 1991) and updated, consolidated, and developed the additional guidance on the control of exposure from radiation sources. The Commission has previously issued recommendations for protection against radon-222 at home and at work in Publication 65 (ICRP, 1993).

(2) The Commission has now reviewed recently available scientific information on the health effects attributable to exposure to radon and its decay products. The Commission’s full review accompanies this Statement. As a result of this review, for radiological protection purposes the Commission now recommends a detriment-adjusted nominal risk coefficient for a population of all ages of $8 \times 10^{-10}$ per Bq h m$^{-3}$ for exposure to radon-222 gas in equilibrium with its progeny (i.e. $5 \times 10^{-4}$ WLM$^{-1}$). The Commission’s findings are consistent with other comprehensive estimates including that submitted to the United Nations General Assembly by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 2009).

(3) Following from the 2007 Recommendations, the Commission will publish revised dose coefficients for the inhalation and ingestion of radionuclides. The Commission now proposes that the same approach be applied to intakes of radon and progeny as that applied to other radionuclides, using reference biokinetic and dosimetric models. Dose coefficients will be given for different reference conditions of domestic and occupational exposure, taking into account factors including inhaled aerosol characteristics and disequilibrium between radon and its progeny. Sufficient information will be given to allow specific calculations to be performed in a range of situations. Dose coefficients for radon and progeny will replace the Publication 65 dose conversion convention which is based on nominal values of radiation detriment derived from epidemiological studies comparing risks from radon and external radiation. The current dose conversion values may continue to be used until dose coefficients are available. The Commission advises that the change is likely to result in an increase in effective dose per unit exposure of around a factor of two.

(4) The Commission reaffirms that radon exposure in dwellings due to unmodified concentrations of radium-226 in the earth’s crust, or from past practices not conducted within the Commission’s system of protection, is an existing exposure situation. Furthermore, the Commission’s protection policy for these situations continues to be based on setting a level of annual dose of around 10 mSv from radon where action would almost certainly be warranted to reduce exposure. Taking account of the new findings, the Commission has therefore revised the upper value for the reference level for radon gas in dwellings from the value in the 2007 Recommendations of 600 Bq m$^{-3}$ to 300 Bq m$^{-3}$. National authorities should consider setting lower reference levels according to local circumstances. All reasonable efforts should be made, using the principle of optimisation of protection, to reduce radon
exposures to below the national reference level. It is noted that the World Health Organisation now recommends a similar approach (WHO, 2009).

(5) Taking account of differences in the lengths of time spent in homes and workplaces of about a factor of three, a level of radon gas of around 1000 Bq m\(^{-3}\) defines the entry point for applying occupational protection requirements for existing exposure situations. In Publication 103, the Commission considered that the internationally established value of 1000 Bq m\(^{-3}\) might be used globally in the interest of international harmonization of occupational safety standards. The Commission now recommends 1000 Bq m\(^{-3}\) as the entry point for applying occupational radiological protection requirements in existing exposure situations. The situation will then be managed as a planned exposure situation.

(6) The Commission reaffirms its policy that, for planned exposure situations, any workers’ exposure to radon incurred as a result of their work, however small, shall be considered as occupational exposure (see paragraph 178 of ICRP, 2007).

References


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Lung cancer risk from radon and progeny

DRAFT

Editor

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This report reviews recent epidemiological studies of lung cancer risk linked to exposure to radon and its progeny. It concentrates on the results from pooled case-control studies of residential exposures and cohorts of underground miners exposed to low levels of radon and radon progeny. Consistent with the approach used in ICRP Publication 65 (1993), recent miner data are used to recommend a revised detriment-adjusted nominal risk coefficient of $5 \times 10^{-4}$ per WLM ($14 \times 10^{-5}$ per mJ h m$^{-3}$), replacing the ICRP Publication 65 value of $2.8 \times 10^{-4}$ per WLM ($8 \times 10^{-5}$ per mJ h m$^{-3}$). Furthermore, pooled analyses of epidemiological studies of lung cancer risk from residential exposures demonstrate a statistically significant increase per unit of exposure below average annual concentrations of about 200 Bq m$^{-3}$. The risk estimates derived from these pooled analysis are consistent with those from underground miners and are sufficiently robust to enable protection of the public to be now based on residential concentration levels. However, for occupational protection purposes, dose estimates are required to demonstrate compliance with limits and constraints. Dose estimates also allow comparisons between various sources of public exposure. ICRP Publication 65 recommended that doses from radon and its progeny should be calculated using a dose conversion convention based on miner epidemiological studies. ICRP now proposes to treat radon and radon progeny in the same way as other radionuclides and will publish dose coefficients calculated using dosimetric models for use within the ICRP system of protection.
A Task Group was appointed by ICRP Committee 1, with representation of several members from Committee 2 and one member from Committee 4 to review risks from alpha emitting radionuclides. The Commission subsequently asked the Task Group to concentrate initially on radon. This report reviews epidemiological studies of lung cancer risk associated with the inhalation of radon and radon progeny in homes and in underground mines.

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EXECUTIVE SUMMARY

(a) Epidemiological studies of occupational exposures of miners and of domestic exposures of the public have provided strong and complementary evidence of the risks of lung cancer following inhalation of radon and radon progeny. In the large cohorts of underground miners, annual occupational exposures were considered for the whole working period of each individual. Consequently, these studies are able to analyse dose-response relationships taking account of time-dependent modifying factors such as age at exposure and time since exposure. Lung cancer risk associated with domestic exposures to radon has been studied in a large number of case-control studies, requiring estimates of radon exposure in houses over a period of 30 years preceding lung cancer diagnoses. A weakness of such studies is that measurements made during the study period are assumed to apply throughout the whole period of exposure. An important strength, however, is that the residential studies often include detailed interviews so that adjustments can be made, in the statistical analysis, for tobacco smoking as well as exposure to other potential lung carcinogens in the home or at work.

(b) In 1998, the BEIR VI report presented a comprehensive analysis of available miner cohorts (NRC, 1998). Recent studies of lung cancer in miners include relatively low concentrations of radon and radon progeny, long duration of follow-up and good quality of individual exposure data (UNSCEAR, 2009). A recent pooled analysis of the French and Czech miner cohorts also provided risk estimates associated with low levels and good quality of individual exposure (Tomášek et al., 2008a). These results, consistent with previous analyses of combined miner studies, demonstrate statistically significant associations between cumulative radon exposure and lung cancer mortality at levels of exposure as low as 50 Working Level Months (WLM), i.e. 180 mJ h m\(^{-3}\). On the basis of calculations of lifetime excess absolute risk (LEAR), using Publication 103 (ICRP, 2007) reference background rates and risk models from pooled analyses (NRC, 1998; Tomasek et al., 2008a), a detriment-adjusted nominal risk coefficient of 5 \(10^{-4}\) per WLM (14 \(10^{-5}\) per mJ h m\(^{-3}\)) is now recommended for radiological protection purposes. This nominal risk coefficient replaces the Publication 65 value of 2.8 \(10^{-4}\) per WLM (8.0 \(10^{-5}\) per mJ h m\(^{-3}\)).

(c) Three comprehensive publications have provided joint analyses of data from domestic case-control studies for Europe (Darby et al., 2005), North America (Krewski et al., 2005; 2006) and China (Lubin et al., 2004). Each joint analysis demonstrated an increased risk of lung cancer with increasing domestic radon concentration, considering exposures over a period of 30 years preceding the diagnosis of cancer. The estimates of an increase of lung cancer per unit of concentration in the three joint analyses are very close to each other and statistically compatible: the values obtained were 1.08, 1.10 and 1.13 per 100 Bq m\(^{-3}\). A combined estimate calculated for Europe, North American and China was 1.09 per 100 Bq m\(^{-3}\) (UNSCEAR 2009). All of these results were obtained after adjustment for smoking habits. It is noted also that the slope of the linear exposure-response relationships increased to 1.11 per 100 Bq m\(^{-3}\) when the analyses focused on those cases and controls with more complete estimation of cumulated individual exposure (UNSCEAR 2009).

(d) The joint analyses also made adjustments to take account of uncertainties associated with variations in radon concentration. For example, in the European pooled analysis (Darby et al., 2005), taking account of such uncertainties increased
the estimate of relative risk from 1.08 to 1.16 per 100 Bq m$^{-3}$. Limiting the European analysis to those cases and controls with a relatively low annual exposure, there is evidence of an increased risk below 200 Bq m$^{-3}$. It is concluded that the residential studies provide a reasonable estimate of lung cancer risk and a basis for risk management related to low protracted radon exposures in homes, considering cumulated exposure during a period of at least 25 years.

(e) Although comparisons are complex, the cumulated excess absolute risk of lung cancer attributable to radon and radon progeny estimated for residential exposures appears to be consistent with that obtained from miners at low levels of exposure.

(f) In the European pooled analysis of domestic exposures, a statistically significant trend in lung cancer risk was observed among smokers and also separately among non-smokers (Darby et al., 2006). Therefore radon has been demonstrated to be a lung carcinogen even in the absence of smoking. However, due to the dominating effect of tobacco use on lifetime risk of lung cancer, the excess absolute risk of lung cancer attributable to a given level of radon concentration is much higher among lifelong cigarette smokers than among non-smokers.

(g) The control of domestic exposures can be based directly on lung cancer risk estimates per unit exposure derived from epidemiological data, that is, in terms of radon concentrations in homes.

(h) However, for the purposes of control of occupational exposures using dose limits and constraints, estimates of dose per unit exposure are required. In ICRP Publications 65 and 66 (ICRP, 1993; 1994), the effective dose per unit exposure to radon and radon progeny was obtained using the so-called dose conversion convention. This approach compared the detriment per unit exposure to radon and its progeny with the total detriment associated with unit effective dose, estimated largely on the basis of studies of Japanese survivors of the atomic bombings (ICRP, 1993). The values given were 5 mSv per WLM (1.4 mSv per mJ h m$^{-3}$) for workers and 4 mSv per WLM (1.1 mSv per mJ h m$^{-3}$) for members of the public.

(i) Doses from radon and radon progeny can also be calculated using different dosimetric models. A review of published data on the effective dose per unit exposure to radon progeny obtained using dosimetric models is included as Appendix B of this report. Values of effective dose range from about 6 to 20 mSv per WLM (1.7 to 5.7 mSv per mJ h m$^{-3}$), with results using the ICRP (1994) Human Respiratory Tract Model (HRTM) in the range from about 10 to 20 mSv per WLM (3 to 6 mSv per mJ h m$^{-3}$) depending on the scenario of exposure.

(j) ICRP has concluded that doses from radon and radon progeny should be calculated using ICRP biokinetic and dosimetric models, including the HRTM and ICRP systemic models. That is, radon and its progeny should be treated in the same way as other radionuclides within the system of protection. ICRP will provide dose coefficients per unit exposure to radon and radon progeny for different reference conditions of domestic and occupational exposure, with specified equilibrium factors and aerosol characteristics. Until dose coefficients are published, the previously recommended Publication 65 values should continue to be used. It should be recognised, however, that the dose coefficients to be published will be larger by about a factor of two or more.
Case-control study
Type of epidemiological study design in which a group of subjects with the disease of interest (the cases with lung cancer) is compared to a group of subjects that are free of this disease (the controls) but have similar characteristics (sex, attained age...). This type of epidemiological design was especially used in indoor radon studies. For each individual, past exposures are estimated from measurements of radon concentration in current and previously occupied dwellings.

Cohort study
Type of epidemiological study design in which a population exposed to radon and radon progeny is followed over time for the occurrence of diseases (including lung cancer). This type of epidemiological design was especially used in underground miner studies. The exposure in time is considered for each individual on an annual basis.

Dose conversion convention
This method defined in ICRP Publications 65 (ICRP, 1993) was used to relate exposure to radon progeny expressed in WLM or J h m\(^{-3}\), to effective dose expressed in mSv on the basis of equal detriment.

Equilibrium equivalent concentration (EEC)
The activity concentration of radon gas, in equilibrium with its short-lived progeny which would have the same potential alpha energy concentration as the existing non-equilibrium mixture.

Equilibrium factor, F
The ratio of the equilibrium equivalent concentration to the radon gas concentration. In other words it is the ratio of potential alpha energy concentration (PAEC) for the actual mixture of radon decay product to that which would apply at radioactive equilibrium.

Existing exposure situations
A situation that already exists when a decision on control has to be taken, including natural background radiation and residues from past practices that were operated outside the Commission’s recommendations.

Human Respiratory Tract Model (HRTM)
Model used in ICRP Publication 66 (1994) to evaluate the deposition and clearance of inhaled particles in the respiratory airways as well as the resulting dose to the lung tissues.

Planned exposure situations
Planned exposure situations are situations involving the deliberate introduction and operation of sources. Planned exposure situations may give
rise both to exposures that are anticipated to occur (normal exposures) and to exposures that are not anticipated to occur.

Potential alpha energy concentration (PAEC)
The concentration of short-lived radon or thoron progeny in air in terms of the alpha energy emitted during complete decay from radon-222 progeny to lead-210 or from radon-220 progeny to lead-208 of any mixture of short-lived radon-222 or radon-220 in a unit volume of air.

Radon progeny
The decay products of radon-222, used in this report in the more limited sense of the short-lived decay products from polonium-218 through polonium-214. Radon progeny are sometimes referred to as “radon decay products”.

Reference level
In existing controllable exposure situations, this represents the level of dose or risk, above which it is judged to be inappropriate to plan to allow exposures to occur, and below which optimisation of protection should be implemented. The chosen value for a reference level will depend upon prevailing circumstances of the exposure under consideration.

Risk
Risk relates to the probability that an outcome (e.g. lung cancer) will occur. Terms relating to risk are grouped together here:

- Excess absolute risk
An expression of excess risk based on the assumption that the excess risk from radiation exposure adds to the underlying (baseline) risk by an increment dependent in dose but independent of the underlying natural or background risk. In this report lifetime excess absolute risk (LEAR) of lung cancer is computed.

- Excess relative risk (ERR)
Relative risk – 1.

- Relative risk
The ratio of the incidence rate or the mortality rate from the disease of interest (lung cancer) in an exposed population to that in an unexposed population.

- Risk coefficient
Increase of risk per unit exposure or per unit dose. In general, expressed as ERR per WLM, per J h m⁻³, per 100 Bq m⁻³ or per Sv.

- Risk model
A model describing the variation of the risk coefficient as a function of modifying factors, such as time since exposure, attained age or age at exposure. It may be related by a factor to the age specific baseline risk (multiplicative) or added to the baseline risk (additive).

- **Lifetime risk**
  Risk cumulated by an individual up to a given age. The estimate used in the present report is the Lifetime Excess Absolute Risk (LEAR) associated to a chronic exposure scenario, expressed in number of death $10^{-4}$ per WLM (also sometimes denominated as the Radiation Excess Induced Death REID). In the present report, except if otherwise stated, the lifetime duration is 90 years as generally considered in ICRP publications, and the scenario is a constant low level exposure to 2 WLM per year during adulthood from 18 years to 64 years, as proposed in ICRP Publication 65 (ICRP, 1993).

- **Detriment**
  Detriment is an ICRP concept. It reflects the total harm to health experienced by an exposed group and its descendants as a result of the group’s exposure to a radiation source. Detriment is a multi-dimensional concept. Its principal components are the stochastic quantities: probability of attributable fatal cancer, weighted probability of attributable non-fatal cancer, weighted probability of severe heritable effects, and length of life lost if the harm occurs.

- **Detriment-adjusted risk**
  The probability of the occurrence of a stochastic effect, modified to allow for the different components of the detriment in order to express the severity of the consequence(s).

**Thoron progeny**
The decay products of radon-220, used herein in the more limited sense of the short-lived decay products from polonium-216 through polonium-212 or thallium-208.

**Unattached fraction**
The fraction of the potential alpha energy concentration of short-lived radon progeny that is not attached to the ambient aerosol.

**Upper reference levels**
Maximum values of exposure under which ICRP recommends national authorities to establish their own national reference levels.

**Working level (WL)**
Any combination of the short-lived progeny of radon in one litre of air that will result in the emission of $1.3 \times 10^5$ MeV of potential alpha energy. 1 WL = $2.08 \times 10^{-5} \text{ J m}^{-3}$.
Working Level Month (WLM)

The cumulative exposure from breathing an atmosphere at a concentration of 1 working level for a working month of 170 hours.

Units

Joules (J) : $1 \text{ J} = 6.242 \times 10^{12} \text{ MeV}$

Potential alpha energy concentration:

Radon progeny :

$1 \text{ Bq m}^{-3} \text{ of radon at equilibrium} = 3.47 \times 10^4 \text{ MeV m}^{-3} = 5.56 \times 10^{-9} \text{ J m}^{-3}$

Thoron progeny :

$1 \text{ Bq m}^{-3} \text{ of thoron at equilibrium} = 4.72 \times 10^5 \text{ MeV m}^{-3} = 7.56 \times 10^{-8} \text{ J m}^{-3}$

Working level:

$1 \text{ WL} = 1.3 \times 10^8 \text{ MeV m}^{-3}$

$1 \text{ WL} = 2.08 \times 10^{-5} \text{ J m}^{-3}$

Working level month (WLM) :

$1 \text{ WLM} = 3.54 \times 10^{-3} \text{ J h m}^{-3}$

$1 \text{ WLM} = 6.37 \times 10^5 \text{ Bq h m}^{-3} \text{ EEC of radon}$

$1 \text{ WLM} = 6.37 \times 10^5/F \text{ Bq h m}^{-3} \text{ of radon}^{(a)}$

$1 \text{ Bq m}^{-3} \text{ of radon during 1 year} = 4.4 \times 10^{-3} \text{ WLM at home}^{(b)}$

$1 \text{ Bq m}^{-3} \text{ of radon during 1 year} = 1.26 \times 10^{-3} \text{ WLM at work}^{(b)}$

$1 \text{ WLM} = 4.68 \times 10^4 \text{ Bq h m}^{-3} \text{ EEC of thoron}$

(a) $F = \text{equilibrium factor}$

(b) Assuming 7000 h per year indoors or 2000 hours per year at work and an equilibrium factor of 0.4 (ICRP, 1993).
1. INTRODUCTION

(1) Radon-222 is a naturally occurring radioactive gas, with a half-life of 3.8 days. It is formed as the decay product of radium-226 (half-life 1600 years), which is a member of the uranium-238 decay chain. Uranium and radium occur naturally in soil and rocks and provide a continuous source of radon. Radon gas emanates from the earth’s crust and as a consequence is present in the air outdoors and in all buildings, including workplaces. There is a large variation of indoor air concentrations of this gas, depending mainly on the geology of the area and factors that affect the pressure differential between the inside and outside of the building, such as ventilation rates, heating within the building and meteorological conditions.

(2) Because radon is inert, nearly all of the gas that is inhaled is subsequently exhaled. However, $^{222}\text{Rn}$ decays into a series of solid short-lived radioisotopes which deposit within the respiratory tract. Because of their relatively short half-lives (less than half an hour), the radon progeny decay mainly in the lung before clearance can take place. Two of these short-lived progeny, polonium-218 and polonium-214, emit alpha particles and it is the energy from these alpha particles that dominates dose to the lung and the associated risk of lung cancer.

(3) The historical unit of exposure to radon progeny applied to the uranium mining environment is the working level month (WLM) which is related to the potential alpha energy concentration of its short-lived progeny. One WLM is defined as the cumulative exposure from breathing an atmosphere at a concentration of 1 working level (WL) for a working month of 170 hours. A concentration of 1 WL is any combination of the short-lived radon progeny in one litre of air that will result in the emission of $1.3 \times 10^5$ MeV of alpha energy. One WLM is equivalent to $3.54 \times 10^{-3}$ J h m$^{-3}$ in SI units. Exposures can also be quantified in terms of the activity concentration of the radon gas in Bq h m$^{-3}$. The two units are related via the equilibrium factor, $F$, which is a measure of the degree of disequilibrium between radon and its short-lived progeny ($1 \text{WLM} = 6.37 \times 10^5 / F \text{Bq h m}^{-3}$; $1 \text{J h m}^{-3} = 1.8 \times 10^8 / F \text{Bq h m}^{-3}$). Thus, an annual domestic exposure of 227 Bq m$^{-3}$ gives rise to 1 WLM, assuming occupancy of 7000 h y$^{-1}$ and $F$ value of 0.4.

(4) Radon has long been recognised as a cause of lung cancer and it was identified as a human lung carcinogen in 1986 by the World Health Organisation (WHO, 1986; IARC, 1988). The main source of information on risks of radon-induced lung cancer has been epidemiological studies of underground miners (ICRP, 1993), and more recent studies have provided informative data on risks at lower levels of exposure (e.g., Lubin et al., 1997b; NRC, 1998; EPA, 1999; 2003, Tomášek et al., 2008a). In addition, recent combined analyses of data from case-control studies of lung cancer and residential radon exposures have demonstrated raised risks (Darby et al., 2005; 2006; Krewski et al., 2006; Lubin et al., 2004).

(5) A complication in the specification and control of doses and risks from radon has been that doses can be calculated in two ways: the so-called “epidemiological” approach and the “dosimetric” approach. Publication 65 (ICRP, 1993) recommended an epidemiological approach in which the risk of fatal lung cancer per unit radon exposure (in J h m$^{-3}$ or WLM) was compared with the total risk, expressed as detriment, per unit effective dose (in Sv). Hence, values of mSv (effective dose) per mJ h m$^{-3}$ or WLM were obtained and referred to as the dose conversion convention. Alternatively, various dosimetric models of the human respiratory tract, including the ICRP (1994) model can be used to estimate
equivalent dose to the lungs and effective dose per unit radon and radon progeny exposure. Given the uncertainties inherent in the estimation of risks from radiation exposure, and in the calculation of doses using dosimetric models, it is not surprising that the two approaches to calculating effective dose per unit radon exposure have resulted in different values. In fact, the differences are remarkably small. However, the use of different values by different organisations, notably by ICRP (1993) and UNSCEAR (2000) suggests the need for clarification and the formulation of a consistent approach. ICRP now intends to treat radon and its progeny in the same way as other radionuclides and publish dose coefficients calculated using models for use within the ICRP system of protection.

(6) This report considers epidemiological data on radon risks published since ICRP Publication 65 (ICRP, 1993) focusing on studies involving low levels of protracted exposure. Results of pooled residential case-control studies are discussed in Chapter 2 and results of recent miner epidemiological studies with low exposures are discussed in Chapter 3. The miner data are used to recommend a revised estimate of lung cancer lifetime risk per unit radon progeny exposure at low protracted levels of exposure to radon and its progeny. Appendixes provide additional information on epidemiological results obtained from miner studies (Appendix A) and review published results of dose per unit exposure to radon progeny and thoron progeny, calculated using dosimetric models of the human respiratory tract (Appendix B).
2. EPIDEMIOLOGY OF LUNG CANCER RISK ASSOCIATED WITH RESIDENTIAL EXPOSURES TO RADON AND RADON PROGENY

2.1. Introduction

(7) In 1988, the International Agency for Research on Cancer (IARC, WHO) classified radon as a human lung carcinogen, based on a review of evidence from experimental data on animals and from epidemiological studies of underground miners exposed to relatively high radon and radon progeny concentrations. This report is focused on those epidemiological studies able to provide information on the dose-response relationship between lung cancer risk at relatively low annual exposures to radon and radon progeny. Studies which include both individual exposure assessment and individual assessment of potential confounding factors or co-factors, such as tobacco use, are given special emphasis. Ecological studies of cancer rates and average exposure per country or per region do not provide individual exposure data and are not considered: they are unable to provide reliable information on risk and are limited due to the unknown effect of confounding factors, including smoking, and the unknown influence of population movement into and out of the study areas (WHO, 1996; NRC 1998).

(8) The applicability of studies of underground miners to estimate radon-induced lung cancer for residential concentrations of radon has been an important uncertainty over the last twenty years. A variety of factors need to be considered in this extrapolation from mines to homes, including the linearity of the dose-response relationship, any differences between risks for adult males and the general population which includes women and children, the difference in other environmental exposures which may include arsenic, quartz and diesel exhaust amongst others, different equilibrium factors between radon and its short-lived progeny, and different breathing rates.

(9) Because of the desirability of having direct information on risks associated with domestic radon concentrations, a large number of residential epidemiological studies were launched in the late 1980s and early 1990s. There was also an awareness that pooling of data may be required to provide the statistical power to demonstrate a significant risk at residential exposure concentrations (Lubin et al., 1997a). Reliable estimates of individual exposure conditions over long periods of time were an important prerequisite of the epidemiological studies, with long-term radon measurement in the current and previous homes of each individual. Individuals’ habits and ventilation conditions in dwellings had to be considered.

2.2. Studies published since 1990

(10) This section includes analytical epidemiological studies that have been published since 1990 that have included at least 200 lung cancer cases, as well as long-term domestic radon measurements. Selected studies are listed in Table 2.1; more extensive details are available elsewhere (UNSCEAR, 2009).

(11) In most of the studies, year-long measurements of radon and its decay products were made using standard methodologies in order to integrate any variations in the specific conditions of the dwellings and any climatic and seasonal changes. Most measurements are of concentrations in air using alpha track detectors. In a few studies, glass-based retrospective detectors were also used.
A number of European studies were designed with the intention of conducting a pooled analysis (see Section 2.3). Considerable efforts were made to have comparable protocols before starting studies in different countries. They were all case-control studies, with face-to-face interviews whenever possible of both the cases (lung cancer patients) and the controls (hospital controls or controls from the general population). The same detailed questionnaire was used to analyse lung cancer risk in relation to domestic radon exposure, adjusting for tobacco consumption, occupational exposures, and indicators of socio-economic status. These studies provide information on lung cancer risks from radon, for smokers and non-smokers, and allow adjustment to be made relating to years as a smoker, the age smoking began, years since stopping smoking, and the number of cigarettes per day. Several large case-control studies were also conducted in Canada and in the USA, as well as two studies in China, one in Shenyang and one in Gansu. Table 2.1 summarizes 20 studies published between 1990-2006.
Table 2.1. Residential radon cases-controls studies and one cohort study with more than 200 lung cancer cases published between 1990 and 2006

<table>
<thead>
<tr>
<th>Reference</th>
<th>Region</th>
<th>Population</th>
<th>No of Cases / Controls</th>
<th>Measurement period</th>
<th>Relative risk per 100 Bq/m³</th>
<th>95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schoenberg et al., 1990</td>
<td>USA (New Jersey)</td>
<td>Women</td>
<td>480 cases, 442 controls</td>
<td>1 year</td>
<td>1.49</td>
<td>0.89-1.89</td>
</tr>
<tr>
<td>Blot et al., 1990</td>
<td>China-Shenyang</td>
<td>Women</td>
<td>308 cases, 356 controls</td>
<td>1 year</td>
<td>0.95</td>
<td>undefined-1.08</td>
</tr>
<tr>
<td>Pershagen et al., 1992</td>
<td>Sweden</td>
<td>Women</td>
<td>201 cases, 378 controls</td>
<td>1 year</td>
<td>1.16</td>
<td>0.89-1.92</td>
</tr>
<tr>
<td>Pershagen et al., 1994</td>
<td>Sweden</td>
<td>-</td>
<td>1281 cases, 2576 controls</td>
<td>3 months</td>
<td>1.10</td>
<td>1.01-1.22</td>
</tr>
<tr>
<td>Letourneau et al., 1994</td>
<td>Canada</td>
<td>-</td>
<td>738 cases, 738 controls</td>
<td>1 year</td>
<td>0.98</td>
<td>0.87-1.27</td>
</tr>
<tr>
<td>Alavanja et al., 1994</td>
<td>USA (Missouri)</td>
<td>Women, never-smokers</td>
<td>538 cases, 1183 controls</td>
<td>1 year</td>
<td>1.08</td>
<td>0.95-1.24</td>
</tr>
<tr>
<td>Auvinen et al., 1996</td>
<td>Finland</td>
<td>-</td>
<td>517 cases, 517 controls</td>
<td>1 year</td>
<td>1.11</td>
<td>0.94-1.31</td>
</tr>
<tr>
<td>Ruoostenoja et al., 1996</td>
<td>South Finland</td>
<td>Men</td>
<td>318 cases, 1500 controls</td>
<td>2 months</td>
<td>1.80</td>
<td>0.90-3.50</td>
</tr>
<tr>
<td>Darby et al., 1998</td>
<td>United Kingdom</td>
<td>-</td>
<td>982 cases, 3185 controls</td>
<td>6 months</td>
<td>1.08</td>
<td>0.97-1.20</td>
</tr>
<tr>
<td>Alavanja et al., 1999</td>
<td>USA (Missouri)</td>
<td>Women</td>
<td>477 cases, 516 controls</td>
<td>1 year</td>
<td>1.27</td>
<td>0.88-1.53</td>
</tr>
<tr>
<td>Field et al., 2000</td>
<td>USA (Iowa)</td>
<td>Women</td>
<td>413 cases, 614 controls</td>
<td>1 year</td>
<td>1.24</td>
<td>0.95-1.92</td>
</tr>
<tr>
<td>Kreienbrock et al., 2001</td>
<td>Germany (West)</td>
<td>-</td>
<td>1449 cases, 2297 controls</td>
<td>1 year</td>
<td>0.97</td>
<td>0.82-1.14</td>
</tr>
<tr>
<td>Lagarde et al., 2001</td>
<td>Sweden</td>
<td>Never-smokers</td>
<td>436 cases, 1649 controls</td>
<td>3 months</td>
<td>1.10</td>
<td>0.96-1.38</td>
</tr>
<tr>
<td>Wang et al., 2002</td>
<td>China-Gansu</td>
<td>-</td>
<td>768 cases, 1659 controls</td>
<td>1 year</td>
<td>1.19</td>
<td>1.05-1.47</td>
</tr>
<tr>
<td>Kreuzer et al., 2003</td>
<td>Germany (East)</td>
<td>-</td>
<td>1192 case, 1640 controls</td>
<td>1 year</td>
<td>1.08</td>
<td>0.97-1.20</td>
</tr>
<tr>
<td>Baysson et al., 2004</td>
<td>France</td>
<td>-</td>
<td>486 cases, 984 controls</td>
<td>6 months</td>
<td>1.04</td>
<td>0.99-1.11</td>
</tr>
<tr>
<td>Bochicchio et al., 2005</td>
<td>Italy</td>
<td>-</td>
<td>384 cases, 404 controls</td>
<td>6 + 6 months</td>
<td>1.14</td>
<td>0.89-1.46</td>
</tr>
<tr>
<td>Sandler et al., 2006</td>
<td>USA (Connecticut + Utah-South Idaho)</td>
<td>-</td>
<td>1474 cases, 1811 controls</td>
<td>1 year</td>
<td>1.01</td>
<td>0.79-1.21</td>
</tr>
<tr>
<td>Tomášek et al., 2001</td>
<td>Czech republic</td>
<td>-</td>
<td>173 cases in a cohort of 000 inhabitants</td>
<td>12</td>
<td>1.10</td>
<td>1.04-1.17</td>
</tr>
</tbody>
</table>

CI: confidence interval; - = men and women

The studies listed in Table 2.1 evaluated the association between lung cancer and domestic radon exposure. Results are presented in terms of the relative risk per 100 Bq m⁻³ averaged for most studies over 20 to 30 years prior to lung cancer diagnosis. Two studies considered only never-smokers; most studies considered males and females, smokers and non-smokers. Risks of radon exposure are adjusted for smoking habits, and in several studies are also adjusted for occupational exposures known to be potential lung carcinogens (e.g. exposure to asbestos). Most of the twenty studies (17 out of 20 independent studies) reported a positive trend in lung cancer risk with increasing exposure, but few of the trends were statistically significant. A few studies were also consistent with the absence of a positive trend. Each study considered alone had low statistical power and provided...
an estimate of the risk per unit of exposure with a large confidence interval. Most of
the studies included only a small number of lung cancer cases that were never-
smokers and thus were limited in evaluating associations between radon decay
products and lung cancer in non-smoking populations.

(14) In most studies, there were some residences in which radon concentration
could not be measured, e.g. if the house had been demolished. Radon concentrations
for such missing periods needed to be estimated for the purposes of the statistical
analyses. Even when radon had been measured in a home, the measurements were
subject to uncertainty in the sense that repeated measurements in the same residence
and in the same period showed a high variability of radon levels. The inability to
detect an association in many individual studies may have been due to poor
retrospective radon exposure assessment and/or to there being only very few cases
and controls living in residences with high radon concentrations over 200 Bq m\(^{-3}\). In
several studies the average time weighted radon concentrations in homes occupied
by cases and controls were low, and only a few studies (e.g. in the Czech republic,
Finland, France, Sweden and Gansu, China) included persons living in relatively
high levels of exposure, above 400 Bq m\(^{-3}\).

2.3. Pooled studies

(15) Since 2000, several joint analyses were published, integrating the basic data
from individual case and control subjects and applying a standard methodology,
both in defining selection criteria and statistical analysis. It is noted that several
informative meta-analyses of radon studies have been conducted but did not have
the strengths of these pooled analyses which handle individual data in the same
manner (Lubin 1997a; NRC 1998; UNSCEAR 2009). Three joint analyses have
been conducted based on data from Europe (Darby et al., 2005), North America
(Krewski et al., 2005; 2006) and China (Lubin et al., 2004) (Table 2.2). Each joint
analysis showed evidence of lung cancer risk, increasing with cumulated domestic
exposure to radon. The exposure period considered was at least 30 years prior to
diagnosis for the North American and Chinese joint analysis and 35 years for the
European joint analysis. In each analysis the radon concentrations estimated for the
5 years prior to diagnosis were not considered since a minimum lag time of 5 years
was assumed from lung cancer induction to diagnosis based on data from studies of
underground miners (NRC 1998). In consequence the estimated risk per unit of
exposure is based on a time weighted average exposure for a window period 5 to 30
years prior to diagnosis (5 to 34 years for the European pooled analysis). The
estimates of the increase of lung cancer per unit exposure in the three joint analyses
are very close to each other and statistically compatible (Table 2.2): the values
obtained were 1.08, 1.10 and 1.13 per 100 Bq m\(^{-3}\) for Europe, America and China,
respectively. The combined estimate for Europe, North American and China was
1.09 per 100 Bq m\(^{-3}\) (UNSCEAR 2009).

(16) The relative risk of lung cancer was shown to be increased among both
smokers and non-smokers. In the European joint analysis, the estimated relative risk
per 100 Bq m\(^{-3}\) was 1.11 (95%CI : 1.00-1.28) for life long non–smokers; in the joint
North American study, the relative risk for non-smokers was of the same level; 1.10,
but not statistically significant (95%CI : 0.91-1.42).

(17) It is noteworthy that the slope of the linear exposure-response relationships
increased when analyses were restricted to those cases and controls with more
precise estimates of cumulated individual exposure, for example, if data were
considered only for individuals resident in the same house for the previous twenty years. In the North American study (Krewski et al., 2005; 2006), analysis restricted on residential stability (i.e. only 1 – 2 houses occupied in the 5 - 30 years preceding diagnosis) and completeness of radon monitoring (measurements for at least 20 years of the considered period), resulted in an increase in relative risk from 1.10 to 1.18 per 100 Bq m\(^{-3}\). In the Chinese analysis (Lubin et al., 2004), when considering only those subjects living in their current homes for 30 years or more, the relative risk increased from 1.13 to 1.32 (1.07-1.91). According to the UNSCEAR 2006 report, for all three joint analyses combined the slope of the linear exposure-response relationships increased to 1.11 per 100 Bq m\(^{-3}\) when the analyses focused on those cases and controls with more precise estimates of cumulated individual exposure (UNSCEAR 2009).

(18) The joint analyses tried also to take account of uncertainties associated with variations in exposure (Fearn et al., 2008). In the European pooled analysis (Darby et al., 2005; 2006), taking account of random uncertainties in radon measurements increased the estimate of the relative risk from 1.08 to 1.16 per 100 Bq m\(^{-3}\).

(19) Limiting the European analysis to those cases and controls with a relatively low annual exposure, there is convincing evidence of an increased risk for those exposed to levels below 200 Bq m\(^{-3}\) (Darby et al., 2006).

(20) One of the strengths of these joint analyses is that efforts were made to collect detailed past smoking habits on the basis of direct interviews in most studies and each analysis included adjustment for smoking. For the European pooling (Darby et al., 2005; 2006), a negative correlation between residential radon and smoking was demonstrated, meaning that failure to take account of smoking would have biased the estimates of risks from radon towards the null. The relative risk of lung cancer per 100 Bq m\(^{-3}\) was 1.02 when stratifying by study, region, age and sex, but not smoking. This estimate increased to 1.05 after additionally stratifying for smoking using seven categories (never-smokers; current cigarette smokers of <15, 15-24 or \(\geq 25\) cigarettes per day; ex-smokers of <10years or \(\geq 10\) years duration; and unknown). A further increase to 1.08 was observed when current smokers were further stratified by age at starting smoking and ex-smokers were stratified by the number of cigarettes smoked.

Table 2.2 Pooled analyses of case-control studies of residential exposure to radon and lung cancer, based on measured radon concentrations

<table>
<thead>
<tr>
<th>Joint analysis (Darby et al., 2006)</th>
<th>Number of studies included</th>
<th>Number of cases</th>
<th>Number of controls</th>
<th>Relative risk per 100 Bq m(^{-3}) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>13</td>
<td>7148</td>
<td>14208</td>
<td>1.08 (1.03-1.16)</td>
</tr>
<tr>
<td>North American</td>
<td>7</td>
<td>3662</td>
<td>4966</td>
<td>1.10 (0.99-1.26)</td>
</tr>
</tbody>
</table>
In conclusion, the joint analyses of lung cancer risk from residential radon exposures show an increase in risk of at least 8% per 100 Bq m$^{-3}$, considering a period of exposure from 5 up to 30 - 35 years preceding the date of cancer diagnosis. Confining analysis to those with presumably more precise exposure measurements, the observed risk is increased in each of the joint analyses. The European pooling reported an excess relative risk increase of 16% per 100 Bq m$^{-3}$ when uncertainties in the measured radon activity concentrations were considered. This value may be considered as a reasonable estimate of the risk associated with relatively low and prolonged radon exposures in homes, considering an exposure over a period of 25 - 30 years.

When the analysis is limited to lifelong non smokers, a statistically significant positive trend is still observed in the European pooling, based on a large number of lung cancer cases: 268 in men and 616 cases in women and on more than 5000 controls (Darby et al., 2006).

On the basis of the results of the European pooling, the cumulative risk of lung cancer up to 75 year of age is estimated for lifelong non-smokers as 0.4%, 0.5% and 0.7% for radon activity concentrations of 0, 100 and 400 Bq m$^{-3}$, respectively. Lifelong cigarette smokers have a much higher baseline risk of lung cancer that is about 25 times higher than for non-smokers. The lifetime cumulative risks of lung cancer by age 75 for lifelong smokers are close to 10%, 12% and 16% for radon activity concentrations of 0, 100 and 400 Bq m$^{-3}$, respectively, and reflect the dominating effect of tobacco use on lifetime risk of lung cancer with or without radon contribution.

A “world pooling” analysis is in progress under the coordination of Sarah Darby (Oxford University), considering more then 13,700 lung cancer cases from 25 studies; it will include three supplementary studies: one from Russia (Urals) and two from North America (Massachusetts and New Jersey). Results from this large joint analysis are expected in the near future. They may provide better adjustments for cofactors, but as the dominant studies included are considered here in the three separate joint analyses from Europe, North America and China, the overall conclusion is expected to be the same: clear evidence of an increased relative risk of lung cancer related to radon exposure cumulated in houses during a residence period of at least 30 years prior to the diagnosis.
3. EPIDEMIOLOGY OF CANCER RISK ASSOCIATED TO RADON AND RADON PROGENY EXPOSURE IN UNDERGROUND MINES

3.1. Review of results since Publication 65

(25) ICRP Publication 65 (ICRP, 1993) estimated the risk of lung cancer death from radon exposure on the basis of studies on seven cohorts of miners (Colorado USA, Ontario Canada, New Mexico USA, Beaverlodge Canada, Western Bohemia Czech Republic, CEA-COGEMA France and Malmberget Sweden) (Table A1 in Appendix A). The total number of miners was 31,486. The weighted average of the excess relative risk (ERR) per 100 WLM for these studies was 1.34 (95% CI = 0.82-2.13). This ERR coefficient applied to a follow-up period of 20 years, taking into account a lag-time (minimum latency) of 5 years, i.e., radon results for exposures experienced 5 years prior to death from lung cancer (or comparable date for other miners) are excluded from the analyses. A model was derived, taking account of the modifying effects of age at exposure and time since exposure (ICRP, 1993).

(26) A comprehensive analysis of epidemiological results based on 11 cohorts of radon-exposed miners was published in 1994 (Lubin et al., 1994). In comparison to the ICRP Publication 65 report, results for some cohorts were updated (Colorado USA, Ontario Canada, Beaverlodge Canada, Western Bohemia, Czech Republic and Malmberget Sweden) and other cohorts were added (Yunnan China, Newfoundland Canada, Port Radium Canada, and Radium Hill Australia). This analysis gave an ERR per 100 WLM of 0.49 (95% CI = 0.2-1.0) (Lubin et al., 1994). After some minor updates of the same 11 cohorts, a new joint analysis was published in the BEIR VI report (NRC, 1998). This joint analysis relied on a total of 60,606 miners, with a total of 2,674 lung cancer deaths (Table A2 in Appendix A). The estimated combined ERR per 100 WLM was 0.59, assuming an exposure lag-time of 5 years. Two models were derived, taking account of modifying effects of age at exposure and time since exposure, as well as either duration of exposure or mean rate of exposure. Analyses on restricted ranges of cumulative exposure of less than 100 or 50 WLM were also performed (NRC, 1998).

(27) Since the BEIR VI report (NRC, 1998), new results have been published for the West-Bohemian cohort (uranium mines) and North Bohemian cohort (tin mines) in the Czech Republic (Tomášek and Placek, 1999; Tomášek, 2002; Tomášek et al., 2003; Tomášek and Zarska, 2004), the Newfoundland cohort (fluorspar mines) (Villeneuve et al., 2007) and the Eldorado cohort (including workers from Port Radium and Beaverlodge) (Howe, 2006) in Canada, the Colorado Plateau cohort (Schubauer-Berigan et al., 2009) in the US, the Wismut uranium mines in Germany (Kreuzer et al., 2002; Grosche et al., 2006; Kreuzer et al, 2008) and the CEA-COGEMA mines in France (Rogel et al., 2002; Laurier et al., 2004; Vacquier et al., 2008; Vacquier et al., 2009).

(28) The UNSCEAR 2006 (2009) report provided a comprehensive review of available epidemiological results from nine studies (the New Mexico USA and Australian studies were not included), including a total of more than 126 000 miners (Table A3 in Appendix A). The weighted mean average ERR per 100 WLM was 0.59 (95% CI = 0.35-1.0) (UNSCEAR, 2009).

(29) Since the UNSCEAR 2006 (2009) report, the results of a joint analysis of the Czech and French miner cohorts have been published. This analysis included 10,100 miners with a relatively long follow-up (mean, about 24 years) and relatively
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Low levels of cumulative exposure (mean, 46.8 WLM). The estimated ERR per 100 WLM was 1.6 (95% CI = 1.0-2.3) (Tirmarche et al., 2003; Tomášek et al., 2008a).

Although other miner studies have been published, they are generally not included here or in other comprehensive summaries since they provide little to no quantitative information on the relationship between radon and cancer risk.

3.2. Summary of estimates of Excess Relative Risk per WLM

The results from combined analyses summarised in Table 3.1 are presented as simple linear estimates of the ERR per WLM. They apply across the whole population of the cohorts under consideration but do not reflect variations of risk between or within the cohorts. Some characteristics of the cohorts may explain variations in the estimated ERR per WLM, including duration of follow-up, attained age, duration of work, exposure levels and background rates of lung cancer. It is important, therefore, to consider such factors in the assessment of the risk associated with radon and radon progeny exposure. Nevertheless, the three large-scale analyses that summarise most currently available information (Lubin et al., 1994; NRC, 1998; UNSCEAR, 2009) provide estimates of the association between cumulated WLM exposure and lung cancer risk that are highly concordant.

Table 3.1 Summary of Excess Relative Risk per WLM published from combined analyses of miners studies

<table>
<thead>
<tr>
<th>Reference</th>
<th>No. of cohorts</th>
<th>No. of miners</th>
<th>Person-years</th>
<th>ERR per 100 WLM</th>
<th>SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICRP, 1993</td>
<td>7</td>
<td>31 486</td>
<td>635 022</td>
<td>1.34</td>
<td>0.82</td>
<td>0.82 – 2.13</td>
</tr>
<tr>
<td>Lubin et al., 1994</td>
<td>11</td>
<td>60 570</td>
<td>908 903</td>
<td>0.49</td>
<td>0.20</td>
<td>0.20 - 1.00</td>
</tr>
<tr>
<td>NRC, 1998</td>
<td>11</td>
<td>60 705</td>
<td>892 547</td>
<td>0.59</td>
<td>1.32</td>
<td>0.35 – 1.00</td>
</tr>
<tr>
<td>UNSCEAR, 2009</td>
<td>9</td>
<td>125 627</td>
<td>3 115 975</td>
<td>0.59</td>
<td>0.35</td>
<td>0.59 – 1.00</td>
</tr>
<tr>
<td>Tomášek et al., 2008a</td>
<td>2</td>
<td>10 100</td>
<td>248 782</td>
<td>1.60</td>
<td>1.00</td>
<td>1.00 – 2.30</td>
</tr>
</tbody>
</table>

SE: standard error CI: confidence interval

(31) All of the combined analyses and some of the individual studies demonstrate a modifying effect of time since exposure (TSE) and, to a lesser extent, of age at exposure (AE) (ICRP, 1993; Lubin et al., 1994; NRC, 1998; Howe, 2006; Tomášek et al., 2008a). An inverse exposure-rate effect (or protraction enhancement effect) has also been observed in most analyses (Lubin et al., 1994; NRC, 1998), although such a modifying effect is not seen at low levels of cumulative WLM exposure (Lubin et al., 1995; Tomášek et al., 2008a) or was no more evident using improved individual dosimetric data (Howe, 2006; Vacquier et al., 2009). Models have been developed to combine the modifying effects of TSE, age and exposure rate. Two models were proposed in the BEIR VI report: the TSE-age-concentration model and the TSE-age-duration model (NRC, 1998). These models provide risk coefficients for different windows of cumulative exposure, with additional modifying effect of age and concentration/duration based on categorical variables. An alternative approach has been proposed in the joint analysis of the Czech and French cohorts, modelling the risk associated with cumulative radon exposure and...
integrating the modifying effects of TSE and AE as continuous variables (Tomášek et al., 2008a).

(32) For current radiation protection purposes, the most relevant results from miner studies are those derived for populations with low levels of cumulative exposure, long duration of follow-up and good data quality. In general, the ERR per 100 WLM estimated from cohorts with low level of exposure (for example, the Ontario, Beaverlodge and French cohorts) are higher than those estimated from cohorts with high levels of cumulated exposures, although the confidence intervals are broader (Table A3 in Appendix A). Some publications have provided estimates based on analyses on restricted ranges of exposure (Lubin et al., 1997b). In the BEIR VI report, such analyses resulted in estimated ERR per 100 WLM of 0.81 and 1.18, below 100 WLM and 50 WLM, respectively (NRC, 1998). In addition, coefficients corresponding to low exposure rates can be obtained from models that take account of modifying factors. In the BEIR VI report, an ERR per 100 WLM of 3.41 was obtained for low exposure rates < 0.5 WL (TSE-age-concentration model, for an attained age of 55 - 64 years and at 15-24 years following exposure) (NRC, 1998). Recent analyses from the French and Czech cohorts have provided risk estimates associated with low levels of exposure and reasonably good quality exposure assessment (“measured exposures”), with values of ERR per 100 WLM varying between 2.0 and 3.4 (Tomášek et al., 2008a; Vacquier et al., 2008). A summary of these risk estimates is presented in Table 3.2, demonstrating significant associations between cumulative radon exposure and lung cancer mortality at low levels of cumulative exposure.

Table 3.2 Estimates of the ERR per WLM based on subgroups with low levels of exposure and low exposure rate

<table>
<thead>
<tr>
<th>Reference</th>
<th>Model</th>
<th>Exposure</th>
<th>ERR per 100 WLM</th>
<th>CI 95%</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRC, 1998</td>
<td>BEIR VI restricted range</td>
<td>&lt; 100 WLM</td>
<td>0.81</td>
<td>0.30 – 1.42</td>
</tr>
<tr>
<td>NRC, 1998</td>
<td>BEIR VI restricted range</td>
<td>&lt; 50 WLM</td>
<td>1.18</td>
<td>0.20 – 2.53</td>
</tr>
<tr>
<td>NRC, 1998</td>
<td>BEIR VI TSE-age-concentration model</td>
<td>rate &lt;0.5 WL</td>
<td>3.41 *</td>
<td>-</td>
</tr>
<tr>
<td>Howe, 2006</td>
<td>Beaverlodge</td>
<td>Mean 23 WLM</td>
<td>0.96</td>
<td>0.56 – 1.56</td>
</tr>
<tr>
<td>UNSCEAR, 2009</td>
<td>Ontario</td>
<td>Mean 31 WLM</td>
<td>0.89</td>
<td>0.5 – 1.5</td>
</tr>
<tr>
<td>Vacquier et al., 2008</td>
<td>French cohort, employed after 1956</td>
<td>Mean 17 WLM</td>
<td>2.0</td>
<td>0.91 – 3.65</td>
</tr>
<tr>
<td>Tomášek et al., 2008a</td>
<td>Joint Czech-French cohort **</td>
<td>Mean 47 WLM</td>
<td>2.7 *</td>
<td>1.7 – 4.3</td>
</tr>
</tbody>
</table>

CI: Confidence interval - WL: Working level - WLM Working Level Month
* for an attained age of 55 - 64 years at 15 – 24 years following exposure
** restricted to miners with measured radon exposures
3.3. Lung cancer risks from radon and smoking

Although smoking is by far the strongest risk factor for lung cancer, most studies of underground miners could not take account of smoking habits. Several studies have partial smoking data, including the Chinese Yunnan cohort, the Colorado Plateau cohort (United States), the Newfoundland cohort (Canada), the Sweden cohort, the New Mexico cohort (United States) and the Radium Hill cohort (South Australia). Case-controls studies among miners have also been conducted to investigate the interaction between radon exposure and smoking on lung cancer risk (Qiao et al., 1989; Lubin et al., 1990; L’Abbe et al., 1991; Thomas, 1994; Yao et al., 1994; Brüske-Hohlfeld et al., 2006; Leuraud et al., 2007; Amabile et al., 2009). More information on the lung cancer risk associated with both radon and cigarette smoking should be available in the future as new datasets from cohort and case-control studies are currently under development in Canada (Ontario cohort) and in Europe (Czech, German and French cohorts) (Tirmarche et al., 2009).

Considering currently available data (up to 2009), the results indicate that the relationship between lung cancer mortality and radon exposure persists when account is taken of smoking habits. The analyses conducted for the BEIRVI report demonstrated a sub-multiplicative interaction between radon exposure and smoking status (NRC, 1998). In a recent French nested case-control study, the ERR for lung cancer related to cumulative radon exposure, adjusted for smoking, was 0.85 per 100 WLM (Leuraud et al., 2007), while the value obtained from the total French cohort, when smoking information is ignored, was 0.82 per 100 WLM (Rogel et al., 2002; Tirmarche et al., 2003). Tirmarche et al. (2003) concluded that presently available models derived from cohort studies of underground miners that do not take account of smoking status appear acceptable for the estimation of radon associated lung cancer risks in a population including both smokers and non-smokers. When the smoking status is known, the estimated ERR generally appears to be larger (even if not significantly) among non-smokers than among smokers (Lubin et al., 1994; Tomasek et al., 2002).
4. ASSESSMENT OF THE DETRIMENT FROM RADON AND RADON PROGENY EXPOSURE

4.1. Risks other than lung cancer

(35) Radon and its progeny deliver substantially more dose to the lung than to systemic organs and the gastrointestinal tract regions. Nevertheless, calculations indicate that small doses may be received by the red bone marrow and other systemic organs (Kendall and Smith, 2002; 2005; Khursheed, 2000).

(36) Studies of underground miners generally have not shown any excess of cancer other than lung cancer to be associated with radon exposure (Darby, 1995; NRC, 1998; UNSCEAR, 2009). There have been some associations suggested in individual studies but they have not been replicated in other studies and no consistent pattern has emerged. For example, recent studies in the Czech Republic indicated an association with chronic lymphocytic leukaemia incidence (Rericha et al., 2006), but this finding was not confirmed by other studies in the Czech Republic (Tomášek and Malatova, 2006) and in Germany (Möhner et al., 2006). An excess of larynx cancer suggested in some analyses was also not confirmed in recent studies (Laurier et al., 2004; Möhner et al., 2008). Specific excesses or trends with radon exposure were noted by recent studies for non-Hodgkin lymphoma, multiple myeloma, kidney, liver and stomach cancers (Vacquier et al., 2008; Kreuzer et al., 2008; Schubauer-Berigan at al., 2009), but such observations have not been confirmed by other studies.

(37) Epidemiological studies have been conducted on the possible association between leukaemia and indoor radon concentrations (Laurier et al., 2001; Raaschou-Nielsen, 2008). For childhood leukaemia, an association with domestic radon exposure has been observed in some ecological studies, including the recent findings of Evrard et al. (2005; 2006). Several large-scale case-control studies which included alpha-track measurements in the homes of all subjects were unable to confirm an association between radon exposure and leukaemia risk (Steinbuch et al. 1999; Lubin et al. 1998; UK Childhood Cancer Study Investigators 2002), but a recent study in Denmark suggested a positive significant association between radon concentrations, estimated on the basis of comprehensive modelling, and acute lymphocytic leukaemia (Raaschou-Nielsen et al., 2008). A recent review concluded that an association between indoor exposure to radon and childhood leukaemia might exist, but the current epidemiological evidence is weak and further research with better study designs is needed (Raaschou-Nielsen, 2008).

(38) In conclusion, the review of the available epidemiological evidence shows no consistent evidence for an association between radon concentrations and cancer other than that of the lung.

(39) It is noted that most available data relate to adult population. While dosimetric calculations indicate that doses per unit exposure should not differ appreciably between children and adults (see Appendix, paragraph B 10), more information is needed to quantify the effects of exposures received during childhood.
4.2. Calculation of lung cancer lifetime risk estimates for underground miners

Most miner studies have demonstrated the existence of time modifying factors of the relationship between cumulated radon exposure and lung cancer risk, such as age at exposure or time since exposure. Due to variations in the characteristics of the study populations (attained age, duration of follow-up), the direct comparison of ERR estimates obtained from different cohorts may be misleading. Account can be taken of such variations in the calculation of the lifetime risk associated with a specific exposure scenario (Thomas et al., 1992). In outline, calculation of lifetime risk requires:

1. Risk coefficients derived from an epidemiological study or studies, with or without modifying factors such as attained age.
2. A projection model, enabling extrapolation of risk outside the range considered by the epidemiological study (exposure range, sex, age) and transport to other populations.
4. A scenario of exposure to radon concentrations.

This approach was used in the ICRP Publication 65 to estimate the risk of lung cancer associated with prolonged exposure to radon concentrations based upon studies of underground miners (ICRP, 1993). Since that, several lifetime risk estimates have been published (NRC, 1998; EPA, 2003; Tomášek et al., 2008a), but cannot be easily compared due to differences in the nature of the estimates or in the underlying assumptions. We focus here on estimates of the lifetime excess absolute risk (LEAR) of lung cancer death from radon and radon progeny, as considered in ICRP Publication 65, and we exclude those derived for background rates corresponding to a specific country. We prioritized models derived from pooled analyses rather than from single studies. The published estimates are summarized in Table 4.1.

Table 4.1 Estimates of the lifetime excess absolute risk (LEAR) of lung cancer associated with radon and radon progeny concentrations in underground mines (ICRP Publication 65 scenario of a constant exposure to 2 WLM/y from age 18 to 64y)

<table>
<thead>
<tr>
<th>Primary risk model</th>
<th>Projection model</th>
<th>Background reference rates</th>
<th>LEAR (10^{-4}/\text{WLM})</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICRP 65 (ICRP, 1993)</td>
<td>Relative risk</td>
<td>ICRP 60</td>
<td>2.8</td>
<td>ICRP, 1993</td>
</tr>
<tr>
<td>ICRP 65 (ICRP, 1993)</td>
<td>Relative risk</td>
<td>ICRP 103</td>
<td>2.7</td>
<td>Tomášek et al., 2008b</td>
</tr>
<tr>
<td>BEIR VI model TSE-age-c (NRC, 1998)</td>
<td>Relative risk</td>
<td>ICRP 103</td>
<td>5.3</td>
<td>Tomášek et al., 2008b</td>
</tr>
<tr>
<td>Czech-French joint model * (Tomášek et al., 2008a)</td>
<td>Relative risk</td>
<td>ICRP 103</td>
<td>4.4</td>
<td>Tomášek et al., 2008b</td>
</tr>
</tbody>
</table>

ICRP 60 reference rates: averaged over males and females and over 5 countries
ICRP 103 reference rates: averaged over males and females and over asian and euro-american populations
* model relying on periods of work with the best quality of exposure assessment

The scenario of exposure considered in estimating the LEAR shown in Table 4.1 is as proposed in ICRP Publication 65 (ICRP, 1993): constant low level exposure to 2 WLM per year during adulthood from 18 years to 64 years, with risk
estimated up to 90 or 94 years of age. Using the reference background rates of lung cancer from Publication 60 (ICRP, 1991), Publication 65 (ICRP, 1993) adopted a LEAR for lung cancer (also denoted as the nominal probability coefficient or fatality probability) of $2.8 \times 10^{-4}$ per WLM for radon exposure. Since detriment was entirely due to lung cancer mortality, the Commission adopted a total detriment coefficient equal to this fatality coefficient (ICRP, 1993).

(43) Applying the same risk coefficient as in Publication 65 (ICRP, 1993) to the reference background rates found in Publication 103 (ICRP, 2007), Tomášek et al. (2008b) calculated a LEAR of lung cancer of $2.7 \times 10^{-4}$ per WLM. This comparison shows that the modification of the reference population for background cancer rates between Publication 60 and Publication 103 has only a small impact on the estimated LEAR.

(44) Using the same scenario of exposure as in Publication 65 (ICRP, 1993) and reference background rates from the Publication 103 (ICRP, 2007), Tomášek et al. (2008b) also calculated the LEAR using the BEIR VI TSE-age-concentration model (NRC, 1998). This model relies on the combined analysis of data from 11 cohorts of miners, and takes into account modifying effects of attained age, TSE, and exposure rate (note that the scenario corresponds only to the lowest category of exposure rate). The LEAR estimate based on this model was $5.3 \times 10^{-4}$ per WLM.

(45) Based on the same assumptions (scenario of exposure of Publication 65 (ICRP, 1993) and reference background rates from Publication 103 (ICRP, 2007)), Tomášek et al. (2008b) calculated the LEAR using the model developed from the combined analysis of the Czech-French cohorts (Tomášek et al., 2008a). This model used exposure data for the periods of work with the best quality of exposure assessment. It took account of the modifying effects of age at exposure and TSE. As the analysis focused on miners with low levels of exposure, no exposure rate effect was observed in this analysis (Tomášek et al., 2008a). The LEAR estimate based on the Czech-French model was $4.4 \times 10^{-4}$ per WLM (Tomášek et al., 2008a).

(46) Table 4.1 shows a substantial increase in LEAR when using both the BEIR VI model and the Czech-French model compared to the LEAR estimated using the model from Publication 65 (ICRP, 1993). Other published lifetime estimates, based on specific national rates and therefore not directly comparable to the LEAR estimated in Publication 65 (ICRP, 1993), also support a tendency for an increase in the estimated lifetime risks compared to earlier values (EPA, 2003). This increase in LEAR estimates is related in part to consideration of chronic low rate exposures and in part to the increase in the estimated ERR per WLM observed in recent studies.

(47) Additional LEAR calculations were performed by the Task Group in order to validate the published results, and to provide a sensitivity analysis of the different underlying hypotheses using different models, scenarios and background rates. Some calculations were performed independently by different experts to provide an internal quality check; we especially thank Doug Chambers and Ladislav Tomášek. Results confirmed the higher LEAR estimated using the BEIR VI model and the Czech-French model. In addition to these models derived from pooled analyses, other recent models obtained from single studies were also considered (French CEA-AREVA cohort (Vacquier et al., 2008; Canadian Eldorado cohort (Howe, 2006), German Wismut cohort (Grosche et al., 2006)). It showed that the estimated LEAR can vary from about $3$ to $7 \times 10^{-4}$ per WLM according to the model used. These results illustrate the sensitivity of the estimate to the choice of the model, and reinforced our preference for models derived from pooled analyses. Other calculations also illustrated the sensitivity of LEAR estimates to background rates. Using the rates for Euro-American males instead of the reference rates averaged...
over males and females and over European-American and Asian populations (ICRP 2007), the estimated LEAR is about $7 \times 10^{-4}$ per WLM. This difference is due to the higher background lung cancer rate among European-American males. Conversely, using lower background lung cancer rates (such as females or non-smokers) would lead to lower estimated LEAR per WLM.

(48) We conclude that a LEAR of $5 \times 10^{-4}$ per WLM ($14 \times 10^{-5}$ per mJ h m$^{-3}$), should now be used as the nominal probability coefficient for radon and radon progeny induced lung cancer, replacing the ICRP Publication 65 value of $2.8 \times 10^{-4}$ per WLM ($8 \times 10^{-5}$ per mJ h m$^{-3}$). Current knowledge of radon associated risks for organs other than the lungs, does not justify the selection of a detriment coefficient different from the fatality coefficient for radon exposure. The estimated lifetime excess absolute risk of lung cancer death, corresponding to the attributable probability of fatal lung cancer (or nominal fatality probability coefficient), is therefore considered to reflect the lifetime detriment associated with radon and radon progeny exposure.

4.3. Comparison of results from underground mine and domestic exposures

(49) The comparison of results obtained from miner studies and from indoor studies is not straightforward. This is due mainly to the use of different epidemiological designs (mostly cohort studies for miners and case-control studies for indoor exposures) as well as different measures of exposure (WLM in mines, radon gas concentrations in homes). The miner studies have the advantage of considering the evolution over time of the individual radon cumulative exposure and therefore enable the consideration of the modifying effects of age and time since exposure, but often are unable to consider the effect of cofactors, such as smoking. The domestic case-control studies have the advantage of providing detailed information about many potential cofactors, but contemporary measures must be used to estimate prior radon concentrations during previous decades. They generally consider only the average radon concentration in a home over a given period and are not able to analyse potential time modifiers of the exposure-risk relationship.

(50) Estimated primary risk coefficients are presented in Tables 2.1 and 2.2 for indoor studies and Tables 3.1 and 3.2 (and Appendix A) for miner studies. According to ICRP Publication 65, assuming an occupancy of 7000 h y$^{-1}$ and an equilibrium factor (F) of 0.4, a concentration of 1 Bq m$^{-3}$ radon gas leads to an exposure of $4.4 \times 10^{-3}$ WLM indoor (ICRP, 1993). Most indoor case-control studies have estimated radon concentrations for periods of 30 or 35 years before diagnosis, with an exposure-lag time of five years. Therefore, considering a period of 30 years (ie. the last 35 years before diagnosis with a lag time of 5 years) and a time weighted averaged concentration of 100 Bq m$^{-3}$, the cumulated exposure of $2.1 \times 10^{7}$ h Bq m$^{-3}$ corresponds to a cumulated exposure of approximately 13 WLM assuming F=0.4. Using these values, an ERR per 100 Bq m$^{-3}$ of 0.16 for indoor exposures (as obtained in the European pooling study with uncertainty correction; Darby et al., 2006) corresponds to an ERR of 1.2 per 100 WLM, which is similar to the value obtained in the BEIR VI analysis restricted to low levels of exposure below 50 WLM (NRC, 1998; see Table 3.2). This approach indicates a reasonably good agreement between the risk coefficients estimated for lung cancer mortality from indoor studies and miner studies at low levels of exposure. The same reasoning has been presented by several authors and led to the same conclusion (Zielinski et al., 2006; UNSCEAR, 2009; Tomášek et al., 2008a).
The above approach does not consider the modifying effects of age and time since exposure on the exposure risk relationship demonstrated by miner studies. Lifetime estimates of lung cancer risk can account for these modifying factors and provide another method for comparing underground miner study results with those from indoor radon investigations. Nevertheless, due to differences in background rates, duration of life considered and scenario of exposure, considerable caution is needed in comparing published lifetime estimates obtained from miner studies (ICRP, 1993; NRC, 1998; EPA, 2003; Tomášek et al., 2008a) and from indoor studies (Darby et al., 2006).

To allow comparison of estimated risks between miner studies and the European indoor study, additional calculations were performed by the Task Group using parameters chosen to respect as closely as possible the characteristics of the available data. A specific scenario was elaborated in order to reflect the characteristics of the subjects included in the European indoor study (attained age of 70 years corresponding to the average age at diagnosis, constant exposure to 100 Bq m\(^{-3}\) over a time window of 5 to 30 years before diagnosis). To reflect the fact that miner studies provide risk estimates for males, we used the ERR per 100 Bq m\(^{-3}\) of 0.25 obtained in the European pooling study for males only (Darby et al., 2006). Using these parameters, the cumulated absolute risk up to 70 years of age estimated for two pooled analyses of miner studies (BEIR VI and French-Czech) and for the European pooled analysis of indoor exposures were 3.5, 2.7 and 2.7 \(10^{-4}\) per WLM, respectively.

In conclusion, the currently available results show reasonably good consistency between lung cancer risk estimates obtained from miner and from indoor studies.
5. CONCLUSIONS

(54) This review and analysis of the epidemiology of radon leads to the following conclusions:

• There is compelling evidence from cohort studies of underground miners and from case-control studies of residential radon exposures that radon and its progeny can cause lung cancer. For solid tumours other than lung cancer, and also for leukaemia, there is currently no convincing or consistent evidence of any excesses associated with radon and radon progeny exposures.

• The three pooled residential case-control studies (in Europe, North America and China) gave similar results and showed that the risk of lung cancer increases at least by 8% for an increase of 100 Bq m\(^{-3}\) in the radon concentration (Darby et al., 2005; Krewski et al., 2006; Lubin et al., 2004).

• After correcting for random uncertainties in the radon activity concentration measurements, the European pooled residential case control study gave an excess relative risk of 16% (5% to 32%) per 100 Bq m\(^{-3}\) increase (Darby et al., 2005). This value may be considered as a reasonable estimate for risk management purposes at relatively low and prolonged radon exposures in homes, considering that this risk is linked to an exposure period of at least 25 years.

• There is evidence from the European pooled residential case-control study that there is a risk of lung cancer even at levels of long-term average radon concentration below 200 Bq m\(^{-3}\) (Darby et al., 2005).

• The cumulative risk of lung cancer up to 75 year of age is estimated for lifelong non-smokers as 0.4%, 0.5% and 0.7% for radon activity concentrations of 0, 100 and 400 Bq m\(^{-3}\), respectively. The lifetime cumulative risks of lung cancer by age 75 for lifelong smokers are close to 10%, 12% and 16% for radon activity concentrations of 0, 100 and 400 Bq m\(^{-3}\), respectively (Darby et al., 2005; 2006). Cigarette smoking remains the most important cause of lung cancer.

• Appropriate comparisons of lung cancer risk estimates from miner studies and from indoor studies show good consistency.

• Based upon a review of epidemiological studies of underground miners, including studies with relatively low levels of exposure, a detriment adjusted nominal risk coefficient of 5 \(10^{-4}\) per WLM (0.14 per J h m\(^{-3}\)) is adopted for the lung detriment per unit radon exposure. This value of 5 \(10^{-4}\) WLM\(^{-1}\) (0.14 per J h m\(^{-3}\)) is derived from recent studies considering exposure during adulthood and is close to twice the value calculated in Publication 65 (ICRP, 1993).

(55) Risk estimates obtained from indoor epidemiological studies are sufficiently robust to enable protection of the public to be now based on residential concentration levels. ICRP Publication 65 recommended that doses from radon and its progeny should be calculated using a dose conversion convention based on miner epidemiological studies. No such conversion convention is proposed in the present report.

(56) For occupational protection purposes, dose estimates are required to demonstrate compliance with limits and constraints. In addition to the review of
epidemiology data, the Task Group also reviewed published dose calculations for radon and progeny (see Annex B). The Commission now proposes to treat radon and radon progeny in the same way as other radionuclides within the system of protection and publish dose coefficients (dose per unit exposure). Doses from radon and its progeny will be calculated using ICRP biokinetic and dosimetric models, including the ICRP Publication 66 Human Respiratory Tract Model (HRTM) and ICRP systemic models. This will apply to thoron (\(^{220}\text{Rn}\)) and thoron progeny as well as radon (\(^{222}\text{Rn}\)) and radon progeny (see Appendix B). Published values of effective dose from radon progeny inhalation derived using the HRTM range from about 10 to 20 mSv WLM\(^{-1}\) (3 to 6 mSv per mJ h m\(^{-3}\)) depending on the scenario of exposure. Reference ICRP dose coefficients per unit exposure to radon and its progeny will be published for different reference conditions of exposure, with specified aerosol characteristics and equilibrium factors. Until these dose coefficients are published, the previously recommended values (ICRP 1993) should continue to be used.
REFERENCES


## Results from epidemiological studies of underground miners

### Table A1 Characteristics of the cohorts used in ICRP Publication 65 (ICRP, 1993)

<table>
<thead>
<tr>
<th>Name-place</th>
<th>Country</th>
<th>Type of mine</th>
<th>Follow-up period</th>
<th>Nb miners</th>
<th>Cumul expo WLM</th>
<th>Person-years *</th>
<th>ERR 100 WLM</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorado *</td>
<td>USA</td>
<td>Uranium</td>
<td>1951-82</td>
<td>2 975</td>
<td>510</td>
<td>66 237</td>
<td>0.60</td>
<td>0.30-1.42</td>
</tr>
<tr>
<td>Ontario</td>
<td>Canada</td>
<td>Uranium</td>
<td>1955-81</td>
<td>11 076</td>
<td>37</td>
<td>217 810</td>
<td>1.42</td>
<td>0.60-3.33</td>
</tr>
<tr>
<td>New Mexico</td>
<td>USA</td>
<td>Uranium</td>
<td>1957-85</td>
<td>3 469</td>
<td>111</td>
<td>66 500</td>
<td>1.81</td>
<td>0.71-5.46</td>
</tr>
<tr>
<td>Beaverlodge</td>
<td>Canada</td>
<td>Uranium</td>
<td>1950-80</td>
<td>6 895</td>
<td>44</td>
<td>114 170</td>
<td>1.31</td>
<td>0.60-3.01</td>
</tr>
<tr>
<td>West Bohemia</td>
<td>Czech Rep.</td>
<td>Uranium</td>
<td>1953-85</td>
<td>4 042</td>
<td>227</td>
<td>97 913</td>
<td>1.70</td>
<td>1.21-2.41</td>
</tr>
<tr>
<td>Cea-Cogema</td>
<td>France</td>
<td>Uranium</td>
<td>1946-85</td>
<td>1 785</td>
<td>70</td>
<td>44 005</td>
<td>0.60</td>
<td>0.00-1.63</td>
</tr>
<tr>
<td>Malmberget</td>
<td>Sweden</td>
<td>Iron</td>
<td>1951-76</td>
<td>1 292</td>
<td>98</td>
<td>27 397</td>
<td>1.42</td>
<td>0.30-9.57</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
<td>31 486</td>
<td>120</td>
<td>635 022</td>
<td>1.34</td>
<td><strong>0.82-2.13</strong></td>
</tr>
</tbody>
</table>

ERR : excess relative risk  
CI : confidence interval  
* < 2000 WLM
Table A2 Characteristics of the cohorts considered in the BEIR VI report (NRC, 1998)

<table>
<thead>
<tr>
<th>Name-place</th>
<th>Country</th>
<th>Type of mine</th>
<th>Follow-up period</th>
<th>Nb miners *</th>
<th>Cumul expo WLM</th>
<th>Person-years *</th>
<th>ERR per 100 WLM</th>
<th>se</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yunnan</td>
<td>China</td>
<td>Tin</td>
<td>1976-87</td>
<td>13 649</td>
<td>286.0</td>
<td>134 842</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>W-Bohemia</td>
<td>Czech Rep.</td>
<td>Uranium</td>
<td>1952-90</td>
<td>4 320</td>
<td>196.8</td>
<td>102 650</td>
<td>0.67</td>
<td></td>
</tr>
<tr>
<td>Colorado</td>
<td>USA</td>
<td>Uranium</td>
<td>1950-90</td>
<td>3 347</td>
<td>578.6</td>
<td>79 556</td>
<td>0.44</td>
<td></td>
</tr>
<tr>
<td>Ontario</td>
<td>Canada</td>
<td>Uranium</td>
<td>1955-86</td>
<td>21 346</td>
<td>31.0</td>
<td>300 608</td>
<td>0.82</td>
<td></td>
</tr>
<tr>
<td>Newfoundland</td>
<td>Canada</td>
<td>Fluorspar</td>
<td>1950-84</td>
<td>1 751</td>
<td>388.4</td>
<td>33 795</td>
<td>0.82</td>
<td></td>
</tr>
<tr>
<td>Malmberget</td>
<td>Sweden</td>
<td>Iron</td>
<td>1951-91</td>
<td>1 294</td>
<td>80.6</td>
<td>32 452</td>
<td>1.04</td>
<td></td>
</tr>
<tr>
<td>New Mexico</td>
<td>USA</td>
<td>Uranium</td>
<td>1943-85</td>
<td>3 457</td>
<td>110.9</td>
<td>46 800</td>
<td>1.58</td>
<td></td>
</tr>
<tr>
<td>Beaverlodge</td>
<td>Canada</td>
<td>Uranium</td>
<td>1950-80</td>
<td>6 895</td>
<td>21.2</td>
<td>67 080</td>
<td>2.33</td>
<td></td>
</tr>
<tr>
<td>Port Radium</td>
<td>Canada</td>
<td>Uranium</td>
<td>1950-80</td>
<td>1 420</td>
<td>243.0</td>
<td>31 454</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>Radium Hill</td>
<td>Australia</td>
<td>Uranium</td>
<td>1948-87</td>
<td>1 457</td>
<td>7.6</td>
<td>24 138</td>
<td>2.75</td>
<td></td>
</tr>
<tr>
<td>Cea-Cogema</td>
<td>France</td>
<td>Uranium</td>
<td>1948-86</td>
<td>1 769</td>
<td>59.4</td>
<td>39 172</td>
<td>0.51</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
<td><strong>60 606</strong></td>
<td><strong>164.4</strong></td>
<td><strong>888 906</strong></td>
<td><strong>0.59</strong></td>
<td><strong>1.32</strong></td>
</tr>
</tbody>
</table>

ERR : excess relative risk  se: multiplicative standard error  * among exposed
Table A3 Characteristics of the cohorts considered by UNSCEAR (2009)

<table>
<thead>
<tr>
<th>Name-place</th>
<th>Country</th>
<th>Type of mine</th>
<th>Follow-up period</th>
<th>Nb miners</th>
<th>Cumulative exposure WLM</th>
<th>Person-years</th>
<th>ERR per 100 WLM</th>
<th>CI 95%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorado</td>
<td>USA</td>
<td>Uranium</td>
<td>1950-90</td>
<td>3347</td>
<td>807</td>
<td>75 032</td>
<td>0.42</td>
<td>0.3 – 0.7</td>
</tr>
<tr>
<td>Newfoundland</td>
<td>Canada</td>
<td>Fluorspar</td>
<td>1951-01</td>
<td>1742</td>
<td>378</td>
<td>70 894</td>
<td>0.47</td>
<td>0.28 – 0.65</td>
</tr>
<tr>
<td>Yunnan</td>
<td>China</td>
<td>Tin</td>
<td>1976-87</td>
<td>13 649</td>
<td>277</td>
<td>135 357</td>
<td>0.16</td>
<td>0.1 – 0.2</td>
</tr>
<tr>
<td>Wismut</td>
<td>Germany</td>
<td>Uranium</td>
<td>1946-98</td>
<td>59 001</td>
<td>242</td>
<td>1 801 626</td>
<td>0.21</td>
<td>0.18 – 0.24</td>
</tr>
<tr>
<td>Malmbgerget</td>
<td>Sweden</td>
<td>Iron</td>
<td>1951-90</td>
<td>1415</td>
<td>81</td>
<td>32 452</td>
<td>0.95</td>
<td>0.1 – 4.1</td>
</tr>
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<td>W-Bohemia</td>
<td>Czech Rep.</td>
<td>Uranium</td>
<td>1952-99</td>
<td>9979</td>
<td>70</td>
<td>261 428</td>
<td>1.60</td>
<td>1.2 – 2.2</td>
</tr>
<tr>
<td>Cea-Cogema</td>
<td>France</td>
<td>Uranium</td>
<td>1946-94</td>
<td>5098</td>
<td>37</td>
<td>133 521</td>
<td>0.80</td>
<td>0.3 – 1.4</td>
</tr>
<tr>
<td>Ontario</td>
<td>Canada</td>
<td>Uranium</td>
<td>1955-86</td>
<td>21 346</td>
<td>31</td>
<td>319 701</td>
<td>0.89</td>
<td>0.5 – 1.5</td>
</tr>
<tr>
<td>Beaverlodge</td>
<td>Canada</td>
<td>Uranium</td>
<td>1950-99</td>
<td>10 050</td>
<td>23</td>
<td>285 964</td>
<td>0.96</td>
<td>0.56 – 1.56</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
<td>125 627</td>
<td>3 115 975</td>
<td>0.59</td>
<td>0.35 – 1.0</td>
<td></td>
</tr>
</tbody>
</table>

ERR : excess relative risk  
CI : confidence interval

References for Appendix A

B.1. Radon

(B 1) The equivalent dose to the lung following the inhalation of radon and its short-lived progeny can be calculated using the ICRP Human Respiratory Tract Model (HRTM) (ICRP, 1994) and other models of the human respiratory tract. Nearly the entire lung dose arises from the inhalation of the radon progeny and not from the gas itself as almost all of the gas that is inhaled is subsequently exhaled. However, a large proportion of the inhaled radon progeny deposits in the respiratory airways of the lung. Because of their short half-lives (less than half an hour), dose is delivered to the lung tissues before clearance can take place, either by absorption into blood or by particle transport to the alimentary tract. Two of the short-lived radon progeny (\(^{218}\)Po, \(^{214}\)Po) decay by alpha particle emission and it is the energy from these alpha particles that accounts for the relatively high dose to the lung. In comparison, doses to systemic organs and gastrointestinal tract regions are low and can be ignored in the calculation of effective dose. The equivalent dose to the extrathoracic region is not small but its contribution to the effective dose is quite small.

(B 2) The radon progeny aerosol in the atmosphere is created in two steps. After decay of the radon gas, the freshly formed radionuclides (\(^{218}\)Po, \(^{214}\)Pb, \(^{214}\)Bi) react rapidly (< 1 s) with trace gases and vapours and grow by cluster formation to form particles around 1 nm in size. These are referred to as unattached particles. The unattached particles may also attach to existing aerosol particles in the atmosphere within 1 – 100 s forming the so-called attached particles. The attached particles can have a trimodal activity size distribution which can be described by a sum of three lognormal distributions (Porstendörfer, 2001). These comprise the nucleation mode with an activity median aerodynamic diameter (AMAD) between 10 nm and 100 nm, the accumulation mode with AMAD values 100 – 400 nm and a coarse mode with an AMAD > 1 \(\mu\)m. Generally, the greatest activity fraction is in the accumulation mode which has a geometric standard deviation of about 2.

(B 3) A dosimetric model for the respiratory tract needs to describe the morphology, the deposition of the inhaled material, clearance from the respiratory tract and the location of target tissues and cells at risk. For radon progeny, it is the dose to the target cells in the bronchial and bronchiolar regions of the lung that are of importance. In comparison, the dose to the alveolar region is significantly lower (UNSCEAR 1982, Marsh and Birchall, 2000).

(B 4) ICRP (1987) used values of dose per unit radon exposure based on an NEA (1983) review of available dosimetric models (Jacobi and Eisfed, 1980, 1982, James et al. 1982, Harley and Pasternack, 1982, Hofmann et al. 1980). UNSCEAR (1982; 1988; 1993) used similar estimates of dose from radon inhalation and the 2000 report retained a value of effective dose of 5.7 mSv WLM\(^{-1}\) (1.6 mSv per mJ h m\(^{-3}\), i.e. 9 nSv per Bq h m\(^{-3}\) of equilibrium equivalent concentration (ECC) of radon) for indoor and outdoor exposures (Table B.1). In the 2000 report, UNSCEAR recognised that more recent calculations with new dosimetric models resulted in higher values of this dose conversion factor. However, because of the lower values calculated using the dose conversion convention (ICRP, 1993), it was concluded that...
the previous value of 9 nSv per Bq h m\(^{-3}\) (ECC) should continue to be used in dose evaluations (UNSCEAR, 2000, 2009).

Table B.1 Published values of effective dose to an adult male from the inhalation of radon and progeny calculated using dosimetric models

<table>
<thead>
<tr>
<th>Publication</th>
<th>Model Type</th>
<th>Exposure scenario</th>
<th>Effective dose mSv WLM(^{-1})</th>
<th>Effective dose mSv per mJ h m(^{-3})</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICRP 50, 1987</td>
<td>NEA, 1983</td>
<td>Indoors</td>
<td>6.4</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Outdoors</td>
<td>8.9</td>
<td>2.5</td>
</tr>
<tr>
<td>UNSCEAR, 2000</td>
<td>NEA, 1983</td>
<td>Indoors and outdoors</td>
<td>5.7</td>
<td>1.6</td>
</tr>
<tr>
<td>Harley et al., 1996</td>
<td></td>
<td>Indoors and mines</td>
<td>9.6(^a)</td>
<td>2.7</td>
</tr>
<tr>
<td>Porstendörfer, 2001</td>
<td>Zock et al., 1996</td>
<td>Home(^b)</td>
<td>8</td>
<td>2.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Work place</td>
<td>11.5</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Outdoor</td>
<td>10.6</td>
<td>3.0</td>
</tr>
<tr>
<td>Winkler-Heil and Hofmann, 2002</td>
<td>Deterministic airway generation model</td>
<td>Home</td>
<td>7.6</td>
<td>2.1</td>
</tr>
<tr>
<td>Winkler-Heil et al., 2007</td>
<td>Deterministic airway generation model</td>
<td>Mine</td>
<td>8.3</td>
<td>2.3</td>
</tr>
<tr>
<td></td>
<td>Stochastic airway generation model</td>
<td>Mine</td>
<td>8.9</td>
<td>2.5</td>
</tr>
<tr>
<td>Marsh and Birchall, 2000</td>
<td>HRTM(^d)</td>
<td>Mine</td>
<td>11.8</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>HRTM(^d)</td>
<td>Home</td>
<td>15</td>
<td>4.2</td>
</tr>
<tr>
<td>James et al., 2004</td>
<td>HRTM(^d)</td>
<td>Mine(^c)</td>
<td>20.9</td>
<td>5.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home(^b)</td>
<td>21.1</td>
<td>6.0</td>
</tr>
<tr>
<td>Marsh et al., 2005</td>
<td>HRTM(^d)</td>
<td>Mine(^b)</td>
<td>12.5</td>
<td>3.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home(^b)</td>
<td>12.9</td>
<td>3.6</td>
</tr>
</tbody>
</table>

\(^a\) A value of an absorbed dose of 6 mGy WLM\(^{-1}\) (1.7 mGy per mJ h m\(^{-3}\)) was calculated for the bronchial region. The effective dose per unit exposure was then calculated with a radiation weighting factor for alpha particles of 20 and a tissue weighting factor of 0.08 (= \(2/3 \times 0.12\)) for lung (ICRP, 1993).

\(^b\) Home without cigarette smoke.

\(^c\) No hygroscopic growth was assumed.

\(^d\) HRTM: Human Respiratory Tract Model (ICRP, 1994).
are given in Table B.1; more comprehensive tabulations of values published between 1956 and 1998 are given by UNSCEAR (2000).

(B 6) The main sources of variability and uncertainty in the calculation of the equivalent dose to the lung per unit radon progeny exposure include:

• the activity size distribution of the radon progeny aerosol,
• breathing rates,
• the model used to predict aerosol deposition in the respiratory tract,
• the absorption of the radon progeny from lung to blood,
• the identification of target cells and their location within bronchial and bronchiolar epithelium,
• the relative sensitivity of different cell types to radiation,
• the regional differences in the radiation sensitivity of the lung.

Marsh and Birchall (2000) performed a sensitivity analysis to identify those HRTM parameters that significantly affect the equivalent dose to lung \( (H_{\text{lung}}) \) per unit exposure to radon progeny under conditions found in houses. Other sensitivity analyses have been reported (NCRP, 1984; NRC, 1991; Zock et al., 1996; Tokonami et al., 2003) and UNSCEAR (1988) noted that equivalent dose may vary by a factor of about 3 according to the target cells considered.

(B 7) Winkler et al. (2007) compared the results of the effective dose for radon progeny inhalation obtained using the HRTM, a deterministic airway generation model and a stochastic airway generation model, with the same input parameter values. Similar results were obtained ranging from 8.3 to 11.8 mSv WLM\(^{-1}\) (2.3 to 3.3 mSv per mJ h m\(^{-3}\)) (Table B.1). The authors noted that one of the important issues affecting the comparison is the averaging procedure for the doses calculated in airway generation models.

(B 8) Porstendörfer (2001) calculated doses from radon progeny exposure for different exposure scenarios using an airway generation model developed by Zock et al. (1996). The effective dose calculated for ‘normal’ aerosol conditions in homes, workplaces and outdoors ranged from 8.0 to 11.5 mSv WLM\(^{-1}\) (2.3 to 3.3 mSv per mJ h m\(^{-3}\)) (Table B.1). However, in places with one dominating aerosol source producing a high number particle concentration (e.g. cigarette smoking or combustion aerosols by diesel engines) the effective dose was calculated to be lower, ranging from 4.2 to 7.1 mSv WLM\(^{-1}\) (1.2 to 2.0 mSv per mJ h m\(^{-3}\)). The activity size distributions and unattached fractions assumed for these calculations were based upon their measurements in indoor and outdoor air, and in the air at different workplaces in Germany.

(B 9) James et al. (2004) calculated effective doses from radon progeny for mines and homes using the HRTM. The activity size distributions given in the BEIR VI report (NRC, 1998) were assumed. The authors calculated a range of values for mines (18 – 21 mSv WLM\(^{-1}\); 5.1 – 5.9 mSv per mJ h m\(^{-3}\)) and homes (16 – 21 mSv WLM\(^{-1}\); 4.5 – 5.9 mSv per mJ h m\(^{-3}\)) depending upon whether or not the attached particles double in size in the respiratory tract due to hygroscopic growth and depending upon the presence or absence of cigarette smoke in homes. These estimates are higher compared with other estimates (Table B.1), mainly because the activity size distribution assumed differed from those used by other investigators. Marsh et al. (2005), also using the HRTM and activity size distributions for mines and homes based upon measurements carried out in Europe, calculated values of about 13 mSv WLM\(^{-1}\) (3.7 mSv per mJ h m\(^{-3}\)) for mines and homes (Table B.1).

(B 10) Calculations performed with the HRTM showed that the equivalent dose to lung per unit exposure is relatively insensitive to age (BEIR VI, 1999, Marsh and
Birchall, 2000; Marsh et al., 2005, Kendall and Smith, 2005). For example the lung dose for an adult compared with that of children (> 1y) differs only by about 10%. The reason for this is that there are competing effects that tend to cancel out. Children have lower breathing rates which decreases the intakes and lung doses, while this is partly compensated by the smaller mass of target tissue which increases the doses. Also children have smaller airways which increase deposition by diffusion, but this is also compensated in part by smaller residence times that decrease deposition by diffusion.

(B 11) The values of effective dose from radon progeny inhalation derived from the HRTM range from about 10 to 20 mSv WLM$^{-1}$ (3 to 6 mSv per mJ h m$^{-3}$) depending on the scenario of exposure. For typical aerosol conditions in home and mines the effective dose is about 13 mSv WLM$^{-1}$ (3.7 mSv per mJ h m$^{-3}$) (Marsh et al., 2005). However, assuming the same aerosol conditions as for a home but with a breathing rate for a standard worker (1.2 m$^{-3}$ h$^{-1}$) the effective dose increases from 13 mSv WLM$^{-1}$ (3.7 mSv per mJ h m$^{-3}$) to about 20 mSv WLM$^{-1}$ (6 mSv per mJ h m$^{-3}$).

(B 12) The Commission has concluded that doses from radon progeny should be calculated using ICRP biokinetic and dosimetric models, including the HRTM and the ICRP systemic models. In other words, radon progeny should be treated in the same way as any other radionuclide within the system of protection. One of the advantages of this approach is that doses to organs other than lung can also be calculated. ICRP will provide dose coefficients per unit exposure to radon progeny for different reference conditions of domestic and occupational exposure (i.e., of equilibrium factor and aerosol characteristics).

B.2. Thoron

(B 13) Thoron ($^{220}$Rn) gas is a decay product of radium-224 and is part of the thorium-232 decay series. Thoron ($^{220}$Rn) has a short half-life (56 s) and decays into a series of solid short-lived radioisotopes, including lead-212 which has a half-life of 10.6 h. Because of the short half-life of thoron, it is less able than radon ($^{222}$Rn) to escape from the point where it is formed. As a consequence, building materials are the most usual source of indoor thoron exposure.

(B 14) As for radon, doses from the inhalation of thoron and progeny are dominated by alpha particle emissions from decay of the progeny (Jacobi and Eisfed, 1980, 1982). Because of its very short half-life, the gas activity concentration of thoron can vary substantially across a room and so it is not possible to use thoron gas concentration in dose evaluation. Therefore, for control purposes, the potential alpha energy concentration of the thoron progeny should be determined for the estimation of thoron exposure. However, it is usually sufficient to control the intake of $^{212}$Pb for protection purposes because the potential alpha energy (PAE) per unit activity inhaled is about 10 times higher for $^{212}$Pb than for other thoron progeny (ICRP, 1987).

(B 15) UNSCEAR (2000) and the BEIR VI committee (NAS, 1999) presented data for the ratio of potential alpha energy concentration (PAEC) arising from thoron ($^{220}$Rn) progeny to that from radon ($^{222}$Rn) progeny. The values ranged from 0.1 to 5. The highest values were for wood-frame and mud houses found in Japan and for some houses in Italy that used building materials of volcanic origin. UNSCEAR also noted that in the UK, a value as high as 30 was observed for a house with a high ventilation rate and an unusually low radon concentration.
The BEIR VI committee concluded that for dwellings with high radon (222Rn) concentrations, it appears that the thoron (220Rn) progeny will not be an important additional source of exposure and dose (NRC, 1999).

A summary of dose coefficients for thoron progeny, calculated using dosimetric models, is given in Table B.2. Values range from 1.5 – 5.7 mSv WLM\(^{-1}\) i.e. 0.42 - 1.6 mSv per mJ h m\(^{-3}\) or 10 – 122 nSv per Bq h m\(^{-3}\) (EEC).

The dose coefficient given in ICRP Publication 50 (ICRP, 1987) is based on the work of an Expert Group of OEC/NEA (NEA, 1983), which reviewed the models of Jacobi and Eisfed (1980; 1982) and of James et al. (1980; 1982). Only doses to the bronchial epithelium and pulmonary tissue were considered.

In its 1982 report, UNSCEAR not only considered the doses to the lung based upon the work of Jacobi and Eisfed (1980) but also considered doses to other tissues by applying the dosimetric models given in ICRP Publication 30 (ICRP, 1979). Values of 1.9 mSv WLM\(^{1}\) (0.54 mSv per mJ h m\(^{-3}\)) and 2.5 mSv WLM\(^{1}\) (0.71 mSv per mJ h m\(^{-3}\)) were recommended for indoor and outdoor exposures, respectively. The effective dose coefficients for thoron progeny given in the 1988 UNSCEAR report were based upon the calculations of Jacobi and Eisfed (1982) and corresponded to an effective dose per unit PAE of 0.7 mSv mJ\(^{-1}\). These values were retained in the 1993 report (UNSCEAR, 1993) and are given in Table B.2.

UNSCEAR (2000, 2009) has since adopted a value 40 nSv per h Bq m\(^{-3}\) (ECC) (i.e. 1.9 mSv per WLM or 0.54 mSv per mJ h m\(^{-3}\)) for indoors and outdoor exposures, which is similar to the value given in ICRP Publication 50 (ICRP, 1987).

The values of the dose coefficients obtained using the HRTM (Marsh and Birchall, 1999; Ishikawa et al., 2007, Kendall and Phipps, 2007) are higher than the values recommended by ICRP (1987) and UNSCEAR (1993). Kendall and Phipps (2007) calculated the effective dose conversion factor for thoron progeny with the HRTM and the most recent biokinetic models for lead (ICRP, 1993) and bismuth (ICRP 1979). The authors showed that typically the dose to the lung contributed...
more than 97% of the effective dose and that the intake from $^{212}$Pb alone contributes to about 85% of the total dose. Calculations for different age groups (> 1 y) showed that the dose per unit exposure differed by 10% or less (Kendall and Phipps, 2007).

(B 20) Following the decision to treat radon isotopes in the same way as other radionuclides for protection purposes, biokinetic and dosimetric models will be used to provide dose coefficients for radon-220 as well as radon-222.

References for Appendix B


Neuherberg, GSF.


