

ANNEX E

Sources-to-effects assessment for radon in homes and workplaces

Contents

	<i>Page</i>
INTRODUCTION	201
I. SOURCES AND LEVELS OF RADON EXPOSURES.....	203
A. Outdoors	203
B. Indoors	203
C. Workplaces	211
D. Measurements of radon and radon decay products.....	211
II. DOSIMETRY	215
A. Dosimetric models.....	215
B. Dosimetry of radon and thoron	215
C. Dose conversion factors	218
D. Uncertainties in dose conversion factors	218
E. Exposures in homes and in workplaces other than uranium mines	219
III. EXPERIMENTAL STUDIES	221
A. Animal experiments.....	221
B. Biomarkers	223
IV. EPIDEMIOLOGICAL STUDIES OF MINERS	229
A. United States: Colorado Plateau miners	229
1. Introduction	229
2. Radon and radon decay products.....	230
3. Exposure estimation.....	232
4. Other hard rock mining exposures	234
5. Epidemiological analyses	236
6. Evaluation	238
B. Canada: Ontario uranium miners	238
1. Introduction	238
2. Radon and radon decay products.....	239
3. Exposure estimation.....	240
4. Epidemiological analyses	241
5. Evaluation	242
C. Czechoslovak miners.....	242
1. Introduction	242
2. Radon and radon decay products.....	243
3. Exposure estimation.....	245
4. Epidemiological analyses	245
5. Evaluation	247

	<i>Page</i>
D. Swedish iron ore miners	247
1. Introduction	247
2. Radon and radon decay products	247
3. Exposure estimation	248
4. Epidemiological studies	249
5. Evaluation	249
E. Canada: Beaverlodge, Saskatchewan, miners	250
1. Introduction	250
2. Radon and radon decay products	251
3. Exposure estimation	254
4. Epidemiological studies	258
5. Evaluation	261
F. Germany: Wismut miners	261
1. Introduction	261
2. Radon and radon decay products	261
3. Epidemiological studies	262
4. Evaluation	265
G. Canada: Port Radium miner study	266
1. Introduction	266
2. Radon and radon decay products	267
3. Exposure estimation	268
4. Epidemiological studies	268
5. Evaluation	268
H. French uranium miners	269
1. Introduction	269
2. Radon and radon decay products	269
3. Exposure estimation	269
4. Epidemiological studies	270
5. Evaluation	271
I. Canada: Newfoundland fluorspar miners	272
1. Introduction	272
2. Radon and radon decay products	273
3. Exposure estimation	273
4. Epidemiological studies	274
5. Evaluation	276
J. Chinese miners	276
K. Australia: Radium Hill uranium miners	277
L. Overall evaluation of miner studies	277
V. EPIDEMIOLOGICAL STUDIES OF RESIDENTIAL EXPOSURES	281
A. Introduction	281
B. Case-control studies of residential radon	281
C. Ecological studies of residential radon	285
D. Overall evaluation of residential radon studies	287
VI. EFFECTS OF RADON ON ORGANS AND TISSUES OTHER THAN THE LUNG	295
A. Dosimetric considerations	295
B. Epidemiology of cancers other than lung	297
C. Effects other than cancer	298

	<i>Page</i>
VII. IMPLICATIONS FOR RISK ASSESSMENT	301
A. General studies of exposure uncertainty	301
B. Biologically based models	303
C. Risk projection	305
VIII. OVERALL CONCLUSIONS	309
APPENDIX	311
REFERENCES	313

INTRODUCTION

1. For many years, the Committee presented its evaluations of information and data on the “Sources” and “Effects” of ionizing radiation separately. These were divided into two volumes in the UNSCEAR 2000 Report [U2]. During its 50th session, in April 2001, the Committee discussed the feasibility of preparing documents in an integrated “Sources-to-Effects” approach. This document provides a “test case” for future UNSCEAR “Sources-to-Effects” assessments.

2. Radon is an inert noble gas. Its most common isotope, and the one that is commonly known as radon, is ^{222}Rn , which arises in the radioactive decay chain of uranium-238. Uranium occurs naturally in varying levels in all rocks and soils. Some fraction of the radon produced in rocks and soils escapes to the air; therefore radon is present in the atmosphere. Thus, simply by breathing, people everywhere are exposed to radiation from radon itself and also from short-lived radon decay products (RDPs).¹ Moreover, radon is soluble in water, and groundwater that passes through uranium-bearing soils and rocks contains radon. When radon-rich groundwater is used as drinking water, people are exposed both through water consumption and by radon being released from the water to the air and being inhaled.

3. Thoron (^{220}Rn) is an isotope of radon, and therefore also an inert noble gas, which arises from the decay chain of thorium-232 (^{232}Th). Thorium is a common element in the earth’s crust and therefore, like radon, thoron is found in air at varying concentrations. Since radon and thoron are members of different decay chains, the ratio between radon and thoron (or between the decay products of radon and thoron) will depend in part on the ratio of uranium to thorium in local soils, rocks or building materials. As discussed in the UNSCEAR 2000 Report [U2], the radioactive half-lives of radon and thoron and their respective decay products are also very important in determining the exposures of people in workplaces and homes. Since thoron has a much shorter half-life ($t_{1/2} = 55$ s) than radon ($t_{1/2} = 3.82$ days), the distance it can travel before undergoing radioactive decay is very much shorter than the distance radon can travel in the same medium, and therefore its expression in the environment is quite different from that of radon.

4. Sources of radon and thoron and of potential exposures to workers and the public are briefly discussed in section I. A more comprehensive discussion of sources of exposures to radon and thoron in the workplace is provided in the

UNSCEAR 2000 Report [U2]. The Committee is presently updating its assessments of these sources.

5. Historically, a wide variety of quantities and units were used to assess radon exposure. Appendix A provides a short summary of the historical units used in this report and the relevant conversion factors. To maintain the integrity of the historical data, the original units used in the papers cited in this annex are preserved. Most historical, and indeed current, measurements of radon in mines are in units of working level (WL) or working level month (WLM). The former is a measure of the potential alpha energy concentration in the air and the latter is a measure of exposure for an assumed working month of 170 hours. The unit of WLM is the traditional unit used to report exposure to RDPs in studies of miners. The modern quantity for expressing concentration of RDPs is the equilibrium-equivalent concentration (EEC), which represents the concentration of ^{222}Rn in equilibrium with its decay products that would have the same potential alpha energy. To convert from an exposure in WLM, it is necessary to multiply by 6.4×10^5 (see appendix A) to obtain Bq h m^{-3} (EEC). Where appropriate, the RDP measurement expressed as EEC is provided in brackets or is otherwise discussed.

6. For many years, radon was recognized as constituting a hazard to underground miners. However, while it was also recognized that domestic exposure to radon might carry a risk, there was no direct evidence of this until recently. The risk from residential exposure to radon and its decay products is of great interest in many countries; thus methods to evaluate exposure to radon and thoron and their decay products as well as the subsequent risks from exposure are of great interest.

7. In the past, exposures to thoron and its decay products were often ignored. As will become evident from the discussion in section I, it has become increasingly clear that the exposure to thoron and its decay products cannot be ignored in some environments (both workplace and residential) as it contributes to the risks otherwise assigned solely to exposure to radon and its decay products (e.g. [S41]). In some epidemiological studies, no distinction is made between exposure to radon and its decay products and exposure to thoron and its decay products. Measurement techniques for discriminating between them do, however, exist (e.g. [C21, T12, T14, T15, T16, T17, Z3, Z4]).

8. The use of recent measurements to estimate exposures received many years ago, for example in the uranium miner studies and in the studies of residential radon exposures,

¹ The term “radon” is used generically in this report to indicate both radon and its decay products, the latter in fact contributing most of the dose to lung tissue.

carries particular difficulties. In the case of mining, ore grade, mining methods, ventilation practices and other factors have changed over time. Residential radon studies experience similar problems in estimating past exposures, because of, for example, changes in heating and ventilation practices over time. It is thus important to recognize factors that have a substantial impact on exposure estimates and to assess the potential magnitude of these impacts. For example, the entry of radon into structures is an important consideration, is well studied and is described in many reports, including references [U2, U5, U6].

9. To understand how radon exposure estimates can be transferred from one miner population to another, or from conditions in mines to conditions in homes, it is important to understand the differences in dosimetry of exposures in mines compared with homes. The 1991 National Research Council companion report [N10] to the report of the BEIR IV Committee [C19] provides a comprehensive discussion of these differences, as do BEIR VI [C20] and the UNSCEAR 2000 Report [U2]. Section II of this annex provides a concise overview of current issues in radon and thoron dosimetry.

10. An understanding of the mechanisms for the carcinogenicity of radon and its decay products and of how radon interacts with other agents is important. Much information is available on these topics (e.g. [C20, N11, U2]). Information from animal experiments and experiments at the cellular and subcellular levels relevant to understanding the mechanisms of radon carcinogenicity is discussed briefly in section III.

11. Until recently, the main basis for estimating risks from residential exposure to radon and its decay products was provided by epidemiological studies of underground miners that extrapolated the results down to the levels of exposure seen in homes. Studies of historical miners require retrospective estimation of exposure conditions many years in the past. Often there are few or even no actual measured exposure data from the early years of mining; the results from such studies are less certain because of this and other factors. Extrapolation from risks estimated in miner studies to residential exposure conditions involves additional assumptions. For example, such extrapolations must consider the impact of the relatively short exposures at high exposure rates seen in mines compared with the longer exposures at lower exposure rates in homes. The different dosimetry of exposures in mines and in homes, as well as other factors, must be considered in selecting the most appropriate exposure–(or dose–) response model for extrapolation down to residential levels.

12. Consequently, direct estimates of residential risk from the exposure of the general population are of great interest, and numerous studies of risk from residential exposure to

radon were made using case–control studies. The results of these studies demonstrate an excess risk at the levels of radon seen in homes and suggest a pattern of increasing risk with increasing exposure that is generally consistent with the experience of epidemiological studies of miners. Section IV discusses the epidemiological studies of miners exposed to radon and section V discusses the epidemiological studies of residential exposure to radon.

13. While an increased risk of lung cancer associated with exposure to radon and its decay products is well established from epidemiological studies of underground miners and more recently from residential radon studies, the potential risks to tissues and organs other than the lung are also of interest and are the subject of section VI.

14. There is great interest in predicting future risks to people who are exposed to radon and its decay products either in the workplace or in the home. It is necessary to understand the limitations of risk projections and how such projections may be affected by consideration of exposures to other agents (cigarette smoke being the most important) in the workplace or in the home. The characteristics and limitations of existing models, including biologically based models, and a recommended approach to risk projection are the subjects of section VII.

15. The reliability of estimates of radiation exposures is one important factor in assessing the risk of cancer following radiation exposure. The sources of uncertainty in epidemiological studies and how these uncertainties affect the dose–response analysis [N5] was discussed in the UNSCEAR 2000 Report [U2]. Uncertainties arise in estimating exposures of miners at work, historically reported in WLM, and of people at home, reported in Bq h m^{-3} .

16. The sources and characteristics of uncertainty in the exposure of miners are of considerable interest and constitute a major focus of this report. The report of the BEIR VI Committee [C20] discusses this subject at length, as does the most recent radon report of the National Council on Radiation Protection and Units (NCRP) [N11], which provides the most comprehensive examination to date of the underground miner data. There are large difficulties in developing reliable estimates of underground radon exposure for epidemiological studies of miners, especially for the pre-1960 miners [C20, L10]. Studies of miners employed more recently have relatively reliable exposure information (e.g. [H35, S12, S14]). Uncertainties in assessment of exposure are similarly important considerations in residential radon studies (e.g. [D15, D17, L8]).

17. Finally, section VIII provides an overall summary of the main observations from this annex.

I. SOURCES AND LEVELS OF RADON EXPOSURES

18. The majority of the dose to the lung arises from exposure to the short-lived decay products of radon and thoron. Concentrations of the potential alpha energy of these short-lived decay products are estimated by considering the state of equilibrium between the parent nuclides and their respective decay products. In practice, an equilibrium factor F_{eq} is used to characterize the state of equilibrium. The equilibrium factor F_{eq} is defined as the ratio of the actual potential alpha energy concentration (PAEC) to the PAEC that would prevail if all the decay products in each series were in equilibrium with the parent radon or thoron, as the case may be. However, as discussed in reference [U2], it is simpler to evaluate this factor in terms of an equilibrium-equivalent radon or thoron concentration.

19. The Committee customarily reports concentrations of radon or thoron (and also the lung dose) in terms of equilibrium-equivalent concentration (EEC). This is defined as the equivalent concentration of the decay products in equilibrium with the parent gas that yields the same potential alpha energy per unit volume as the existing mixture.

$$\begin{aligned} \text{EEC}^{(222\text{Rn})} &= 0.105 (^{218}\text{Po}) + 0.516 (^{214}\text{Pb}) + 0.379 (^{214}\text{Bi}) \\ \text{EEC}^{(220\text{Rn})} &= 0.91 (^{212}\text{Pb}) + 0.087 (^{212}\text{Bi}) \end{aligned}$$

where ^{218}Po , ^{214}Pb , etc., and EECs are in Bq/m^3 . Older publications often report activity in curies (Ci). The Appendix presents relevant conversion factors.

20. Radon and thoron are ubiquitous in the air at ground level and are significant contributors to the average dose from natural background sources of radiation [U2]. In homes, in underground mines and in other situations where radon (and thoron) may be present and where ventilation may be limited, the levels of these radionuclides and their decay products can accumulate to high levels. Soils and rocks are often the main sources of radon. In unsaturated soils or rocks, radon moves with air through pores and fractures. In saturated zones, radon moves with groundwater to underground openings, such as mines and caves, and to buildings [N9].

A. Outdoors

21. Concentrations of radon in the outdoor environment are affected not only by the magnitude of the release rate from the ground to the atmosphere but also by atmospheric mixing phenomena. Solar heating during the daytime induces turbulence, so radon is more readily transported upwards and away from the ground. Doi and Kobayashi [D18] provide information on the vertical distribution of outdoor radon and

thoron in Japan. At night and in the early morning hours, atmospheric (temperature) inversion conditions are often found; these tend to trap the radon closer to the ground. This means that outdoor radon concentrations can vary diurnally by a factor of as much as 10. Seasonal variations, related to the effects of precipitation or to changes in prevailing winds, also exist [U2]. An evaluation of exposure to outdoor concentrations of radon in Iowa and Minnesota [S40] in the United States concluded that outdoor exposure to radon in some areas can be a substantial fraction of an individual's exposure to radiation and moreover is highly variable across the population. Outdoor levels of radon provide a baseline for indoor levels of radon. In tropical climates, indoor and outdoor concentrations are essentially the same because of rapid exchange between indoor and outdoor air [C43].

22. The UNSCEAR 2000 Report [U2] suggests that typical outdoor levels of radon and thoron gas are each of the order of 10 Bq/m^3 . There is, however, a wide range of long-term average concentrations of radon, from approximately 1 Bq/m^3 to more than 100 Bq/m^3 , with lower levels typical of isolated small islands or coastal regions and higher levels typical of sites with high radon exhalation over large surrounding areas. Although data are relatively sparse for thoron, considerable variability from place to place would be expected because of thoron's short half-life, which amplifies the effect of local variations in exhalation rate. Thoron decay products were measured continuously outdoors in suburban New Jersey, United States, for a 2-year period [H20]. The average outdoor concentration of ^{212}Pb was 0.09 Bq/m^3 and the variability over seasons was a factor of 2. Bismuth-212 was not detectable. The average outdoor concentration of thoron gas was 15 Bq/m^3 , yielding a value of 0.005 for the equilibrium factor outdoors [C21].

B. Indoors

23. In buildings with high radon levels, the main mechanism for entry of radon is pressure-driven flow of soil gas through cracks in the floor. This arises because the air inside buildings is normally at a slightly lower pressure than the air outdoors. This underpressure is the consequence of the air inside the building being warmer than that outside. In temperate zones especially, this causes a convective flow ("chimney effect"), which, together with the effect of the wind blowing over chimneys and other openings ("Venturi effect"), draws soil gas and hence radon into the building. However, in addition to pressure differences, other factors, including relative humidity and soil moisture, can also influence radon levels in buildings [S66].

24. While most building materials produce some radon, certain materials can act as significant sources of indoor radon. Such materials have a combination of elevated levels of ^{226}Ra (the radioactive parent of radon) and a porosity that allows the radon gas to escape. Examples are lightweight concrete with alum shale, phosphogypsum and Italian tuff.

25. Groundwater, particularly in granitic areas, can have high levels of radon. Workplaces such as laundries and restaurant kitchens can have high radon levels from the use of such water. Because municipal water supplies are often from rain catchment surface reservoirs, radon levels in public water supplies are normally not high, and any problems are normally limited to wells in geological formations containing naturally elevated levels of uranium. In the United States, groundwater supplies were reported to contain radon at levels of as high as 10^6 Bq/m³ or more [N9]. In Germany, treatment and distribution workplaces for groundwater supplies also were found to contain elevated radon concentrations in air, with up to several hundred thousand becquerels per cubic metre [U2].

26. There is a considerable amount of data available on radon concentrations in indoor air, and new information is becoming increasingly available on thoron concentrations indoors. Substantial compilations of radon measurements appeared in the UNSCEAR 2000, 1993 and 1988 Reports [U2, U5, U6]. On the basis of current data, the UNSCEAR 2000 Report [U2] deduced values of 40 and 30 Bq/m³ for the arithmetic and geometric means of indoor radon gas concentrations worldwide, with a geometric standard deviation of 2.3. On the basis of the information collected for the present report, these values are still appropriate.

27. Published literature provides additional insight into the sources and levels of radon and thoron. Examples include publications on the assessment of radon in drinking water [N9, T22] and numerous papers on residential radon levels in Iowa, United States [F3, S40], Italy [B15, F4, P6], Poland [Z9], the former Yugoslavia [Z2], China [W13, W16], the Isle of Man [G6] and the Republic of Korea [C33], among others. The influence of groundwater on radon concentrations in drinking water and subsequently on residential radon levels in air from the use of potable water indoors was also investigated in radon-prone areas of Japan [K27, I6, N17].

28. A great deal of information is available from radon measurements in homes in the United Kingdom. A national survey carried out in the early 1980s provided results of gamma ray dose rate and radon concentration measurements in more than 2,000 dwellings selected systematically according to postal codes [W14]. The mean radon level from the survey, after adjustment for dwelling types, was 20.5 Bq/m³. The same study also reported data from studies carried out in regions of the United Kingdom with elevated uranium mineralization. The regional surveys showed average radon levels of up to 300 Bq/m³ for areas of south-west England, which are about 15 times the national average. In addition, simultaneous measurements of thoron decay

product levels in high-radon regions showed a mean thoron decay product level of about 0.6 Bq/m³, though the national average was estimated to be 0.3 Bq/m³ [W14]. Subsequently, more than 400,000 measurements were made throughout the United Kingdom aimed at identifying dwellings with elevated radon levels; this information is available for England and Wales [G10], Scotland [G11] and Northern Ireland [G12].

29. A nationwide indoor radon survey carried out since 1982 in France has updated estimates of the population exposure [B38]. Indoor radon measurements were performed using passive dosimeters left in place for 2 months in the main room. A questionnaire was completed regarding housing characteristics. In total, the survey included 12,261 radon measurements distributed over the whole country. Corrections for seasonal variations [B36] and housing characteristics were applied. The crude average of indoor radon concentrations was 89 Bq/m³, and the average corrected for season and housing characteristics was 83 Bq/m³ (the range over French districts was 19 to 297 Bq/m³). Weighting by district population density yielded a national average of about 63 Bq/m³.

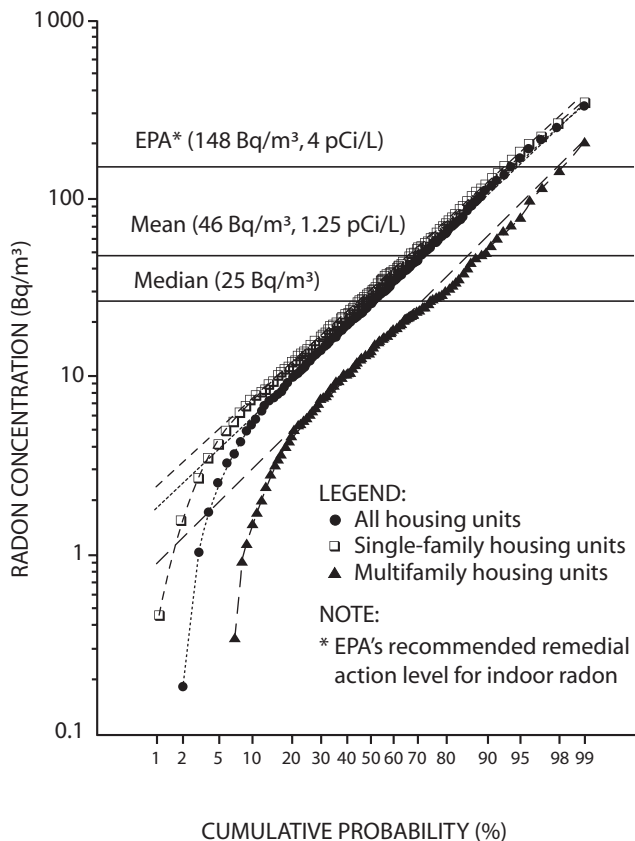
30. Bochicchio et al. [B40] report a national radon survey of some 5,631 dwellings distributed in 232 towns across Italy. The authors report a national population-weighted average of 70 Bq/m³ and a geometric mean and geometric standard deviation of 52 Bq/m³ and 2.1, respectively. The authors also report seasonal differences. On a national scale, over the 21 regions studied, the authors report a winter radon to summer radon ratio with a geometric mean and standard deviation of 1.23 and 1.71, respectively.

31. European Union (EU) efforts to develop a radon map of European countries are under way as part of an EU project to develop a European atlas of natural radioactivity [D20].

32. A major survey of radon concentrations in United States homes measured radon levels in homes in 125 counties in 50 states with the results shown in figure I (after reference [M1]). Approximately 6.1% of the homes surveyed exceeded the United States Environmental Protection Agency (EPA) action level of 148 Bq/m³ (4 pCi/L). This study indicated that the distribution of radon concentrations in homes could be reasonably described by a log-normal distribution. The overall geometric mean (median) of the radon concentration data was 25 Bq/m³ with a geometric standard deviation of about 3.1.

33. Information on radon levels in Latin American countries is also becoming available (e.g. [C41, M41, S65]). According to reference [C41], most indoor radon levels in Latin America are below 100 Bq/m³. However, levels can be quite variable in different regions within a country, as evidenced by data for 17 states in Mexico, which report mean radon levels across the 17 states surveyed ranging from less than 40 Bq/m³ to near 200 Bq/m³. The overall mean value reported across the 17 states surveyed was 111.6 Bq/m³.

Figure I. Distribution of indoor radon levels in homes in the United States (after reference [M1]).



34. A study of indoor and outdoor radon levels in Brazil reported that a variability in radon levels of about 50% in a single day could be measured on an hourly basis, with the highest values in the morning and the lowest values in the afternoon. The authors also reported a seasonal variability of two orders of magnitude in outdoor levels, with the highest levels in the dry winter season and the lowest levels in the wet summer months [M41]. The mean indoor radon concentration within the urban area of Rio de Janeiro is 40 Bq/m³, while the indoor radon concentrations within urban and rural areas in Poços de Caldas are 61 and 204 Bq/m³, respectively [M41].

35. Several Japanese studies, including two nationwide indoor radon surveys, showed significant differences [F14, F15, S64]. The first survey of more than 7,000 dwellings, using passive radon detection, reported a mean radon concentration of 20.8 Bq/m³ [F14]. To investigate potential confounding by thoron, a survey of 900 dwellings used a detector that could discriminate between radon and thoron. The mean radon concentration from the second survey was 15.5 Bq/m³ [T12]. The authors noted a gradient in thoron concentration in the homes, the concentration decreasing by nearly a factor of 2 with increasing distance, up to about 1.5 m, from walls made of soil. Radon concentrations are reported as generally homogeneous in rooms, except for places near the inlet and outlet of indoor airflow [Z8]. This result should be considered when choosing locations for detection.

36. In the 1980s and 1990s, two nationwide surveys [R12, W21] of indoor radon decay products were performed in China using various grab sampling measurements (e.g. scintillation cell method and two filter methods) or short-term measurements (2–4 days of activated charcoal adsorption). The arithmetic mean (AM) of the indoor radon concentration was 24 Bq/m³ and the geometric mean (GM) was 21 Bq/m³ [U2]. Since 1996, China's housing situation has greatly changed. A new survey of indoor radon in 26 cities and regions during 2001 and 2005 used alpha track detection (exposure period of 3–6 months) [S67]. The AM of indoor radon concentration was 43.8 ± 37.7 Bq/m³ and the GM was 34.4 ± 1.95 Bq/m³, with the median of the concentration at 32.9 Bq/m³. These results are significantly higher than those of previous surveys. The radon concentration in some high-rise buildings exceeds the national action level of 400 Bq/m³.

37. Traditional soil-brick and mud-wall structure houses are still popular in the countryside of China. At least 100 million people are currently living in such houses. Surveys of thoron and its decay products in Chinese traditional soil structure houses have been conducted. Fan et al. observed a thoron EEC of 4.0 Bq/m³ ($n = 56$) in 1991 in Shaanxi soil houses [F20]. More recently a survey using improved alpha track monitors was completed in Guangdong, Gansu, Yunnan, Xinjiang, and Guizhou provinces: thoron and its decay product concentrations (AM) were 318 Bq/m³ and 3.8 Bq/m³ ($n = 148$, maximum value 15.8 Bq/m³), respectively [S68]. Applying a dose conversion factor of 40 nSv/(Bq h m⁻³), the average annual effective dose from inhalation of ²²⁰Rn was estimated to be in the range 1.1–4.4 mSv. The dose contribution from thoron is obviously significant in this situation.

38. Surveys of radon and thoron decay products were carried out in Fujian province in China, where the natural levels of ²³⁸U and ²³²Th in soil are elevated (about 53.4 Bq/kg and 116.8 Bq/kg, respectively) [Z5]. Radon and thoron decay products were measured in homes and various other locations, including outdoors, in mineral processing plants, underground and in railway tunnels. The average radon and thoron decay product concentrations (expressed as EEC) measured in 204 dwellings were 12.9 Bq/m³ and 0.87 Bq/m³, respectively. The mean outdoor level of radon decay product concentrations (expressed as EEC) derived from measurements made at 180 locations was 9.69 Bq/m³. The authors also assessed potential exposure of people living in Fujian province, and concluded that thoron (and decay products) contributed about 20% of the estimated effective dose of 1.28 mSv/a from radon and thoron combined.

39. Measurements of outdoor (1,100 samples) and indoor (1,050 samples) levels of radon and thoron decay products were made at a university campus in northeastern Japan over a period of 4 years [K20]. The authors analysed the data using a variety of statistical methods. A seasonal variation was observed, with higher radon concentrations for autumn and winter, both outdoors and indoors. A seasonal variation of thoron levels was not as clear.

40. Shang et al. [S67] report the results of a countrywide survey of 3,098 homes in China carried out with alpha track detectors between 2001 and 2005 by the Chinese National Institute for Radiological Protection. Measurement sites were statistically distributed in the municipalities (Beijing, Shanghai and Tianjin) and 15 provinces, using a sampling rate of 0.09 in 10,000 houses across the country. The authors report an arithmetic annual average radon concentration of 43.8 ± 37.7 Bq/m³ and a geometric mean of 34.4 ± 2.0 Bq/m³, and note that about 6.4% of the measured houses had levels of above 100 Bq/m³, although only 0.7% were above 200 Bq/m³. The authors also report that in dwellings made of soil or mud, exposure from thoron may be significant, and that a study of thoron has been designed.

41. Additional data on indoor levels of thoron in Europe and Asia are also reported in reference [S41], which notes that the doses from thoron and its short-lived decay products can be comparable to, or even larger than, the dose from radon and its short-lived decay products. Others also reported data on thoron and thoron decay products for some areas of East Asia (e.g. [T21, Z5]).

42. It was not practical to calculate lung dose directly from thoron gas measurements because the equilibrium factor (F_{eq}) between the gas and decay products was not well established. Past dose estimates for thoron were made mainly from filtered air measurements of the thoron decay product ²¹²Pb. However, much work on methods of measuring radon and thoron and their decay products was carried out in Japan (e.g. [T12, T13, T14, T15, T16, T17, T18, T19, T20, T34, T41, Z3]). A Japanese study [I15] observed that it was important to consider the influence of thoron on the measurement of RDPs. A great deal of work was also carried out in Japan concerning the measurement of radon and thoron and their decay products, including passive and continuous systems, and the measurement of various factors (e.g. F_{eq} and the fraction f_p of decay products attached to particulates) which are important in assessing lung dose [K28, T42, T43, T44, T45, T46, T47, T48]. Shang et al. also investigated the effect of thoron on the measurement of RDPs in Chinese cave dwellings [S69]. Since thoron will be present in many homes, if a detector is used that responds to thoron and radon without distinguishing between them, the contribution from thoron will be attributed to radon. For example, in testing the sensitivity of one thoron monitor, the average thoron level in 20 homes in southern India was 168 Bq/m³ [Z3]. More detailed information on the average thoron levels in these homes is provided in reference [T33].

43. Evaluation of exposure to radon and thoron and their decay products thus must take account of the actual activity concentrations of the various alpha-emitting radionuclides from the two series in the air that is inhaled. As noted previously, the total alpha particle energy yet to be released by the decay of inhaled radon or thoron is reported in terms of potential alpha energy concentration (PAEC), with units of either J/m³ or WL (working level). This quantity can be calculated once the activities of the individual radionuclides are

determined. In most cases, the individual activities are not directly measured. Thus the exposure rate must be indirectly determined using assumptions on concentration ratios, i.e. equilibrium factors, which lead to the determination of the EEC. The environmental factors that influence concentration ratios in each of the radioactive series are of great significance for assessments of both exposure and dose [M33, U2].

44. Many measurements of RDPs have been reported. These suggest that a rounded value for the equilibrium factor of 0.6 may be appropriate for the outdoor environment [U2]. Ramachandran and Subba Ramu [R6] reported variations in indoor equilibrium factors. The UNSCEAR 2000 Report also noted that there is a wide range of values from individual measurements. This is understandable given the many environmental factors, including exhalation rates and atmospheric stability conditions, that influence the various activity ratios. The range of the equilibrium factor for outdoor radon is from 0.2 to 1.0, indicating a high degree of uncertainty in the application of a typical value to derive EECs [U2].

45. Measurements of both thoron and radon gas using passive alpha track detectors have been reported (e.g. [B16, D11, G13, I8, I9, L37]). Measurements were obtained over a 2-year period for both thoron gas and its ²¹²Pb decay product in four locations — three indoors and one suburban outdoor location [C22, H20, H21]. Tokonami et al. [T48] provide information on the contribution from thoron for several radon detectors. The authors also provide a comparison of small indoor surveys in the Gunma prefecture in Japan and in Kovagoszlos, Hungary. The average radon/thoron ratio from the Japanese survey was 1.3, compared with a mean of 4.5 in the Hungarian survey. The authors concluded that measurements without discrimination of radon isotopes have the potential to affect risk estimates. Sugino, Tokonami and Zhuo [S70] report radon and thoron concentrations in offices and dwellings of the Gunma prefecture. The average radon concentrations in offices were about 29 Bq/m³, higher than the 17 Bq/m³ reported for dwellings. As for reference [T48], the mean ratio of thoron and radon was estimated as approximately 1.3.

46. The UNSCEAR 2000 Report [U2] assumed average equilibrium factors obtained from ²¹²Pb/²²⁰Rn ratios of 0.003 outdoors and 0.02 indoors to derive estimates of a dose conversion factor for thoron EEC and recommended a value of 40 nSv/(Bq h m⁻³) for dose estimation.

47. Determinations of the equilibrium factor for radon indoors generally confirm the typical value of 0.4 previously assessed by the Committee [U5, U6]. While indoor measurements show a range from 0.1 to 0.9, most are within 30% of the typical value of 0.4 [H22, R6]. A study [H22] in seven North American houses showed that the equilibrium factor varies significantly with time, typically by a few tens of per cent. Measurements were carried out over a 4-year period to gain an understanding of the characteristics of radon and its decay products in air-conditioned office buildings in Tokyo, Japan. The equilibrium factor was evaluated during working

hours and over the whole day in this survey; there was little difference between the two conditions. These values were within 30% of the assumed typical value of 0.4 [T34]. Although the measurement of radon gas concentration may be a surrogate for direct measurement of the decay product concentration in the determination of exposure, EECs or PAECs estimated using this assumed typical value may be in error, frequently by several tens of per cent, though rarely by as much as a factor of 2.

48. More caution should be exercised in assuming the average values of the equilibrium factor for dose assessment from inhalation of thoron decay products. An objection to the use of thoron gas measurements for dosimetric purposes is that thoron may not be well mixed in the indoor air because of its short half-life. As indicated previously, some data indicate that indoor thoron concentrations vary with the distance from walls and floors [Z8]. In many samples, the thoron concentrations in the centre of the room or more than 1 m from the surface of building material containing ^{224}Ra were as low as in outdoor air, while the thoron concentration near the surface of the building material was more than 10 times that in the centre of the room. Only where a room fan is used would thoron be well mixed and a large variation of the thoron concentration in the room not be found [M40].

49. Equilibrium factors for estimating ^{220}Rn EEC are given above as single values. Because of the large spatial variations in thoron concentrations in a room, these single values should be regarded as being subject to large uncertainties. Thus the use of an equilibrium factor for thoron should be limited to situations where large spatial variation is not found. On the basis of the results of simultaneous measurements of thoron concentration and thoron EEC by long-term passive methods, Yonehara et al. were unable to find a good correlation between thoron concentration and the EEC [Y9]. However, Yamada et al. were able to find good agreement in a study of 265 cave dwellings [Y10].

50. Although only a limited number of measurements of thoron in indoor air are available, several investigations reported both radon and thoron EECs. While acknowledging the uncertainty noted earlier, this allows some generalizations to be made from the derived ratios of the radon and thoron EECs. On the basis of the physical characteristics of radon and thoron, model entry rates to buildings and a ventilation rate of 0.7 h^{-1} , the International Commission on Radiological Protection (ICRP) estimated expected concentrations in buildings [I5], which in terms of EEC are 2–50 Bq/m^3 for radon and 0.04–2 Bq/m^3 (mean = 0.5 Bq/m^3) for thoron. This corresponds to a thoron/radon EEC ratio of 0.03 [U2].

51. Table 1 provides a summary of concentrations of radon in indoor air determined from surveys. Many of the data in table 1 are from annex B of the UNSCEAR 2000 Report [U2]. Data carried forward from reference [U2] are indicated as such. Original sources of data in [U2] are from UNSCEAR surveys of Natural Radiation Exposure and literature as cited in [U2]. Since the publication of [U2], the Committee has carried out three surveys of the natural radiation environment, in 2001, 2004 and 2006. Summary results from these surveys are provided in table 1. Not surprisingly, many of the indoor radon data were collected for areas or regions where indoor radon levels were thought to be elevated. Thus, where the survey results provided to the Committee make such a judgement possible, a note is given in the last column of table 1 indicating whether the data provided in the table may be considered as some form of national average or whether they are more indicative of a regional or local area of elevated levels. In some instances the national authorities provided this judgement, while in others the judgements were made by the Committee. In all instances, precedence was given to data provided to the Committee, as opposed to literature values. The data in table 1 show considerable variability both within countries and from country to country, with, for example, reported nominal geometric mean indoor levels ranging from $<10\text{ Bq/m}^3$ in Egypt and Cuba, to more than 100 Bq/m^3 in a number of European countries, and to above 600 Bq/m^3 in parts of the Islamic Republic of Iran.

52. The UNSCEAR 2000 Report [U2] gives worldwide arithmetic mean values of 46 Bq/m^3 (unweighted) and 39 Bq/m^3 (population-weighted). Worldwide geometric mean values of 37 Bq/m^3 (unweighted) and 30 Bq/m^3 (population-weighted) with corresponding geometric standard deviation of 2.2 (unweighted) and 2.3 (population weighted) are also presented in [U2]. Given the wide variety and disparity of data currently available, no attempt was made to update the nominal values from [U2], and the values provided in [U2] are retained for the purposes of this report.

53. The UNSCEAR 2000 Report [U2] gives an annual per caput dose estimated at 1.15 mSv from exposure to natural sources of radon. This value is still appropriate. The UNSCEAR 2000 Report also gives an annual dose of 0.1 mSv from natural sources of thoron [U2]. While this value is still reasonable, data collected for the present study indicate that the levels of thoron (and hence doses from exposure to thoron and its decay products) are highly variable and that thoron may provide a larger contribution to natural background dose than previously thought. Doses from radon and thoron represent approximately half of the estimated dose from exposure to all natural sources of ionizing radiation.

Table 1 Concentrations of radon in indoor air

Region/country	Population (10 ⁶)	Indoor radon (²²² Rn) (Bq/m ³)				Notes
		Arithmetic mean	Geometric mean	Maximum value	Geometric standard deviation	
Africa						
Algeria [U2]	28.78	30		140		–
Egypt [U2]	63.27	9		24		–
Ghana [U2]	17.83			340		–
North America						
Canada [U2]	29.68	34	14	1 720	3.6	
Canada [L47]	32.27	28.35	11.2	1 720	3.9	
Mexico	107.03	140	90	1 193		[M39]
United States [U2]	269.4	46	25		3.1	National survey [M1, U18]
South America						
Argentina	38.75	35	25	211	2	Countrywide average ^e
Brazil	186.40	81.95		310.0		[C41]
Chile [U2]	14.42	25		86		–
Cuba	11.20	7.7	5.2	15.3	3.3	Countrywide average ^f
Ecuador		200				[Z11]
Paraguay [U2]	4.96	28		51		–
Peru	27.97	32.29		50.20		[C41]
Venezuela	26.75	52.50		346		[C41]
East Asia						
China	1315.84	43.8	34.4	596		National average based on sampling 3098 dwellings [S67]
China [U2]	1 232	24	20	380	2.2	–
Hong Kong SAR [U2]	6.19	41		140		–
Taiwan	22.89	10.0	8.5	63.5	0.6	Countrywide average ^f
India [U2]	944.6	57	42	210	2.2	–
Indonesia	213.67	35.1	35.1	165	1.2	Countrywide average ^e
Japan [U2]	125.4	16	13	310	1.8	–
Kazakhstan	14.83			5 000		Countrywide average ^e
Republic of Korea	48.85	53.4	43.3	1 350	1.8	Countrywide average ^e
Malaysia [U2]	20.58	14		20		–
Pakistan [U2]	140.0	30		83		–
Philippines	75.90	23	22	62	1.13	Countrywide average ^e
Philippines	76.57	23	23	62	±6	Countrywide average ^g
Russian Federation		50–60				[Z11]
Thailand [U2]	58.7	23	16	480	1.2	–

Region/country	Population (10 ⁶)	Indoor radon (²²² Rn) (Bq/m ³)				Notes
		Arithmetic mean	Geometric mean	Maximum value	Geometric standard deviation	
West Asia						
Armenia [U2]	3.64	104		216	1.3	
Islamic Republic of Iran	63.76	82		3 070		Countrywide average
Islamic Republic of Iran	795	2 745		31 000		High-background areas
Islamic Republic of Iran		600		1 000		High-background areas
Kuwait	1.69 [U2]	14 [U2]	10.6	119.2	0.74	Countrywide average
Palestine (Gaza) [Y7]	0.95	34		105		–
Saudi Arabia [A25]		16		36		–
Syrian Arab Republic [U2]	14.57	44		520		
North Europe						
Denmark	5.2	59 ^a	39 ^a	1 200	2.2	Countrywide average
Estonia [U2]	1.47	120	92	1 390		
Finland	5.2	120	84	20 000 ^d	2.1	Countrywide average
Iceland	0.3	10		26		Countrywide average ^g
Lithuania	3.73	49	38	1 900		Countrywide average
Lithuania	3.49	55	36.5	636		Countrywide average
Norway [U2]	4.35	73	40	50 000		–
Sweden	8.88	108	56	84 000		Countrywide average
West Europe						
Austria [U2]	8.11		15	190		–
Belgium	10.22	48	38	12 000	2	Countrywide average
France [U2]	58.33	62	41	4 690	2.7	–
		89.3	53.5	4 964		Population-weighted mean of 63 Bq/m ³ based on 12,261 measurements in dwellings
Germany [U2]	81.92	50	40	>10 000	1.9	Countrywide average
Ireland	3.84	89	57	7 000	2.4	Countrywide average
Liechtenstein	0.03	80		1 098		^e
Luxembourg	0.22	110	70	2 500	2.0	Countrywide average
Netherlands [U2]	15.58	23	18	380	1.6	–
Switzerland	6.71	75	41	10 000		Countrywide average
Switzerland		142 ^b 73 ^c	81 ^b 59 ^c	15 000 ^b 15 000 ^c	2.6 ^b 1.8 ^c	Countrywide average
United Kingdom [U2]	58.14	20	14	17 000	3.2	[C26]
England [G10]		90	50			Average (20 Bq/m ³) population- weighted average
Wales [G10]		84	48			

Region/country	Population (10 ⁶)	Indoor radon (²²² Rn) (Bq/m ³)				Notes
		Arithmetic mean	Geometric mean	Maximum value	Geometric standard deviation	
East Europe						
Belarus		31.8		221		Countrywide average
Bulgaria	8.10		22	250	2.1	Countrywide average
Czech Republic		118	94.4	70 000	1.84	Countrywide average
		442		20 000		High-background area
		214		20 000		High-background area
		124		70 000		High-background area
		112		20 000		High-background area
		136		6 000		High-background area
		214		6 500		High-background area
Hungary [U2]	10.05	107	82	1 990	2.7	–
Poland	38.12	49.1		1 300		Countrywide average ^e
Poland	38.17	49	31	3 260	2.3	Countrywide average ^f
Romania	22.55	25.0		564		Countrywide average ^e
Slovakia [U2]	5.35	87		3 750		–
South Europe						
Albania [U2]	3.40	120	105	270	2.0	–
Croatia [U2]	4.50	35	32	92		–
Cyprus [U2]	0.76	7	7	78	2.6	–
Greece [U2]	10.49	73	52	490		–
Greece		55	44	1 700	2.4	Countrywide average ^g
Italy [U2]	57.23	75	57	1 040	2.0	–
Italy	57.3	70	52	1 036	2.1	Countrywide average ^g
Montenegro (Yugoslavia)	0.60	184	110	1 128	2.74	Countrywide average ^e
Portugal [U2]	9.81	62	45	2 700	2.2	–
Slovenia [U2]	1.92	87	60	1 330	2.2	–
Spain	36.72	90.38	45.69	15 400		Countrywide average ^e
Spain	0.001	748.5	242.64	15 400		High-background areas ^e
Spain	40.84	90.4	45.7	15 400	2.9	Countrywide average ^g
Spain	0.02	610.0		1 400.0		High-background areas ^g
Oceania						
Australia [U2]	18.06	11	8	420	2.1	–
New Zealand	3.81	21.5	19.5	80		Countrywide average ^e

^a Population-weighted average.^b Upper value, unweighted data.^c Lower value, data weighted for floor dependence, population distribution, error of exposition and mobility.^d Annual mean.^e Data from *UNSCEAR Global Survey on Exposures to Natural Radiation Sources (2001-2006)*, submitted in 2001.^f *Ibid.*, submitted in 2004.^g *Ibid.*, submitted in 2006.

C. Workplaces

54. The spectrum of workplaces other than mines where radon can present a hazard is large. While it includes below-ground workplaces such as subways, tunnels, underground parking, stores, caves, spas and closed-out mines open to visitors, the majority of such workplaces, such as factories, shops, schools and offices, will be above ground.

55. Other workplaces where large quantities of materials with elevated radium concentrations are stored or processed, for example phosphate fertilizer production [R11] or monazite sand mining, can similarly exhibit elevated radon and thoron levels [A30, C42]. One study [S71] reporting on radon and thoron concentrations in the monazite production area of a rare earth facility in Kerala, India, found radon levels to be below the detection level of about 1.7 Bq/m³ (see reference [S72] for discussion of monitoring techniques), but the corresponding thoron levels were 5.9 kBq/m³.

56. Underground workplaces, including mines other than uranium mines, especially coal mines (e.g. [B17, J8, S43, U16, V5]), can accumulate high radon levels in the same way as natural caves or abandoned mines. High radon levels in underground workplaces will not be limited to only those areas where elevated levels were found in above-ground workplaces. The experience of the Newfoundland fluorspar miners illustrates this. The levels of ²²⁶Ra in the host rock were low, but the miners were exposed to elevated levels of radon and short-lived decay products arising from radon in the groundwater, which entered the mine and subsequently the mine atmosphere (e.g. [A14, M16, M17]).

57. Exposure to environmental sources of radon from mining and mineral processing is common, and the inhalation of RDPs can be a significant exposure pathway. For

workers involved in the nuclear fuel cycle, radon exposure from mining and milling is a relatively important contributor to the per caput dose.

58. The total number of workers exposed to human-made sources and enhanced natural sources was given in the UNSCEAR 2000 Report [U2] as 11.1 million. Approximately 4.6 million workers were exposed to human-made sources at an annual average (effective) dose of about 0.6 mSv. Some 6.5 million workers were exposed to enhanced natural sources at an annual average dose of about 1.8 mSv, of which approximately half was from radon. The estimate for radon in above-ground workplaces (for example in the phosphate industry) is still considered to be crude.

59. In the case of occupational exposure, there are several situations where workers who have the highest radiation doses receive a significant contribution from radon. These include the situations listed in table 2 (from reference [U2]). For workers involved in nuclear power production, those involved in the mining of uranium typically receive the highest collective doses; a significant part of that exposure is from radon inhalation. The group of workers in the category of above-ground workplaces (see table 2) are the second largest group identified in annex B of the UNSCEAR 2000 Report [U2]. These workers were estimated to receive an average annual effective dose of 4.8 mSv. This is the largest average annual dose received by any type of worker and was due entirely to radon. According to the UNSCEAR 2000 Report [U2], radon inhalation is also a significant contributor to the doses to the other categories of workers in table 2. While information to fully update table 2 is not available, a paper by Liu et al. [L46] indicates that there are about 6 million miners in Chinese coal mines alone, which nearly doubles the number of coal miners reported in reference [U2].

Table 2 Situations where doses from radon are significant [U2]

<i>Source/practice</i>	<i>Number of monitored workers</i>	<i>Average annual effective dose (mSv)</i>
Nuclear fuel cycle (including uranium mining)	800 000	1.8
Mining (other than coal and excluding uranium mining)	760 000	2.7
Coal mining ^a	3 910 000	0.7
Mineral processing	300 000	1.0
Above-ground workplaces (radon)	1 250 000	4.8

^a See paragraph 59.

D. Measurements of radon and radon decay products

60. It is well established that the inhalation of the short-lived decay products of radon (²²²Rn) and their subsequent deposition along the walls of the various airways of the bronchial tree are the main pathways of radiation exposure of the lungs [U2]. As discussed elsewhere in this report, the lung

exposure arising from the decay products of thoron (²²⁰Rn) is also of increasing interest. Traditionally, the potential alpha decay energy per litre of air (referred to in units of working level month (WLM)) was the measure of exposure to RDPs used in evaluations of exposures in mines. Miner epidemiological studies use data based on measurements of this type. Later, time-integrated radon measurement techniques were

developed and are the method of choice for modern studies of residential radon. The discussions of each miner cohort (section IV) or residential case-control (section V) epidemiological study include a brief description of the methods used for measuring radon and its decay products; in addition, a few general comments are provided below. Further information is available in published reports (e.g. NCRP Report No. 97 [N15]).

61. Various techniques were used in the past to assess residential radon exposure, including: instantaneous grab samples and subsequent analysis in a scintillation cell; accumulation on a charcoal absorber with subsequent gamma spectroscopy; various solid state detectors; and a variety of other techniques, including continuous measurements [I12, N15]. Overall, long-term track etch radon measurements are widespread and are almost universally used for residential epidemiological studies. In recent years, increasing attention was given to measurements that assist in the reconstruction of past exposures, including the measurement of polonium (^{210}Po) activity on glass surfaces or in volume traps (e.g. [F1, F9, L31, M33, N11, N15, P15]). Bochicchio et al. [B41] describe a comprehensive quality assurance programme for radon measurements carried out as part of a residential radon case-control study.

62. A paper on the use of (outdoor) radon levels in meteorology provides additional information on environmental radon monitoring and experimentally available lower limits of detection [Z7]. Tokonami et al. [T42] conducted an inter-comparison of measurement methods for radon, RDPs and particle size using a radon/aerosol chamber. The authors report good agreement (typically within $\pm 5\%$) across institutions and methods for continuous radon monitoring. However, the ratios of the radon concentrations measured by various institutions to those measured by alpha track detectors ranged from about 0.71 to 1.34. The authors noted that these types of detector (alpha track detectors) are often used in surveys and that quality control is always needed.

63. Much of the miner epidemiology is based on exposures characterized by relatively short-term (grab) measurements

of WL in one or more areas of a mine (combined with an evaluation of the hours worked in the same location). According to reference [L13], the first radon gas measurements in United States mines were in 1949, and the first RDP measurements were in 1951. In 1973, the American National Standards Institute (ANSI) published a consensus standard for radiation protection in uranium mines in the United States [A22]. According to this standard, a monitoring system for RDPs must be capable of measuring the annual accumulated exposure in WLM within an uncertainty interval of 50% at the 95% confidence level. To satisfy the ANSI criterion, uranium mining companies in the United States adopted a procedure based on periodic measurements of the air concentration of RDPs. All of the measurement methods in routine use at the time involved drawing a known volume of air through a filter. After a specified time delay, the activity on the filter was analysed. The concentration of short-lived RDPs, in units of working levels (WL), was calculated from the measured activity on the filter. The most common method was that of Kusnetz [K23], who devised a procedure for estimating WL from a single alpha count. (The Kusnetz method generally involves a 5-minute sample of air drawn through a glass fibre filter paper at a rate of 1–2 L/min; the filter paper is counted for total alpha activity after a delay of 40–90 min and the concentration in WL is determined from the total alpha count using a correction factor based on the decay time [K23].) For control purposes, a single measurement was used to establish whether or not a work area was safe for occupancy by miners. For routine dosimetry, the concentration estimated from the grab sample was assigned to the appropriate work area. As discussed later in section IV, similar practices were followed in many uranium mines outside the United States.

64. Twelve grab sampling methods for measuring airborne radon decay product concentrations were analysed by Schiager et al. [S56] to determine whether they would satisfy the ANSI standard [A22]. The evaluations considered six independent sources of uncertainty that together, according to reference [S56], comprised the overall uncertainty of the method. The results of their evaluation are summarized in table 3.

Table 3 Uncertainties in grab sampling (adapted from reference [S56])

<i>Nature or source of uncertainty</i>	<i>Total uncertainty (Percentage of measured value)</i>
Variations in airborne RDP concentrations	36
Inherent errors of the method	4–20
Precision based on counting statistics (dependent on concentration)	1.4–9.3 ^a
Human error	1–4
Estimation of occupancy time	4
Record-keeping and data transcription	1.5
Overall uncertainty	37–41

^a Range for 10 out of 12 methods analysed at 0.3 WL.

65. For comparing the uncertainties involved in different grab sampling methods, Schiager et al. [S56] assumed that all of the sources of uncertainty were independent, multiplicative and normally distributed. The total relative uncertainty in an individual measurement was estimated, by conventional propagation of error techniques, as the square root of the sum of the squares of the individual relative uncertainties. The result was an approximation, since the shape of the distribution of errors was not determined. In spite of the limitations of the analysis, Schiager et al. [S56] determined that the estimated uncertainties are entirely adequate to indicate the nature and magnitude of the various sources of potential errors and the adequacy of the sampling methods used for meeting the ANSI criterion.

66. Makepeace and Stocker [M35] presented a statistical analysis of WL measurements in Canadian uranium mines. No attempt was made to establish the accuracy or precision of the individual measurements. A total of 2,427 observations were obtained at 33 mine locations. The number of

observations per location varied from 22 to 188, and the sampling periods ranged from 4 to 12 days. The combined data at each location had a coefficient of variation ranging from 5 to 95%, with an arithmetic mean value of 30%, consistent with the ANSI standard. Francis et al. [F18] evaluated the effects of autocorrelation on workers' daily exposures and concluded that sampling programmes that rely on measurement on consecutive days (a possibility in the uranium mine environment) can result in biased estimates of exposure if autocorrelation is present. They suggested a random sampling strategy be employed where day-to-day correlation is high.

67. The possible role of exposure to thoron and its decay products is of increasing interest, and a number of authors report that the contribution of thoron and its decay products can be a significant component of the total exposure (radon plus thoron); thoron can thus be a source of error in residential radon studies that do not distinguish the two contributions to exposure (e.g. [C21, T11, T17]). Future measurement studies should therefore consider the contribution of both radon and thoron.

II. DOSIMETRY

68. The health risk associated with radon arises from the inhalation of the short-lived decay products and the consequent dose to critical cells of the respiratory tract. Estimates of the absorbed dose to the critical cells of the respiratory tract per unit radon exposure can be derived from an analysis of information on aerosol size distribution, unattached fraction, breathing rate, fractional deposition in the airways, mucous clearance rate and location of the target cells in the airways. Such estimates are model-dependent and necessarily subject to all the uncertainties associated with the input data as well as with the assumptions built into the particular model. The dose calculation procedure and assumptions are described in the UNSCEAR 2000 Report [U2]. The magnitude of the risk from exposure to RDPs was quantified in epidemiological studies of the increased rate of lung cancer among uranium mine workers and more recently in residential case-control studies.

69. ICRP Publication 65 [I2] recommended the use of the risk factors determined from epidemiological studies of uranium miners as the preferred method for converting RDP exposure to effective dose. Since that time, as discussed in section VI, numerous case-control studies of residential exposure to radon suggest risk factors that are generally consistent with those from miner epidemiology. These case-control studies provide direct evidence of risk from residential radon, removing the dependence on dosimetric adjustments from conditions in mines to conditions in homes. Thus, as discussed in section V, the assessment of risks from exposure to radon in the home can now be based on the evidence from residential case-control studies.

70. The ICRP has provided guidance on a dose conversion convention for radiation protection purposes [I2]. For radon, the ICRP recommends the RDP conversion convention that all exposures are combined on a dose- and risk-equivalent basis. The use of a single value for the dose conversion factor implicitly assumes: (a) that the distribution of RDP particle size for occupational exposure, in particular the fraction and particle size of the ultrafine mode, is not too different from the particle size distribution in uranium mines; and (b) for exposures of the general public, that the differences in aerosol conditions are offset by lower breathing rates for members of the public, particularly children. The current ICRP conversion convention is not applicable to thoron decay products.

A. Dosimetric models

71. Dosimetric models can be used to estimate the radiation doses arising from the inhalation of airborne

radioactive material. Such models incorporate data on respiratory tract deposition, including data on deposition of ultrafine particles in nasal passages (e.g. [H22, I3, I13, J1, J2, N8, N10]). These models, widely used for the assessment of dose for most inhaled radionuclides, show that the dose per unit intake of RDPs is dependent upon the site of deposition within the respiratory tract. The site of deposition in turn is strongly dependent on the particle size of the airborne RDP, particularly for those particles of below 10 nm diameter (typically the “unattached fraction” of the RDP, i.e. the fraction not attached to ambient particulates). A weighted dose conversion factor for a particular exposure location can be derived from measurements of RDP size distribution combined with the particle-size-dependent dose conversion factors, calculated using one of these dosimetric models.

72. Various techniques are used to measure size distributions of RDPs associated with fine (5 nm) and ultrafine (0.5 nm) particles. These are extensively reviewed elsewhere (e.g. [N8]). Many of these methods rely on fractionation of the sub-micrometre radioactive particles by diffusion processes, using sets of wire screens, or by inertial and impaction processes, using cascade impactors. The latter systems are also capable of resolving particle sizes exceeding 1 μm . These systems have been used to measure RDP size distributions in homes, workplaces and uranium mines [G1, G2, R7, S44, S57, T23, T25]. A simpler technique involving the measurement of the unattached fraction is widely used [H23]; this method relies on the separation of the ultrafine and accumulation modes (particles in the size range 50–500 nm, which grow on inhalation, affecting the pattern of deposition in the lungs) using a diffusion sampler, usually a single wire screen. When used with the optimized configuration, a system with a set of wire screens can measure size distributions in both modes concurrently [F16, F17]. Tokonami et al. [T49] describe two methods of measuring the particle size distribution of RDPs and report data measured in a mine in Japan, as well as in a well-controlled radon chamber.

B. Dosimetry of radon and thoron

73. A number of researchers have used the ICRP dosimetry model to assess the doses from radon and thoron and their decay products (e.g. [B35, I7]). The ICRP advises against using the human respiratory tract model for risk estimation [I3].

74. Tokonami et al. [T36] report measurements of physical parameters related to dose assessment in an actual room where environmental conditions such as ventilation rate, air-conditioning and the operation of an air cleaner were varied. Using the data and the ICRP Publication 66 respiratory tract model, dose conversion factors were calculated under different situations and compared with each other. The paper investigated the wide variation of dose conversion factors with environmental conditions and concluded that the most sensitive parameter is the unattached fraction of decay products (f_p).

75. Other dosimetric models have also been described [H3, N10], and current models are relatively consistent in their dose estimates, particularly in showing a strong dependence of the radiation dose per unit intake on the size of the inhaled radioactive particles (aerosol in the range 0.8 nm to $>1 \mu\text{m}$).

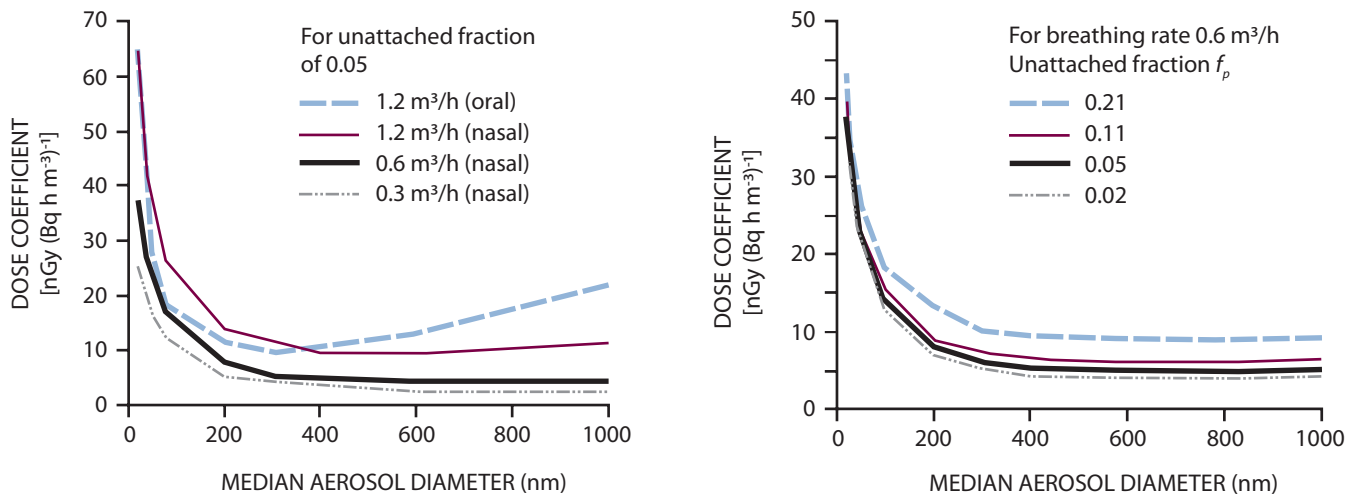
The publication of James [J5] contains an illustration of the dose conversion factors according to various models.

76. The UNSCEAR 2000 Report [U2] listed, in table 26 of annex B, the principal dosimetric assessments for the lung dose from deposited RDPs. For convenience, this table is reproduced here as table 4. Graphs of the dose coefficient as a function of median inhaled aerosol diameter, breathing rate and unattached fraction are reproduced from reference [U2] in figure II. The current RDP dosimetric models use the standard values for tissue and radiation weighting factors to convert tissue dose to effective dose. The effective dose estimates vary, but are within a factor of 3 higher than the estimates derived from the epidemiological approach used by the ICRP conversion convention. Considering the uncertainties in both the epidemiological and dosimetric approaches, this agreement is remarkable.

Table 4 Principal dosimetric assessments of lung dose from deposited RDPs [U2]

Year	Investigator	Parameter values		Target region	Model type	Dose factor ^a [nGy(Bq h m ⁻³) ⁻¹]
		Unattached fraction	Breathing rate (m ³ /h)			
1956	Chamberlain and Dyson [C1]	0.09	1.2	Average in 45 μm epithelium	Cast of trachea and bronchi	11
1959	ICRP [I4]	0.1	1.2	Mean tracheobronchial region	Deposition retention assumptions	6.7
1964	Jacobi [J1]	0.25		Basal cells (30 μm)	Findeisen–Landahl 6-region anatomical model	24
1964	Altshuler et al. [A3]	0.085	0.9 basal	Cells (22 μm)	Findeisen–Landahl 6-region anatomical model	32
1967	Haque and Collinson [H3]	0.35	Basal	Cells (30 μm)	Weibel dichotomous airway model	71
1972	Harley and Pasternack [H5]	0.04	0.9 basal	Cells (22 μm)	Weibel dichotomous airway model	5.7
1980	Jacobi and Eisfeld [J2]	0.1	1.2	Mean epithelium	Weibel dichotomous airway model, correction for upper airway turbulent diffusion [M2]	8.9
1980	James et al. [J6]	0.1	1.2	Mean epithelium	Yeh–Schum anatomical model [Y2]	14
1982	Harley and Pasternack [H6]	0.07	1.1 basal	Cells (22 μm)	Same as Jacobi and Eisfeld [J2]	6.4
1982	Hofmann [H10]	0.2	0.9	Mean epithelium	Same as Jacobi and Eisfeld [J2]	11
1991	National Research Council [N10]	0.16	1.2 basal	Cells (35–50 μm)	Yeh–Schum anatomical model [Y2], correction for upper airway turbulent diffusion	21
1996	Harley et al. [H4]	0.1	1.2 basal	Cells (27 μm)	Nikiforov and Schlesinger [N12] anatomical model, airway deposition from empirical data from human airway casts	9
1998	Marsh and Birchall [M2]	0.08	0.8	Bronchial cells: basal (35–50 μm), secretory (10–40 μm) Bronchiolar cells: secretory (4–12 μm)	ICRP lung model [I1]	8.5 19 14

^a Per unit ²²²Rn concentration (EEC). WLM converted to Bq h m⁻³ using 0.27×10^{-3} WL (Bq/m³)⁻¹ and 170 h per working month.

Figure II. Absorbed dose in bronchial epithelial cells per unit exposure to radon decay products as a function of aerosol size [U2].

77. It is not possible to assess the radiation dose from inhalation of thoron decay products by epidemiological means, and therefore it must be estimated using dosimetric modelling. In annex A of the UNSCEAR 2000 Report [U2], a conversion factor for thoron decay products of 40 nSv (Bq h m^{-3}) was used. According to reference [U2], this value was intended to include the dose to organs other than the lungs due to the transfer of ^{212}Pb from the lungs. Table 5 provides a summary of the principal dosimetric

assessments of lung dose from deposited thoron decay products and supports the continued use of a conversion factor of 40 nSv (Bq h m^{-3})⁻¹. Marsh and Birchall [M42], in a comment on the review of thoron dosimetry issues [N16], report thoron dose conversion factors based on the latest ICRP biokinetic models and provide a range of dose conversion factors from 1.1 mSv/WLM to 3.8 mSv/WLM, which encompasses the full range of previously estimated thoron dose conversion factors.

Table 5 Principal dosimetric assessments of lung dose from deposited thoron decay products

Year	Investigator	Parameter values		Target region	Model type	Effective dose ^a [nSv(Bq h m^{-3}) ⁻¹]
		Unattached fraction ^{212}Pb	Breathing rate (m^3/h)			
1956	Chamberlain and Dyson [C1]	0.02	0.3	Average in 45 μm epithelium	Cast of trachea and bronchi	30
1959	ICRP [I4]	0.02	1.2	Mean tracheobronchial region	Deposition retention assumptions	43
1973	Harley and Pasternack [H31]	0.02	0.9	Basal cell generations 2–15	Weibel	43
1980	Jacobi and Eisfeld [J2]	n.a.	1.2	Basal cell generations 2–15	Weibel	35
				Bronchial from whole lung $\times 0.06$	Weibel	64
1981	ICRP 32 [I10]	n.a.	1.2	Bronchial from whole lung $\times 0.06$	Based on Jacobi and Eisfeld [J2]	73
1982 (1993)	James et al. [J16] (James [J5])	n.a.	1.2	Bronchial basal cell	Birchall–James model using either Weibel or Yeh–Schum [Y2] model	34–103
1983	NEA 1983 [N8]	0.02	1.2	Mean bronchial	Jacobi and Eisfeld [J2]	36
2000	UNSCEAR [U2]	n.a.	1.2	Whole body	ICRP 50 [I5]	40
2001	Porstendorfer [P12]	0.005–0.02	0.75	Bronchial	Modified from ICRP 66 [I3] weighting by basal and secretory cell density	53

^a Per unit ^{220}Rn concentration (EEC). WLM converted to Bq h m^{-3} using 3.6×10^{-3} WL (Bq/m^3)⁻¹ and 170 h per working month.

C. Dose conversion factors

78. The health risks from exposure to radon and thoron are principally due to the inhalation of the short-lived decay products and alpha particle irradiation of the bronchial airways. Radon and thoron decay product exposure rates are specified by the measure of potential alpha energy concentration (PAEC), given in units of J/m^3 or working levels (WL), and the equilibrium-equivalent concentration (EEC), given in Bq/m^3 . The potential alpha energy concentration is derived from a linear combination of the activities of the short-lived decay products in each radon decay series (see paragraph 122, annex B, UNSCEAR 2000 Report [U2]). The constants in the linear combination are the fractional contributions of each decay product to the total potential alpha energy. The EEC (in Bq/m^3) can be converted to the PAEC by the relationships:

$$1 \text{ Bq}/\text{m}^3 = 5.56 \times 10^{-9} \text{ J m}^{-3} = 2.7 \times 10^{-4} \text{ WL (}^{222}\text{Rn);}$$

and

$$1 \text{ Bq}/\text{m}^3 = 7.6 \times 10^{-8} \text{ J m}^{-3} = 3.64 \times 10^{-3} \text{ WL (}^{220}\text{Rn).}$$

79. For occupational exposure to inhaled ^{222}Rn decay products, the ICRP 65 [I2] recommended the use of a single factor (conversion convention) to relate the ^{222}Rn decay product exposure to the effective dose to an individual. This conversion convention is based on a comparison of the risk to a uranium miner, based on epidemiological studies, with the risk to a radiation worker from an effective dose of 1 Sv, in other words, comparison of the radiation detriment coefficient (risk per unit dose) with the miner detriment (risk per PAEC exposure). For worker exposure, this factor is $1,430 \text{ mSv (J h m}^{-3})^{-1}$ (rounded to 1,400), 5.06 mSv/WLM (rounded to 5 mSv/WLM), or $7.95 \text{ nSv (Bq h m}^{-3})^{-1}$ (rounded to $8 \text{ nSv (Bq h m}^{-3})^{-1}$) EEC (tables 7 and 8, ICRP Publication 65 [I2]).

80. In recommending that a similar approach be used for members of the public, ICRP Publication 65 [I2] assumed that the lung cancer risk per unit exposure in a home was the same as that in an underground uranium mine, in order to derive a conversion factor for members of the public. Since the detriment coefficient for the public is greater than for workers ($7.3\% \text{ Sv}^{-1}$ against $5.6\% \text{ Sv}^{-1}$), the derived conversion convention for members of the public was calculated to be $1,100 \text{ mSv (J h m}^{-3})^{-1}$, or 3.88 mSv/WLM (rounded to 4 mSv/WLM), or $6.1 \text{ nSv (Bq h m}^{-3})^{-1}$ EEC.

81. In developing ICRP Publication 65 [I2], data from epidemiological studies of seven miner cohorts were used to derive a central estimate of the excess relative risk (ERR) of exposure to RDPs. The ICRP (table A.2 of reference [I2]) estimated a mean ERR coefficient of $3.79 \text{ (J h m}^{-3})^{-1}$ ($1.34\% \text{ WLM}^{-1}$), by averaging the results from the seven selected cohorts, weighted according to person-years of risk for each cohort. Lowe and Chambers [L7] considered three additional epidemiological studies (the Port Radium uranium miners, the Chinese tin miners and the Newfoundland fluorspar miners, as discussed in section IV) that were not

included in ICRP's calculations, though cited in them. Their inclusion yielded a mean ERR coefficient of $3.08 \text{ (J h m}^{-3})^{-1}$ ($1.09\% \text{ WLM}^{-1}$), about 80% of the $3.79 \text{ (J h m}^{-3})^{-1}$ ($1.34\% \text{ WLM}^{-1}$) value estimated by the ICRP.

82. Alternative methods to ICRP's weighting by person-years can also be used to arrive at a mean risk estimate. One alternative considered by Lowe and Chambers [L7] was based on the confidence interval (CI) associated with each estimate: in this approach, the estimates with the least assigned uncertainty are given the most weight in estimating an overall mean. The method of combining values with different CIs was to weight each value by the inverse of its variance. It was assumed that the 95% CI about each estimate approximated to a range of ± 2 standard deviations about the estimate, and that the variance was the square of the standard deviation. This weighting method gave a mean ERR of $0.73 \text{ (J h m}^{-3})^{-1}$, or $0.26\% \text{ WLM}^{-1}$.

83. Stather [S62], in a discussion of the dosimetric and epidemiological approaches, arrives at a similar conclusion and suggests that the difference of about a factor of 3 is "surprisingly good". The Committee agrees with this view and simply notes that the calculated doses are in reasonable agreement with risk factors derived from epidemiology, uncertainties in both approaches considered.

D. Uncertainties in dose conversion factors

84. Uncertainties are present in both epidemiological (see sections IV and V) and dosimetric approaches, and the dosimetry of inhaled RDPs is quite complex and depends on many factors (biological, physical and behavioural). Many authors have reported estimates of doses, including references [A3, C1, H4, H5, H6, H10, J1, J2, J5, J6, N1, Y2, Y3, Y4, Y5], and both sensitivity (e.g. [M2]) and uncertainty analyses (e.g. [M32]) have been conducted. It may be that a single dose conversion factor cannot adequately cover the variety of natural and occupational exposure situations.

85. Marsh et al. [M32] described a detailed parameter uncertainty analysis for the weighted equivalent lung dose (absorbed dose averaged over the lung that is weighted for the relative biological effectiveness of alpha radiation) per unit exposure to RDPs in the home. The authors commented that the ICRP (para. 356 of reference [I3]) recommended that the risks from residential radon be based on epidemiological studies of miners, from which a conversion factor (effective dose per unit exposure to radon) of 5 mSv/WLM for workers was estimated. In contrast, Birchall and James [B18] calculated a conversion factor of 13.4 mSv/WLM for miners based on the ICRP's human respiratory tract model [I3], a factor of 2–3 times larger than that estimated from miner epidemiological studies. In carrying out the parameter uncertainty analysis, Marsh et al. [M32] assumed a dosimetric tissue weighting factor of 0.12 for the lung and an alpha radiation weighting factor of 20. Their analysis considered various aerosol parameters, target cell parameters,

and parameters such as breathing rate and fraction of breathing through the nose, related to the characteristics of individuals at home. Using the ICRP's weighting factors for exposure at home, the mean ratio of the distribution of millisieverts per working level month was found to be about 15. It was further concluded that a conversion factor of as low as 4 mSv/WLM was extremely unlikely from a dosimetric perspective [M32].

86. In a separate analysis of the physical parameters and (dose) conversion factors for RDPs, Porstendorfer [P12] calculated a range of (dose) conversion factors of 4.2–11.5 mSv/WLM, depending on aerosol concentration and other factors. Homes with higher aerosol concentrations had lower dose conversion factors. Porstendorfer [P12] noted that, while dose conversion factors calculated for homes with “high” aerosol levels were comparable to those derived from miner epidemiological studies, the discrepancy between the dose conversion factors derived from miner epidemiology and dosimetry remained for homes with “normal” aerosol concentrations. Dose conversion factors for exposure to thoron decay products of 2–3 mSv/WLM were also calculated.

87. Nikezic et al. [N13] discussed the importance of the absorbed fraction (of alpha particles in sensitive cells) when estimating dose conversion factors for RDPs. The authors noted that the most important parameter in estimating the absorbed fraction was the depth of the sensitive cell layers. In another paper [N14], the ICRP's human respiratory tract model [I3] and microdosimetric considerations were used to investigate the dose conversion factors for RDPs. The authors concluded that having the alpha particles deposit their energy only in the nuclei of sensitive cells reduced the dose conversion factor from 15 mSv/WLM to about 11 mSv/WLM.

88. All of the above calculations assumed the standard alpha radiation weighting factor of 20. Brenner et al. [B19] suggested that a quality factor of 20 may be too large, on the basis that RDP alpha particles deposit most of their energy in a region of relatively low biological effectiveness, and recommended a value of about 10 for residential radon exposure. The suggestion of Brenner et al. [B19] seems quite reasonable on the basis of their review of the extensive data available at present on the *in vitro* transformation of cultured mouse cells. However, Brenner's suggestion would also be likely to raise questions at the tissue level, where the cell loss by killing may stimulate cell renewal that eventually promotes the development of cancer (i.e. increasing the weighting factor). In ICRP Publication 92 [I11], the ICRP noted that the current radiation weighting factor (w_R) of 20 for internally deposited alpha emitters can serve as a guideline, and suggested that for specific situations such as exposure to radon and its progeny “more meaningful weighting factors can be derived”, whether based on specific assumptions about target cells and dosimetric models or on epidemiology.

89. Little [L21] compared lung cancer risk in the survivors of the atomic bombings in Japan (using data from reference [P13]) and the Colorado Plateau uranium miners (using data

from references [R8] and [H9]). Models of ERR were used, and time since exposure, smoking and sex were considered. Little found that, although there are statistically significant differences between the two data sets in how ERR varied with time since exposure, these differences were no longer statistically significant when only male atomic bombing survivors were used as the basis for comparison with the Colorado Plateau miners. Little concluded that the conversion factor based on the atomic bombing survivors was 18 (95% CI: 6.1, 110) mSv/WLM, using a model with exponential adjustments for the effects of radiation with time since exposure and age at exposure, and 19 (95% CI: 6.2, 160) mSv/WLM, using a model with adjustments for the effects of radiation proportional to a power function of time since exposure and attained age. The absence of smoking data for the Japanese atomic bombing survivors was acknowledged to be a potentially important confounding factor. Little's estimates compare with the range developed by Birchall and James [B18] of 17.2–22.5 mSv/WLM (with 95% CI extending from at least two times smaller to at least two times larger) using the ICRP lung model [I3] and with the ICRP's estimate of 5 mSv/WLM [I5] based on the Japanese atomic bombing survivor data and the uranium miner data. Overall, Little concluded that the various estimates of the dose conversion factor are “very close” to that predicted by dosimetry and “statistically compatible” with the epidemiological derivation of the conversion factors [L21].

E. Exposures in homes and in workplaces other than uranium mines

90. Both the BEIR IV and the BEIR VI Committees [C19, C20] used dosimetric models to examine the comparative doses for exposures of miners and people exposed at home. As a follow-up to the BEIR IV report, the United States National Academy of Science compared the dosimetry of radon in mines with that in the home [N10]. This study expressed differences in exposure–dose relationships in terms of a “K-factor”, defined in reference [C19] as the ratio of dose per unit exposure at home to the dose per unit exposure to a male miner. Thus, for $K < 1$, the dose per unit exposure at home is less than in a mine, and for $K > 1$, the dose per unit exposure at home is larger than for exposure in a mine. As reported in reference [N10], the K-factors for children and infants, calculated for a wide range of exposure scenarios, were somewhat larger than those calculated for adults, but nevertheless did not exceed 1. BEIR VI [C20] used the concept of the K-factor and included various environmental and physiological factors. BEIR VI reviewed measurements of RDP activity size distributions for data collected in Germany, the United Kingdom and the United States (annex B of reference [C20]). These size distributions were used with the RDP dosimetric model to estimate the K-factor for exposure in homes. The values obtained were close to 1 for male and female adults and for children. James et al. [J15] described the technical basis for the comparative radon dosimetry applied to BEIR VI [C20], including a thorough review of the basis for the BEIR VI

[C20] Committee's choice of unity for the K-factor. James et al. concluded that a K-factor of unity is appropriate, but noted that it is not yet established that the conditions in the houses considered in the BEIR VI analysis are representative of conditions in houses in other regions across the United States (including the effects of seasonal and climatic conditions) [C20].

91. No similar review of RDP activity size distributions has been undertaken for occupational exposure. While it might be expected that RDP exposure in uranium mines would be consistent with the ICRP conversion convention, namely $8 \text{ nSv (Bq h m}^{-3}\text{)}^{-1}$, there are a significant number of workers exposed to elevated levels of environmental radon in workplaces other than uranium mines (buildings, other kinds of mine, caves, etc.). A series of RDP aerosol measurements carried out in Australia in a range of workplaces [S45] showed that some workplaces have aerosol conditions that differ markedly from those found in operating uranium mines, particularly those operating during the 1950s and 1960s. Adjustment factors for these workplaces, also referred to as K-factors, were derived from the Australian measurements for occupational exposures and are in the range 1–2.6. Use of the ICRP conversion convention in these

exposure situations would lead to a significant underestimation of radiation doses. Thus it is important to understand the factors that affect the estimation of dose in workplaces other than uranium mines.

92. As discussed in the UNSCEAR 2000 Report [U2], the Committee adopted the dose conversion factors set out in ICRP Publication 65 [I2], which had been based on epidemiological evidence. Dosimetric evaluation of the absorbed dose to the basal cells of the bronchial epithelium per unit exposure gave values in the range $5\text{--}25 \text{ nGy (Bq h m}^{-3}\text{)}^{-1}$, and a value of $9 \text{ nGy (Bq h m}^{-3}\text{)}^{-1}$ was estimated for average indoor conditions. Using a tissue weighting factor of 0.08 for the bronchial and bronchiolar regions and a radiation weighting factor of 20 for alpha particles, the effective dose per unit equilibrium-equivalent concentration (EEC) became $15 \text{ nSv (Bq h m}^{-3}\text{)}^{-1}$. The epidemiological approach provides a value of $6 \text{ nSv (Bq h m}^{-3}\text{)}^{-1}$, a factor of $2\frac{1}{2}$ times lower, and the dosimetric evaluations provide dose coefficients in the range $6\text{--}15 \text{ nSv (Bq h m}^{-3}\text{)}^{-1}$. The UNSCEAR 2000 Report concluded that the value used by the Committee in earlier evaluations [U5, U6], $9 \text{ nSv (Bq h m}^{-3}\text{)}^{-1}$, is within this range, and recommended that this value continue to be used in dose evaluations [U2].

III. EXPERIMENTAL STUDIES

93. Information from animal experiments and from cellular and molecular biology is helpful in understanding the underlying mechanisms of cancer induction, potential interactions among agents and the uncertainties associated with extrapolating risks from exposure in mines to residential exposure. The UNSCEAR 2000 Report [U2] discussed the biological effects of low doses of ionizing radiation (annex G) and the combined effects of exposure to radiation and other agents (annex H), and provided much information from animal experiments, studies of DNA, and cellular and molecular responses to various forms of radiation. This section provides a concise summary of experimental results, including more recent data than reported in reference [U2].

A. Animal experiments

94. Animal studies have been conducted for several decades to identify the nature and levels of the uranium mine air contaminants responsible for producing the lung cancers observed among uranium miners (e.g. [C40, M29]). Many of the initial studies were concerned with early effects or short-term pathological changes (e.g. [R4]). Exposures were based primarily on radon gas concentrations, and provided little or no information on the radon decay product concentrations that contribute the greatest radiation dose to the lung. The early studies (e.g. [K10]), in which lung tumours were produced, were methodologically or statistically inadequate to show an unequivocal association of lung tumours with exposure to radon and/or RDPs.

95. In the 1950s, there was growing concern that the increased incidence of respiratory cancer observed in European uranium miners would also be found in United States miners. This led to the initiation of systematic studies in the United States to identify the agents responsible for increased incidence of lung cancer in miners and to develop exposure–response relationships in animals. Investigators at the University of Rochester began to focus attention on the biological and physical behaviour of RDPs as well as on their contribution to the radiation dose to the respiratory tract [B1, M12, M13]. Shapiro [S33] exposed rats and dogs to several levels of radon alone and in the presence of RDPs attached to room dust aerosols. He also showed that the degree of attachment of RDPs to carrier dust particles was a primary factor influencing the alpha radiation dose to the airway epithelium. This dose was further demonstrated to be due primarily (>95%) to the short-lived RDPs rather than to the parent radon. Cohn et al. [C8] reported the relative levels of radioactive material found in nasal passages, trachea

and major bronchi, and in other portions of rat lungs after exposure to radon and/or RDPs. The respiratory tracts of animals that inhaled radon plus its decay products contained 125 times more radioactive material than those of animals that inhaled radon alone [C44].

96. Beginning in the mid-1950s, at the University of Rochester in the United States, Morken and Scott initiated a pioneering series of experiments (e.g. [M13]) to evaluate the biological effects of inhaled radon and radon decay products in mice; later experiments used rats and beagle dogs. The essentially negative biological results of these studies suggested that alpha irradiation alone was relatively inefficient in producing tumours in the respiratory system. NCRP Report No. 78 [N7] provides a comprehensive summary of many of the early animal data.

97. In the late 1960s and early 1970s, other studies in France (Compagnie Générale des Matières Nucléaires (COGEMA)) and the United States (Pacific Northwest Laboratory (PNL, later PNNL)) were initiated and later proved successful in producing lung tumours from RDPs. The French investigators exposed rats to RDPs, either alone or in combination with stable cerium, uranium ore dust or cigarette smoke, to produce tumours in the lung (e.g. [C4, P4]). Later, the potential co-carcinogenic effects of various environmental and industrial airborne pollutants, e.g. minerals from metallic ore mines and diesel exhausts, combined with radon and/or radon progeny exposure were also investigated [M31]. The later United States studies were designed to systematically determine the pathogenic role of RDPs, either alone or in various combinations with uranium ore dust, diesel engine exhaust and cigarette smoke. These studies involved lifespan exposures of beagle dogs and Syrian golden hamsters, and chronic exposures of rats (e.g. [C13, C14]). A joint review of PNNL (United States) and the Commissariat à l'énergie atomique (CEA)–COGEMA (France) animal experimental data was published in 1999 [C40]. Bronchial dose models were published for the Syrian golden hamster, rats (Long–Evans, Wistar, Sprague–Dawley, Fischer) and beagle dogs [D23, H39, H40].

98. A review of the animal studies through 1970 appeared in the final report of subgroup IB, Interagency Uranium Mining Radiation Review Group [R4]. That report, which addressed the early acute radon toxicity studies, concluded (as had an earlier Federal Radiation Council report [F2]) that experimental work prior to the 1970s had not demonstrated that pulmonary carcinomas could be produced in animals in a systematic way from controlled exposures to radon and its

decay products. Since that review, discussions of the biological effects of inhaled radon and RDPs in animals have appeared in ICRP Publication 31 [I1] and NCRP Report No. 78 [N7]. A more detailed review of animal studies was provided in the most recent NCRP SC65 Report [N11].

99. Gilbert et al. [G3] analysed data on the risk of lung tumours in rats exposed to radon. Male, specific-pathogen-free rats were exposed starting at about 90 days of age to RDPs (and uranium ore dust) at levels of from 20 WLM to more than 10^5 WLM, and at exposure rates of 10 WL to 10^3 WL. (The experiments are reported by the authors to have resulted in an estimated dose of about 5 mGy/WLM at the cellular level.) The authors used a time-dependent hazard model to accumulate risk and examined several exposure-response functions, the simplest being a simple linear model. They concluded that the rat data were in “reasonable agreement with a linear exposure-response model” and estimated an overall linear (risk) coefficient of 237 per 10^6 WLM. In addition, their analysis showed “no observed evidence of exposure-rate effect below 1,000 WLM”.

100. Bijwaard et al. [B20] used equations based on a two-mutation biological model for two large data sets of radon-exposed rats (10,000 in total). The improvement in fitting these data sets separately compared with fitting them jointly was statistically insignificant, indicating that a joint fit represented the data well. The joint solution exhibited a first mutation rate two orders of magnitude larger than the second mutation rate, which was strongly suppressed by a mutation killing term acting at high exposure rates. Maximum cancer incidence occurred for exposure rates of 1–10 WLM/d, which is in good agreement with reference [M10]. An inverse exposure-rate effect was evident at higher exposure rates, with incidence orders of magnitude larger than for lower exposure rates. A very pronounced effect of age at exposure was also observed. In all cases, ERR ranged between 0.007WLM^{-1} and 0.025WLM^{-1} . The proposed model compared reasonably well with a similar model derived for Colorado uranium miners [L23], when the data for rats were scaled by the ratio of human to rat lifetimes.

101. Heidenreich et al. [H8, H26] used a two-step clonal model, based on Luebeck et al. [L9] and Moolgavkar et al. [M11], to investigate the risk of lung cancer induction in rats exposed to radon. The authors concluded that only fatal lung tumours among the rats could be used for generalizations to models for lung cancer induction in humans. This model for fatal tumours showed an inverse dose-rate effect at average exposure rates of above 20 WL, but below 10 WL the lung cancer risk per unit exposure decreased with increasing duration of exposure. Finally, on the basis of their analysis, the estimated ERR for rats at low exposure rates was in the range from 0.003WLM^{-1} to 0.012WLM^{-1} , depending on the exposure periods; this is of the same order of magnitude as the ERR seen in humans. Because a statistical test had given a strong indication that the results from different rat strains should not be pooled together, Kaiser et al. [K25] carried out separate risk analyses for two rat cohorts: the PNNL cohort

of Wistar rats, and the combined cohort of Sprague–Dawley rats at the CEA and at AEA Technology Plc (AEAT), United Kingdom. The study was restricted to fatal tumours. Using a refined technique of age adjustment [H34], the lifetime absolute risk was standardized with the survival function for competing risks in the control population. The age-adjusted excess risks for both strains of rats were of similar magnitude, despite the higher lifetime excess absolute risks per unit exposure (LEAR at 1 WLM) in the European cohort because of the very low mortality in the control group.

102. Rats were exposed to tobacco smoke and radon (1,000 WLM) at the CEA. When animals were exposed to cigarette smoke prior to radon exposure, a slight decrease in lung carcinoma incidence was observed compared with rats exposed to radon only, but when the cigarette smoke exposure occurred after the radon exposure, there was a highly significant factor of 4 increase in lung carcinoma incidence. This resulted mainly from an increase in squamous-type tumours. These data were used to estimate smoke-dependent parameters in the biologically based two-stage clonal expansion model [H36]. Although smoke had no effect on tumour initiation, an effect was seen on tumour promotion. Promotion by cigarette smoke was also seen in levels of adenomatosis in the PNNL studies. It appears that preneoplastic lesions induced by radon are promoted by cigarette smoke.

103. Monchaux and Morlier [M28] reported the results from a study of the effect of exposure rate on lung cancer induction in radon-exposed Sprague–Dawley male rats. The study was conducted at relatively low cumulative exposures of about 100 WLM, which is comparable to lifetime exposures in high-radon houses or to current underground mining exposures. The risk of lung cancer in rats decreased with potential alpha energy concentration (PAEC), i.e. exposure rate, confirming the results obtained at lower exposures [M30]. These results and those from former experiments [M31] indicated that the risk of lung tumour induction in rats was maximal for cumulative exposures ranging from 25 to 200 WLM and PAEC ranging from 50 to 150 WL, i.e. exposure rates ranging from 5 to 25 WLM per week. These data suggest that there is a “watershed” at cumulative exposures of about 50 WLM. Below this exposure, decreasing the exposure concentration (WL) or protracting the time over which the dose is delivered results in a reduction in the lung tumour risk. Above this level, the reverse is true: decreasing exposure concentrations or protracting the exposure time results in an elevated lung cancer risk [M29]. These results were confirmed by Collier et al. [C38] in a series of lifespan experiments in which the effects of radon and its decay products were investigated at different total doses, dose rates and unattached fraction. Collier et al. [C16, C34] also reported on studies of the factors that affect the risk of inducing lung tumours in rats exposed to radon and RDPs.

104. The results of rat experiments conducted in parallel at the CEA and AEAT (as described in Work Package 4 of reference [T30]) have been reviewed by various individuals. The studies were carried out specifically to investigate the

effect of exposure rates on induction of lung cancer at cumulative exposures of about (100 WLM) (0.36 J h m^{-3}). Except for the fact that rat exposures in the CEA experiments were carried out for a working day without a carrier aerosol and the AEA Technology experiments were for continuous exposure with carnauba wax as a carrier aerosol, the experimental designs were similar. The joint analysis comprised more than 4,000 exposed rats and 1,500 non-exposed control rats. The authors calculated both relative and absolute risks and found, in general terms, increased risks of lung cancer both with increasing cumulative exposure and with increasing exposure rate. These results indicated that, at low exposures comparable to those in modern mines or high-radon homes and for cumulative exposures of up to about 100 WLM, the risk of lung cancer in rats decreased with decreasing exposure rates, confirming earlier results [L43, M30] at lower cumulative exposures. A parallel analysis of these experimental data with data from European uranium miner cohort studies was performed [L44, T30]. It confirmed that epidemiological and animal data are consistent in showing an increase of risk with cumulative exposure protracted at low exposure rates.

105. Mitchel et al. [M9] reported an experimental study using a nose-only breathing system with male Sprague-Dawley rats exposed to one of two concentrations of natural uranium ore dust (44% U, at 50 mg/m^3 or 19 mg/m^3) without significant radon exposure. They concluded that chronic inhalation of ore dust alone posed a risk of lung cancer in rats that was directly proportional to dose rate.

106. Although it was established that the rat constitutes a valuable model to study radon-induced cancers, Petitot et al. proposed a system which is designed to allow direct exposure of isolated cell populations cultured *in vitro* to radon and its decay products [P18]. One advantage of this is that cells cultured *in vitro* are irradiated directly by a natural radon emanation and a mixture of RDPs in exposure conditions similar to those used for inhalation exposures of rats (in *in vitro* studies). This new method could help to identify biological markers of irradiated cells in radon-induced cancers by an *in vitro* approach.

107. Overall, animal data support the conclusion from epidemiology that exposure to radon and its decay products is carcinogenic. Moreover, animal data also confirm the observations from epidemiology that the risk from exposure to radon and its decay products increases with increasing cumulative exposure, even for protracted exposures at low exposure rates.

B. Biomarkers

108. Traditionally, biomarkers have been used to determine exposures or doses. Jostes [J14] provides an overview of the use of biomarkers as measures of effects of exposure to radon. It is becoming increasingly apparent that biomarkers of effect are potentially of great value in evaluating potential health impacts. The following discussion provides an overview of selected studies of biomarkers involving exposure

to RDPs. Readers interested in a more extensive discussion of this subject are referred to annex C, "Non-targeted and delayed effects of exposure to ionizing radiation".

109. In biodosimetry, the emphasis has traditionally been on assessing doses *per se*; however, an important perspective nowadays is to think of these biomarkers as possible surrogates for measuring the body's integrated response to radiation damage, rather than as simply indicators of absorbed dose. The focus of this section is on the potential use of biomarkers of effects of radiation exposure rather than on biological dosimetry *per se*.

110. In addition to interactions with DNA, ionizing radiation also damages other cellular components. Cellular responses to various forms of radiation include structural and functional changes to cells and cell organelles. In addition to the morphological signs related to cell death, several reversible alterations are seen in the structure of different cell organelles. Radiation-induced changes in the supramolecular organization of the membranes, including the plasma membrane as well as different cell organelle membranes, can play a significant role in the development of radiation effects. Various morphological alterations of nuclear chromatin (e.g. changes of fine structure, development of chromosome aberrations, etc.) are thought to originate from radiation-induced damage to the supramolecular organization of DNA and/or nuclear proteins [N3, S35]. Changes in chromosomes are considered to be useful as biological indicators or even biological dosimeters of radiation injury [A26, A27, A28, A29, B2, C3], and can be evaluated qualitatively and/or quantitatively by various techniques, such as morphological analysis of metaphase chromosomes, fluorescence *in situ* hybridization (FISH) and the scoring of micronuclei. Brenner et al. [B32] argued that there was a need for a biomarker to distinguish between the effects of exposure to high-linear-energy-transfer (LET) radiation and other carcinogens. They suggested that exposure to high-LET radiation produced a distinctly low ratio of stable interchromosome to intrachromosome aberrations, and recommended this ratio as a candidate for such a biomarker.

111. Miller et al. [M7] exposed cultures of C3H 10T $\frac{1}{2}$ cells either to microbeam irradiation using the Columbia University microbeam or to broadbeam irradiation in order to investigate oncogenic transformation rates, and demonstrated that cells exposed to exactly one alpha particle each had a significantly lower response than cells exposed to a Poisson mean of one alpha particle each. Miller's group concluded that cells intersected by multiple alpha particles (as may occur in high-exposure miner studies) contributed most of the response. This suggested that extrapolating from the high-exposure conditions of miners could result in an overestimation of the risk of cancer induction at domestic levels of radon exposure, in which situation essentially no target cell is ever traversed by more than a single alpha particle.

112. Brenner and Hall [B10] commented on a phenomenon known as the inverse dose-rate effect. They noted that it is

widely observed for radiation with medium to high LET, such as neutrons and alpha particles, and that a given dose delivered over a longer time (i.e. protracted) had a greater effect than an acute exposure at the same total dose. They suggested that the dose-rate effect was significant only when a certain combination of dose, dose protraction and radiation quality was present. When uranium miners are exposed to low WLs for a long period of time, the possibility of the (inverse) dose-rate effect occurring exists, but such dose-rate effects in cases of typical domestic exposures to radon are unlikely, because the average cell is traversed by one or zero alpha particles in a lifetime. Hence when risk estimates for domestic exposure are extrapolated using data from miners, the radon risk may be overestimated.

113. Similar to the conclusion reached by Brenner and Hall [B10], Jostes [J14] noted that epidemiological studies have shown that radon is a risk factor for both smoking and non-smoking miners, and that it is reasonable to suppose, on the basis of molecular and cellular considerations, that exposure to RDPs in the home also poses a risk of cancer induction. He also suggested that, while an inverse dose-rate effect has been seen in miners, such an effect may not apply to residential exposures, since the majority of lung cell nuclei would experience no alpha hits and only a few cells would receive even a single alpha hit.

114. Mutations induced in mammalian cells following irradiation with alpha particles have been studied using microbeam methods to irradiate the cytoplasm of individual human-hamster hybrid cells (A_L cells) [W11]. The aiming point of the microbeam was 8 μm from the ends of the major axis of each cell nucleus. The probability of a scattered alpha particle hitting the nucleus was 0.4%. Irradiation of the cytoplasm produced gene mutations in the nucleus in a process mediated by free radicals. While the microbeam irradiation induced minimal toxicity to the irradiated cells, it was effective in inducing mutations in the nucleus. An analysis of the mutational spectra induced by nuclear versus cytoplasmic irradiation suggested that different mechanisms of cancer induction were operative. Two approaches were used to investigate whether reactive oxygen species (ROS) mediated the process of mutagenesis through cytoplasmic radiation. The first involved the use of an antioxidant (dimethyl sulphoxide) to scavenge ROS, and the second involved the addition of a drug (buthionine-S-R-sulphoximine) to deplete endogenous scavenger sulphhydryl groups. In the first approach, the induction of mutations was suppressed; in the second, the induction of mutations was enhanced. Indicators of cellular ability to scavenge ROS may have potential as a biomarker of effect. Observations from *in vitro* studies further demonstrate that cytoplasmic irradiation is clearly a risk factor.

115. An earlier study had also demonstrated that radiation damage from alpha irradiation reflected in the patterns of sister chromatid exchange (SCE) in human diploid lung fibroblast cells was independent of the number of alpha particles traversing a nucleus [D3]. The much larger cross-sectional

area of the cell relative to that of the nuclear target was suggested to be an important consideration. It was hypothesized that the effect was mediated by the free radicals formed in the cytoplasm as the result of traversal by alpha particles. In support of this hypothesis, a paper subsequently described the production of superoxide anions and hydrogen peroxide by alpha particles traversing the cytoplasm [N1].

116. In recent years, evidence has accumulated that suggests that both directly hit and bystander cells may show effects of exposure to alpha radiation (e.g. [M36]). Goodwin and Lehnert [G8] found from *in vitro* studies that a low dose of alpha radiation increased levels of SCE, an indicator of genetic damage in the lungs of mice and humans. In addition, the amount of ROS increased. SCE and ROS levels were also increased in non-irradiated cells. The authors concluded that harmful effects of radiation are induced in bystander cells to the same extent as in irradiated cells. There was evidence that the ROS response triggered the up-regulation of cytokines that mediate the bystander effect [G8]. The authors anticipated that an important next step would be to show whether this work applied *in vivo* and whether carcinogenesis and other disease processes were a result of the bystander effect from ionizing radiation.

117. Azzam et al. [A15] postulated that the reaction of non-irradiated cells is due to gap junction-mediated inter-cellular communication (GJIC) of a damage signal between irradiated cells and their neighbours. The levels of a protein ($p21^{\text{Waf1}}$) induced by stress were compared in cells competent in GJIC and not competent in GJIC. In the competent cells, more cells showed elevated levels of the protein than could have been directly intersected by an alpha particle. The expression of $p21^{\text{Waf1}}$ correlated with micronucleus formation (indicating DNA damage) and with increased phosphorylation of the $p53$ gene. While GJIC is certainly one mechanism by which a signal is communicated from an irradiated cell to a non-irradiated cell, there is also evidence for soluble factors being secreted into the culture medium to communicate the bystander signal [M43, S73].

118. A bystander effect and gene induction in non-irradiated cells occurred for situations where only a small fraction of the cell nuclei were traversed by an alpha particle [L5]. Oncogenic transformation can arise from the passage of single alpha particles through cell nuclei [M7]. The role of ROS in mediating DNA damage and cell-related effects is of great interest in understanding bystander effects. The bystander effect and other non-targeted effects are discussed at greater length in annex C, "Non-targeted and delayed effects of exposure to ionizing radiation".

119. Little and Wakeford [L35] discussed radon-induced lung cancer and the bystander effect in C3H 10T $\frac{1}{2}$ cells exposed to alpha radiation. These authors fitted a model of the bystander effect [B21] to experimental data [M7, S47] and to epidemiological data for various residential radon studies, for Colorado Plateau miners [L10] and for the combined analysis of BEIR VI [C20]. The best estimate of the number

of neighbouring cells that contributed to the bystander effect was between 0 and 1.0 (with an upper 95% confidence limit of between 1 and 6.5), and therefore the bystander effect seen in the experimental C3H 10T½ cell system probably did not play a large role in the radon-induced lung cancer seen in the epidemiological studies of humans. The authors also found that the ERRs in the Colorado Plateau miner data were statistically indistinguishable from those derived from residential radon studies.

120. One potential biomarker of radon exposure is interleukin-8 [N2]. When alpha particles hit a cell, the production of ROS increases; this causes the production and release of interleukin-8 to increase. However, there are triggers for ROS production other than alpha radiation; asbestos, ozone and cigarette smoke can all cause an increase in ROS levels and therefore in interleukin-8 levels.

121. In an early study of chromosome aberrations in uranium miners, Brandom et al. [B30] studied cultures of peripheral blood leucocytes in 15 uranium miners and 15 normal age-matched non-miner male controls. The miners' work experience ranged from 1 to 20 years, and cumulative exposures ranged from 10 to 5,400 WLM. Chromosome abnormalities were observed in 3 (0.28%) of the control cells and 37 (2.3%) of the miner cells. The differences were statistically significant and were considered by the authors to be biologically important.

122. Smerhovsky et al. [S58] described a study of chromosome aberrations in radon-exposed miners in the Czech Republic. The study included 1,323 cytogenetic assays of peripheral blood lymphocytes from 225 mine workers exposed to RDPs at levels ranging from about 1.7 to 662 WLM. Seventy-five of the workers were reported as non-smokers. In total, some 36 lung cancers were observed in this group. Kaplan–Meier survival analysis was used to investigate cancer incidences and Cox regression was used to model associations between chromosome aberration frequency and cancer incidence. The Kaplan–Meier survival analysis indicated a significant decrease in survival times dependent on frequency of aberrant cells and on frequency of chromatid breaks. The Cox regression showed that the frequency of aberrant cells was significantly related to the risk of cancer.

123. A pilot study on German miners determined the presence of biological markers related to radon exposure in uranium mines [P3]. The researchers looked at two categories of markers: markers in the blood and markers in bronchoalveolar lavage fluid. They found that former miners' leucocytes had a decreased ability to repair DNA. In addition, there were chromosome aberrations in their lymphocytes and an increased frequency of micronuclei in lung macrophages. There was an increase in the levels of tumour necrosis factor alpha in the miners compared with the control group, and this was weakly correlated to their radon exposure. Further study is needed to establish whether these measurements are reliable biomarkers and whether individual cancer risk

can be predicted on the basis of the markers. A study of the relationship between residential radon and the occurrence of chromosome aberrations in peripheral blood lymphocytes was reported in reference [O3]. This study demonstrated an excess in the number of cells containing dicentric and/or centric rings for 61 people living in dwellings with radon concentrations of above 200 Bq/m³ compared with a control group from the researchers' laboratory (53 people). However, no statistically significant difference was seen between the control group and people exposed to radon concentrations of between 230 and 13,000 Bq/m³. Uncertainty in exposure was suggested as a possible confounding factor for this result [O3].

124. Another study done on German uranium miners tried to identify a specific genetic defect caused by alpha radiation that led to lung cancer [W4]. The authors were unable to find a mutation of the tumour-suppressor gene *p53* that was characteristic of a radon-induced cancer.

125. Hussain et al. [H28] described studies of the mutability of codons 249 and 250 of the *p53* gene patterns in normal human bronchial epithelial cells from a 15-year-old male who had never smoked. The cells were either unexposed or irradiated to a total dose of 4 Gy (equivalent, according to the authors, to 1,460 WLM of exposure to RDPs). In this study, exposure was from alpha particles from ²³⁸Pu in six equal fractionated doses. The authors found that alpha radiation selectively increased mutation frequency in both codons 249 and 250 but noted that interindividual variability argues against extrapolation of their results based on a single donor.

126. Yngveson et al. investigated the association between residential radon exposure and *p53* mutation in lung tumours [Y6]. Their study included 83 lung cancer cases in non-smokers and 250 lung cancer cases in smokers. Lung cancer cases were selected with time-weighted average radon concentrations of below 50 Bq/m³ or exceeding 140 Bq/m³. Molecular analysis was carried out on samples obtained from the pathology departments where the cancer cases had been diagnosed. Statistical analysis was carried out to investigate associations between exposure to radon, tobacco consumption and the presence of *p53* mutations. A non-statistically-significant odds rates (OR) for increased mutation prevalence was indicated for those exposed to high levels of residential radon (OR = 1.4; 95% CI: 0.7, 2.6), especially among non-smokers (OR = 3.2; 95% CI: 0.5, 15.5). Mutations of *p53* were also found to be associated with smoking status and, in the case of non-smokers, with exposure to environmental tobacco smoke.

127. Although biomarkers such as mutations in the *TP53* gene (which encodes the *p53* protein) are recognized as important biomarkers of radiation damage, not all studies arrive at this conclusion. Besides the study in [W4], a more recent study of *p53* in serum samples from former uranium miners found no correlation between *p53* protein concentrations in serum and exposure to ionizing radiation (measured

in WLM), and the authors concluded that there was no benefit in screening for *p53* or *p53* antibodies at the present time [S7]. A study by Vahakangas et al. [V3] of lung tumours in miners found that their *TP53* gene mutations were different from those typically seen in lung cancers caused by tobacco smoke: there were no G:C to A:T transitions in the coding strand, and the mutations were mostly transversions and some small deletions, which are very uncommon in human lung cancers.

128. Albertini et al. [A2] studied the viability of using *HPRT* mutations in human T-cells as markers of the quality of radiation to which a person was exposed. They looked at the different mutations produced by high- and low-LET radiation. With high-LET radiation such as that from radon, the mutations were mostly small partial deletions, with fewer than 2% being total gene deletions. There were relatively more breaks in the DNA strand because of the energy transferred from the alpha particle to the strand. A high proportion of the breaks were lethal, so alpha radiation is efficient at killing cells. In the case of low-LET radiation, 10% of the mutations were total gene deletions and most of the damage to the DNA molecule occurred because of secondary ionization, not from the initial collision. These or related differences may eventually allow researchers to differentiate between high- and low-LET radiation.

129. Alpha particles, like other high-LET particles, induce the tumorigenic phenotype into BEP2D cells in the lungs [Z1]. This is achieved by deleting suppressor genes. Thus, by looking at the levels of the products of these genes, it may be possible to detect the early development of cancerous cells.

130. Jostes [J14] suggested that the cellular response to alpha radiation may depend on the repair status of the affected cell. This was based on studies by Schwartz et al. [S8] and Shadley et al. [S32], which compared the induction of chromosome aberrations in repair-proficient versus repair-deficient cells. In reference [S8], it was found that a cell line that was repair-deficient for single-strand DNA breaks was more prone to aberration induction than the parental repair-proficient cell line. In reference [S32], the cell line used was repair-deficient with respect to double-strand DNA breaks, and it was less prone to aberration production when alpha particles were used than when X-rays were the inducers. It also appears that there is an adaptive response to radiation. Jostes [J14] reported that when human peripheral blood lymphocytes were exposed to a small priming dose of low-LET radiation and later to a challenge dose from radon, the number of chromosome aberrations was smaller than expected [W8, W9]. However, when alpha particles and X-rays were used at the same time, the number of micronuclei was higher than the anticipated additive effect [B4]. Taylor et al. [T1] suggested a *p53* mutation hot spot in radon-associated lung cancer, noting that Jostes [J14] had also compiled results from various studies on mutations at the *HPRT* locus in Chinese hamster ovary cells exposed to alpha radiation. In general, half of the mutations were complete gene deletions, with the

other mutations composed roughly equally of partial deletions and rearrangements (~25%) and undetectable changes (~25%). The proportions changed depending on cell type [J14].

131. In a study using comparative genomic hybridization, Dano et al. investigated gains and losses of genetic material in a series of radon-induced rat lung tumours [D13, D19]. Frequent losses occurred at various locations homologous to human chromosome bands. These regions are frequently (30–80%) deleted in human lung cancer and contain tumour suppressor genes or proto-oncogenes such as *MET*, *CDKN2A*, *CDKN2B*, *FHIT* and *RBI*, and genes yet to be identified. Frequently observed gains involved chromosomes homologous to those in human encoding *MYCN* and *MYC* oncogenes. The genetic similarities between rat and human lung cancer suggest common underlying mechanisms for tumour evolution in both species and provide an opportunity to study early events in carcinogenesis. Moreover, cytogenetic and molecular genetic analyses of radon-induced rat lung tumours could help to better understand the development and progression of radon lung cancer in humans.

132. Smoking remains the predominant cause of lung cancer. The effects of smoking per se and of exposure to environmental tobacco smoke (ETS) [A24] are of great interest for both miner and residential epidemiological studies of radon [A23]. On the basis of in vitro studies, Piao and Hei [P7] suggested that the combined effects of radiation and smoking are additive for both low- and high-LET radiation.

133. Bennett et al. [B31] reported a molecular epidemiological study of gene–environment interactions in promoting lung cancer in women who never smoked. The study was designed to assess the risk of lung cancer from exposure to ETS, radon and dust, as well as family history and occupational exposure in 106 white women with lung cancer who never smoked. The authors also carried out genetic analysis for cancer susceptibility genes. Odds ratios and 95% CIs were calculated by multiple logistic regression. A dose–response relationship was found between ETS exposure and increased lung cancer risk in women with a common genetic deficiency, loss of GSTMI enzymatic activity, with the trend test significant at the 2% level. This suggested that ETS exposure could possibly double the risk of lung cancer in nearly half of the white women in Western nations. According to Bennett, loss of GSTMI enzymatic activity occurs in about 50% of the white population in Europe and North America.

134. Alavanja [A23] reviewed biological damage from exposure to tobacco smoke and from radon, and concluded that strong similarities existed between the biological damage caused by the two agents. He also noted a protective effect arising from cruciferous vegetable consumption, at least in part attributable to their antioxidant properties, in both smokers and non-smokers [A23].

135. Traditionally, biological effects from irradiation of a population of cells are considered to arise as a result of

unrepaired or misrepaired damage to DNA in irradiated cells. It has been noted previously in this section that radiation effects can also occur in non-irradiated cells (e.g. [L5, L35, M7]), a phenomenon commonly referred to as the bystander effect (e.g. [M36, M37, M38]). As the bystander effect is thoroughly discussed in annex C, “Non-targeted and delayed effects of exposure to ionizing radiation”, a detailed discussion here is unnecessary, other than to note that while the existence of the bystander effect raises many questions about traditional dosimetric modelling, risk factors derived from epidemiology will already implicitly take into account any contribution from the bystander effect, thereby supporting the use of epidemiological evidence for risk estimation.

136. At the present time, considerable uncertainty remains about the effect on risk estimation of the “new” biology outlined above and discussed in greater detail in annex C. Future dosimetry will need to consider the effect of mechanisms of carcinogenesis such as those discussed above. In the meantime, it should be noted that the effects of such mechanisms are implicitly included in the results of epidemiological studies. Although several potential biomarkers of radon exposure have been studied, chromosome aberrations appear to be the most promising at this time, owing particularly to the possible “signature” of high-LET exposures and the correlation with cancer risk.

IV. EPIDEMIOLOGICAL STUDIES OF MINERS

137. Studies of underground miners exposed to radon form the current basis for estimating risks from radon and its decay products. The UNSCEAR 2000 Report [U2], BEIR VI [C20] and others have reviewed the epidemiological studies of miners, but scant attention was given to the basis for the exposure estimates. Miner studies are reviewed in this section. Particular attention is given to the sources and effects of uncertainty in estimates of risk to miners.

A. United States: Colorado Plateau miners

1. Introduction

138. The discovery of radioactive ores in the Colorado Plateau dates to between 1881 and 1887. The ores contained vanadium, uranium and a small quantity of ^{226}Ra [H13]. Before there was a demand for uranium and radium, vanadium was mined on a small scale. Radium production became more important during the period 1916–1923. However, United States radium production eventually lost its competitiveness owing to the availability of radium from high-grade ores from the Belgian Congo, and during the period 1930–1945, the vanadium content of the Colorado Plateau ores was the principal objective of their mining. Subsequently, through the defence initiatives associated with the Second World War, the emphasis turned more towards the mining of uranium [L13].

139. The uranium mining industry had expanded somewhat by 1949, and by 1950, some 500 miners worked in the Colorado Plateau area mining uranium ores, mostly in small underground workings with an average production of about 1 ton (907 kg) per person-day [H12]. According to Holaday [H12], employment peaked in 1960, when about 5,800 underground miners were employed. By 1967, employment had declined to approximately 2,800 miners, who were then producing approximately 3 tons per person-day. Holaday noted that by 1967, the occupational health field station had some information on exposures in over 1,200 different mining operations; however, an unknown additional number of operations were never surveyed.

140. Cooper [C11], writing in a special supplement of the *Journal of Occupational Medicine*, noted that in 1953 or 1954, large deposits of primary uranium ores were discovered in the Moab, Utah, area and in the Grants area of New Mexico.

141. In 1949, as a result of concern about the possible health hazards of uranium mining, the United States Public Health

Service (USPHS), in cooperation with the United States Atomic Energy Commission and the state health departments of Arizona, Colorado, New Mexico and Utah began studies of exposures in uranium mines. By 1950, medical studies had been initiated [A12], and uranium miners were subject to routine medical examinations. Beginning in 1954, medical examinations were performed every three years on all uranium miners who could be reached and who agreed to undergo examination [C11].

142. The initial study population, some 90% of these so-called “Colorado Plateau” miners, consisted of all miners examined in 1954. The USPHS began to collect data on radiation exposure, smoking history and mortality for these miners. Several analyses of these data, with different periods of follow-up, were published [A4, A10, A12, A16, A17, H17, L13, L14, L15, W1, W2, W3, W12].

143. Holaday [H11] recommended that all states adopt a tentative “working level” of 10^{-10} Ci/L of radon in equilibrium with its decay products [S31]. For practical purposes, the recommended standard was equivalent to 1 WL, or 12 WLM per year of exposure. Holaday indicated that this recommendation was adopted as a guide by official agencies in most of the uranium mining states. Holaday [H11] documented decreasing exposure to RDPs in the Colorado Plateau mines: in 1961, only 21% of the mines studied had WL measurements of below 1 WL, but by 1967, 70% of the mines fell in the <1 WL range.

144. Before 1964, the road systems serving the mines in the Colorado Plateau area were inadequate, and since most mines were located in remote areas, supplies of fresh water and electricity were often insufficient [H13]. Holaday and Doyle hypothesized that this insufficiency of electrical power contributed to high RDP levels during the early days of mining [H13]. In those days (early 1950s), the mines were typically shallow [H11], were usually entered by a horizontal adit or incline, and were almost always ventilated by natural draught only. In most mining operations, the miners would work throughout the mine in a variety of activities — drilling, mucking, handling the ore or setting timbers, etc. This pattern of working was established when the mining was for vanadium.

145. Uranium was identified in ores in the Shiprock area of New Mexico around 1918. At that time, it received little attention. By 1950, however, there was considerable interest in uranium, and various uranium outcroppings were discovered in limestone and sandstone areas in the Grants

mineral belt [S5]. At various times during the period 1950–1960, some 60 mines were in operation. Uranium market fluctuations starting in the early 1960s resulted in various expansions and shutdowns. In the period 1966–1978, the annual output of uranium in the Grants area of New Mexico represented approximately 45% of United States uranium production.

146. The New Mexico Health Department began monitoring RDP levels in New Mexico mines in the late 1950s, with the aid of the state mine inspector's office. According to Samet et al. [B47, S1, S3, S4, S5, S74], the maximum permissible concentration of RDPs decreased from 25 to 10 WL in 1960, to 5 WL in 1963, to 3 WL in 1967, to 1.75–2 WL in 1969, to 1.4 WL in 1973, and finally to 1 WL in 1976. The Grants clinic, which opened in 1957 to serve the miners, handled 80–90% of the pre-employment and follow-up medical examinations of miners, and kept records of the movements of most miners in this area. The majority of the investigations of New Mexico miners were examinations of morbidity and mortality among uranium miners and parallel investigations of the miners' exposures, with information on the exposures being published in the third annual report of the study by the University of New Mexico [S4]. A 1989 report provided the results of a case-control study to investigate lung cancer risk in a cohort of the New Mexico underground uranium miners [S1].

2. Radon and radon decay products

147. According to Lundin et al. [L13], radon gas measurements in United States mines were first made in 1949, and RDP measurements were first performed in 1951. In 1952, an effort was made to survey all operating uranium mines, and during that year, RDP measurements were made in 157 mines and radon gas measurements in 79 mines. In 1953–1954, the number of surveys was limited, but by 1955, many of the larger uranium mining companies initiated their own air sampling programmes. By 1956, the mining companies were performing most of the mine survey work, while agencies continued their own control programmes [L13]. These measurements were carried out across the four states of Arizona, Colorado, New Mexico and Utah.

148. Lundin et al. [L13] emphasized the large number of measurements made in the mines: "From the entire 1951–1968 period nearly 43,000 measurements of RDP concentrations were available to characterize the approximately 2,500 uranium mines from which ore was shipped" (p. 31). However, this represented typically less than one measurement per mine-year averaged over 18 years of study. The distribution of these WL measurements, moreover, was far from uniform (from no measurements to a fairly large number of measurements in a single mine). Lundin et al. tabulated the number of mines in which five or more RDP measurements were made in any one year. This amounted to 116 mine-years for the period 1951–1954, during which more than half of the cumulative exposure of miners was received. For the period

1955–1968, the total number of mine-years for which there were more than five measurements was 1,313 [L13]. An earlier study by the Advisory Council of the United States National Academy of Sciences [N4] observed, "Exposure values assigned to the period before 1956 are highly unreliable, being based almost entirely on estimates rather than measurements of concentrations" ([N4], p.7).

149. The exposure estimates used by Lundin et al. [L13] were based on RDP concentrations derived in one of three ways:

- "Measured". Values were derived directly from one or more measurements of RDP concentrations in any given mine in a given calendar year. Before 1955, such measured data included only a few of the work areas in the mine.
- "Extrapolated". Extrapolated concentrations were obtained by extrapolating between measurements made in the same mine in other years, if not more than two years before or after the year to which the extrapolation applied. In some cases, regional extrapolations were also made for mines having no direct measurements; this was done by assigning a concentration value equal to the average for the other mines in the vicinity.
- "Guesstimates". These were provided by Holaday for mines in which RDP concentrations could not be obtained by any of the extrapolation techniques discussed in Lundin et al. [L13]. The "guesstimates" were based on general knowledge of the airborne concentrations that occurred in similar mines during the same time period and on expert knowledge of long-term trends.

150. The miner database of the United States Public Health Service/National Institute of Occupational Safety and Health (USPHS/NIOSH) contains a great deal of information on miners' exposures [S13]. There are significant temporal and spatial trends in RDP concentrations. Figure III illustrates the decrease in RDP concentrations over time. The figure is a plot of annual RDP concentration (WL) by years, estimated using the different methods described by Lundin et al. [L13]. The WL values estimated by the different methods are approximately comparable and show a similar temporal decrease. The high measurement average in 1951 was due to a high WL (895 WL) averaged over 8 samples at the Freedom Mine in Salt Lake, Utah. Figure IV illustrates the extent of variability in measured WL values for Colorado Plateau miners.

151. Given the uneven distribution of measurements on a per-mine basis and the large variability between mines, it may not be possible to estimate exposures for a specific year in those mines where no measurements were taken. SENES [S13] estimated variabilities at the four levels of "area estimate" by the pooled standard deviation of measurements, under the assumption that there was no spatial or temporal trend in the variation of exposure rate. At each level (i.e.

Figure III. Average radon decay product concentration for underground uranium mines in the Colorado Plateau area from 1950 to 1968, inferred using different methods [L13].

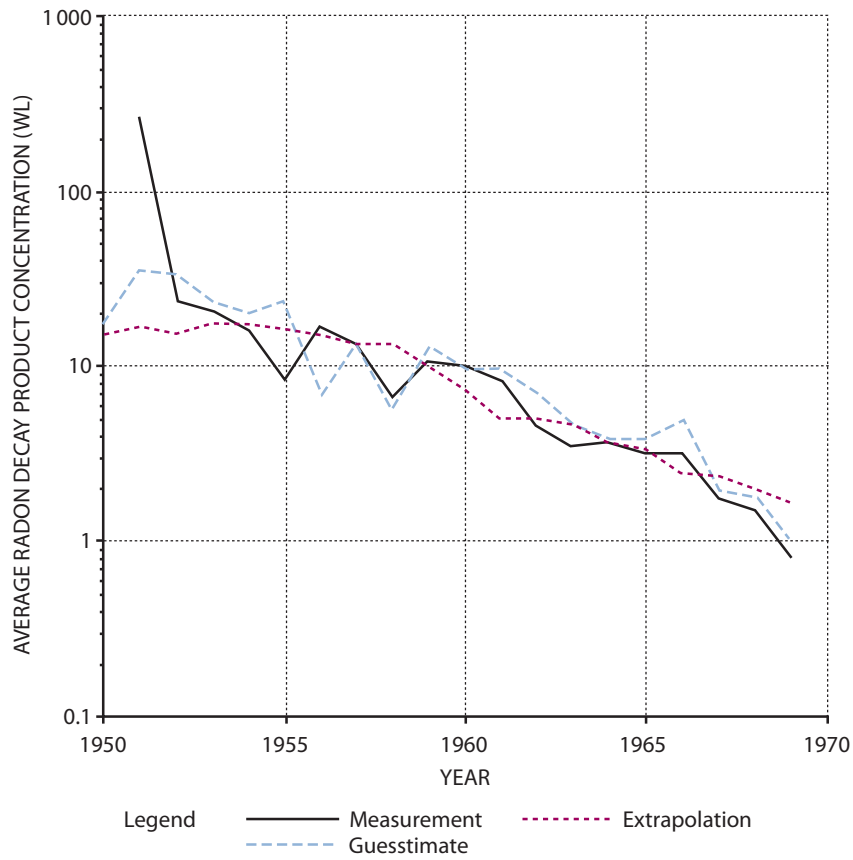
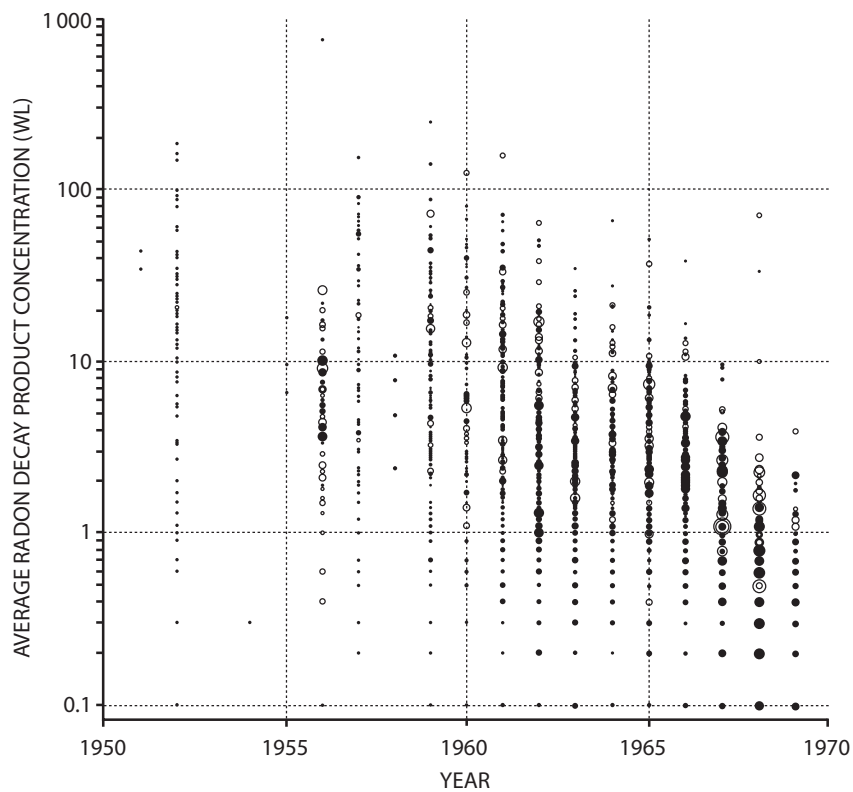


Figure IV. Range of average radon decay product concentrations measured in underground uranium mines in the Colorado Plateau area [S13].



local, district, etc.), only areas that met the criteria of having more than three mines and 10 samples were included in the calculation; the natural logarithm of measured concentration

was used with sample size as a weighting factor. The results are listed in table 6 together with the equations used in the calculations to rate the large variability in WL values.

Table 6 Estimated variabilities at different levels of area estimate [S13]

Level of area estimate	Symbol	Standard deviation of natural logarithm (WL)
Locality ^a	S_L	3.16
District ^b	S_D	3.23
State ^c	S_S	3.39
Colorado ^d	S_C	3.66

$$S_X^2 = \frac{\sum_m \sum_\alpha (E_{m\alpha} - \bar{E}_\alpha)^2}{\sum_\alpha (N_y - 1)}$$

where
E is the natural logarithm of measured concentration (WL);
 \bar{E} is an average over *E* weighted by sample size;
N is the number of measurements;
X = *L*, *D*, *S* and *C*, denoting locality, district, state and Colorado levels, respectively;
 $\alpha = (ly), (dy), (sy); m = \text{mine}; y = \text{year}$.

Summation over α denotes summation over both indices indicated in parentheses. In the case of the Colorado level, summation over α reduces to a single summation over variable *y* only.

^a Locality: reflects variability among mines within the local mining area.

^b District: reflects variability between mines within an entire mining district.

^c State: reflects variability between states (Arizona, Colorado, New Mexico, Utah).

^d Colorado: reflects variability among Colorado mines.

152. Schiager and Hersloff [S6], reporting on interviews with Holaday, noted that, while large uncertainties existed in the estimates of the RDP concentrations, these were the best that could be made and there was no conscious bias injected into the estimates of the early exposure conditions. This is in contradiction to other statements, which indicated that, where uncertainties existed, WL were deliberately overestimated [L13]. If, on average, exposures were overestimated, then on average the risk per unit exposure would be underestimated. As noted previously, mining companies began conducting the majority of exposure measurements after 1956. Lundin et al. [L13] felt that during this period, i.e. the 1950s and 1960s, there was a possibility of a bias towards underestimation of exposure, and therefore these authors tried to avoid this potential bias: "...efforts were made to exclude company measurements from data after 1960 from use in the epidemiological study of uranium miners" (p. 31). However, since the bulk of the exposures upon which risk estimates are based occurred prior to 1960, this selection had very little impact on the results overall.

153. In summary, most WL exposure estimates for Colorado Plateau miners for the period before 1950 seem unreliable, as essentially no data exist and all the estimates were based on extrapolation or "guesstimates". Exposures estimated for the period 1950–1956 were considered more reliable, although still highly uncertain. After 1956, the mining companies themselves started to make measurements on a systematic basis and, at least in the large mines, measurements became

more reliable. However, there is still likely to be considerable uncertainty in exposure assessment up to what one might refer to as the "modern period", i.e. 1967 to the present.

3. Exposure estimation

154. Wagoner et al. [W2] concluded that the excess respiratory cancer rates among uranium miners were not attributable to age, smoking activity, heredity, urbanization, self-selection, diagnostic accuracy, prior hard rock mining or ore constituents. They attributed the excess risk to airborne radiation.

155. Miners who were examined in the Colorado Plateau area during the period 1951–1960 and for whom sufficient records existed made up the cohort studied by Wagoner et al. [W2]. The exposure calculation was performed individually for each miner; the miners were then categorized into groups according to exposure levels. The average RDP concentration (WL) and duration of work underground in working months (one working month is taken as 170 hours) were multiplied to arrive at average exposures (WLM) for each group of miners; additional details were provided in a subsequent paper by Wagoner et al. [W1]. At that time, approximately 12,000 RDP measurements were available for the approximately 1,200 mines under study. The RDP concentrations used in the calculations were derived as follows:

“When there were multiple measurements in the mine during a calendar year, an average annual exposure was calculated. Measurements from non-work areas were excluded from all calculations. When no measurements were available for a mine during a calendar year, estimates were made from the average of measurements for the same mine during the preceding and following calendar year or other mines on the basis of geographic proximity, similar ore bodies, physical layout, ventilation and control efforts of regulatory agencies” ([W1], p. 184).

156. More detailed measurements began in 1967 with the intention of presenting these data for review by the Federal Radiation Council (FRC) [F2] so that the FRC guidelines for the control of radiation hazards in uranium mining could be updated. The results of this update were presented in a paper by Lundin et al. [L14], who examined a cohort of 3,414 white and 761 non-white underground uranium miners who had undergone medical examinations in 1950–1961. The results indicated that uranium miners who smoked had an excess lung cancer risk ten times greater than non-smoking miners. Prior hard rock mining experience had little effect on lung cancer mortality overall but was suspected of contributing more significantly to risks in the lower exposure categories.

157. In 1971, Lundin et al. [L13] reported on the cohort with exposures updated from the start of mining up to the end of September 1969. The cohort contained 3,366 white and 780 non-white uranium miners with at least one month of underground mining prior to 1 January 1964. Procedures were also developed by Lundin et al. [L14] for estimating RDP exposure from hard rock mining other than uranium mining.

158. As previously noted, radiation levels in uranium mines dropped sharply after 1967. This fact, combined with a drop-out rate of 10%–50% per year for the original cohort after 1960, justified the assumption that exposures received after September 1969 contributed only a relatively small additional exposure to this cohort.

159. According to Lundin et al. [L13], more than 50% of the collective exposure of some 2.8×10^6 person-WLM was received prior to 1955. For the calculated cumulative exposures (expressed in person-WLM) received up to 30 September 1960 by 3,325 men, more than 25% was received by 1,325 men and was based on actual measurements [L13]. Although the converse was not explicitly stated, it may be inferred from reference [L13] that, for the other 2,000 men in this cohort, less than 25% of the cumulative exposure was based fully on measured data. This emphasizes strikingly the uncertainties in the early exposure data; however, it does not necessarily imply any bias in exposure estimates and hence in risk estimates. The duration and periods of exposure of individual miners were determined in some cases from employment records, but in most instances they were determined from interviews with the miners themselves. Annual exposures for full-time miners were assigned on the basis of

RDP concentrations averaged throughout the mine in which each miner worked. For calculating annual exposures, “it was assumed, unless we had information to the contrary, that a man worked in the mine at which he was found for six months before and six months after the census or questionnaire date (6 month rule). When a man was known to work in two different mines at less than a one year interval, the period of employment during the interval was equally divided between the two mines” [L13].

160. Full-time mining was assumed to consist of 12 full months underground with no adjustment for vacation or illness. This assumption was an important reason for believing that exposures were overestimated; however, no adjustments were made for overtime work or ‘moonlighting’ by any of the miners [S6]. People familiar with the uranium mining industry in the 1940s and 1950s recalled that many miners worked exceptionally long hours. A standard working week until 1960 was at least 48 hours. Many miners working entirely under production contracts spent 50–60 hours underground each week. It was not unusual for a miner to work a regular full-time shift for an established company and then spend his days off developing a mining claim of his own. Since estimates of cumulative exposures (i.e. WLM) were based on months rather than actual hours worked, the method tended to underestimate exposures. It is likely that any potential bias in exposure estimates resulting from not making allowances for vacation and sickness was more than compensated by not making allowances for underground time exceeding the normal (48 hour) working week.

161. The 1968 report of the National Academy of Sciences [N4] suggested that the problem of determining radiation exposure for individual uranium miners was also complicated because official mine records did not necessarily show the actual job assignment. Only the miner himself, and to a lesser extent his immediate supervisor, knew the areas in which he worked. The report also raised the possible problem of exposure from previous mining, and further noted, “there is some uncertainty in the average working values even in mines in which numbers of measurements have been made.” Measurements consisted of spot sampling at a particular time and location, and therefore reflected only the conditions that existed at that time and location. (This is important since exposure in WLM is the product of time in working months, a working month nominally being taken as 170 hours, and RDP concentration in WL.) The report recognized that workplace conditions (dust levels, WL, etc.) varied with the nature of the operations being carried out, e.g. blasting, the ventilation provided and the amount of ore uncovered.

162. According to a review by the USPHS epidemiological study ([J10], p. 1,266), the basic information examined for individual uranium miners was the following:

- Mines worked;
- Dates of employment in mines;
- Fractional time in mines;

- Radon decay product concentration levels measured or estimated for each mine as a function of calendar year.

From these data, cumulative WLM values were calculated for each individual miner. Cumulative WLM values were calculated for both underground uranium mining alone and for uranium mining plus other hard rock mining. The average exposure of the group of miners in the USPHS study was of the order of 800 WLM [C18].

163. Some effort was made to estimate the exposure of miners to RDPs accumulated since 1967. As reported in reference [S13], exposure data were collected and examined at 23 separately managed large mining operations and also for more than 180 small mines representing in total some 25,000 miners. The major conclusions of this study (for 1967–1985) were: the average working time for the underground uranium miners studied was 3.3 years, and was 5.9 years for those who worked more than 2 years; the average lifetime exposure (i.e. 1967–1985) of underground uranium mine employees was 3.6 WLM; and the average yearly exposure per employee who worked in underground uranium mines in this period was 1.2 WLM.

164. Many New Mexico miners were included in the cohort study above. An independent epidemiological study of New Mexico uranium miners was initiated in 1977. This study was performed by the University of New Mexico under the direction of J.M. Samet. The focus of Samet's study group was retrospective analyses of a cohort of 3,055 underground uranium miners whose first underground experience occurred prior to 1971. An outline of the approach used to estimate exposure in the New Mexico study is provided in references [M34, S4, S75]. According to Samet et al. [S75], two large databases were developed to profile exposures to RDPs of Grants area miners:

“The first comprises WLM measurements from 1957 to 1967. A total of 20,086 individual readings are available from the 186 visits made during the 11 years; after 1960, mine index values, which weight individual measurements by number of persons exposed, were generally reported. The second includes all individual WLM reports by companies for 1967 through 1982” [S75].

165. The mean WL values reported by Butler et al. [B47] were 42 in 1954, decreasing greatly to about 4.3 in 1955, and subsequently generally declining to about 1.7 in 1967. These authors also reported that the mean annual WLM exposure was below 10 from 1969 onward [B47]. It is perhaps worth noting that the (weighted) mean of the WL measurements was found to provide the best indicator of the total mine index values, which weight measurements by numbers of personnel exposed [B47, L35, S1, S4].

166. The employment history of United States miners was documented by various means. Miners were interviewed

about mining experiences during medical examinations. In addition, supplemental information from subsequent annual uranium miner censuses, records of official agencies and mail questionnaires was available [L13]. Efforts were also made to account for other hard rock (OHR) mining experience, as many of the underground uranium miners in the Colorado Plateau had previous mining experience. For example, some had hard rock mining experience from other western mines, while others were coal miners from the eastern United States. However, the lack of radon or RDP measurements from these other mining activities may result in an underestimate of the miners' actual exposure.

4. Other hard rock mining exposures

167. Radon is a normal constituent of mine atmospheres, including those of non-uranium mines. For example, Holaday [H14] reported the results of several early measurements of radon in non-uranium mines in Colorado and New York. Radon levels surveyed in 35 metal and clay mines in Colorado ranged from 10 to 2,100 pCi/L. In the New York mines, radon concentrations ranged from 2 to 110 pCi/L. According to SENES [S9], about 40% (1,433 out of 3,359) of white male Colorado Plateau miners accumulated radiation exposures from OHR mining. To investigate this, exposures from OHR mining were calculated for miners whose exposures in uranium mines were less than 120 WLM. Following the procedure employed in the USPHS investigations as described by Lundin et al. [L14, L15], a miner was assumed to have completed his OHR mining activity by the year preceding the start of his underground uranium mining. Exposures were based on assumed RDP concentrations in OHR mines of 1.0 WL prior to 1935, 0.5 WL from 1935 to 1939 and 0.3 WL from 1940 onward.

168. When these OHR mining exposures were added to the exposures from uranium mining, the dates used in the previous analysis (i.e. the dates when each miner reached the limit of each exposure category) had to be recalculated. The assumed starting date of OHR mining was calculated on the basis of the total exposure and the appropriate rates. Next, from the OHR mining exposure rate and the known dates at which different uranium mining exposure levels were reached, the miner's exposure history was classified according to exposure rates and the number of months at each rate. The exposures were assumed to be accumulated at a constant rate during each period at a particular exposure level. The dates on which each miner reached a certain level of accumulated exposure were seen to be earlier when the OHR data were taken into account. The addition of OHR mining exposures caused a shift of the miners, and hence the lung cancers (and person-years), into the higher exposure categories. The average uranium and OHR mining exposures from the NIOSH cohort (with follow-up through 1985) are shown in table 7 [S13].

Table 7 Average uranium and OHR mining exposures by exposure category — white miners [S13]

<i>Exposure category for uranium mining (WLM)</i>	<i>Average time spent underground (years)</i>	<i>Average exposure for uranium mining (WLM)</i>	<i>Average exposure for uranium and OHR mining combined (WLM)</i>	<i>Average exposure rate while underground (WLM/year)</i>
<120	1.11	51.7	68	46.58
120–359	3.33	233.4	249	70.09
360–839	5.24	572.6	594	109.27
840–1799	7.47	1229.6	1252	164.61
1800–3719	10.66	2515.5	2534	235.98
3720+	14.22	5787.3	5808	406.98

169. Although the impact of OHR mining on the overall cohort average WLM is small, the effect for individual miners can be appreciable. Table 8 summarizes the estimated exposures of miners from uranium mining only and from OHR mining, for those whose uranium-only exposure was estimated at less than 120 WLM [S9]. When OHR mining exposure was added, 5 of the 10 miners shifted from the 0–120 WLM category into the next higher exposure category. The average exposure of this group increased

from 51 to 101 WLM. These data suggest that, particularly in the low exposure categories that are of greatest interest to present-day miners, a potential bias towards underestimation of exposure is introduced if OHR mining exposure is neglected. In the absence of an agreed procedure for estimating OHR mining exposures, studies could be done either on miners with no pre-1950 exposure (which minimizes this effect) or with the use of categorical variables denoting the presence or absence of pre-1950 exposure.

Table 8 Effect of OHR mining for lung cancer cases with less than 120 WLM uranium mining exposure [S9]

<i>SENES ID number</i>	<i>Uranium mining exposure (WLM)</i>	<i>OHR mining exposure (WLM)</i>	<i>Total exposure (WLM)</i>
1911	8	0	8
1011	13	136	149
1092	28	0	28
4017	30	0	30
3321	33	0	33
3633	42	0	42
3297	42	187	229
3588	44	62	106
2023	68	29	97
1563	73	68	141
50	82	0	82
534	83	122	205
560	119	44	163
Average	51	50	101

170. Limited data are available on the characteristics of mine atmospheres in the past. For example, the attached fraction of RDPs will depend on a number of characteristics of the mine atmosphere, among them WL, dust levels, dust particle size and humidity. Such data are important for both dosimetry and epidemiology. Useful data are summarized in various publications, including references [C10,

G1, N7, N10, S23]. A 1957 report on the control of radon and its decay products [H27, U16] compared the operating conditions in uranium mines with those in conventional hard rock mines. While few data were presented, the report states that “dust counts in uranium mines indicate concentrations from 5 to 20 million particles per cubic foot of air” and “the silica content of uranium ore (carnotite) ranges from 50 to

75 percent” [H27]. It is important to remember that the majority of measurements in mines are from the mid-1960s onward, and that the major portion of exposure of miners dates from earlier times when few actual measurements were made.

5. Epidemiological analyses

171. In 1950, the United States Public Health Service (USPHS) began to collect various data on uranium miners in Arizona, Colorado, New Mexico and Utah, including radiation exposure, smoking history and mortality. Numerous analyses of the data have been published [G4, H9, H17, L13, L16, W12]. The miner data were incorporated into analyses carried out by BEIR IV [C19], Lubin et al. [L10] and BEIR VI [C20], among others.

172. Hornung and Meinhardt [H17] reported a proportional hazards analysis of the Colorado Plateau cohort, originally described by Lundin et al. [L13], with follow-up to 31 December 1982. The cohort consisted of 3,366 (white) miners with 256 lung cancer deaths, a median (cumulative) exposure of 10.3 WLM and a median duration of employment of 48 months underground. This study evaluated several risk models and chose a power function model since it provided the best fit to the data, and permitted analysis of the effects of several temporal factors and smoking. The study estimated excess relative risk (ERR) to be 0.9–1.4% WLM⁻¹, compared with a previous smoking-related estimate of 0.31% WLM⁻¹ reported by Whittemore and McMillan [W12]. The relative risk increased with age at exposure and decreased with increasing time since exposure (a reduction of about 55% 10 years after cessation of mining compared with miners with the same exposure, smoking history and age) [H17]. Hornung et al. [H9] conducted further studies of modifiers of lung cancer risk in the Colorado Plateau miner cohort; follow-up to 31 December 1990 added an additional 121 lung cancer deaths, bringing the total number of lung cancer deaths to 377. This analysis confirmed the earlier finding of a strong interdependence of relative risk and age. It also suggested an exposure-rate effect resulting in a concave downward dose response; i.e. the relative risk was reduced at low exposure rates and low cumulative doses. For risk estimation, the authors recommended the use of ERR after stratification to lower exposure rates (<10 WL) and cumulative

exposures (<800 WLM). The authors also briefly discussed the potential for errors in the exposure estimates to bias epidemiological analysis, and suggested that such errors might result in the underestimation of the ERR.

173. A 1992 report [S9] described exploratory analyses performed on white male underground uranium miners with follow-up to 31 December 1985. The purpose was to investigate the effect of other hard rock (OHR) mining experience and smoking on the risk of lung cancer. The report also included an analysis of a subgroup of miners with cumulative exposures of below 2,000 WLM, using Poisson regression to estimate model parameters using iteratively reweighted non-linear regression. Simple linear ERR and absolute risk models were among the dose responses evaluated. The results are shown in table 9. While the regressions in table 9 are all significant ($p < 0.01$), on the basis of the F-ratio (a statistical test to see whether the amount of variation explained by the regression is significant), the absolute risk model accounts for more of the variability in the data for all miners and smoking groups than the relative risk model. The effect of OHR mining and the joint effect of OHR mining and smoking were also investigated (table 10). The miner groups included all of the miners with no exposure from OHR mining, those with OHR mining experience but no uranium mining exposure prior to 1950, those with neither OHR mining experience nor pre-1950 exposure in uranium mines, and those with only OHR mining experience. All of the regressions in table 10 are significant ($p < 0.01$). On the basis of the F-ratio, the results in table 10 show that, in this case, the relative risk model accounted for more of the variability in the data than the absolute risk model. The absolute risk to miners who never smoked ($2.8 \times 10^{-6} \text{ a}^{-1} \text{ WLM}^{-1}$) was about half that calculated for “all” smokers ($6.6 \times 10^{-6} \text{ a}^{-1} \text{ WLM}^{-1}$). In this analysis, the risk was calculated relative to the baseline risk in a non-smoking reference population. Finally, the authors fitted the parameters of the BEIR IV model with the exposure–response model using the data for white Colorado Plateau uranium miners. The parameter estimates for time since exposure and attained age were comparable to factors presented by BEIR IV; however, the basic risk factors (b2 in BEIR IV terminology) estimated in the regressions of Colorado Plateau data alone (not correcting for exposure in OHR mining) were an order of magnitude lower than those estimated for the combined cohorts by BEIR IV [C19].

Table 9 Summary of regression analyses by smoking category [S9]

Category	Number of miners	Number of lung cancers	Excess absolute risk			Excess relative risk		
			Slope (PY/WLM) ^a	Intercept (PY) ^a	F-ratio ^b	Slope (WLM ⁻¹)	Intercept	F-ratio ^b
All miners	3359	305	5.0×10^{-6}	-0.18×10^{-3}	3960	2.3×10^{-3}	0.30	619
Non-smokers	493	14	3.1×10^{-6}	-0.61×10^{-3}	139	21.1×10^{-3}	-3.8	203
Ex-smokers	318	38	7.5×10^{-6}	1.1×10^{-3}	45	9.2×10^{-3}	1.2	55

Category	Number of miners	Number of lung cancers	Excess absolute risk			Excess relative risk		
			Slope (PY/WLM) ^a	Intercept (PY ⁻¹) ^a	F-ratio ^b	Slope (WLM ⁻¹)	Intercept	F-ratio ^b
All smokers	2754	282	5.7×10^{-6}	-0.30×10^{-3}	1954	2.2×10^{-3}	0.30	607
Light smokers	501	29	5.2×10^{-6}	-0.35×10^{-3}	63	3.5×10^{-3}	1.3	11
Heavy smokers	1910	215	5.5×10^{-6}	-0.0035×10^{-3}	7882	1.8×10^{-3}	0.29	264

^a PY = person-year.

^b A statistical test to see whether the amount of variation explained by the regression is significant.

Table 10 Summary of regression analyses by smoking category for miners with no OHR mining exposures [S9]

Category	Number of miners	Number of lung cancers	Excess absolute risk			Excess relative risk		
			Slope (PY/WLM) ^a	Intercept (PY ⁻¹) ^a	F-ratio ^b	Slope (WLM ⁻¹)	Intercept	F-ratio ^b
All miners with no OHR mining	1926	152	5.5×10^{-6}	-0.92×10^{-3}	599	2.6×10^{-3}	-0.031	1266
Never smoked	338	9	2.8×10^{-6}	-0.64×10^{-3}	344	20.7×10^{-3}	-4.1	438
Ex-smokers	177	22	8.6×10^{-6}	-1.7×10^{-3}	56	9.7×10^{-3}	3.4	27
Light smokers	318	15	5.1×10^{-6}	-0.80×10^{-3}	108	3.3×10^{-3}	1.1	11
Heavy smokers	1015	99	5.9×10^{-6}	-0.84×10^{-3}	977	2.4×10^{-3}	-0.15	498
All smokers	1528	138	6.6×10^{-6}	-1.3×10^{-3}	310	2.6×10^{-3}	-0.033	1034

^a PY = person-year.

^b A statistical test to see whether the amount of variation explained by the regression is significant.

174. Stram et al. [S60] reported an analysis which used a measurement error correction of lung cancer risk based on fitting a multilevel statistical model to the Colorado Plateau uranium miner cohort data within the same mine, locality and mining district. The authors used two subcohorts from the cohort of 3,347 white miners employed for at least one year in the period 1950–1960 as defined by Roscoe [R8]. The first cohort (referred to as the 1950 cohort) included 2,074 miners with 263 lung cancer deaths who had their initial mining experience commencing in 1950 or later. The second cohort (referred to as the 1952 cohort) included 2,388 miners with 209 lung cancer deaths. The authors noted that the reason for the selection of the 1952 cohort was that systematic measurement of radon in mines did not start until 1952. The approach to (exposure) error correction was based on a computation of the exposures (WLM) for each year and mine of interest. The authors investigated a number of models for lung cancer mortality, including a simple linear ERR model of the form $1 + \beta\chi(t)$, where β is the ERR per 100 WLM and $\chi(t)$ is the miner's cumulative workplace radon exposure up to 2 years prior to the attained age. In addition, the authors investigated several models, including a simplified BEIR VI model, of the effect of smoking, attained age and time since exposure. For

the simple linear risk model, the authors report $\beta = 0.28$ (SE = 0.075), and for the exposure error adjusted model, $\beta = 0.44$ (SE = 0.14), both for the 1950 cohort. Similarly, for the 1952 cohort, the authors report $\beta = 0.33$ (SE = 0.1) using the uncorrected exposure and $\beta = 0.54$ (SE = 0.2) for the exposure error adjusted model. For both cohorts, the error correction increased the risk estimates by about 60%. The authors found a submultiplicative relationship between radon exposure and smoking, both with and without error correction. The authors also observed an exposure-rate effect; this, however, diminished after correction for exposure measurement error. With their simplified BEIR VI model, the effect of low exposure rate (0–15 WL) was essentially the same with and without measurement error correction.

175. Gilliland et al. [G4] reported a study of the exposure to RDPs and lung cancer risk in non-smoking uranium miners. The authors used case-control methodology and conditional logistic regression analysis to investigate the relative risk of death as a function of cumulative exposure to RDPs. Their findings are in close agreement with a parallel analysis of miners reported by Lubin et al. [L10]. The authors concluded that non-smoking miners were indeed at increased risk of developing lung cancer.

176. Luebeck et al. [L16] noted that with their biologically based model and parameters applied to the Colorado Plateau miner cohort, an inverse dose-rate effect was not seen with levels of exposure typical in residences. The ERR was estimated to be about 0.0078 (95% CI: 0.0036, 0.0165) per WLM for 25 years of residential radon exposure at a level of 150 Bq/m³. This is consistent with the value reported from a ratio analysis of eight epidemiological analyses of residential radon exposure [L4]. Finally, Luebeck et al. [L16] noted that the comparable risks in the BEIR VI report, expressed as lifetime risk, were higher than their own estimates by a factor of 2–4.

177. The Navajo of the south-west United States were involved in the mining and milling of uranium ores in the Colorado Plateau area from the 1940s to the 1970s. The 1995 study of Roscoe et al. [R9] updated an earlier study [A4] of mortality among Navajo uranium miners. The 1995 study reported on a cohort of 757 Navajo miners with vital status followed from 1960 to 1990 and a mean cumulative exposure of 755 WLM accumulated over an average of 8.3 years of underground work. The exposures were based on the work of Lundin et al. [L13]. A life table approach based on mortality data for non-white men in New Mexico and Arizona, direct standardization of rates and internal comparisons between the exposure categories were used to analyse the cohort. All exposures were lagged five years, to represent a reasonable minimum period for the induction of lung cancer. A Cox regression analysis was used to account for simultaneous risk factors and the use of external mortality rates. The time-dependent regressors considered in the model included cumulative exposure, log cumulative exposure, duration of exposure, log exposure rate, log cumulative pack-months of smoking, time since first exposure and others.

178. Standardized mortality ratios for a number of causes of death (heart, circulatory and digestive diseases) were lowered. Of the diseases examined, only the values for lung cancer, pneumoconiosis and “other respiratory diseases” were elevated. The mean exposure to RDPs among the 34 deaths observed from lung cancer (versus 10.9 expected) was 1,517 WLM. Smoking status for the miners with lung cancer was similar to that for the entire cohort of Navajo miners. A log-linear model and a linear model in cumulative exposure fitted the data equally well, and yielded ERRs of 13.8 and 9, respectively, for a cumulative exposure of 400 WLM relative to no exposure. Unlike the case of the white miners, smoking was not strongly associated with lung cancer risk in the Navajo cohort. The authors attributed the excess non-malignant respiratory disease (standard mortality ratio of 1.4) to be due mainly to pneumoconiosis and exposure to silica and other workplace contaminants rather than to radon.

179. Latency is an important consideration in evaluating potential lung cancer risk following exposure to RDPs (e.g. [C20, L10, N7, N11]). Langholz et al. [L39] reported on an investigation of methods to assess latency effects and an analysis of latency in Colorado Plateau uranium miners using a nested case–control methodology with 263 lung cancer deaths. Of these, 239 cases were matched to 40 controls

each and the remaining 24 cases with fewer than 40 controls (all controls were used). The relative risk of lung cancer increased for about 8½ years and then decreased, reaching background levels after about 34 years. The decline in risk with increasing time since exposure was much more pronounced in persons over 60 years of age. Hauptmann et al. [H30] reported on the use of splines (piecewise polynomial functions) to analyse latency in the Colorado uranium miner cohort and reported similar results, with ERR > 0 for the period from 9 to 32 years prior to the identification of lung cancer. The ERR reached a maximum of about 0.6 for 100 WLM about 14 years after exposure and decreased to about 0.02 thereafter [H30].

6. Evaluation

180. Estimated exposure rates for individual miners in the Colorado Plateau area have large uncertainties due to: variations between workplaces within mines (even for those mines where RDP concentrations were measured); the necessity to use “guesstimates”, extrapolations or estimates from other mines in the area; uncertainties in OHR mining exposures before (and possibly after) employment at uranium mining facilities; and discrepancies in the work histories of those in the sample group examined. There are significant discrepancies in the work histories of workers who worked underground part-time. Furthermore, the average WL values may not apply to this type of worker, as they may have worked in areas subject to lower ventilation rates than those for the other workers. Reconstruction of exposure histories from company records is probably not feasible, because workers tended to work in numerous mines, for which few or no records are available. However, it should be possible to study a statistically valid sample of miners and from these investigations to draw conclusions concerning the uncertainty associated with the estimates of exposure for the cohort per se.

181. Overall, notwithstanding limitations in the exposure data, the Colorado Plateau cohort of uranium miners is an extremely valuable resource for risk estimation. It provides one of the most substantial bases for risk estimation for groups exposed to RDPs, and the best information on smoking histories. The prominent strengths of this group include: the size of the cohort; the extent of follow-up; the considerable amount of exposure information for the periods of interest (prior to the mid-1960s); information on smoking; and the possibility to assess the effect of OHR mining. Although no systematic bias was identified in the estimates of the exposures for this cohort, the uncertainties in the exposures of individual miners are very large, particularly for the early years of mining.

B. Canada: Ontario uranium miners

1. Introduction

182. Uranium mining in Ontario started in the early to mid-1950s in the Elliot Lake area. Uranium production developed

rapidly to reach a peak during 1957–1960, and declined just as rapidly after 1960. To illustrate this, there were 2 operating uranium mines in 1955, 15 in 1958 and 5 in 1964. The period during which the largest exposures occurred for Ontario uranium miners was relatively short (10 years or so). In total, more than 16,000 men were employed in the Ontario uranium mines at various periods between 1955 and 1977 [M8]. By 1988, only two uranium companies, both in Elliot Lake, remained in operation, and by 1993, only one of these, Stanleigh, remained in operation. It ceased operations in 1996.

183. Muller and co-workers have carried out a number of studies (e.g. [K13, M3, M8, M15, M19, M21, M22, M23, M24, M25, M26]) on some 50,000 men who worked in one or more mines in Ontario. The Ontario miner population was subdivided into gold miners, nickel/copper miners, iron ore miners, uranium miners, other ore miners and a mixture of miners. Men who worked at the Eldorado Mining Company's Port Hope plants or in Eldorado mines in Saskatchewan or the Northwest Territories, as well as those with documented exposure to asbestos, were treated separately. This left a cohort of about 15,000 miners with exposure in Ontario uranium mines. Men were considered to have entered the study at the time of their first medical examination (between 1 January 1955 and 1 January 1978) if they reported having worked for a half-month or longer in dust exposure in an Ontario uranium mine. Miners left the study on 31 December 1981, or at death if this occurred earlier. Miners with uranium mining exposures outside Ontario and asbestos miners were excluded.

2. Radon and radon decay products

184. A certain number of workplace exposure data for operating uranium mines in Ontario became available starting in 1955, although systematic measurements were not started until 1958. According to Ham [H1], the Ontario Department of Mines issued codes in 1957 requiring that various measurements, including measurements of RDPs, be taken in the mines. Thus, however infrequent, measurements were made in each operating mine from that time on. Records of RDP levels for 1954–1955 show that average RDP concentrations ranged from 3 to 7 WL and the average exposures of full-time underground miners from 36 to 84 WLM/a. The levels varied (up and down) over time as new mines developed and new

methods of mining and ventilation were incorporated. Considerable information about exposure conditions in Ontario mines is given in Ham [H1] and in Muller et al. [M19].

185. Ventilation flows changed in Ontario mines over the same time period. McCrodan [M4] reported ventilation rates for two of the Elliot Lake mines (table 11).

186. The Ontario investigators recognized that estimates of RDP concentrations involved some uncertainty, especially for the early years of mining for which relatively few reliable measurements were available. The epidemiological investigators worked together with mine ventilation engineers who were familiar with the Ontario uranium mines over the early years of operation to develop two estimates of RDP concentrations, "standard WL" and "special WL", in an attempt to bracket the uncertainty [M19]. Muller and his co-workers suggested that the standard WL estimate was more representative of a miner's exposure, while the special WL estimate was probably on the high side.

187. In 1985, a reconstruction of early underground uranium mining environments was undertaken [D7]. Mining practices during the late 1950s and early 1960s in the Elliot Lake area were reproduced in reconstructed underground mine areas. Extensive measurements were made during these tests. RDP levels were recorded under a variety of operating conditions. Under continuous ventilation with compressed air (the most favourable condition), exposures during the 1950s were likely to have been not more than double the exposures received by miners today. However, in unventilated areas, the exposure levels could be as much as 10 times higher than in mines today [D7]. While this work reduced the uncertainties associated with the concentrations of RDPs, the uncertainties in ventilation practice and in the times spent in different locations in the mine remained. The 1985 study concluded that a typical raise miner in the 1950s, relative to miners of around 1985, would have been exposed to RDP levels three times higher, about equal levels of thoron decay products and gamma radiation, and much higher levels of uranium and quartz dust. Further information concerning the atmospheres of the Elliot Lake mines and the levels of radon and thoron decay products is given in reference [D8]. The measurements made during the reconstruction also confirmed that the exposure values used for the early years (specifically for raise miners) did not overestimate the working exposures at that time.

Table 11 Ventilation rates at Elliot Lake mines (adapted from reference [M4])

Year	Denison		Quirke I	
	cfm ^c	cfm/ton ^d	cfm ^c	cfm/ton ^d
1957–1958	200 000	33	300 000	54
1960	–	–	300 000	70 ^a
1961	350 000	59	–	–

Year	Denison		Quirke I	
	cfm ^c	cfm/ton ^d	cfm ^c	cfm/ton ^d
1965	450 000	75	–	85
1965	530 000	88		
1965	530 000	150 ^a		
1968	–		250 000	125 ^a
1969	–			
1971	650 000	108	400 000	200 ^b
1975	650 000	108		

^a Reduced tonnage.

^b Mine reopened.

^c Cubic feet (of ventilation air) per minute (1 cfm = 4.72×10^{-4} m³ s⁻¹).

^d Cubic feet (of ventilation air) per minute per short ton (907.19 kg) hoisted to surface.

3. Exposure estimation

188. According to Muller et al. [M19], some 131,000 measurements of RDP concentrations were made over the period 1955–1981, using the Kusnetz method. These data were obtained over a period representing some 141 mine-years of operation, which corresponds to 929 measurements per mine-year of operation. Up to 1977, there were approximately 55,000 measurements representing some 126 mine-years of operation; this corresponds to approximately 430 measurements per mine-year of operation over this period. Expressed another way, in the period 1955–1977, approximately 1.7 measurements were made per average man-year worked, or, on average, one WL measurement for every 9 WLM accumulated by the study cohort.

189. About 23% of the assigned WLM exposure of the cohort was based on extrapolated values of RDP concentrations, particularly during the early years of mining. The mean period of extrapolation was approximately 1.1 years.

The key observations concerning exposure estimations for the Ontario study taken from references [M23, M24] are summarized below:

- 1955: 2 uranium mines; sporadic measurements;
- 1958: 15 uranium mines; systematic measurements, assuming 80% of time spent in working areas (headings, stopes, raises) and 20% in travelways;
- 1955–1981: 131,000 RDP concentration measurements over 141 mine-years of operation (an average of 929 per mine-year, though fewer in the early years); mean extrapolation period (pre-1958) of 1.1 years (representing 23% of the total collective exposure expressed in person-WLM); “standard WL” and “special WL” by mine and calendar year, with “standard WL” considered more representative.

Table 12 shows the distribution of collective exposure based on extrapolated and measured WL values.

Table 12 Exposure (WLM) of Ontario miners based on extrapolated WL values [M23, M24]

Calendar year	Extrapolated standard WLM	Total standard WLM	Per cent WLM based on extrapolation
1954	843	843	100
1955	4 276	4 276	100
1956	20 806	20 806	100
1957	52 263	52 263	100
1958	9 736	87 048	11
1959	10 617	81 733	13
1960	12 902	55 275	23
1961–1977	0	185 079	0
TOTAL	111 443	487 323	23

190. It is not clear how the upper bounds for the exposures of uranium miners from 1955 to 1977 in reference [M19] were generated. Unfortunately, no justifications or explanations were given; however, as noted earlier, the exposures used for the early years were not likely to have been overestimated [D7].

191. The Ontario mining industry experienced frequent changes in market conditions. Mining companies and miners moved from mining one type of ore to mining another, and from one location to another [M19]. According to Muller et al. [M15], the miners were all requested to fill in detailed employment information concerning their first 60 months of mining experience so that those involved in mining other ores could be identified and excluded from the study.

192. For the period 1955–1977, full-time miners were assumed to spend 80% of their working time underground and 20% on travelways. Part-time miners were assumed to spend 50% of their time underground in areas with higher than average concentrations of RDPs. Up to the end of 1967, and in one mine up to 1 April 1968, WLM exposure values for each miner were estimated as $WLM = WL \times WHF \times Months$, where WL is the weighted average of WL measurements in stopes, raises and travelways; WHF is a work history factor introduced to account for overtime or work stoppages; and Months are the total number of actual months worked underground. The Ontario investigators set WHF equal to unity if normal working hours were maintained. The WHF was increased or decreased as appropriate if overtime hours were worked or if work stoppages occurred in a particular calendar year. After 1967, the individual exposure estimates were based on time cards, filled out daily by miners, and on WL measurements made in the particular work locations reported by the miner on his time card.

193. Smoking information on the Ontario uranium miners is very incomplete. The most recent studies of Muller et al. [M23, M24] reported the results of a 3% sample of men born prior to 1939. Smoking information was sought on 226 uranium miners who had no former gold mining experience. Smoking information was also sought on 80 lung cancer deaths that occurred between 1955 and 1981 in the cohort of uranium miners with no former gold mining experience. Of the 80 lung cancer deaths, smoking information was obtained on 73 men; of these, 72 were smokers or former smokers [M22].

194. Studies of lung cancer incidence and mortality in Ontario gold miners reported by Kusiak et al. [K11, K12] suggested that both radon and arsenic might be causative factors in lung cancer. Kusiak et al. [K12] noted that available data indicated that dust concentrations in some gold mining occupations in the 1930s and 1940s were often above 1,000 particles/mL (p/mL), decreasing over time to an average of 400 p/mL by 1959 and 200 p/mL by 1967. Geological data confirmed the “anomalously high arsenic levels where gold is found...” and “that arsenic concentrations...are regionally enriched”. The authors noted, however, that no

excess of lung cancer could be identified in gold miners who began mining gold after 1945 [K12].

195. According to reference [K12], no RDP concentration measurements were made in Ontario gold mines prior to 1961. RDP levels in gold mines were variable. In some mines, average WL values in inactive areas were 0.3 WL, while in other mines, levels were below 0.02 WL.

196. Increased ventilation rates and related practices introduced since the 1950s may have had less effect on ore dust concentrations than on the concentration of RDPs in the mine air. According to Ham, dust levels in Ontario uranium mines decreased by only about a factor of 2 between 1960 and 1975 [H1], while the estimated concentrations of RDPs decreased by about a factor of 5 over the same period [M21]. On the other hand, data reported by DSMA Atcon Ltd. [D7] and by Duport and Edwardson [D10] suggested that the levels of ore dust and/or RDPs in the mine air diminished by comparable amounts over the years in the Ontario mines.

197. In the Elliot Lake mines, thoron decay products also contributed an appreciable radiological exposure; their concentrations, however, were less affected by increased ventilation than the concentrations of RDPs [J9]. Data for two Elliot Lake mines reported by Chambers et al. [C2] showed thoron decay product levels ranging from 0.1 to 0.3 WL, with parallel RDP levels ranging from 0.2 to 0.5 WL. This suggests a ratio of thoron decay product exposure to RDP exposure of about 0.5.

198. A great deal of information is available concerning the working environment in the Elliot Lake mines. Some data were developed by the mining companies for engineering or regulatory purposes, while other data were developed through the various research activities of the Elliot Lake Mining Research Laboratory operated by Energy Mines and Resources Canada, the Atomic Energy Control Board (AECB) of Canada and the mining companies themselves. Data are available on various subjects, including: the effects of using diesel equipment on the characteristics of mine air [B8, K2, K3]; the composition of the mineral dust (70% quantity) [B7, K4]; particle and activity size distributions [B6, B9, D10]; gamma radiation levels [C5, C6]; and arsenic levels in Ontario gold mines [O1]. A 1986 study [S11] examined the potential to use electrostatic precipitation to reduce radioactive aerosols in underground uranium mine atmospheres. This study further summarized available data on dust loadings, particle size distribution and the attached fraction of the RDPs. Overall, detailed information is available to characterize the mine environment for the 1970s onward, but little information is available for earlier times.

4. Epidemiological analyses

199. In the Ontario uranium miner cohort, the average miner had, in 1984, 1½ years of mining experience and a median age of 39 years. Overall, there was an average of

15.1 person-years at risk per man. Muller et al. [M26] reported mean exposures for this group in the range 40–90 WLM.

200. The present cohort consists of men who worked a half-month or longer in an Ontario uranium mine between 1 January 1955 and 31 December 1977 [M19]. The exclusion of those with known asbestos exposure or with exposure in uranium mines other than in Ontario reduced the cohort size to 15,984 men. It was discovered that 66% of these miners had OHR mining experience and that OHR mining, particularly gold mining, increased the risk of lung cancer significantly [M23, M24, M26]. Exclusion of miners with OHR mining experience reduced the cohort size to 5,443 [M19]. The Ontario studies demonstrate the importance of identifying OHR mining experience, since uranium miners with previous gold mining experience exhibited excess risk of lung cancer even at zero exposure from uranium mining. In men who worked as gold miners before becoming underground uranium miners, 92 deaths from lung cancer occurred, compared with the 55.7 expected [M22].

201. In a follow-up analysis of mortality from lung cancer in Ontario uranium miners, Kusiak et al. [K13] re-examined the Ontario uranium miner cohort; mortality follow-up was extended from 1981 to 1986. An association between excess lung cancer and RDP exposure was found in the miner cohort; this was similar to that found in the same cohort with follow-up to 1981. The study found that lung cancer mortality in Ontario uranium miners who also mined gold was related to exposure from both RDPs and arsenic.

202. In a cohort and case-control analysis of Ontario miners, Finkelstein [F13] investigated the presence of silicosis as a risk factor for lung cancer. A cohort of 382 miners with silicosis and 970 controls were developed from the 68,000 workers in the Ontario database. Data were available for 94% of the silicosis cases and for 99% of the controls. In discussing his cohort analysis, Finkelstein noted that there was a significant excess of cancer (mainly lung cancer) in miners with silicosis and that men with normal radiographs had a lower cancer incidence than the Ontario average. Finkelstein used a case-control methodology and logistic regression to assess the risk of cofactors. He found that silicosis is a highly significant risk factor for lung cancer. In an analysis of cumulative risk, Finkelstein calculated a weak association between silicosis and lung cancer with $OR = 1.004$ (95% CI: 0.9967, 1.011), while in his model of the joint effect of cumulative radon exposure and silicosis, he found no association of lung cancer with radon ($OR = 0.995$; 95% CI: 0.986, 1.004) and a strong association with silicosis ($OR = 6.99$; 95% CI: 1.91, 25). BEIR VI [C20] and the UNSCEAR 2000 Report [U2] reviewed the available data on the effects of exposure to silica in underground miners and commented on various studies, including that of Finkelstein [F13]. Overall, there is a range of opinions on the effects of exposure to silica in the mining environment, and some uncertainty remains concerning the influence of silicosis on risk of lung cancer.

203. One of the most important observations from the Ontario miner study was the use of a “time since exposure” effect, where the risk of lung cancer decreased with increasing time since exposure [M22]. NCRP Report No. 78 [N7] previously reported a dose-response model where the excess absolute risk of lung cancer was assumed to be zero before 40 years and then, following an initial latent period of five years, to decline exponentially with time since exposure. The concept of declining risk with increasing time since exposure was subsequently incorporated in the analyses of others, including Lubin et al. [L10], BEIR IV [C19] and BEIR VI [C20].

5. Evaluation

204. The Ontario miner study is a large well-defined study cohort with considerable information available on which to base exposure estimates. Researchers have attempted to assess the effect of uncertainty in exposure through the use of standard and special working levels, which are mainly estimates. While more could be done in quantitatively evaluating the effect on dose response of uncertainty in exposure estimates, the cohort provides one of the highest-quality studies available for radon risk estimation. Future updates will further increase the value of this cohort.

C. Czechoslovak miners

1. Introduction

205. Mining in Jachymov (Joachimsthal), Bohemia (now in the Czech Republic), started at the beginning of the 16th century. Thousands of silver miners were involved. Mining for cobalt, bismuth and arsenic started later in the 16th century. In the middle of the 19th century, uranium was mined for use in the glass and porcelain industry. Between 1909 and 1925, Jachymov was devoted to pitchblende mining in pursuit of ^{226}Ra , with an average annual production of about 26 g of radium [L6].

206. Mining conditions in the early years were poor. The mines were usually damp (especially in the spring when the snow was melting) and cold. Miners had to descend ladders hundreds of metres to their working areas. Natural ventilation was provided by a gallery between all the mines, and, according to Lorenz [L6], it was generally “sufficient”, except in dead-end shafts.

207. Although high death rates among miners in their prime years caused by lung-related diseases were recognized and recorded as early as the 1550s, no detailed studies of this were performed. In 1879, Harting and Hesse (as reported in reference [L6]) became the first to conduct organized investigations on workers at the Schneeberg mines, which are across the border from Bohemia, in Saxony (Germany). They found that 75% of the deaths were caused by malignant growths in the lung, and that the incidence was greater among miners than among masons or carpenters working

in the mines [L6]. From 1869 to 1877, 150 deaths due to “miner’s disease” were identified in a workforce of 650 men. The onset of the disease occurred after about 20 years of work in the mines. According to Lorenz [L6], Harting and Hesse were the first to diagnose miner’s disease as lung cancer but, owing to the high levels of dust in the mines, they assumed that the inhaled arsenic and poor nutrition were the predisposing factors for the disease.

208. A more recent paper by Greenberg and Selikoff [G7] reappraises the data reported by Harting and Hesse in 1879, and of the early efforts to identify the cause of lung disease among the miners and to measure how much dust the miners inhaled. One experiment (as reported in reference [G7]) concluded, “a miner inhaled 0.231 g [sic] of dust in a 7 h shift”. Unfortunately, it is not clear whether the studies of Harting and Hesse led to any improvement in working conditions. The identification of RDPs as the primary causal agent was left to later investigators.

209. More investigations followed. Rostoski and Saupe, in a 1921 study of selected miners and non-miners (507 people in all), identified lung cancer as a common cause of death among miners [L6].

210. Pirchan and Sikl [P5] observed the first case of lung cancer in Czechoslovak radium workers in 1926. In 1929, Lowy [L48] reported two deaths of Jachymov miners from lung cancer. Further study of the issue was commissioned by the Czechoslovak Ministry of Public Works. In 1929–1930, Pirchan and Sikl [P5] examined necropsies in 13 of the 19 miners who died during that period. They stated that lung cancer incidence was highly prevalent in Jachymov miners and suggested that “radium emanation” (radon) was the most probable cause. Lorenz [L6], however, later suggested that genetic susceptibility to lung cancer in miners must be unusually high.

211. Once radioactivity was recognized as a major cause of lung cancer deaths among miners, measures were taken to reduce exposures. Drilling with water wash-out was introduced around 1930 to reduce the amount of airborne radioactive dust [S24].

212. Prior to 1932, the mines were ventilated using natural ventilation alone. Beginning in 1932, this was in some cases reinforced by mechanical ventilation. After the Second World War, mining started again. As early as 1946, the Czechoslovak Ministry of Health started collecting miner mortality data to better understand the hazards of exposure to RDPs. Up until 1952, ventilation continued to be mainly by natural means; however, in the 1950s, artificial ventilation, both general and local, was systematically introduced into all mines. From 1955 onward, all mines were mechanically ventilated. From 1956 onward, auxiliary ventilation was also provided in selected areas where appropriate. In 1954, the Czechoslovak Hygienic Service started undertaking inspections of working conditions in uranium mines. Increased efforts to reduce radioactive contaminants by increased ventilation and tighter safety regulations commenced in 1966 [V1].

213. Working conditions were improved as the knowledge of the risks of lung cancer in miners from exposures to short-lived RDPs increased. According to Vesely and Sada [V1], a dramatic reduction in radon concentrations took place in Czechoslovak mines during the 1950s. Mean cumulative exposures of about 310 WLM were reported for a cohort of miners who started work in 1948–1952. The mean cumulative exposure of miners starting work in the years 1968–1972 was 40 times lower [S24].

2. Radon and radon decay products

214. Pirchan and Sikl [P5] reported three active pits in Jachymov prior to 1930. The quantity of radium emanation (radon) found in the air discharged from the Svornost (Harmony) pit (depth 500 m) was 4 Mache units, from the Werner pit (depth 476 m) 15 Mache units and from the Saxon Nobility pit (depth 120 m) 10 Mache units. (One Mache unit is approximately 10 Bq/L, see Behounek [B3]). Pirchan and Sikl realized that radioactivity levels varied throughout the mine and suggested taking measurements at various locations to assess the exposures to miners [P5]. Lorenz [L6] made a similar observation.

215. Water is a significant source of radon in underground mines. The Czechoslovak mines were no exception. Behounek [B3] reported radon in groundwater at levels of up to 426,000 pCi/L in the Jachymov mining district.

216. Systematic radioactivity measurements were not performed in Jachymov until the late 1940s. From 1949 to 1960, radon concentration measurements were recorded. Hundreds of readings for each uranium mine for each year during this period are available [S18]. The “classical” method was used to measure radon, i.e. measuring the current with an electrometer in an ionization chamber [S20]. The measurement of RDPs did not begin in Jachymov until 1960 [S20]. In 1968, personal exposure records, which took working place and work time into consideration, were established. The records were reported quarterly for each individual [S29].

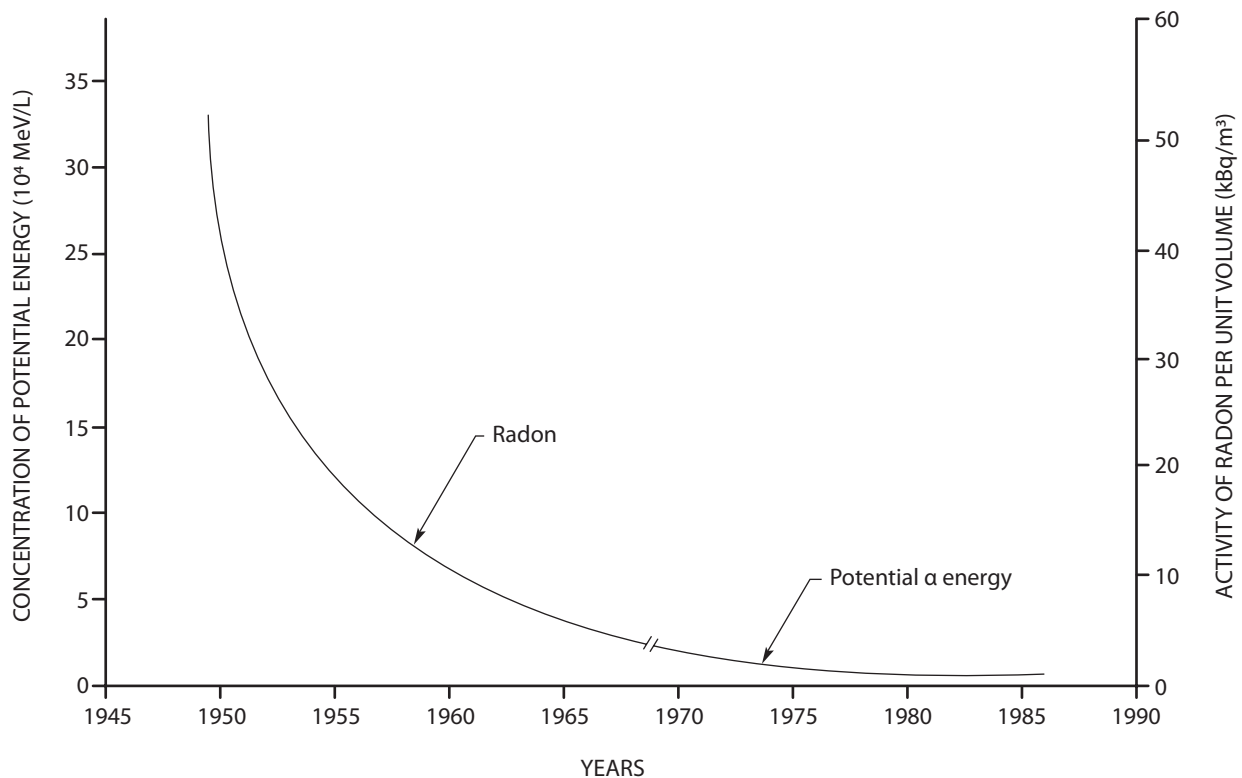
217. Data on attached/unattached fractions are sparse. Some data were collected from 1963 to 1965 by the Czechoslovak Academy of Sciences. These measurements indicated that, typically at that time, the fraction of free unattached radium A (historical name for ^{218}Po) atoms was approximately 10% in the Czechoslovak mines. The AMAD (activity median aerodynamic diameter) was estimated to be between 0.05 and 0.2 μm , with the mean at about 0.1 μm [H2].

218. In 1968 and again in 1973, there were “ventilation incidents” in underground uranium mines in Czechoslovakia [H2]. In both incidents, mechanical ventilation stopped for a period of time. During these periods, radon/radon decay product levels were measured to assess the equilibrium conditions that might have existed in the early days of mining, prior to the introduction of mechanical ventilation. On the basis of data collected during these

two incidents, an overall equilibrium factor of approximately 86% for the period 1948–1952 was estimated. It was noted, however, that in some of the newer mines, the situation was better. From 1953 to 1959, a nominal equilibrium factor of approximately 55% was assumed. For the period 1960–1966, a radon equilibrium factor of 36% was assumed; some of the older mines had higher equilibrium factors and some of the newer mines had lower equilibrium factors.

219. During the period 1948–1952, when the uranium mines in western Bohemia were naturally ventilated, 40% of the measurements indicated radon levels in excess of 1,000 pCi/L. The early high radon levels and the improvements that took place are illustrated in figure V. The break in the curve shown in figure V indicates the change from routine radon measurements to routine RDP measurements as the primary basis for the estimation of exposure.

Figure V. Evolution of the average radon concentrations, 1949–1968 (right ordinate axis), and the average potential (alpha) energy concentration, 1969–1981 (left ordinate axis) [H2, K7].



220. The free unattached fractions of RDPs shown in table 13 were obtained from a series of measurements in uranium mines in Příbram, Czechoslovakia, in 1988 and 1989 [H2].

221. Measurements made more recently in an eastern Slovak iron ore mine with high dust levels (poorly

ventilated) showed low unattached fractions. In one series of measurements, $1.6\% \pm 0.8\%$, and in a second, larger series, $2.3\% \pm 1.3\%$, of (equivalent) RDPs were unattached [H2]. According to investigators, Jáchymov mines were thought to have unattached fractions of 6–10% [H2].

Table 13 Unattached fraction of RDPs in Czechoslovak mines (adapted from reference [H2])

	Range (%)	Mean (%)
^{218}Po	12.0–43.0	27.5
^{214}Pb	4.0–15.7	9.4
^{214}Bi	0.6–10.0	5.3
Overall	5.9–23.0	14.4

3. Exposure estimation

222. Sevc et al. carried out the main Czechoslovak epidemiological studies on uranium miners. The first study was in 1971 [S17] and there have been several updates [K8, K9, S18, S19, S20, S25], the most recent being in 2004 [T40]. The study involved miners who started uranium ore mining between 1948 and 1957. Sevc et al. [S20] reported that “estimates of working levels of RDPs (WL) or radon gas concentrations were made on the basis of records of the ventilation conditions and practices, emanation rates from different types of ores, and RDPs measurements made in 1960 and later” and also “the values for working level months (WLM) were estimated on the basis of radon gas measurements and from data on the number of months of employment with each mine and within each calendar year of the whole employment period for each miner.” No further details were given of how each factor was accounted for. However, the authors estimated the coefficients of variation for WL estimates to be <27%, and <30% for WLM estimates.

223. The average annual RDP concentration (WL) in each shaft was used to calculate each man’s exposure on the basis of his working time. Payroll cards were available for all men in the study groups. Beginning in 1968, individual personal dosimetry cards recorded each miner’s exposure. Discussions with the Czechoslovak investigators indicate that the exposures estimated using time-weighted area measurements are unlikely to differ by more than a factor of 2 from those obtained from personal dosimetry [H2].

224. Radon measurements were obtained for four types of working area: mine workplaces in close proximity to the ore (stopes), where the levels were the second highest in the mine; hallways and corridors where there was no ore; chimneys (raises), where the levels were the highest; and transport ways. Where no data existed on a working area, the average value for the entire mine in a given year was used. Typically, 20% of the total number of measurements was made in transport ways and 80% of the measurements were made in the other working areas.

225. Individual miner exposures were calculated on the basis of job descriptions recorded in personnel cards for all miners (the use of which was begun in 1948 for payroll purposes). The estimates of time spent in the workplace are thought to be reasonable, since the miners were under surveillance by a controller and a mine technician, the latter being responsible for rating the workers’ pay.

226. Very few measurements of the concentrations of radon in the workplace are available for the years before 1948. Estimation of the exposures of miners before this date would require consideration of many factors, including the radon levels recorded in later years and knowledge of early mining practices and ventilation systems. However, according to Hamilton et al. [H2], Group S consists of Czech miners who began mining in the period 1948–1959 and worked for four years or more; it does not include

miners with pre-1948 exposures, and therefore this aspect is not important.

227. The employment history of the miners is another very important factor in estimating exposures. Jachymov is situated in the Erzgebirge (Ore Mountains), where abundant minerals are found. As discussed earlier, there was a long history of mining prior to 1909, when pitchblende was first mined. The early miners probably had previous mining experience (e.g. [L6, P2]). On the other hand, tin mines in the Erzgebirge were not operated between 1931 and 1939, and during the Second World War, the mines employed mainly prisoners [T40]. These facts limit the possibility of previous mining experience among cohort members, and this is consistent with Sevc’s claim that less than 2% of the epidemiological study group mined non-uranium ores before they mined uranium [S24].

228. By definition, a “working month” conventionally now means 170 hours of work. If a miner held more than one job with a mine in the 1920s–1950s, the working hours spent in each would need to be taken into account in order to determine his exposure. Sevc et al. [S20] stated, however, that only the number of months of employment during a year was taken into consideration. There was no evidence that the miners’ actual working patterns were incorporated in the WLM estimations. In discussions held in 1988, the Czechoslovak investigators commented that the workers spent about 80% of their time in the workplace and 20% in the transport ways. In the early days, the men worked 8 hour shifts (3 shifts per day), 6 days per week. After 1968, the normal working week was reduced to 5 days. Uranium miners had a total of 5 weeks of leave per year. By 1990, retirement from uranium mining was mandatory at age 50, but some retired uranium miners often continued to work in other (especially coal) mines. After 1966, people over 40 years of age were not accepted into uranium mining as new miners. Later reports [T37, T38] on the Group S cohort gave more details on how information on jobs was used.

229. On the basis of more recent (preliminary) surveys [T29], Czech investigators estimate the exposure of the general population to RDPs in typical areas of the Czech Republic to be about 0.34 WLM/a, whereas some residents of the Jachymov area could be exposed to 3–4 WLM/a. However, preliminary evaluations suggest that correction for at-home exposure, which would have shifted miners to higher exposure categories, did not affect the risk estimates significantly. The effect of exposure away from work is subject to ongoing investigation.

4. Epidemiological analyses

230. Czech investigators have studied several groups of underground uranium miners. The most studied group is the Group S cohort. These miners represent approximately 11% of the underground miners employed in the Jachymov and Horni Slavkov mines. The cohort originally included

4,364 men [S17]. Reported differences in the size of this cohort are due to whether or not emigrated miners were included. The most recent results for the Group S cohort included 4,320 miners [C20, T37].

231. In discussing the accumulation of WLM exposure, Sevc et al. [S20] indicate that person-years at risk were assigned totally to the final WLM category reached by each individual miner, rather than being distributed across each WLM interval as they accumulated. This affected the estimation of the expected number of lung cancer cases in each exposure category. This difference was discussed by Kunz et al. [K8], who re-evaluated the epidemiological data of reference [S20] and concluded that the distortion caused by earlier methods was not large. Since 1978, all analyses [K8, K9, S25] have used the conventional approach.

232. In earlier papers (1971–1988), the observed numbers of deaths from lung cancer were compared with the numbers expected from general mortality data. This was justified by an investigation of a random group of 700 miners that showed that 70% were smokers, similar to the general male population of Czechoslovakia. However, in publications since 1991, the expected numbers were modified by incorporating a multiplicative parameter that allowed the background mortality to

differ from that of the general population. This approach is close to the “internal approach” if additional stratification for age and calendar year is used [T2, T26].

233. In the past, papers on epidemiological studies of Czech miners did not usually indicate the numbers of miners involved, and therefore it has sometimes been necessary to back-calculate from reported data to obtain the number of miners in the various groups. However, in the first paper [S17] and since 1988 [S25], the numbers of miners were reported.

234. In 1988 [K7, S25], investigations of other study groups were reported (table 14). These include Group S, with subgroups A and B covering the underground uranium miners whose exposure began during 1948–1952 and 1953–1957, respectively. These two subgroups represent the main cohort for epidemiological investigations. In addition, investigations of a number of other study groups are also reported: study Group N, which comprised uranium miners who started exposure at levels lower than those in study Group S; a small study group, Group K, which comprised miners in iron mines in eastern Slovakia; and study Group L, which comprised miners from the Czech shale clay mines. Studies of non-uranium miners were completed with a Czech study of tin miners [T40].

Table 14 Czech and Slovak studies of miners exposed to radon [S25, T40]

<i>Study group</i>	<i>Type of mine</i>	<i>Location</i>	<i>Cohort size</i>	<i>Exposure (WLM)</i>	<i>Latest reference</i>
S* (= A + B)	Uranium	Western Bohemia	4 320	152	[T38]
N* (= C + D)	Uranium	Central Bohemia	5 622	7	[T38]
K	Iron	Eastern Slovakia	1 056	40	[S25]
L	Shale clay	Central Bohemia	916	25	[S25]
C	Tin	Northern Bohemia	2 466	54	[T40]

235. A more recent study by Tomasek and Placek [T2] investigated risks to a subgroup of miners whose exposures were restricted to lower exposure rates. This subcohort had a total of 419 lung cancers to the end of 1995. A decrease in relative risk with time since exposure and age at exposure was observed. Differences in the risk estimates for epidermoid and small cell cancers were also identified, although each had a pattern of risk similar to that of lung cancer overall. The authors found no evidence for non-linearity or dependence on exposure rate (at RDP concentrations of below 8 WL), although the average dose-rate effect was seen in the Group S cohort as a whole.

236. A subsequent study by Tomasek [T26] investigated lung cancer risk in a cohort of 5,002 miners exposed in two different periods. Exposures of the 2,552 miners in the older cohort (S) were derived from workplace radon measurements commencing in 1949. For the 2,450 miners in the newer group of miners (N), exposures were based

on individual dosimetric records. For the newer subcohort (N), smoking data were available for most (about 85%) of the miners. For the older subcohort (S), smoking data were available retrospectively for 279 cases and 410 (nested-in) controls. Follow-up was to the end of 1999. The analysis was based on a relative risk model that allowed consideration of time since exposure and attained age or age at exposure. Excess relative risk (ERR) was linearly dependent on cumulative exposures received more than 5 years previously. The ERR was 0.045 (90% CI: 0.017, 0.140) per WLM among non-smokers (42 cases) and 0.02 (90% CI: 0.011, 0.035) per WLM among smokers (309 cases); the differences between the two estimates were not statistically significant. The lung cancer risk in miners who smoked was about 10.8 times that in non-smoking miners (this included those who had not smoked for the previous 20 years). The ERR was found to decrease by more than 60% per decade of time since exposure and simultaneously by more than 40% per decade of age at exposure.

5. Evaluation

237. In comparison with studies reported in BEIR VI, the S-cohort had (by 1990) the second largest number of lung cancer deaths. Exposure information in the S-cohort is among the most extensive ([C20], p. 322). Extensive measurements of radon in all shafts are available almost from the first years of exposure. In results published in 2003 [T30], only 4% of all exposure years are not based on radon measurements. The most recent results on the Czech uranium miners [T39, T40] are based on a total of 929 lung cancer cases. This combined cohort includes a large proportion of miners with exposures based on detailed personal dosimetric data and provides an opportunity to investigate the role of potential modifiers of effect.

D. Swedish iron ore miners

1. Introduction

238. Iron ore has been mined in Sweden since medieval times. Originally, there was only open-pit mining, but around 1910 underground mining started [E1, E2]. Before 1945, ventilation was entirely natural. According to Axelsson [A11], some mechanical ventilation was developed during the 1940s and 1950s to prevent water freezing underground. Air was warmed by taking it through old shafts before it reached the workplaces. Snihs [S36] commented that in some mines, ventilation air was brought into the mine through crushed rock; this method, although reducing airborne dust and raising the inlet air temperatures, also picked up any radon emitted from the old shafts or crushed rock. In addition, travel time resulted in the ingrowth of RDPs.

239. According to Snihs and Ehdwall [S36, S39], the primary sources of radon in the Swedish iron mines were incoming radon-rich water and, to a lesser extent, radon from radioactive minerals. The uranium content in the waste rocks in the iron mines was of the order of 15–20 ppm [S39]; however, relatively high emanation coefficients (30–40%, measured in accordance with the procedures set out in reference [A20]) for some of the rock were a contributing factor to elevated radon levels in these mines. The radon problem having been identified in about 1968, ventilation in the Swedish mines was gradually improved. The ventilation path was changed to bypass crushed rock and incoming groundwater, thereby leading to reductions in exposure [S37].

240. Snihs [S16] summarized the status of knowledge about RDP levels in Swedish mines around 1972:

“We measured only the radon concentration in many mines to get a rough idea of the radiation problem in the mines. To get the corresponding radon decay product concentration we then applied the factor 0.5. The reason why we measured radon only, is that the mine companies were asked to send samples (in pre-evacuated bottles) by post. By that method we were able to make

the survey in a relatively short time. I agree that the error may be great but it should not be more than $\pm 50\%$, which is acceptable compared to other potential sources of error, even with a very sophisticated method, as local variations, seasonal variations etc. But we have tried to make all necessary corrections for these errors too as far as we know them. The result of that survey is seen in the table.

<i>Radon decay products</i>	<i>Number of mines</i>	<i>Number of workers</i>
<0.1 WL	25	1121
0.1–0.3 WL	13	1740
0.3–1 WL	17	1739
1–3 WL	5	133

This was the situation in 1969 and 1970. My part of the work is to make a “qualified estimate” of the radon decay product exposure during the last 20 years, which will be rather problematic I suppose.”

241. The Malmberget mine, as it now exists, is actually a combination of several mines that initially were separate. The iron ore deposit at Malmberget consists of about 20 distinct, large ore bodies that outcropped to the surface; in these ore bodies several open pits were started. Most of these mines were in line with each other, separated by low-grade iron ore formations.

242. Open-pit mining in the Malmberget mine area first began in about 1890. Even later, when the depth required for mining forced a change from open-pit mining to underground mining (by about 1930 all mines were underground), the bottoms of the mines were still above the general level of the surrounding terrain. The adits could therefore be driven from the side of the mountain to the bottoms of the underground mines, which in turn were connected to the bottoms of the open pits, thereby permitting natural ventilation of the mines. By 1955, the bottom levels of the mines reached below the level of the country surrounding the mountain, and the efficiency of natural ventilation declined as progressively lower levels were developed.

2. Radon and radon decay products

243. Early Swedish mine and miner data are summarized in annex G of the UNSCEAR 1977 Report [U9].

244. According to Snihs [S36], the first radon measurements were made in the early 1950s in the Boliden mine. However, limited knowledge about radon problems in non-uranium mines and a lack of experience in taking measurements delayed the institution of routine radon or RDP measurements. A general awareness that many non-uranium mines had significant radon levels arose in about

1968. This led to radon surveys in which the general procedure was to take 3–12 radon gas samples per mine during both winter and summer periods. Samples were collected in evacuated bottles and sent to the National Institute for Radiation Protection (NIRP) in Stockholm for analysis.

245. Each mine with RDP levels of 0.3 WL or greater was investigated further by NIRP staff. They visited the mine and took RDP samples using the Kusnetz method, as well as many additional radon gas samples. Typically, more measurements of radon gas concentrations than of RDP concentrations were taken. According to reference [S28], the equilibrium between radon and RDPs was found to vary greatly but was typically 50%.

246. To provide a basis for estimating the equilibrium factor, simultaneous measurements of radon and RDP concentrations were taken. Typically, an average equilibrium factor was applied to all working areas in a mine or parts of the mine [S37]. According to Snihs and Ehdwall [S39], the equilibrium factor varied widely, ranging from 0.15 at the air inlet of the mine to nearly 1 at the air outlet. Average equilibrium factors in workplaces were typically between 0.4 and 0.6 [S39]. Axelson [A11], however, felt that a more appropriate typical equilibrium factor was 0.7.

247. Swedish measurements of radon were typically made by the NIRP using 4.8 L conventional propane containers. The containers were evacuated by the NIRP and subsequently opened at places of interest in the mines. After sampling, they were sealed and mailed back to the NIRP for analysis in ionization chambers [S39].

248. Following the first measurements of radon, the mines were divided into zones by radon or RDP level and subsequently checked according to the following frequency:

- Zone 1: <10 pCi/L (<0.1 WL), once every two years;
- Zone 2: 10–30 pCi/L (0.1–0.3 WL), every year;
- Zone 3: 30–100 pCi/L (0.3–1 WL), once every six months.

For areas with levels of greater than 1 WL, measurements were to be taken every three months, according to Snihs and Ehdwall [S37].

249. In 1986, Radford and St. Clair Renard reviewed the history of mining methods and of general ventilation in the mines (reported in reference [S28]). When the mines were first converted from open pits to underground mines, the underground method of extraction was by shrinkage stoping. Small pillars were left between the large shrinkage stopes. After the shrinkage stopes were drawn empty of broken ore, the hanging wall was allowed to cave in and fill the opening. Mining methods evolved from shrinkage stoping, followed by sublevel stoping, and in 1965 by sublevel caving, which allowed even larger quantities of wall rock to cave in. Until 1973, fresh air was drawn through the broken rock left above

the mining areas by these two methods; while the principal source of radon in the Malmberget mine was likely to have been radon-rich mine water, it is likely that additional radon entered the mine air because of this and the method of ventilation.

250. Extensive recirculation of air was widely practised in the Malmberget mine in the 1950s and 1960s. This recirculation could have permitted the buildup of both radon and the equilibrium factor during that period. Changes in mining methods in the mid-1960s made it necessary to introduce diesel equipment, which in turn led to the requirement to improve mechanical ventilation. Overall, Swent and Chambers [S28] concluded that the pre-1969 WL values given by Radford and Renard [R2] were likely to have been underestimated by a factor of above 2, as they did not take into account: the earlier practice of recirculating air in the mine; the lower volumes of air circulated through the mine in earlier decades; periods of stagnant ventilation airflow, which occurred during the years when natural ventilation was the only ventilation method; and the pattern of decline in the incidence of silicosis in later years, confirming the improvement in ventilation.

3. Exposure estimation

251. Snihs [S36] developed his estimate of risk of lung cancer by assuming that RDP levels in Swedish mines measured since 1969 were representative of earlier years. Snihs [S36] made “qualified guesses” for exposures that may have occurred in relation to the observed mortality from lung cancer. Snihs and Ehdwall [S39] provided further discussion of the measurements of radon and RDPs that were started only in the late 1960s in Swedish non-uranium mines. These authors noted that the earliest measurements of radon in mine air at Malmberget were in 1968 but that subsequently extensive measurements of radon and radon decay product concentrations in air were made by the NIRP and the Swedish mining company LKAB. Radford and Renard [R2], noting that the new ventilation system for mines became operational in 1972, stated that the reconstruction of past concentrations depended on the measurements made during the period 1968–1972 and on knowledge of the natural and mechanical ventilation used previously. However, the authors noted that the reconstruction of Malmberget exposure data depended on the “assumption” that ventilation conditions in the mines in 1968–1972 were not greatly different from those in the past [R2].

252. Key features of the exposure estimation for Swedish iron miners in the study of Radford and Renard [R2] were:

- 1930: nominal start of the study;
- 1969: first (NIRP) radon measurements;
- Until 1973: fresh air drawn through broken rock (preheated);
- 1955: mechanical ventilation introduced to replace natural ventilation;

- Until about 1965: some air recirculation occurred;
- 1965: diesel engines introduced into the mines;
- All exposures “guesstimated”.

Radford and Renard [R2] assumed constant exposures prior to 1968. This is unlikely because of the changing ventilation practices (e.g. increased volume and elimination of recirculation) and the introduction of mechanical ventilation prior to the first radon measurements.

253. As discussed above, the data available from measurements of radon and radon decay product concentrations make it possible to estimate relatively well the exposure in mines after 1970; however, few if any measurement data are available from the period before 1969, and early data must be estimated from a combination of later exposure data, reconstruction of measurements and consideration of ventilation practices, radon sources and other factors. A paper by Bergdahl et al. [B45] provides a comprehensive re-evaluation of radon exposures in the Kirunavaara and Malmberget iron ore mines. The re-evaluation suggests that radon levels were higher than in the estimate by Radford and Renard [R2], but not as high as suggested by Swent and Chambers [S28].

254. In the epidemiological study of miners in Malmberget by Radford and Renard, undertaken in 1984, the historical exposure was estimated from data from 1968–1972 [R2]. Also in this study the equilibrium factor F_{eq} was assumed to be 0.7. The same exposure estimate was used for all underground workers except those that worked in Koskullskulle (these workers seldom changed their working place; Koskullskulle was a separate mine that was not a part of LKAB until 1953). The radon exposure (expressed as WLM/a) was estimated in reference [R2] for Malmberget as follows: 1970–1972: 3.2; 1960s: 4.9; 1950s: 6.2; 1940s: 6.9; 1930s: 4.6; 1920s: 4.8; and 1910 and earlier: 4. For Koskullskulle, the authors [R2] estimated the exposure for the period 1920–1969 as approximately 2 WLM/a and for 1970–1972 as approximately 1 WLM/a.

255. Bergdahl et al. [B45] estimated the exposure during the period 1925–1972 to have been constant at between 0.8 and 17 WLM/a, depending on the ore body in which the work took place. In cases where the location of the work was unknown, the exposure was estimated to have been 6 WLM/a. In Koskullskulle, the exposure in the period 1925–1972 was estimated to have been 5 WLM/a, with values before 1925 gradually increasing from the 1910 value of 0.8 WLM/a.

256. The principal differences between the exposure matrix proposed by Bergdahl et al. [B45] and the earlier exposure estimates of Radford and Renard [R2] are that Bergdahl et al. used a lower exposure value for 1970–1972, a higher value primarily during the 1940s and 1950s, and a linear extrapolation back to 1890. Furthermore, Bergdahl et al. [B45] used substantially higher estimates of exposure for Koskullskulle than did Radford and Renard [R2].

4. Epidemiological studies

257. A number of authors reported epidemiological studies of Swedish miners, including references [A6, A7, A8, A9, A11, A13, C7, D1, D2, E1, E2, J11, J12, J13, L2, R1, R2, S21, S22, S36, S38, S39]. However, the basic reference for the study of Malmberget iron miners is the paper by Radford and Renard [R2], which included a description of the basis for the estimates of exposure to RDPs. This was a retrospective study of lung cancer mortality in a group of 1,415 Swedish iron miners. The total cohort represented 24,083 person-years at risk, with an average exposure of approximately 81.4 WLM. The study cohort included men born between 1880 and 1919, who were alive on 1 January 1970, and who had worked for more than a calendar year between 1897 and 1978. Follow-up of these miners was reported for the period 1 January 1951 to 31 December 1976. The authors estimated an ERR of 3.6% WLM⁻¹, and an excess absolute risk of lung cancer of 19 per 10⁶ person-years per working level month. Miners were identified from company and union records of active and pensioned miners, which were available for the years since 1900, as well as from medical records and, in a few cases, from parish records. The Swedish Government gives every person a code at birth; this code is included in all work and hospital records. Every citizen is also required to register in a local parish of the state church. These requirements helped in locating the miners and their records. Also available from company records dating back to 1900 was the total number of man-hours worked underground each year in each section of the mine. However, the exact location of the work within the mine was not available. There is some uncertainty as to work status for miners who started or stopped work or who changed work function midway through the year. Miners were assumed to work 173 hours per month on average in the period 1890–1930; 162 hours per month during the period 1930–1950; and 144 hours per month from 1950 onward.

5. Evaluation

258. There were few radon measurements in Swedish mines and none in Malmberget mines prior to 1969. It was only after this date that investigations were initiated by the National Institute of Radiation Protection, Stockholm. Therefore epidemiological studies of Swedish miners were based on reconstruction of the radon and radon decay product concentrations to determine the exposure of miners. Since underground work began in 1932, investigators undertook this reconstruction for a period of 36 years. During this period, the conditions in the mines would have changed owing to changes in the mining methods and ventilation procedures. Two attempts were made to estimate the exposures [B45, R2]. The estimates obtained in reference [B45] for the Malmberget iron miners were higher than those used in the epidemiological study of Radford and Renard [R2]. However, to date, no epidemiological re-evaluation of this group of miners using the updated exposure data has been published. These updated exposure data represent a great

improvement in the quality of the data available on the exposure of Swedish miners and will provide an opportunity to update epidemiological analyses of Swedish iron miners.

E. Canada: Beaverlodge, Saskatchewan, miners

1. Introduction

259. The uranium mineralization at Beaverlodge in northern Saskatchewan, Canada, was discovered in 1946, and a prospecting mine shaft (the Ace shaft) was started in 1949. By 1951, the company concerned, Eldorado, had identified ore reserves sufficient to proceed with uranium production. By that time, ore bodies to the west of the Ace shaft had also been identified. In 1951, the Fay shaft was started as a production shaft serving the western ore bodies. An underground haulage way also provided access to the Ace ore body. By the 1970s, the mine was over 1.6 km deep and extended more than 5.6 km horizontally. In addition, a number of satellite mines, including two underground mines, were developed. Mining production in the area increased rapidly during the 1950s, but fell off in the 1960s as the demand for uranium declined. Eldorado recruited experienced miners from the area to work at Beaverlodge. The Beaverlodge operations were closed in June 1982. As the Beaverlodge mine developed, all three shafts were connected underground, and two winzes (shafts that do not come to the surface) were also constructed.

260. Mill construction started in 1952, and the first uranium concentrate was produced in early 1953. By this time the Verna ore body to the east of the Ace shaft was identified; work started on the Verna shaft in 1953, with production commencing in 1956.

261. Twelve small satellite mines, most being small open pits, were also developed during the 1960s and 1970s [G9]. Two underground mines, Hab and Dubyna, were also developed, both located several kilometres north and east of the Verna shaft.

262. Bloy [B11] noted that the first mining method used was shrinkage stoping. This was changed to cut and fill, using waste rock and surface sand as the fill material. In turn, this method was replaced by hydraulic tailings fill. The initial ventilation in working headings was mainly by compressed air from the drills, with some surface air being supplied by a 16 inch (~40 cm) metal vent pipe located in the shaft. Because of the lack of knowledge of the hazards of radon and radon decay products, the standards used to control the mine atmosphere conditions were the same as those used in gold mines at the time. This involved the measurement of airborne dust by the konimeter dust sampling method as well as radon measurements. An annual objective of the ventilation programme was to maintain a ventilation rate of 10–25 cubic feet per minute (cfm) of air per square foot² of working face.

263. In the cut and fill operation, ore was removed from the bottom, and mining advanced upward from one level to the next. The sand fraction of the mill tailings was used as backfill in the mine, with miners working off the backfill to remove the next lift. Simpson et al. [S30] investigated this activity as a possible source of radon and concluded that “no positive evidence was found that backfill was a major source of radon in the mine”.

264. In addition to the Beaverlodge mine, Eldorado developed and operated a number of satellite mines. In addition, according to Garbutt [G9], there were a number of other uranium mines operating in the Beaverlodge Lake area of northern Saskatchewan. Eldorado’s policy was to recruit experienced miners wherever possible. Consequently, as other local uranium mines closed and Eldorado remained in production, there were opportunities for miners with non-Eldorado working experience to migrate to the Beaverlodge operation.

265. The other (non-Eldorado) uranium mines in the Beaverlodge area operated for only a few years, and all had been shut down by the late 1960s. Most of these mines were small operations without mechanical ventilation (the exception being the Gunnar mine) and hence there was a potential in these operations for high exposures even in a short time period.

266. Estimation of individual employee exposures did not begin at the Eldorado Beaverlodge uranium mine until 1967. While some measurements of the concentrations of radon and radon decay products were made between 1954 and 1967, these were intended for monitoring ventilation rather than for personal dosimetry. Eldorado began routinely recording individual exposures in 1968, but considered that sufficient workplace data were available to assess individual exposures back to November 1966. The radon and radon decay product measurements from the earlier periods were summarized and utilized to provide exposure rate estimates by occupational grouping and year [F7]. These were point estimates, primarily based on the median concentrations recorded during the year, and were subsequently used in the original epidemiological analysis of Beaverlodge miners [H19].

267. The employee exposures were estimated by merging the annual exposure rates with information from the nominal roll. The nominal roll contained information on age, duration of employment and type of employment for Eldorado employees. The resultant exposure estimates were the basis for many epidemiological studies, including the original cohort analysis [H19], a case–control study investigating the effects of smoking and previous work experience [L17], the analysis of four underground mining cohorts performed by BEIR IV [C19], and the joint analysis of 11 underground miner cohorts in reference [L10] and BEIR VI [C20].

268. On the basis of these exposure estimates, the apparent lung cancer risk observed in the Eldorado Beaverlodge

² 1 cfm per square foot = $5.24 \times 10^{-3} \text{ m}^3 \text{ s}^{-1} \text{ m}^{-2}$.

cohort was substantially higher than the lung cancer risk observed in the Eldorado Port Radium cohort [H18], where the estimated exposure rates were typically much higher than those ostensibly observed for employees who worked at the Beaverlodge mine. Given the substantially different lung cancer risks observed between the two cohorts, and the importance of epidemiology for estimation of risk from RDP exposure, the Atomic Energy Control Board (AECB) of Canada commissioned re-evaluations of the exposure rates at both the Beaverlodge [S12, S14] and the Port Radium uranium mine [S15].

269. Exposure conditions were re-evaluated for underground work areas at Beaverlodge between 1949 and 1968, when individual employee exposure estimates began. The re-evaluation of Eldorado Beaverlodge exposure rates suggested that previous estimates were underestimated by about 50% [S12]. The re-evaluation study noted that exposure rates varied significantly (i.e. by more than a factor of 10) between different areas of the mine. As a result of this workplace variability, substantial uncertainty in individual employee exposures resulted when an average mine-wide estimate was assigned.

270. A detailed investigation of Eldorado Beaverlodge records was conducted to further improve exposure estimates for a case-control group [S12]. Previous mining experience (including experience in gold and other uranium mines) was noted for several of the underground employees; however, these records were largely incomplete, and exposure estimates for this experience were not calculated. Improvements to the Eldorado Beaverlodge exposure estimates involved reviewing stope production records for the presence of individuals from the case-control group. Exposure rates specific to the area and the time where the individuals worked, rather than the mine-wide estimates, were then assigned to the identified individuals. The individual exposures based on specific mine areas were higher by a factor of 2–3 than those based on mine-wide conditions.

2. Radon and radon decay products

271. The first measurements of RDP concentrations were performed in 1954 at Beaverlodge; further measurements were made in 1956 as part of surveys of radiation levels, dust levels and general ventilation conditions [F7]. These initial surveys eventually led to a programme of radon and radon decay product measurements. Data from the 1954 survey indicated that simply turning the compressed air on or off gave rise to substantial changes in workplace concentrations. Whether a miner worked within this envelope of fresh air or in the “unventilated” region outside it was thus a very important factor in estimating his true exposure.

272. Early measurements were for the purpose of providing data for ventilation control. Originally, only the Tsvoglu method was available for RDP measurements. However,

because this method is complicated, most samples taken were analysed for radon only.

273. According to Bloy [B11], a few measurements of RDP concentrations were made in the 1954 survey using the Kusnetz method. Over time, an increasing proportion of the measurements were done in terms of RDPs. By the mid-1960s, the Beaverlodge ventilation department was relying primarily on RDP measurements to assess ventilation conditions in the mine.

274. During the period 1954 to mid-1962, mining engineers also began measuring radon gas concentrations in the Beaverlodge mine as a means to determine the adequacy of mine ventilation, and not necessarily to determine miner exposures to RDPs. Radon concentrations were converted to RDP concentrations by use of equilibrium factors determined in the years 1954, 1956, 1959 and 1961 from the simultaneous measurement of radon and radon decay product concentrations. A large amount of data from the mine operating statements and radiation logbooks was captured in an electronic database by the 1991 SENES analysis [S12].

275. Prior to mid-1962, most of the radiation measurements were for radon. In the years 1954, 1956, 1959 and 1961, as mentioned above, paired measurements were taken where radon decay product and radon concentrations were measured at the same time in the same workplace. The average radon/radon decay product equilibrium factors were calculated from these data. Equilibrium factors for the early years (1954–1956) appear to be generally lower than for later years (1966–1968), and the equilibrium factors for the later years at high RDP concentrations (>2 WL) approached and sometimes exceeded the theoretical maximum value of 1.0. It was expected that the equilibrium factors for later years would be lower because of generally improved ventilation conditions. Radon concentrations without RDP measurements were multiplied by the equilibrium factor to estimate the corresponding RDP concentrations [S12].

276. The total number of radon and radon decay product measurements taken per workplace per year was generally less than 12 and frequently as low as one. The average during the period 1954–1968 was about four measurements per workplace per year.

277. The mine-wide underground estimates of exposure in units of WL produced in the 1991 SENES study [S12] are compared with the previous estimates in figure VI on the basis of various interpretations of exposure data, either WL measurements or WL inferred from radon measurements. The estimates are somewhat higher than those calculated using the medians of available exposure (WL) data, which included the estimates used by Howe et al. [H19], which in turn were based on those determined by Frost [F7]. Figure VII shows that there is considerable variation in WL values throughout the year — in winter, levels are higher than in summer — and in different years.

Figure VI. Comparison of radon decay product concentration estimates with the previous estimates for the Eldorado Beaverlodge mine [S12].

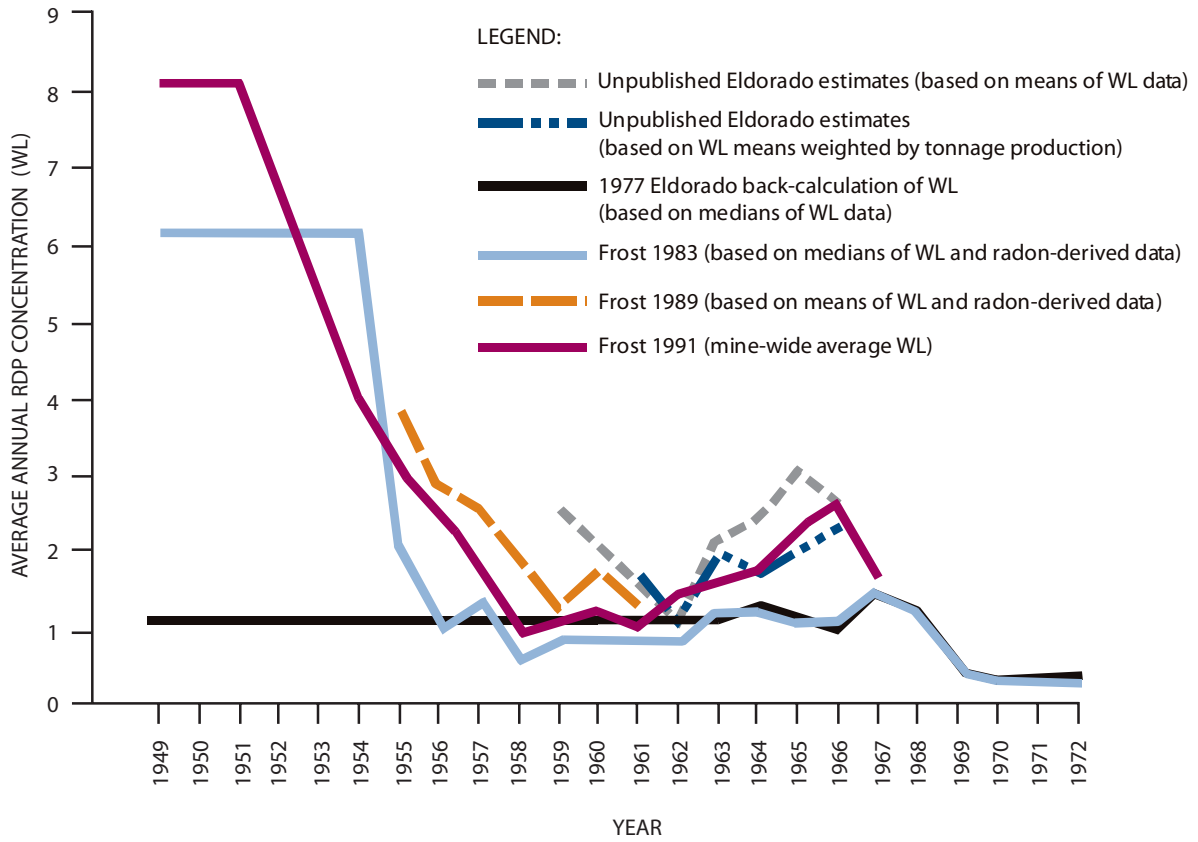
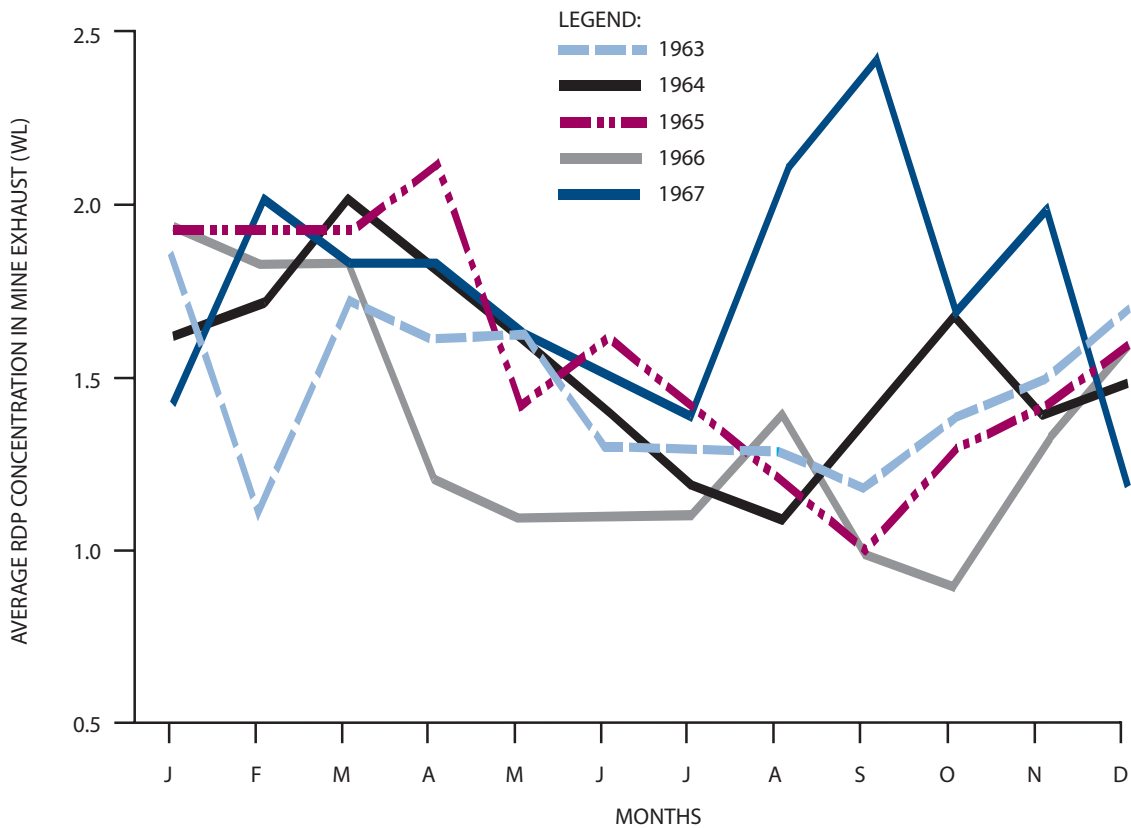


Figure VII. Seasonal variation of radon decay product concentration in Beaverlodge area mines operated by Eldorado [S12].
The high concentration in 1967 coincided with a fan failure.



278. Several factors changed over time at the Eldorado Beaverlodge mine. The calendar years from the start of operation (1949) onward can be subdivided into five periods based on a combination of the production and ventilation characteristics. The period 1949–1953 covered the development of the mine from early shaft sinking to the startup of the mill. The period 1954–1957 was a period of rapid expansion of the Eldorado Beaverlodge mine as well as of many other uranium mines in the Beaverlodge area; mine ventilation increased over the period concurrent with substantial development work. The period 1958–1962 was one of relatively little development at Eldorado, with most of the other area mines closing. The period 1963–1967 saw the development of two areas in the Beaverlodge mine that were not ventilated by flow-through methods; during this time, all other mines in the area were closed. The period from 1968 onward covers the remainder of mine operation, during which the ventilation was increased and individual personal exposure estimates were maintained.

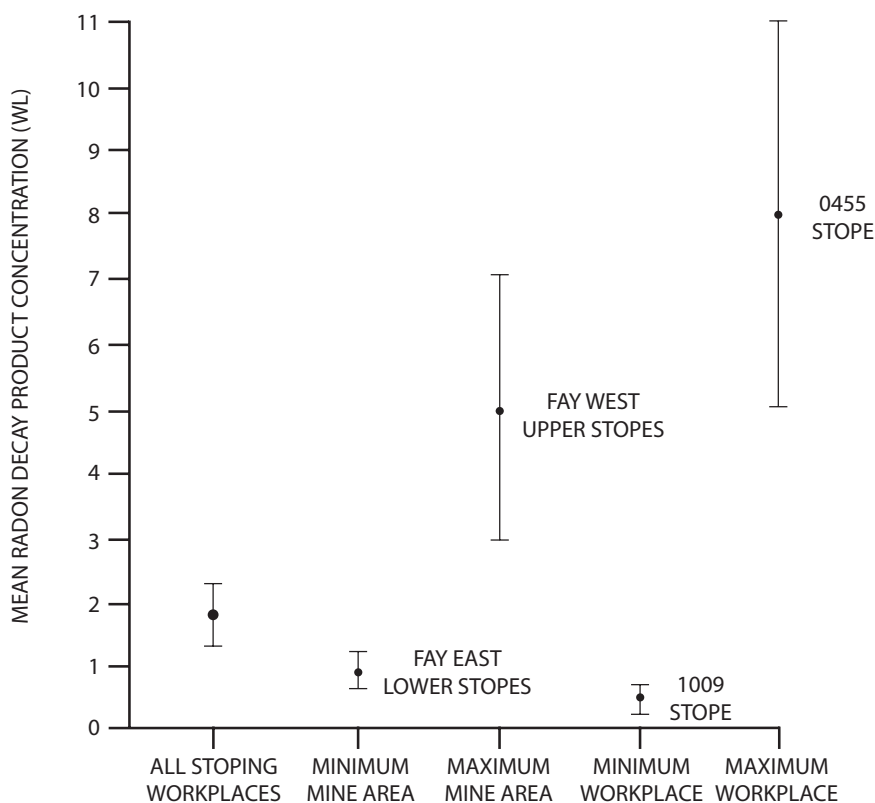
279. The 1991 SENES report [S12] discussed several sources of uncertainty in the WL estimates, including the lack of measurements in some of the early years (pre-1954). For these years, the workplace WL values were assumed to be the same as those estimated for 1954 [S12]. For the years in which no data were recorded, a linear interpolation of the data from the nearest years for which data were available was assumed. Even when measurement data are available, the measurements were focused on detecting deficiencies in ventilation as opposed to evaluating worker exposure.

280. An attempt was made [S12] to correct for these effects by excluding data for stopes and development headings where the mine operating statements did not identify any work activity. This did not mean that work was not going on in those areas. Therefore this procedure underestimated the exposures of radiation technicians and other workers who might, for various reasons, have been in those areas.

281. To correct for this, SENES [S12] weighted the reported WL values by the recorded level of activity as measured in man-shifts. However, there was oversampling in stopes relative to other areas with respect to the number of man-shifts worked. Other problems in assessing exposure were: the reporting, in early measurements, of radon concentrations rather than WL values; the uncertainty about the radon/radon decay product equilibrium factor, which led to uncertainty in the actual WL value; and the considerable variability in data within and among workplaces and with time.

282. Figure VIII illustrates, using data for the Fay area of the mine before 1963, the variability in the workplace data and the effects of agglomeration of the data. Moving from a mine-wide stope average (left side of the figure) to individual work areas, a dramatic increase in the difference between the minimum and maximum values (a factor of greater than 10) is evident. Agglomeration of data reconstruction relied on work history files from Eldorado. It was possible that the work histories of the study cohort were deficient with regard to non-Eldorado employment. This observation is important, because miners

Figure VIII. Estimates of mean radon decay product concentration (WL) by level of aggregation of stoping workplaces, for 1963, Fay area of Beaverlodge mine in northern Saskatchewan [S12].



could have worked in other mines for which no radiation exposure data were available. According to Howe et al. [H19], all exposures prior to 1 November 1966 were recorded as single lifetime totals rather than as separate annual exposures; this makes them less useful for epidemiological purposes. Howe et al. [H19] further indicated that the (annual) median value was used to describe (annual) average WLs.

3. Exposure estimation

283. In 1967, Eldorado began maintaining personal records of RDP exposure for full-shift underground workers. Workers' time cards indicated the hours spent in each workplace. These cards were consolidated into monthly printouts of manpower in each working place. These printouts, together with measurements made in the workplaces and travelways, were used to estimate monthly RDP exposures.

284. In 1970, the computer record system was expanded to include cumulative (i.e. lifetime total) exposure in the exposure summary reports. At this time, records of the previous measurements made for ventilation purposes (as opposed to the measurements made for the purpose of estimating miners' exposures) were used to back-calculate exposures to 1 November 1966.

285. In September 1971, all maintenance, technical, supervisory and other personnel who had received some RDP exposure were added to the exposure roll. Their exposures were calculated back to 1 November 1966 using mine average working levels, hours worked per year, and a factor to account for the portion of time spent underground.

286. In the mid-1970s, work started on the estimation of exposure prior to November 1966. Only RDP measurements were used in this estimate, thus excluding the early survey data and most of the measurements made during the 1950s,

which had been for radon only. Because of the paucity of pre-1964 data, Eldorado used an annual averaging process. Owing to the known variability of workplace conditions, Eldorado decided to use the median, as opposed to arithmetic mean, to describe the central tendency of the exposures. On the basis of a review of work histories for individuals employed prior to November 1966, a system of 22 job categories was devised; classification into a particular category was based on potential RDP exposure. Each person was assigned to a category for each job held throughout his or her employment. An effective RDP concentration (WL) was calculated for each job category for each year based on the fraction of working time spent in each area (underground, office, etc.). These calculations [F7], recorded as single lifetime totals rather than separate annual totals, provided the basis for the exposure estimates in Howe et al. [H18].

287. SENES [S12] used the raw radon and radon decay product survey data from the Beaverlodge mine to assign WL values to individual stopes. Production data were then used to retain only those measurements that were taken when work was in progress in each area. The production data were used further to "weight" the data by the number of man-shifts worked in each area at the measured RDP concentration. The effect of this re-evaluation is shown in figure IX, which illustrates, for the case-control sample of 195 miners, revised exposure estimates plotted against the original exposure estimates. The 45° line corresponds to the situation when the two estimates of exposure are equivalent. Figure IX shows clearly that the majority of employees had revised exposure estimates that are higher than the original estimates. Although a general correlation exists between the two methods of estimating exposure, the revised estimates are substantially higher, up to an order of magnitude for some employees. For example, two employees originally in the 5–49 WLM category were reclassified to the 250+ WLM category. Table 15 shows the extent of movement from one exposure category to another due to the revision of exposure estimates.

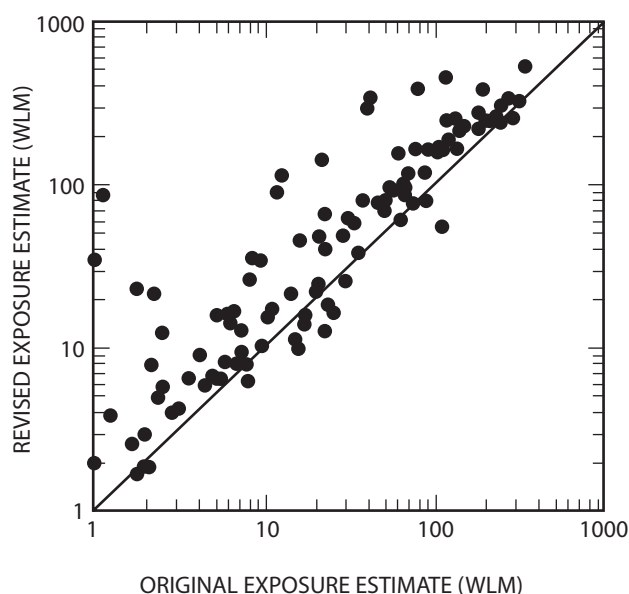
Table 15 Changes in exposure categories of Beaverlodge miners based on revised estimates of cumulative exposures [S12]

Original exposure (WLM) category	Revised exposure (WLM) category							Number of employees
	<5	5–49	50–99	100–149	150–199	200–250	250+	
<5	61 (78)	16 (21)	1 (1)	0 (0)	0 (0)	0 (0)	0 (0)	78
5–49	7 (13)	36 (67)	7 (13)	2 (4)	0 (0)	0 (0)	2 (4)	54
50–99	0 (0)	0 (0)	9 (60)	2 (13)	3 (20)	0 (0)	1 (7)	15
100–149	0 (0)	0 (0)	1 (8)	0 (0)	5 (42)	5 (42)	1 (8)	12
150–199	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (33)	2 (67)	3
200–250	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	4 (80)	1 (20)	5

Original exposure (WLM) category	Revised exposure (WLM) category							Number of employees
	<5	5–49	50–99	100–149	150–199	200–250	250+	
250+	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	5 (100)	5
Number of employees	68	52	18	4	8	10	12	172

Note: Number in brackets is the percentage of employees in the original exposure category that are also in the revised exposure category. Revised exposures were estimated for 172 of the 195 employees in the case-control group. The remainder were not traceable in company records.

Figure IX. Comparison between revised and original estimates of exposure for Beaverlodge uranium miners (note that both axes have logarithmic scales) [C17].



288. The 1991 exposure estimates [S12] did not include non-Beaverlodge mining exposure. Because of the remote location, the cost of recruiting at Beaverlodge was high; consequently, Eldorado's policy was to recruit experienced miners wherever possible. Inclusion of non-Beaverlodge exposure would probably result in further movement of the miners into higher WLM categories.

289. Beaverlodge miners were also exposed to airborne dust. Information on workplace dust levels (konimeter data) that had been recorded coincident with the WL measurements made at Beaverlodge was also entered into the new computer database [S12]. For active stopes, an analysis was conducted to determine if a correlation existed between RDP measurements and dust. The analysis revealed significant correlations between the measured values of dust, the RDP concentration and the calculated equilibrium factor. Dust levels were significantly (99% confidence level) positively correlated with radon and radon decay product concentrations and with equilibrium factors. However, the association between the dust concentration and the equilibrium factor was the strongest of the associations. A correlation coefficient of 0.31 was statistically significant at >99% level. This

suggested an association of higher equilibrium factors with the higher dust levels in stopes with work activity; this seems reasonable, because high dust levels and high equilibrium factors both result from low ventilation rates. For the years where both RDP concentrations and dust levels were captured in the SENES database, the mean dust concentrations by year and type of workplace were calculated. The data indicated high dust concentrations in stopes in 1954 and 1956, with mean concentrations of more than 500 ppcc (particles per cubic centimetre) in stopes. The mean dust concentration was <100 ppcc for the years 1963–1965, and was 133 ppcc in 1966. The highest mean dust concentrations were in the raising workplaces; this is reasonable, because raises are generally more difficult to ventilate during development.

290. SENES [S12] performed a detailed review of WLM exposures for a case-control group defined by Howe. Subsequently, Howe and Stager [H16] carried out an analysis of the case-control group using the revised exposures and observed that the inverse dose-rate effect was no longer present. The revised [S12] methodology for calculation of RDP concentrations was based on estimating annual mean concentrations, taking account of the time spent by an individual employee (employee duration) at specific workplaces. Annual workplace mean RDP concentrations were weighted averages of monthly RDP averages, and the weights were estimates of employee duration based on monthly production statistics.

291. Annual mean RDP concentrations for individual workplaces were agglomerated on the basis of the hierarchy of workplace, mine area and mine-wide estimates. Annual mean concentrations for mine areas were calculated as weighted means of the annual workplace means, where the weights were representative of the estimated employee duration in each workplace. Similarly, the mine-wide annual mean concentrations were weighted means of the mine-area annual mean RDP concentrations.

292. This methodology provided estimated annual mean RDP concentrations for individual workplaces and average mine-wide RDP concentrations. As previously noted, substantial differences in RDP concentration existed between different areas of the mine; this was related both to worker activity and the ventilation infrastructure development in those areas. Even within these mine areas, substantial differences in RDP concentrations existed. Although the method worked well for stoping workplaces, application of the

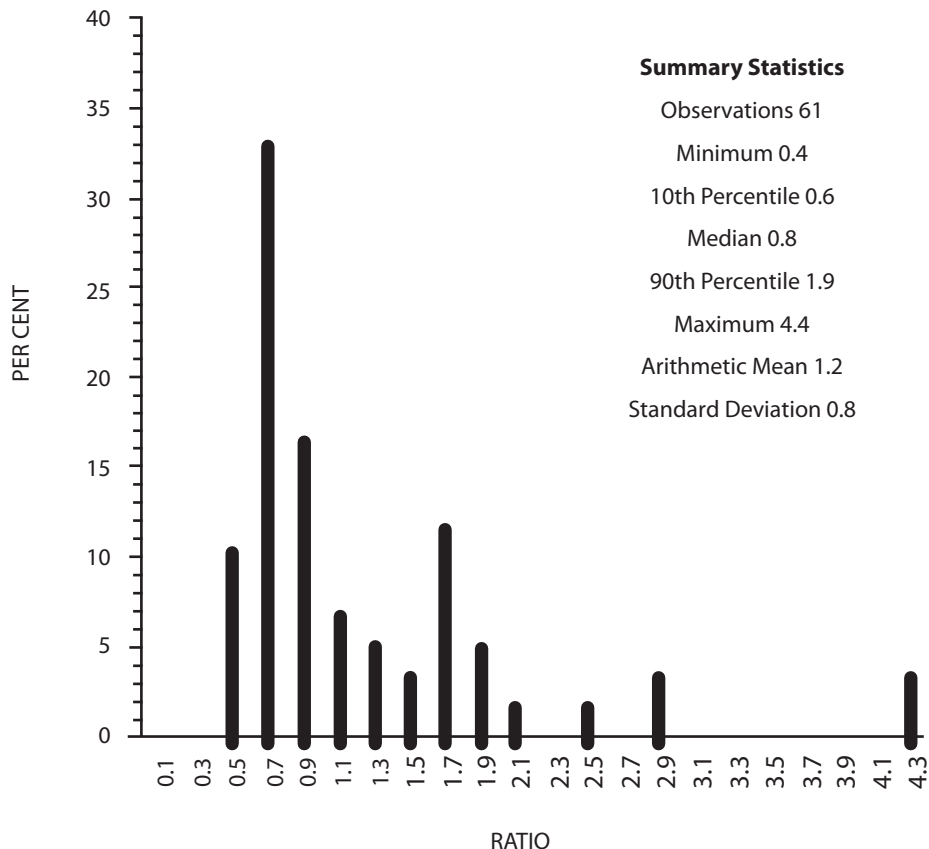
method to drifting, raising, travelways and shaft area workplace categories was not as successful.

293. Available bonus contract data were examined for the case-control group of 195 miners. Bonus contracts were available for the periods 1955–1957, May 1960 and 1963–1965. These records were searched for information on the 195 miners in the case-control group, of whom 129 had underground work experience. For 61 of these employees, information was located on specific workplace, and exposure durations were extracted from the bonus contracts. Total cumulative exposures were calculated for the 191 employees with occupations and exposure durations described in the personnel files. The total RDP exposure over 4,149 worker-months underground was 7,298 WLM for the miners with bonus contract information, based on the use of mine-wide

average concentrations. When mine-area average concentrations were used instead of mine-wide average concentrations only for months with bonus contract information, the estimated exposure to the group was 7,570 WLM. A third estimate of exposure, using the ratio of mine-area to mine-wide average RDP concentrations over all months with bonus contract information, was 8,120 WLM.

294. Figure X shows the distribution for these 61 individual employees of the ratios of exposures calculated by mine-area concentrations to those calculated by mine-wide average concentrations [C17]. This distribution is indicative of the variability in exposure estimates due to RDP concentration differences between the areas in the mine for those underground employees with no bonus contract information.

Figure X. Distribution of ratios of exposures calculated by mine-area concentrations to those calculated by mine-wide average concentrations for Beaverlodge employees for whom bonus contract information was available [C17].



295. An increase in average exposure for the case-control group was seen when mine-area-specific concentrations rather than mine-wide average concentrations were used. This suggested the presence of a bias towards low individual exposure estimates when mine-wide average RDP concentrations were used.

296. For a given cohort, the total exposure calculated by summing the individual exposures that are based on

workplace concentrations and durations should equal the mean exposure calculated using the total exposure duration multiplied by the duration-weighted mine-wide mean concentration, irrespective of the variation or uncertainty in concentrations between workplaces. However, this is not necessarily so for nested case-control groups, where the proportion of cases is higher than in the cohort group. This type of differential uncertainty is likely to be present in many of the other cohorts, since mine-wide average concentrations

were used for estimating the earlier (and generally higher) exposures.

297. Substantial additional RDP exposures, both from non-Beaverlodge work and from other environmental sources (particularly radon in dwellings), along with exposure to other lung cancer risk factors such as arsenic and additional radon exposure that might occur in gold mining, were considered as possible confounders. Of the 195 employees in the case-control study who joined the Eldorado company with recorded previous mining experience, 9 had worked in gold mines and 8 had worked in Beaverlodge area uranium mines. Kusiak et al. [K12] reported RDP levels of the order of 0.3 WL (or greater) in gold mines in Ontario during the 1960s, along with exposure to other risk factors, including exposure to silica and arsenic. The mean duration of recorded mining experience was 6.9 years. It is of interest to note that 5 of the employees selected from the nominal roll, the restrictions on which were to have excluded persons with work experience at other Eldorado facilities, actually had recorded work experience at Eldorado's Port Radium underground uranium mine.

298. Using the revised exposure estimates, Chambers et al. [C17] investigated how uncertainty in exposure might affect the dose-response relationship in the Beaverlodge miners. A file describing a cohort with characteristics similar to that previously studied for determining the dose-response relationship was constructed on the basis of partial information from the nominal roll of Eldorado Beaverlodge employees. This information included an employee identification code and the duration of each occupation for each time period worked at Beaverlodge. Birth year or ages were not available, nor was the vital status of individual employees. Ages at the start of employment were assigned on the basis of random sampling from a uniform distribution of ages between 20 and 40 years. The cohort was similar in size and was assumed to be similar in characteristics to the cohort studied in the epidemiological study [H19]. RDP exposures and doses were assigned to underground employment up to 1967 on the basis of the revised exposure estimates and the algorithm developed for estimating exposures. This provided estimates of nominal, or mine-wide average, annual exposures for each employee plus an estimate of the range of the exposures or variability due to differences in exposure rates between different areas of the mine. Exposures were calculated by multiplying the exposure for a given year by the number of months that the employee worked in that year. Exposures for other occupational groups and for post-1967 time periods were based on previous estimates of exposure rates and an assumed variability between workplaces [C17].

299. Expected and simulated observed numbers of lung cancer deaths were calculated using a life table with reference age-specific total mortality and lung cancer mortality rates. The probability of lung cancer during each year of follow-up was calculated for every employee in the cohort using both the reference lung cancer rates and the lung

cancer rates based on the RDP exposure and the true reference (unexposed) lung cancer rate for the employee. The lung cancer status was assessed for each year of the follow-up on the basis of the probability of lung cancer for that employee during the year, conditional on that employee being alive at the start of the year [C17].

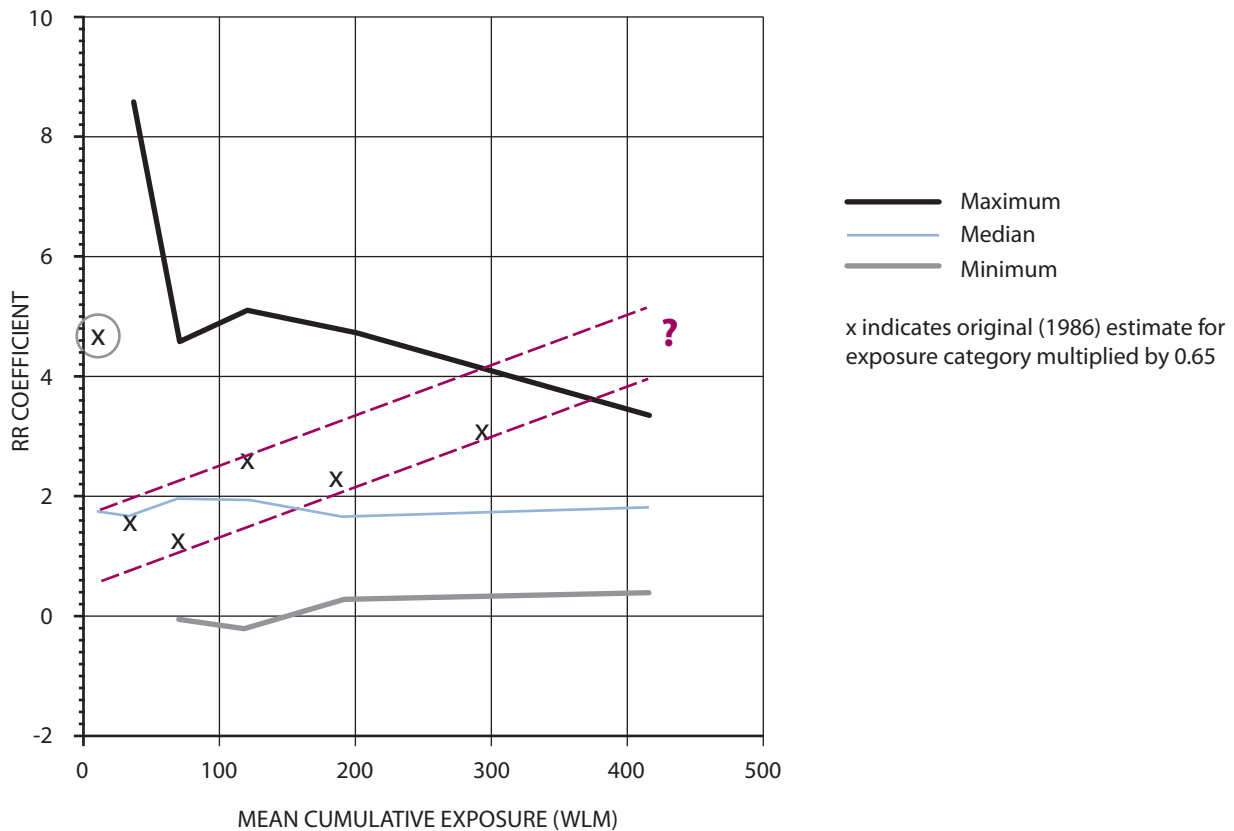
300. The exposure rates for an individual were probabilistically drawn from log-normal distributions that reflected the variability in exposure rate for that occupation and time period of employment. The mean of this distribution was equal to the mine-wide (or occupation) average conditions and was retained as a nominal estimate. The true exposure rate reflected the variation in exposure rates within the mine (or occupation). True reference lung cancer rates were based on the nominal reference lung cancer rate but included a modification based on individual employee variations in this reference rate. These variations could reflect interindividual variability in background rates or varying exposures to other lung cancer risk factors such as may have existed in other types of mine, or variability in the extent of smoking [C17].

301. Employees were assigned to the exposure categories used in the 1986 cohort analysis, and the simulated number of lung cancer deaths was determined. The expected number of lung cancer deaths was determined by summing the annual probabilities of lung cancer death during the follow-up. Relative risks (RRs) for the exposure category were determined by dividing the simulated number of lung cancers by the expected number. Relative risk coefficients were determined by dividing the excess relative risk ($RR - 1$) by the average cumulative exposure in the exposure category. The relative risk estimates and the relative risk coefficients were then summarized by scenario to show the variability in exposure response [C17].

302. Figure XI represents a summary of the simulated dose response based on the simple relative risk model with a risk coefficient of 2 per 100 WLM, no uncertainty in dose and no confounding factors. The three lines show the maximum, median and minimum simulated values of the relative risk coefficient, with the median relative risk coefficient following the 2 per 100 WLM value assumed to be "true" in the simulation. Any observed dose response falling within the two outer lines would be consistent with the assumed model for the scenario. The separation between the outer lines reflects solely the statistical variation in the outcome of a random process, since for this scenario there is no uncertainty in exposure and no confounding factors. For example, between 3 and 7 lung cancer deaths would typically be realized if 5 lung cancer deaths were expected from the risk model. The distance between the bounds is related to the statistical power in that either a large effect or a large study population is required for statistical significance. A mortality update, such as that reported in reference [H35], decreases the distance between the upper and lower confidence bounds, since the predicted number of lung cancers deaths is higher and the relative variability is reduced [C17].

Figure XI. Simulated exposure response for Beaverlodge miners [C17].

Shown are the maximum, median and minimum values of RR per unit exposure, based on a median RR of 2 per 100 WLM.



4. Epidemiological studies

303. The Eldorado epidemiological cohort had 19,370 work records for only 18,424 persons, meaning that up to 946 individuals worked at more than one Eldorado site [N6]. Many of these miners worked at Port Radium, which was Eldorado's first mine; therefore some miners in the Beaverlodge study are likely also to have received exposure while at Port Radium.

304. The mortality study of Ontario miners [M19] had also identified 1,430 former Eldorado employees from Saskatchewan who had worked in an Ontario mine; of this number, 726 had worked in Ontario uranium mines. Consequently, Muller et al. excluded the Eldorado employees from their analysis of Ontario uranium miners because of the lack of exposure data for the time when they worked in the Eldorado mines. Conversely, the Beaverlodge study [H16] included many of these individuals but failed to take account of their non-Eldorado (Ontario) exposure.

305. Howe and Stager [H16] reported on a study of Beaverlodge miners that was part of a larger study of some 18,000 Eldorado employees. The Eldorado epidemiology project has been followed in a series of papers published by Eldorado [A1]. Owing to the size of the study population,

Howe et al. were not able to interview the Eldorado employees. The exposure reconstruction had to rely on work history files from Eldorado. It was therefore possible that the work histories of the study cohort were deficient with regard to non-Eldorado employment. This observation is important, because miners could have worked in other mines for which no radiation exposure data were available.

306. Howe [H35] reports an updated analysis of a cohort of 17,660 individuals known to have worked for Eldorado sometime in the period 1930–1999 [H35]. One of the subcohorts of Eldorado employees includes underground miners employed by Eldorado at the Beaverlodge uranium mine in northern Saskatchewan. The study design for the updated analysis was very similar to that used in the original study [H16] with the addition of workers who had joined the Beaverlodge operation between the cut-off of the original study (31 December 1980) and the final shutdown of the mine in 1982. A considerable effort was made to improve the quality and quantity of the data that were extracted from the nominal roll. This resulted in some deletions and additions for the pre-1980 period [F19]. Exposure estimates and estimates of gamma ray doses were accumulated for the cohort, partly from the original cohort records available at Eldorado supplemented

by records from the Canadian National Dose Registry. Also, work histories and dose records for non-Eldorado uranium mining employment were added. This included miners who worked in Ontario mines as well as a few from Newfoundland. Thus, if a Beaverlodge miner had exposure from RDPs from work at other locations (i.e. Port Radium mine, Port Hope uranium processing plant, non-Eldorado mines around Uranium City, Ontario mines and

Newfoundland fluorspar mines), this exposure was added to the miner's total exposure. Exposures received up until 1999, if a miner continued to work, were also included. The updated study adds a further 19 years of mortality data for the Eldorado cohort and also includes lung cancer incidence results for 31 years, i.e. 1969–1999 [H35]. The basic characteristics of the updated cohort, including the Beaverlodge uranium miners, are summarized in table 16.

Table 16 Basic characteristics of the updated Eldorado cohort [H35]

<i>Characteristics</i>		<i>Number</i>	<i>Per cent</i>
Sex	Males	16 236	91.9
	Females	1 424	8.1
Site	Port Hope	3 003	17.0
	Port Radium	3 300	18.7
	Beaverlodge	10 050	56.9
	Other sites	1 307	7.4
Birth year	1900	414	2.3
	1901–1910	1 028	5.8
	1911–1920	1 803	10.2
	1921–1930	4 030	22.8
	1931–1940	3 913	22.2
	1941–1950	2 790	15.8
	1951–1960	3 118	17.7
	1960+	564	3.2
<i>Cohort/subcohort</i>			
		<i>Mean RDP exposure (WLM)</i>	<i>Standard deviation</i>
Entire cohort		48.0	182.6
Port Hope		12.5	43.4
Port Radium		174.2	369.1
Beaverlodge		23.2	81.7
Other Eldorado sites		1.9	32.9

307. Two general types of comparison were used in the analysis of the Eldorado cohort data. Firstly, observed and expected values were used to estimate standardized mortality ratios (SMRs) and standardized incidence ratios (SIRs). Expected values were derived from Canadian national population rates for mortality between 1950 and 1999 and for cancer incidence between 1969 and 1999. A second series of comparisons were based upon internal comparisons between subgroups within the cohort, i.e. with no reference to an external population.

308. The analysis of mortality rates showed that, while mortality from lung cancer was elevated, the cohort as a whole and the various subcohorts had reduced risks relative to the Canadian population for most of the other causes of death. The analysis of mortality from lung cancer among men in the cohort with respect to RDP exposure was based on 639 lung cancer deaths. (This compares with previous analyses of the Eldorado cohort where the total number of such deaths was 122.) For the Beaverlodge underground miner subcohort, there were 198 lung cancers observed compared with

120.7 expected. The SMR for lung cancer was estimated at 1.6 (95% CI: 1, 1.9) and was statistically significant ($p < 0.001$).

309. Comparisons of the cancer incidence rates between 1969 and 1999 for the cohort with those for the general Canadian population showed that the only cancer which is consistently elevated is lung cancer. For cancer as a whole and for specific cancers, incidence rates for the cohort were generally lower than those for the general population, which was considered to be a manifestation of the healthy worker effect. Howe also investigated mortality and cancer incidence for diseases other than lung cancer and found no evidence of any causal relationship between exposure to RDPs or gamma exposure and an increased risk of any other diseases [H35].

310. The application of the BEIR VI type of risk model, which allows for effect modification with time since

exposure, exposure rate and age at risk, was also investigated. Using the same approach as the BEIR VI Committee [C20], Howe estimated parameters from the present study for a “full interaction model”, which accounts for the influence of age at exposure, dose, dose rate and time since exposure (see table 17). The BEIR VI Committee’s analysis was based on 11 studies of underground miners, including the previous analysis of the Port Radium and Beaverlodge cohorts. In this study, Howe found that the addition of both the time since exposure terms and the six exposure categories resulted in a statistically significant improvement in fit, but that the addition of age at risk terms did not. He suggested that these results may be regarded as essentially independent of the data used by the BEIR VI Committee [H35]. Howe’s 95% confidence limits on estimates of time since exposure (WLM5, WLM15 and WLM25) parameters include the BEIR VI estimates.

Table 17 Parameter estimates for full interaction model and comparison with BEIR VI model estimates for males in the Eldorado cohort (1950–1999) [H35]

<i>Parameter</i>	<i>Estimate</i>	<i>95% lower limit</i>	<i>95% upper limit</i>	<i>Estimate for BEIR VI</i>
WLM 5	5.23	1.33	14.52	7.68
WLM 15	2.5	0.63	7.05	5.99
WLM 25	1.37	0.36	3.99	3.92
Rate(1)	1			1
Rate(2)	1.02	0.39	2.67	0.49
Rate(3)	0.49	0.2	1.21	0.37
Rate(4)	0.35	0.12	1.01	0.32
Rate(5)	0.33	0.13	0.84	0.17
Rate(6)	0.16	0.06	0.44	0.11
Age(1)	1			1
Age(2)	1.94	0.77	4.89	0.57
Age(3)	1	0.37	2.72	0.29
Age(4)	0.05	0	6266.67	0.09

Parameters as specified below:

WLM = total WLM (per 100 WLM) lagged by 5 years

WLM 5 = WLM 5–14 years previously (per 100 WLM)

WLM 15 = WLM 15–24 years previously (per 100 WLM)

WLM 25 = WLM 25 years+ previously (per 100 WLM)

Rate (2) = WL 0.5–1.0

Rate (3) = WL 1.0–3.0

Rate (4) = WL 3.0–5.0

Rate (5) = WL 5.0–15.0

Rate (6) = WL 15+

Age (2) = age at risk 55–64

Age (3) = age at risk 65–74

Age (4) = age at risk 75+

5. Evaluation

311. The Eldorado Beaverlodge cohort was updated with the revised dosimetry [S12, S14] to add a further 19 years of mortality data and to include cancer incidence results for 31 years, i.e. 1969–1999 [H35]. The study design for the updated analysis was very similar to that used in the original study [H16]. The nominal roll was that used in the original study with the addition of workers who had joined the Beaverlodge operation between the cut-off of the original study (31 December 1980) and the final shutdown of the mine in 1982. The updated analysis found little evidence of departure from a simple linear exposure–response model.

312. For the Beaverlodge cohort, an ERR of 0.96 (95% CI: 0.56, 1.563; $p < 0.001$) per 100 WLM was reported [H35]. This can be compared with the previous Beaverlodge estimate of ERR of 3.25 per 100 WLM [H16]. The estimate of ERR for the Beaverlodge cohort has decreased substantially in the new analysis. Howe suggests that this could in part be accounted for by the time-dependent effect modifiers on the ageing Beaverlodge cohort [H35]. Extensive work was undertaken in this update to add the exposures received in non-Beaverlodge mines to those received in the Beaverlodge mines. Thus the decreased ERR could also be partly explained by the addition of these non-Beaverlodge mine exposures.

F. Germany: Wismut miners

1. Introduction

313. The Erzgebirge (Ore Mountains) of Saxony (Germany) and Bohemia (Czech Republic) have a long history of underground mining. As early as the 12th century, silver mining was performed, while later other metals, such as iron, bismuth, cobalt, nickel and tungsten, were mined. The mining of uranium started at the beginning of the 19th century in the Schneeberg area. Miners often died of what was called Schneeberger lung disease, named after the town in the Erzgebirge. By the end of the 19th century, this disease was recognized as lung cancer.

314. Shortly after the Second World War, the Wismut mining company carried out uranium mining in Saxony and Thuringia in the former German Democratic Republic (GDR). According to Jacobi and Roth [J3, J4], large-scale uranium mining started in the Erzgebirge of Saxony in 1946 and later was extended to the eastern parts of Thuringia. The Wismut mines in Saxony were high in arsenic, while the Thuringia mines were low in arsenic. Overall, Wismut produced about 220,000 tons of uranium between 1946 and 1990, making it the world's third largest producer of uranium [K15]. Mining for uranium per se ended in 1990 following the reunification of Germany.

315. Various papers discuss the potential exposure conditions in the uranium mines in the GDR [E3, E4, K5, K15,

K16, L3]. Enderle and Friedrich [E4] characterized the workplace situation in the post-war years (to 1955) as compulsory labour, use of prisoners of war (almost 50% in 1947) and a high rate of illness and accidents. In August 1953, a treaty was signed to convert Wismut from a Soviet enterprise to a Soviet–GDR company, and this resulted in improvements in the working conditions [E4]. From 1946 to about 1955, the underground mine conditions in the Erzgebirge of Saxony were characterized by dry drilling, no mechanical ventilation, very heavy manual work, the absence of industrial health and safety standards, and very long working hours.

316. According to Jacobi and Roth [J3, J4], three time periods can be distinguished: the years 1946–1954, which were referred to as the “wild years”; 1955–1970, during which time, there was ongoing improvement in the conditions through the introduction of wet drilling and improved ventilation; and the period after 1970, when individual exposures were recorded and compared with ICRP limits. Some 20,000 cases of silicosis and 7,000 cases of lung cancer are reported among the Wismut miners. Jacobi and Roth [J4] noted that radiation exposures in the years preceding 1970 can only be “roughly” estimated. For 1955, on the basis of the assumptions of a mean radon concentration of 120 kBq/m³ for drilling and ore exploration areas and 50 kBq/m³ for other worksites, and the nominal time spent in workplaces, a nominal workplace value of about 80 kBq/m³ was estimated. On the basis of a nominal equilibrium factor of 0.5, workplace exposure was estimated at 160 WLM/a. Prior to 1960, the concentrations of long-lived alpha activity in the air could have been higher by a factor of 100 to 1,000 than in post-1960 conditions. Finally, the mean external dose to miners in the last 10–20 years of mining were about 5 mSv/a.

2. Radon and radon decay products

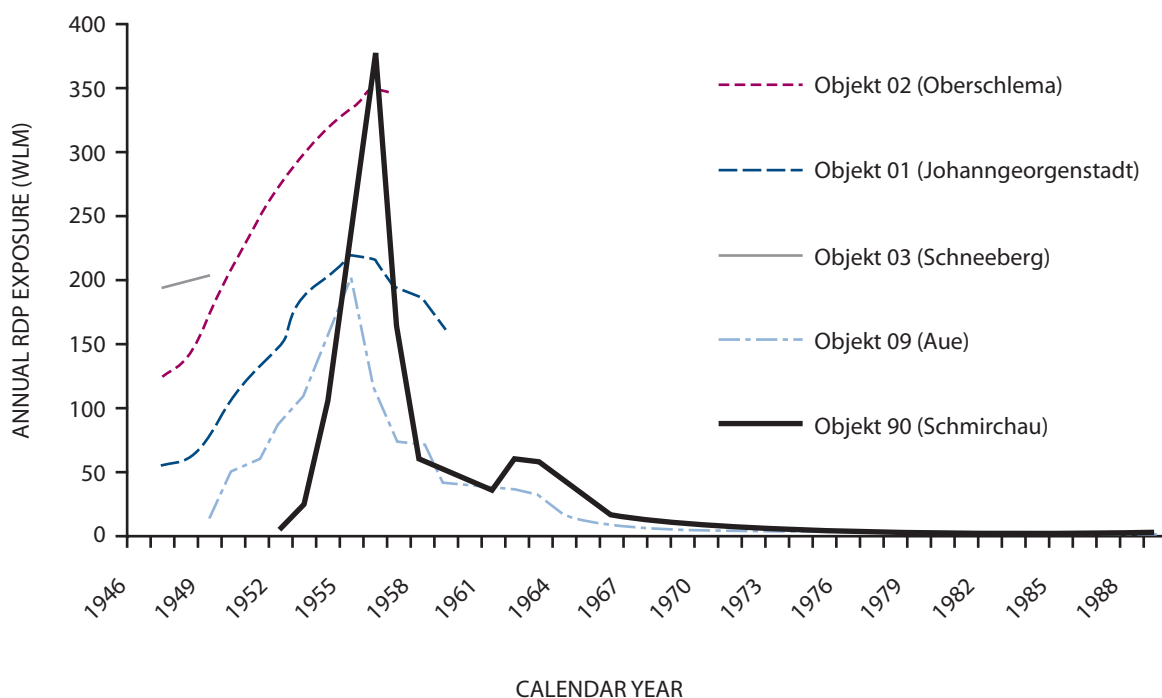
317. The first radon measurements in workplaces in the Wismut mines were carried out in 1955 [J3]. The mean of more than 2,000 measurements carried out in five mining operations was 110 kBq/m³, which according to Jacobi [J3] showed good agreement with measurements carried out in 1937 and 1938 in the Schneeberg mines. This author [J3], on the basis of 1955 data, reported a mean annual miner exposure of 150 WLM, with a range of 30–300 WLM. (The conversion from Bq/m³ to WLM depends on the equilibrium factor F_{eq} . Assuming an F_{eq} of approximately 0.4, exposure to a radon concentration of 110,000 Bq/m³ for 2,000 h corresponds, very roughly, to an annual exposure of 150 WLM.)

318. As reported by Lehmann [L3], from 1946 to 1955 there was no dosimetric recording of radon by the Wismut company. In 1955, radon gas monitoring commenced. Measurement of radon and its decay products was introduced in 1966 in Saxony and in 1975 in Thuringia. Formerly, for purposes of compensation, an average annual exposure to radon decay products of 150 WLM was assumed by the Wismut company for underground workers for the period 1946–1954 [B5]. This was used independently of conditions and led to

both over- and underestimation of individual exposures to RDPs in the early years. A working group of experts was convened in 1993 to develop a job–exposure matrix (JEM) in order to improve the estimation of exposure. The results of their work were published in 1998 [L3]. Radon concentrations for 1946–1954 were estimated retrospectively on the basis of the first available radon measurements in 1955. These estimates took into account previous working conditions in the mines, mine architecture, historical measurements and data gathered by the Czech ore mining industry. Based on these estimates and the measurements available

since 1955, the annual exposures to radon, RDPs, long-lived radionuclides and external gamma radiation were evaluated for each year of employment between 1946 and 1989, each mining facility and each place of work (underground, milling or processing, open-pit mining or surface mining). This evaluation (see figure XII) was performed for a reference job for each place of work (there were fewer reference jobs for underground than for above-ground workers), while the exposures received in other types of job (of which there were more than 200) were derived using weighting factors for the specific reference job (ranging from 1.0 to 0.0).

Figure XII. Annual exposure to radon decay products as estimated using the job–exposure matrix for the job of “hewer”, in five mining facilities typical for the Wismut cohort [K16].



319. The improved estimates of the JEM showed that RDP exposures in the early years depended strongly on the number of old shafts in a given mine and on mining activity. In newly established mining facilities, such as Objekt 09 (Aue) and Objekt 90 (Schmirchau), radon values in the very first years of operation were rather low, while in old reopened mining facilities, such as Objekt 02 (Oberschlema), Objekt 03 (Schneeberg) and Objekt 01 (Johanngeorgenstadt), radon concentrations were already high at the beginning of the operating period. Generally the levels of radon increased with uranium mining and the area of the worked vein to a maximum in 1955–1956, and decreased later owing to the introduction of different ventilation and sealing measures.

320. The JEM [L3] provided estimates of exposures not only to radon and its decay products but also to long-lived radionuclides and gamma radiation. Owing to improved working conditions, the exposure to radon and its decay products decreased, while gamma exposures still remained

relatively high. A JEM for arsenic, fine dust and quartz is being developed on the basis of estimates given in reference [B22].

3. Epidemiological studies

321. Several epidemiological studies of radiation exposures among the Wismut miners are under way. These include a large cohort study [K5], a nested case–control study on lung cancer mortality [T11] and a cohort study among the offspring of miners [T11], among others [G17, K15, K16, K29, K30, K31]. There is also an independent case–control study on the incidence of lung cancer [B23, B24, B39]. About 400,000 people worked for Wismut between 1946 and 1990. For about 130,000 of them, complete working histories, including start and end of work, job specification and places of work (with dates) are available. From these, a stratified random sample of about 64,311 people was drawn [G17,

K15, K16, K30, K31]. In order to reflect the different mining conditions in the Wismut company mines, the sample was stratified by the date of first employment (1946–1954, 1955–1970, 1971–1989), place of work and area of mining. Criteria for inclusion in the cohort study were as follows: (a) minimum duration of employment of at least 6 months; (b) date of first employment between 1946 and 1989; (c) year of birth after 1899; and (d) male. After collection and subsequent

evaluation of the occupational data, which were extracted from the original payrolls, a total of 5,150 individuals were excluded from the initial cohort, because they did not meet the criteria. The final cohort for the analysis thus consisted of 59,001 men [G17, K30, K31]. On the basis of year of first employment, three subcohorts were defined to reflect the different mining conditions: 1946–1954 (subcohort A), 1955–1970 (subcohort B) and 1971–1989 (subcohort C).

Table 18 Characteristics of the Wismut cohort study [G17, K30, K31]

<i>Characteristics</i>	<i>Number of cohort members</i>	<i>Per cent</i>
Year of start of employment	59 001	100.0
1946–1954	23 917	40.5
1955–1969	17 950	30.5
1970–1989	17 134	29.0
Year of end of employment		
1946–1954	2 720	4.6
1955–1974	19 593	33.2
1975–1984	12 963	21.9
1985+	23 725	40.2
Vital status as of 31 December 1998		
Alive	39 255	66.5
Deceased	16 598	28.1
Cause of death available	14 646	88.2
Cause of death not available	1 952	11.8
Lost to follow-up	3 148	5.3
Duration of follow-up in years		
<10	3 764	6.4
10–19	11 225	19.0
20–29	12 536	21.2
30–39	12 704	21.5
40+	18 772	31.8
Year of death		
<1960	224	1.3
1960–1969	1 255	7.6
1970–1979	3 132	18.9
1980–1989	5 368	32.3
1990–1998	6 619	39.9

<i>Characteristics</i>	<i>Number of cohort members</i>	<i>Per cent</i>
Cause of death ^a		
Malignant cancers (C00–C99)	4 800	32.8
Circulatory diseases (I00–I99)	5 417	37.0
Respiratory diseases (J00–J99)	1 559	10.6
Digestive system (K00–K99)	815	5.6
Injuries and poisoning (S00–S99 and T00–T99)	1 284	8.8
Others	771	5.3
Exposed to radon		
Never	8 244	14.0
Ever	50 707	86.0
Cumulative exposure to radon in WLM		
Mean (maximum)	241 (3 244)	
Median	18	

^a Codes from the International Classification of Diseases.

322. In the first mortality follow-up, the vital status for the cohort was determined as at 31 December 1998. At that time, 66.5% were alive, 28.1% had died and follow-up was not complete for 5.3%. The mean age of subjects alive at the end of 1998 was 54 years for the total cohort and 71, 59 and 40 years in subcohorts A, B and C, respectively. A total of 2,388 lung cancer deaths occurred in the first follow-up period (1946–1998), which comprised 1,801,626 person-years. The general characteristics of the Wismut cohort are summarized in table 18 [K15, K16].

323. Data on smoking habits were available for about a third of the total cohort. This proportion was considerably lower for subcohorts A (20%) and B (33%) than for subcohort C (64%), reflecting the fact that smoking habits were recorded only after 1970. More than 50% of the miners with known smoking habits were heavy smokers, while the proportion of non-smokers was about 26% [K16].

324. Within the cohort study, a nested case–control study on lung cancer deaths was conducted that included individuals born after 1927. Two controls per case were matched by date of birth. Controls could be either alive or deceased. Questionnaires were sent either to next of kin or, in the case of the controls, to the miners themselves, if they were still alive. Information was gathered on smoking habits and occupational exposures outside the Wismut facilities, which might be related to lung cancer. Next, data were abstracted from the Wismut health archives in relation to smoking habits, medical radiation exposures and jobs prior to Wismut employment [G14, T11].

325. From the cohort, a subsample of 6,000 miners was drawn as the basis for an offspring cohort study. The lifetime exposure of the 6,000 miners to radon and radon decay products varied between 0 and >3,000 WLM. The first stage

of the study, which is in progress, was to identify the children of miners to be included in the study and to investigate their health status, life expectancy and causes of death. The most important outcome variables are genetic anomalies, infant mortality and childhood cancers. The offspring cohort consists of 7,855 children.

326. Another case–control study on lung cancer incidence among former Wismut employees was conducted between 1991 and 2001 [B23, B24, B39]. Patients with histologically confirmed primary lung tumours were recruited from several study hospitals in Thuringia and Saxony. Controls were randomly selected from the personnel files of the Wismut company and were frequency-matched to the cases according to birth year in 5-year groups. Inclusion criteria for cases and controls were: male workers; employed underground at the Wismut Company at some time between 1947 and 1990. Occupational exposure to radon, its decay products, gamma radiation and long-lived alpha emitters was estimated by using the JEM described in reference [L3]. All subjects were personally interviewed about occupational and smoking history. Lung cancer risk was calculated by using the ERR model and conditional logistic regression. A total of 505 cases and 1,073 controls were included in the study. The cumulative exposure from RDPs ranged from 1 to 2,911 WLM (an average of 552 WLM for the cases and 420 WLM for the controls). The exposure rate ranged from 0.1 to 31.4 WL (an average of 8.2 WL for the cases and 7.2 WL for the controls). The odds ratios (OR; adjusted for smoking, year of birth and asbestos exposure) in the two highest categories, compared with the reference category of 50 WLM, were significantly increased: ≤ 800 –1,599 WLM, OR = 2.08 (95% CI: 1.40, 3.08); and 1,600–2,911 WLM, OR = 3.68 (95% CI: 1.92, 7.03). More than half of the study subjects had been exposed more than 35 years earlier. Assuming a linear exposure–response relationship, there was a significant increase in the

relative risk of 0.10 (95% CI: 0.05, 0.17) per 100 WLM after adjusting for smoking and asbestos exposure. After correcting, in a sensitivity analysis, for the fact that the controls of this study had a higher average exposure than the population of Wismut workers from which they had been recruited, the ERR increased to 0.24 per 100 WLM. For those still smoking, the increase in relative risk was lower (0.05 per 100 WLM), whereas it was higher (0.20 per 100 WLM) among non-smokers and long-time ex-smokers. Lung cancer risk declined with time since exposure, except for those miners who had been exposed 45 or more years in the past. No inverse dose-rate effect was observed.

327. More recently, the autopsy data in the archives of the Central Institute of Pathology of the Wismut Company were analysed for 19,271 persons; these included 12,926 uranium miners (WLM > 0, group 3), 1987 control cases of non-exposed Wismut workers (WLM = 0, group 2) and 4,358 control cases most likely never employed by Wismut (group 1) [W17]. The mean age at the start of exposure was 33.5 years, mean duration of exposure 11.8 years and mean age at death 62.7 years. The autopsy data investigated comprised about 152,300 person-years of uranium mining work with radon exposure. Mean RDP exposure was about 725 WLM, with a maximum of more than 3,000 WLM. Mean exposure to long-lived radionuclides (LRN) was about 7.3 kBq h m⁻³ (²³⁸U), with a maximum of more than 50 kBq h m⁻³. On the basis of the main cause of death or concurrent diseases, 8,882 cases of malignant tumours were found (6,403 for group 3, 889 for group 2 and 1,581 for group 1). For primary malignant lung tumours, a higher incidence was found for exposed Wismut workers (4,526 (35%) in group 3, 377 (19%) in group 2 and 2,472 (11%) in group 1). For primary malignant lung tumours, a significant correlation between OR and WLM category was shown. Depending on the chosen category, the OR for groups with high exposure increased to 10. In categories of high LRN exposure, the OR increased to 8.4 (95% CI: 6.19, 11.56). Below the 400–599 WLM category, no significant difference of OR related to the leading histomorphological tumour types could be determined. In higher WLM categories, small cell carcinomas and squamous cell carcinomas showed an OR nearly twice as high as that of adenocarcinomas. For non-exposed Wismut workers, 6% of non-smokers and about 19% of smokers showed lung tumours, and for exposed workers, 18% of non-smokers and 34% of smokers had developed lung tumours. The relative share of small cell carcinomas and squamous cell carcinomas was higher for smokers than for non-smokers. In turn, the relative share of adenocarcinoma was lower in smokers than in non-smokers. According to autopsy diagnosis, the relative share of silicosis cases increased with the WLM category. This mirrored, at least for the mining sites in Saxony, the parallel exposures to quartz-containing dust and ionizing radiation. The available data gave no indication of a causal connection between silicosis as the cause of death and primary malignant lung tumours for the Wismut workers. Molecular biological investigations revealed no evidence for repeated mutations of codon 249 of the *p53* tumour suppressor gene in the Wismut workers, as had

been shown for uranium workers of the Colorado Plateau in reference [T1].

328. Pathological findings on 243 Wismut uranium miners with lung cancer, recruited between 1991 and 1995 into the case-control study on lung cancer incidence [B24], are reported elsewhere [K14]. The frequencies of all tumour cell types were found to be associated with increasing RDP exposure, but high radiation exposures tended to increase the relative proportion of small cell lung cancers and squamous cell carcinomas [K14]. This effect was more pronounced among those who had stopped smoking or had never smoked, and it seemed to be masked among those still smoking [K14]. The first evaluation of the pathology archive of the Wismut company showed a shift from small cell lung carcinomas as the predominant cell type in the first follow-up years to squamous cell carcinomas in the later years [W4]. At present, 5,215 lung carcinoma cases have been identified among former Wismut employees, showing extremely high proportions of small cell lung carcinomas (69%) in the early years (1957–1965), which declined to 34% up to 1990 [W4].

4. Evaluation

329. A number of reports have presented information on the predicted numbers of lung cancers in former Wismut miners [B5, J3, J4]. On the basis of a sample of 3,654 persons drawn from the Wismut database, which resulted in a final data set consisting of 2,282 men, and using 1985 death tables for the German Democratic Republic and various risk projection models, one analysis [B5] predicted a further 1,700 to 4,800 additional lung cancer cases from 1995 onward. The peak incidence was predicted to occur between 1985 and 1991. Another study on pathological findings among Wismut uranium miners was published in 2006 [T50].

330. The main strengths of the Wismut cohort study are its size, a wide range of exposure levels, a long duration of exposure and a large number of cases of lung cancer and other diseases, as well as the availability of information on dust and arsenic exposure. A joint analysis of 11 miner cohort studies [L10] was based on a total of 60,606 exposed miners, including 2,674 with lung cancer, and a mean radon exposure of 164 WLM. Data were combined from different cohorts of miners around the world. Heterogeneity with respect to the quality of exposure assessment, the presence of relevant covariates such as arsenic, dust and tobacco smoke, as well as lifestyle and genetic factors, was likely to be present in this combination of cohorts. The Wismut cohort provides a data set for analysis that is similar in size (59,001 miners and 2,388 lung cancer cases) to the data set used in the combined analysis, yet it is more homogeneous with respect to data collection and estimation of exposure. Therefore it represents a unique opportunity to verify the results of the combined analysis in an independent data set. The potential limitations of the Wismut cohort are the limited information on smoking and the limited validity of the exposure assessment, particularly in the years before 1955.

331. A number of reports have already presented data on the feasibility of epidemiological studies of Wismut miners and have provided some early results [K5, K15, K16, T11]. Recent observations suggest a linear smoking-adjusted ERR of about 0.10 (95% CI: 0.05, 0.17) per 100 WLM [B24, B39]. As noted in reference [T11], the results of future epidemiological studies will depend greatly on the quality of the exposure assessment and the information on vital status and causes of death.

G. Canada: Port Radium miner study

1. Introduction

332. The Eldorado Port Radium mine has a long history extending back to 1930, when a prospector by the name of Gilbert LaBine identified pitchblende on the north shore of the Great Bear Lake in Canada's Northwest Territories. Open-pit mining started shortly thereafter, and the first pitchblende was shipped in 1931. Underground operations were at a high level of activity by 1932. Mining continued until 1940, when a decreasing demand for uranium ore led to the mine being shut down. In 1942, the Port Radium mine was reopened at the government's request, and it continued in production until 1960, when it was shut down. Annual average ore grades in the 1940s were 0.5–1.2%, with occasional pockets of high-grade pitchblende being encountered.

333. Bloy [B11] noted that ventilation in the Port Radium mine was somewhat unusual. At the start of underground work, the mine had only natural ventilation; this was greatly reduced in winter, as all openings to the surface were kept closed because of the extremely cold temperatures. Openings to the surface other than the shaft, such as raises from stopes through the surface pillar, were covered and tightly sealed in the winter. The only air entering the mine, in winter was compressed air used to power the drills [T3]. Thompkins conducted a ventilation survey at the Port Radium mine in 1945 [T4]. There was only a relatively small amount of air circulating through the mine. This condition, together with there being no definite routing of the air currents and the lack of dust-reducing features on mine machinery, resulted in generally high dust concentrations in the mine. As part of the 1945 ventilation survey, Thompkins [T4] estimated the balance of the air entering and leaving the Port Radium mine by natural means. Airflow from the surface entering through the 921 Raise and the 722 Raise was estimated at 455 and 1,500 cfm,³ respectively. Compressed air entering the mine was estimated to be 1,400 cfm. Therefore the total volume of air entering the mine was approximately 3,350 cfm. The volumes of air leaving through the shaft and the manway/pipe raise were estimated as 2,350 and 850 cfm, respectively, for a total of 3,200 cfm. Only 150 cfm of the air that entered the mine remained unaccounted for [T4].

334. By 1944, the need for heated ventilation at the Port Radium mine was recognized. A ventilation unit and steam

plant for heating air going into the mine were installed in 1946 and were in operation by early 1947.

335. By 1956, ore zones at the Port Radium mine had reached a stage where it was necessary to relocate the mine surface ventilation unit and steam heating unit to accommodate active workings. Owing to the apparent short life expectancy of the mine, driving a complex ventilation raise system was deemed impractical. As an alternative, old manways and workings were used to bring the air to active areas. Air was forced into the mine down 136 Raise into 136 Stope and down to the first level through the stope raises. From there, the air was channelled to the lower levels through a series of old raises. The air on the levels was controlled by a series of vent doors [B14]; there were, however, substantial air losses between the first and fifth levels as a result of air escaping through old stopes filled with broken material. For instance, at the first level, a loss of 7,000 cfm was noted. Bloy [B14] concluded that it was virtually impossible to adequately ventilate the winze section with the existing set-up, since air volumes were substantially reduced and any air reaching the area was highly contaminated.

336. In 1956 and 1957, the main fan on the surface was stepped up from 22,000 cfm to 35,000 cfm. Heating facilities were increased to handle the extra airflow in winter. By then, a definite air route was established underground. However, when the installation was completed and the air volume checked, only 6,000 cfm of the approximately 35,000 cfm of air put into the mine actually reached the active workings, and this was contaminated with radon and dust. The rest of the air escaped through leaking doors, bulkheads and coarse backfill. To solve this problem, the airways were lined with two types of plastic: polyethylene plastic sheeting of about 4 mil thickness (0.1 mm) and a liquid spray plastic called cocoon [B12].

337. The plastic sheeting lined the raises from the first level to the surface fan inlet. Although the sheeting worked very well, it was difficult to handle and tore easily. By the time these raises were completed, the liquid spray plastic method (cocoon) was being tried, and the results were so encouraging that the system was used for all remaining airways [B12].

338. An auxiliary fan delivering approximately 11,000 cfm was located on the 11th level to ventilate the winze area, thus supplying this area with much better ventilation than had been found in the 1956 survey. A raise system was also completed in the winze area, which greatly improved the ventilation [B13]. In addition to the cocoon method, three additional auxiliary fans were added in the winter of 1957–1958, resulting in a significant lowering of radon concentrations by the spring of 1958 [F5]. No ventilation data for the periods after 1958 were found in the records of Cameco, the later owner, or the literature review.

339. Kupsch [K6] provided a detailed history of the Eldorado company, including its Port Radium mine. Additional interesting information on the history of the Port Radium mine was provided by McNiven [M6]. He reviewed the

³ 1 cfm = 4.72 × 10⁻⁴ m³ s⁻¹.

operations at Port Radium from the reopening of the mine in 1942 to its final shutdown in September 1960.

2. Radon and radon decay products

340. Port Radium was the first mine in Canada where radon and radon decay product sampling was performed. According to Bloy [B11], the first radon samples at this mine were taken in February 1945. The reason for the study was to investigate the “suffocating gas” noticed by the miners. (The suffocating gas turned out to be simply oxygen deficiency.) Radon levels were found to range from 13,000 to 47,000 pCi/L.

341. Representatives from the Chalk River Nuclear Laboratories visited the Beaverlodge and Port Radium operations and carried out preliminary surveys for radon in 1951 and 1952 [S15, S30]. The surveys established that both Beaverlodge and Port Radium had serious radon problems. By 1954, Chalk River Nuclear Laboratories had designed and built new radon sampling equipment which they used in another survey of the Port Radium mine [B11].

342. It was the practice in those years to perform konimeter dust sampling from time to time in parallel with the radon and radon decay product sampling. In addition, an external gamma radiation survey of the Port Radium mine in 1952 showed gamma exposure levels in a few areas of 95 mR (approximately 0.95 mSv) per shift [B11].

343. The radon and radon decay product data were discussed by Frost [F6]. Initially, only radon was measured. By the mid-1950s, both radon and radon decay product concentrations were measured (the Kusnetz method was used for the latter). The equilibrium factor between radon and its decay products was found to be approximately 30% in 1957. Frost [F5, F6] concluded that the equilibrium factor was likely to have been higher in the early years of mining when there was no forced ventilation.

344. Average RDP concentrations were estimated by Frost [F5] to be of the order of 77 WL in 1945, 38 WL in 1952, 10 WL in 1956 and 8 WL in 1957, on the basis of the available measurements and assuming an equilibrium factor of 30%. Unfortunately, these data and the corresponding estimates of miner exposure have not been published.

345. In a 1996 re-evaluation [S15] of RDP concentrations, radiation measurements recorded by Eldorado staff were compared with the duration of employee exposure for each radiation measurement. All but a few of the radiation measurements were solely for radon concentrations, as the three paired measurements of radon and radon decay product concentrations in 1957 did not provide enough information to estimate equilibrium factors. Thus a modelled equilibrium factor based on the ventilation rate was used to convert radon levels to WL RDP concentrations.

346. WL estimates for individual workplaces were aggregated as a function of the workplace classifications to which the 171 employees selected for this study were assigned by Howe [S15]. At each level of aggregation, efforts were made to provide a consistent estimator of WL. Since there were no individual measurements of RDP concentrations, individual WLM exposures could not be calculated directly. An estimate of the exposures of individual employees was based on the mine-wide average.

347. The duration-weighted arithmetic mean was chosen in this analysis as the estimator to characterize RDP concentrations. This statistic facilitated the calculation of a mine-wide average for a variety of employee classifications. This mine-wide average (mine index) provided a WL value that would be the expected WL concentration over all the individuals in the classification. From a preliminary review of the data for Port Radium provided by Cameco, the amount of downcast ventilation into the mine varied substantially (by about a factor of 2) by season of the year, especially from 1947–1957. This was because air needed to be heated in the winter to prevent the upper portion of the shafts from freezing. The limited capacity of the heating plant required the ventilation volume to be reduced during the colder winter months (mid-December to mid-March) to avoid freezing the workings.

348. On the basis of the ventilation characteristics, the entire operational period of the mine was divided into three subperiods: pre-1947, 1947–1955 and post-1955. Table 19 summarizes the available radon data for shaft stations. Similar data are available for active stopes and other underground workplaces. A large winter/summer difference is evident.

Table 19 Radon concentration in shaft stations of the Port Radium mine [S15]

<i>Period</i>	<i>Season</i>	<i>Radon concentration (pCi/L)^a</i>
Pre-1947	Summer	8318 (6)
	Winter	8318 ^b
1947–1955	Summer	2084 (10)
	Winter	4760 (6)
Post-1955	Summer	3434 (16)
	Winter	593 (4)

^a Arithmetic mean of measured radon levels with number of observations given in round brackets; 1 pCi/L = 37 Bq/m³

^b Indicates an estimated value, since measurements were not available.

3. Exposure estimation

349. Information provided by Howe allowed a comparison of exposure duration and exposure (WLM) for a 171-member case-control group. There were significant differences in the estimates of months worked between the 1996 re-evaluation and the original epidemiological study [H15]. Estimates of total WLM for the case-control group as a whole are not significantly different between the two approaches. However, as for exposure duration, individual differences in exposure (WLM) could be very large. To assess the implications of this re-evaluation for the epidemiological study, it would be necessary to look at the differences in the individual miner exposures between the two studies. It is not known from the evaluation whether the cases follow the general pattern or if a difference exists between the cases and the controls (which was the expectation of the authors).

4. Epidemiological studies

350. Since early miners had a potential excess risk of lung cancer, Eldorado sponsored a pilot epidemiological study of Port Radium workers. This study [G5] found an excess of lung cancers in miners who had 5 years or more of underground experience.

351. Consequently, Eldorado initiated a more detailed epidemiological study [A1] that included the radiation exposure data, and that involved Statistics Canada and the National Cancer Institute of Canada, which performed the actual epidemiological analyses.

352. This study by Howe et al. [H18] investigated some 2,103 miners employed between 1942 and 1960. In this group, 57 lung cancer deaths were observed compared with 24.73 expected. Employment records were not available before 1940 and hence exposures before that date were not estimated. As a consequence, the exposures of the Port Radium miners may well have been underestimated. Risk coefficients estimated from the Port Radium analysis should therefore be regarded as upper limits [H18].

353. Radon gas samples were collected for seven of the years between 1945 and 1958, with between 9 and 71 samples per year and a total of 251 samples. The range of concentrations was reported as 50–300,000 pCi/L. Howe et al. [H18] cite Frost [F5] as the source of their RDP exposure data. The Port Radium study, unlike the Beaverlodge study by the same investigators, in at least some circumstances, used the annual average rather than the median as representative of workplace RDP levels. While radon gas samples were made as early as 1945, early data are sparse and the uncertainties in the exposures are likely to have been very large. Howe et al. [H18] indicated that weighted average equilibrium factors were calculated on the basis of the known labour distribution and type of workplace. The highest factor used for many work groups was 0.5, although it was discovered that there could have been substantially

higher values. Working time was based on a 40-hour week and 48 weeks per year. Howe et al. [H19] acknowledged that there were many potential sources of error in the procedure for estimating RDP exposure.

354. The average exposure of the 2,103 miners was 183.3 WLM. About 42% of the person-years at risk were in the <5 WLM exposure category. The ERR was estimated to be 0.27 per 100 WLM and the excess absolute risk was estimated to be 3.1 cases per 10⁶ person-years per working level month [H18].

355. The ERR per 100 WLM for Port Radium from the updated study [H35] is essentially unchanged from the previous assessment [H18], whereas the ERR for Beaverlodge has decreased substantially. Howe suggests that this could be accounted for in part by the early exposures of Port Radium workers, for whom the time-dependent effect modifiers (time since exposure and age at risk) may be of less importance than for the younger Beaverlodge subcohort [H35].

356. More recently, the Eldorado cohort was updated to add a further 19 years of mortality data (1950–1999) and to include cancer incidence results for 31 years (i.e. 1969–1999) [H35]. The study design for the updated analysis was very similar to that of the original study [H18]. The updated estimate of the ERR for Port Radium miners was 0.37 (95% CI: 0.23, –0.56; $p < 0.0001$) per 100 WLM, which may be compared with the ERR of 0.27 per 100 WLM from the 1980 mortality analysis [H18]. As noted previously, the current estimate of ERR for Beaverlodge is 0.96 per 100 WLM [H35], compared with the previous estimate of 3.25 per 100 WLM [H16].

5. Evaluation

357. During the early years of mining (pre-1947), WL values in the Port Radium mine were of the order of 60, and annual exposures of miners were likely to have been of the order of 600–1,000 WLM. By the end of mining (around 1959), the calculated WL values declined to 2–3, and hence annual exposures were likely to have been of the order of 20–40 WLM. The estimates have large uncertainties, perhaps a factor of 10, for both pre-mechanical-ventilation and post-mechanical-ventilation periods [M5]. Few data are available for their estimation, and the quality of the sparse data available is suspect.

358. Exposure estimates were particularly uncertain for a number of work types where the proportion of time spent underground was unknown. This included mechanics and electricians who may have worked underground for extended periods. The uncertainty in risk estimates if these employees are included in an epidemiological analysis could be substantial, and exclusion of these workers should be considered.

359. In addition to the sparsity and limitations of radiation and ventilation data for the early years, there exist other

recognized sources of uncertainty. Based on the sample of 171 miners provided by Howe, many pre-Port-Radium employment histories are incomplete. Many Port Radium miners are likely to have worked in other mining environments (notably gold mining), which provided a further possible risk factor. Epidemiology for Port Radium miners only included miners' work after 1941, as work records prior to that time were not available. Prior, unrecorded experience at Port Radium may be a risk factor in some instances. The Port Radium ores contained significant concentrations of other elements, including arsenic, nickel and cobalt. The degree of confounding arising from these elements in workplace dust is not known. Overall, the Port Radium cohort provided evidence of the risk of exposure to RDPs. The 1996 exposure re-evaluation [S15] represents the best available data for epidemiological assessment of the Port Radium cohort. However, the exposure uncertainties are very large, and quantitative estimates of dose–response relationships must be viewed as having substantial uncertainty.

360. Notwithstanding the re-evaluation of miners' exposures, the Port Radium data set provides a much weaker basis for dose–response investigation than, for example, the Beaverlodge cohort, and is of lower reliability for use in a quantitative assessment of risk.

H. French uranium miners

1. Introduction

361. Uranium prospecting began in 1947 in France, and the production of the first tonne of uranium occurred in 1949. Extraction was located in four main mining divisions: Crouzille (Limousin) and Forez from 1947, Vendée from 1953 and Hérault from 1977. It continued up to 1999, when the last mine closed.

362. The first radon measurements were taken in 1953. In 1956, forced ventilation was introduced in the mines, leading to a sharp decrease in exposure levels, and systematic control of individual exposures began to be applied in the mines. Individual exposure was assessed for each miner on the basis of ambient measurements. Monthly individual records of RDP and gamma exposures were kept in the mining divisions. By 1958, a regulation decreed limits for internal and external exposures. From 1959 onward, systematic records of uranium ore dust exposure were kept for each miner. After 1983, the system of assessment of exposure was replaced by the use of personal alpha and gamma dosimeters [Z10]. Pradel and Zettwoog [P8] and Bernhard et al. [B46] describe the radiation protection practices in French uranium mines.

2. Radon and radon decay products

363. A 1955 paper by Jammet and Pradel [J7] provided insight into the early conditions in the French uranium mines. This paper reported radon concentrations

of 100–10,000 pCi/L in mine air (based on 40 samples). Groundwater from mineralized zones was an important source of radon. The paper noted that, in one mine, a cross-cut was driven into barren rock and air was blown through the cross-cut. The air came into contact with radon-rich water, and within a distance of 300 m exceeded the workplace concentration limit for radon, referred to as the tolerance level (50 pCi/L of air in French mines at that time). The paper also noted high levels of radon in the smoke generated by blasting (levels of as high as 50,000 pCi/L).

364. The 1974 paper by Pradel and Zettwoog [P9] showed that RDP measurements started in 1955 with about 65 samples being taken per mine per year. Beginning in 1955, miners were required to wear an individual dosimeter to record their exposure to gamma radiation. Monthly exposures to RDPs and long-lived radioactive dust were calculated, and all results of exposure were reported on personal cards. Annual and lifetime exposures for all three components of radiation dose — RDPs, gamma radiation and long-lived radioactive dust — were obtained by summing monthly exposures over time. From Tirmarche et al. [T9, figure 6], about 2,500 person-years of exposure in the 1947–1956 period had to be reconstructed, about 11% of the total person-years.

365. Pradel and Zettwoog [P8] commented that, prior to 1953, only 40 measurements of radon had been made. Large numbers of radon measurements were made later, and this enabled a close approximation to be made of the inhaled quantities of radon. The authors also discussed the possibility of relatively elevated exposures for short periods of time when mining was taking place in high-grade-ore areas. Data from Duport [D9] showed that, in the period 1956–1982, there were typically more than 30 radon measurements per person-year. This is very high compared with the number available for the other miner cohorts used for risk assessment.

366. After 1983, the system of exposure monitoring based on area measurements was replaced by personal alpha and gamma dosimeters. The portable device is described by Zettwoog [Z10]. It comprises an active dosimeter with a spectrographic head for the measurement of alpha radiation (ionograph track detection) and a thermoluminescent dosimeter for the measurement of external gamma radiation. It allows the measurement of the number of ^{222}Rn atoms inhaled, the energy of the alpha particles emitted by the three short-lived decay products of radon and thoron (^{218}Po , ^{214}Po , ^{212}Bi), the alpha activity of the five long-lived alpha emitters (^{238}U , ^{234}U , ^{230}Th , ^{226}Ra , ^{210}Po) contained in ore dust (after decay of the short-lived products) and the gamma radiation dose.

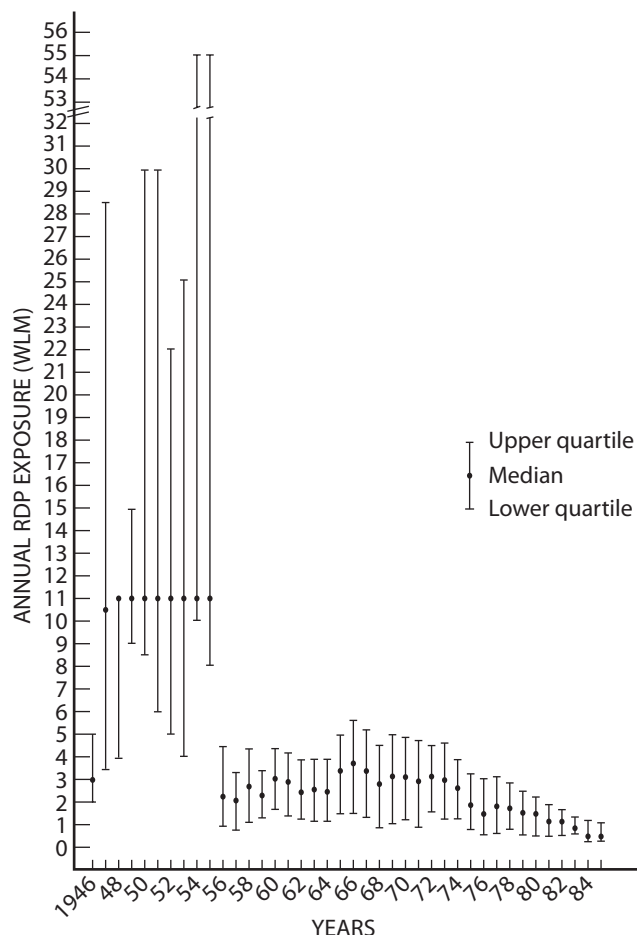
3. Exposure estimation

367. Information about RDP exposure in the initial cohort of French uranium miners is found in references [L10, T5, T6, T7, T9]. This group includes 1,785 miners who began underground work between 1946 and 1972 and who were exposed

for at least 2 years. The mean cumulative RDP exposure was relatively low (70 WLM). The reports describe the reassessment of the RDP exposure of miners by an expert group, notably for the period 1947–1956, for which retrospective estimation was necessary. Working conditions changed dramatically in 1956, with a large reduction in the exposure of miners [T8]. Tirmarche et al. [T7, T8] provided a distribution of individual annual exposure (WLM). Prior to 1956, median annual exposures were estimated to be of the order of 10–11 WLM. After 1956, annual exposures were in the range 1–3 WLM until 1980 and were <1 WLM thereafter, as illustrated in figure XIII. The figure also shows that exposures prior to 1956 had a 5-fold range of interindividual variability.

368. Rogel et al. [R10] and Tirmarche et al. [T30, T31] provided an update of the exposures of the enlarged cohort of French uranium miners. This group included 5,098 males, employed as miners by CEA-COGEMA for at least 1 year between 1946 and 1990. Among the 4,134 miners who were exposed to RDPs, the mean cumulative exposure was 36.5 WLM (with a range of individual exposures of 0.1–960.1 WLM) over a mean duration of 11.5 years (with a range of 1–37 years). The mean annual exposure was 23.9 WLM before 1956, whereas after 1956 it was only 1.5 WLM [R10].

Figure XIII. Distribution of annual exposures to radon decay products by calendar year in the initial cohort of French uranium miners [T7].



4. Epidemiological studies

369. The initial cohort included 1,785 uranium miners who began underground work between 1946 and 1972 and were exposed to RDPs for at least 2 years. Tirmarche et al. gave a status report on the epidemiological follow-up of these uranium miners in a 1985 paper [T9]. A first analysis of this cohort, based on follow-up to December 1985, was published in 1993 [T6]. Compared with national rates, significant excesses of deaths from lung cancer (observed = 45, expected = 21) and cancer of the larynx (observed = 17, expected = 7) were observed.

370. For lung cancer only, a linear dose–response relationship was described with respect to the cumulative exposure to RDPs [T6]. The ERR coefficient was relatively low (0.35 WLM^{-1}) in comparison with those found in other miner studies, but as the number of lung cancer deaths was also low ($n = 45$), the CIs of this coefficient included most of the values of the other studies, as well as estimates from international committees [L10]. The authors cautioned that, since the mean age of the cohort was only 56 years, the cohort was too young for full expression of the lung cancer risk.

371. Laurier et al. described the results obtained from the initial cohort after extension of the follow-up to 1994 [L44]. The mean age at study exit was then 63 years. Causes of death were obtained from the National Mortality Database, which collects information from all death certificates in France. Compared with previous analyses, the use of the National Mortality Database as the principal source of information on the causes of death allowed a reduction in the potential bias in the calculation of standardized mortality ratios (SMRs). The analysis showed, however, that this had little impact on the relationship between RDP exposure and lung cancer risk. Compared with the earlier study [T6], the number of person-years was increased by 25% ($n = 56,372$) and the number of deaths by 74% ($n = 612$). The analysis confirmed the existence of an excess risk of death from lung cancer among French uranium miners (85 observed deaths, $\text{SMR} = 1.9$; 95% CI: 1.5, 2.3), and an increase of this risk with cumulative RDP exposure ($\text{ERR} = 0.6$ (95% CI: 0.1, 1.2) per 100 WLM). An excess risk of laryngeal cancer, noted in the 1993 paper by Tirmarche et al. [T6], was not confirmed in the later study (12 observed deaths, $\text{SMR} = 1.1$; 95% CI: 0.6, 1.9) [L44, T5].

372. The French cohort of uranium miners was enlarged by the inclusion of additional miners with lower radon exposures, and the follow-up was extended to December 1994. The enlarged cohort is described in references [L40, R10, T30, T31]. The study was limited to males who had been employed as miners by CEA-COGEMA for at least 1 year between 1946 and 1990. The main characteristics of the enlarged French cohort of miners are summarized in table 20. This cohort comprised 5,098 miners followed-up from 1946 to 1994, with a total of 133,521 person-years. The percentage of miners lost in the follow-up was 2.3%. The average age at the end of the study was 55 years. A total

of 1,162 deaths were observed. The cohort included a group of 964 non-exposed individuals (but who had the status of a miner and were working in the same mining divisions as the

miners). Among the exposed miners, the mean cumulative exposure was 36.5 WLM, accumulated over a mean duration of exposure of 11.5 years.

Table 20 Characteristics of the enlarged French cohort of uranium miners [R10]

<i>Period of follow-up: 1946–1994</i>	
Number of workers	5 098
Non-exposed miners	964
Number of person-years	133 521
Average cumulative exposure (WLM) ^a	36.5
Average annual exposure (WLM)	
Before 1956	23.9
1956 and later	1.5
Average duration of exposure (years) ^a	11.5
Person-years by lagged cumulative exposure (WLM)	
0	49 408
0–10	35 817
10–50	27 778
50–100	11 358
100–200	6 213
>200	2 947

^a Among 4,134 exposed miners.

373. The total number of lung cancer deaths observed between 1946 and 1994 among the enlarged cohort of French uranium miners was 125. On the basis of the reference rates for the general French male population, the number of lung cancer deaths expected was 83.1. The analysis confirmed a significant excess of lung cancer deaths (SMR = 1.51; 95% CI: 1.25, 1.79). A significant excess was also observed for all cancer mortality (SMR = 1.14; 95% CI: 1.03, 1.25), but this disappeared after exclusion of the lung cancer deaths (SMR = 1.02; 95% CI: 0.90, 1.10). Thus no significant excess of deaths from any other cancer was observed [T30].

374. Rogel et al. [R10] reported on how factors such as time since exposure and exposure rate modified the lung cancer risk in the enlarged cohort of French uranium miners. The statistical analyses were based on a linear relative risk model using Poisson regression to fit the models, maximum-likelihood methods to estimate parameters and likelihood ratio tests for nested models. A linear exposure–response relationship with an ERR of 0.8 (95% CI: 0.3, 1.4; $p < 0.001$) per 100 WLM was found. No inverse exposure–rate effect was observed in the extended French cohort. The strongest modifier was the period of exposure. Analysis showed an ERR that was 10 times higher per WLM for exposures received before 1956 than for exposures received

in 1956 and thereafter. This could be explained by a better quality of exposure assessment after 1956. The ERR for exposures after 1956 was 2.4 (95% CI: 1.1, 4.6, $p < 0.0001$) per 100 WLM [R10, T30].

5. Evaluation

375. The assessment of RDP exposure among French uranium miners is of good quality. Beginning in 1956, monthly records of individual exposure were systematically kept in the mining division. Furthermore, compared with the initial cohort involved in the international joint study in 1994 [L10], the size of the cohort was increased almost 3-fold, and the follow-up was extended by 10 years. Also, it is worth noting that, after 1956, the exposures of the French miners to RDPs were of the same order of magnitude as those received in some homes. Therefore the potential contribution of the French uranium miner cohort data to the estimation of the risk coefficient for lung cancer could be especially relevant to the estimation of the risk for populations exposed to RDPs in their homes. A European project that includes the French, Czech and German cohort studies is in progress [T27, T30]. The combined data, which are of good quality and relate to miners with low levels of exposure, will allow an analysis to be undertaken with a large statistical power.

376. The quantification of the relationship between cumulative RDP exposure and risk of lung cancer mortality required elaborate statistical methods. Different models were applied, and modelling was performed independently by different researchers [L10, R10, T6, T27]. All analyses confirmed an increase of lung cancer mortality with cumulative exposure. In the framework of a European programme, data from the French and Czech miner cohorts were made available to researchers involved in biologically based modelling, with the aim of comparing the approaches and results of the different models [T30]. The different biologically based solutions gave a reasonably good fit of the data and confirmed a linear increase of risk with cumulative exposure [B27, H32]. The comparison of the different approaches provided very interesting discussions regarding the biological validity of the models, the means for testing various hypotheses about the processes of radiation carcinogenesis, the selection of the best fitting model, and the comparison of these biologically based models with the empirical approach that uses a statistical model for describing the data [B37, H33, L42].

377. Data on yearly gamma and long-lived radioactive ore dust exposure are also available since 1956 and 1959, respectively, for each miner of the enlarged cohort. The information will allow all three components of radiation exposure — RDPs, gamma radiation and long-lived radioactive dust — to be considered in an analysis of the dose–response relationship. In addition, a nested case–control study investigating the joint effect of RDP exposure and smoking on lung cancer risk among French uranium miners was published [L44]. It confirms the existence of a significant effect of RDP exposure when smoking information is taken into account.

I. Canada: Newfoundland fluorspar miners

1. Introduction

378. The 1969 report of the Royal Commission Respecting Radiation, Compensation, and Safety at the Fluorspar Mines, St. Lawrence, Newfoundland [A14] provided a wealth of interesting historical data, and is the source of much of the following information.

379. For many years, St. Lawrence, Newfoundland, was an isolated fishing community. Shortly after the First World War, the community was devastated by the drop in price of salt-cured fish (the main source of income) and, in 1929, all of the fishing equipment was destroyed by a tidal wave. Mining eventually took over as the principal occupation [D4]. Fluorspar, which is used in the production of steel, aluminium and high-octane gasoline, is the only mineral resource known to be of economic quality in the St. Lawrence area.

380. The earliest mine to start operation at St. Lawrence was the Black Duck mine, which belonged to the St. Lawrence Corporation. This mine opened in March 1933. Originally, mining was by open-cut methods. By 1937, however, the open cut had reached a depth of about 90 feet. At this

stage, pumps could not cope with the amount of water in the open cut, and it was necessary to sink a shaft, which eventually went to a depth of 250 feet [A14]. This mine ceased operations in 1942. In 1937, the St. Lawrence Corporation started work on a vein called Iron Springs. Originally, mining there was also by the open-cut method, but by 1938, the work had moved underground, with the mine eventually descending to a depth of 970 feet. This mine was closed in December 1956.

381. Standard underground mining procedures were adopted with the first underground mine in 1936. Wet drilling (which resulted in reduced dust levels) was generally adopted in 1942. Shrinkage stoping and cut-and-fill methods were not practised until after 1964. The underground mines were in general very wet. Ventilation was mostly provided by natural means. Except in one case, supplementary blowers were not used until 1946 [D5].

382. The report of the Royal Commission [A14] noted that the St. Lawrence Corporation had 16 veins of fluorspar on its mining properties, and at one time or another most of these were mined by the company. Practically all miners employed by the St. Lawrence Corporation who worked underground prior to 1960 had at some point also spent time working in the Iron Springs mine. According to the report of the Royal Commission [A14], the working conditions in the Black Duck mine were unpleasant. The Commission noted, that prior to 1942, drilling was done with a dry hammer, and that dust and smoke were always such that the driller could only be seen at close quarters. “He was always like a snowman and also had to shut off his machine to clear out his eyes and nostrils.” Clearly, the mines were poorly ventilated, and any radon brought into the mines with mine water would likely remain in the mine for a long period of time, resulting in high radon/radon decay product equilibrium conditions.

383. Until 1942, there were three shifts working underground at the Iron Springs mine. Late in 1942, the mine was put on two shifts. This change was most beneficial: the four hours between shifts could be used for blowing out the smoke with compressed air. There was practically no forced ventilation in any of the mines before 1960, natural ventilation having been the only source of ventilation until that time.

384. Mines were inspected annually by inspectors brought in by the Newfoundland government; however, it was not until 1951 that mining regulations were in place. Consequently, prior to this date, the inspectors had to rely solely on persuasion to achieve improvements in the conditions in the mines.

385. Morrison et al. [M16, M17] noted that, although more than 40 fluorspar veins were located in the St. Lawrence area, most of the ore produced came from only two mines, namely the Iron Springs and the Director mine, mined by the St. Lawrence Corporation and the Newfoundland Fluorspar Corporation, respectively. According to Morrison et al.

[M16, M17], the Iron Springs mine was believed to have had the worst ventilation of any mine in the St. Lawrence area. In contrast, the Director mine, from 1955 onward, employed some forced ventilation. Both mines were extremely wet.

386. Morrison et al. [M16, M17] postulated that, since operations were converted to underground mining procedures in the mid-1930s, in the middle of the Depression, work was done with antiquated equipment, and it was possible that the health and safety conditions were poor as a result of the financial situation at that time. From the mid-1950s, it was clear that miners in St. Lawrence were suffering from various respiratory troubles, some of which had been diagnosed as silicosis. For this reason, the Newfoundland Department of Mines requested the federal Department of National Health and Welfare to carry out a survey of dust conditions in the St. Lawrence mines. Subsequently, the Industrial Hygiene Division of the Department of National Health and Welfare (now Health Canada) conducted a dust survey in the period 1956–1957. By the end of 1957, it was clear that the miners of St. Lawrence were suffering from a respiratory ailment that was not caused by the excessive quantities of siliceous dust. The dust survey was therefore expanded into a broader epidemiological study. By the end of 1959, Windish and Sanderson had completed two brief radiation surveys in the mine [W6, W7]. The results of these surveys established that airborne radioactivity in the form of radon and radon decay products was present in the two mines surveyed in excess of the maximum permissible concentration (indicated in the Royal Commission report to be 1 WL). One of the suggestions made by Windish [W5] was that radon was carried into the mine by mine water and then released to mine air.

387. As described in Aylward et al. [A14], the Occupational Health Division of the Department of National Health and Welfare began a detailed clinical investigation of the St. Lawrence miners in August 1960, under the direction of Dr. A.J. de Villiers. Current epidemiological studies have evolved from this foundation. It should be noted that, prior to 1959, there were no measurements of radon or radon decay product concentrations in the Newfoundland fluorspar mines.

2. Radon and radon decay products

388. On the basis of work done described in the report of the Royal Commission [A14], the source of radon was eventually identified as the water that poured into the mines [D5], the radon itself apparently originating from the host granite. The report of the Royal Commission ([A14], table II) reported levels of radon of 300–13,000 pCi/L in mine water.

389. Interestingly, high radon levels in water were not limited to the mine. According to data of the Royal Commission [A14], measurements of municipal water supplies in the St. Lawrence area revealed radon levels ranging from 1,800 to 14,370 pCi/L. Radon levels in other water supplies in the St. Lawrence area ranged from 6,000 to 12,000 pCi/L.

Radon levels in neighbouring communities close to St. Lawrence were found to range from 1 to 1,140 pCi/L.

390. Morrison et al. [M17] noted that in 1960, because of the high levels of radon identified, mechanical ventilation was introduced into all levels of the mine that were still operating, and the RDP levels subsequently fell below the then current limit of 1 WL. In 1978, mining operations in St. Lawrence ceased, and the last fluorspar mine was closed. By this time, 78 cases of lung cancer had already been identified [C12].

391. Surveys conducted by Windish and Little in 1959 and 1960 collected 17 radon and 80 RDP readings (the Kusnetz method was used for measuring the RDP concentrations) and several gamma radiation readings (reported in reference [A14]).

392. A retrospective study of early mining conditions and working level exposures to RDPs was carried out by Corkill and Dory of the Atomic Energy Control Board (AECB) of Canada [C12]. On the basis of a detailed study of each mine, including measurement data, mining records, ventilation data, interviews and simulation studies, every mine was assigned a high, medium or low RDP exposure level for each operating year (1933–1960). No RDP measurement data were available prior to 1959. Before 1960, when additional ventilation and control measures were introduced, miners were likely to have been exposed to average RDP concentrations of 2.5–10 WL, depending upon the type and place of work. From 1960 onward, estimates of RDP exposures were available for miners by calendar year [M14].

3. Exposure estimation

393. Sources of RDP exposure data include: values estimated and assigned to each mine and each calendar year for 1933–1960 in the retrospective study by Corkill and Dory [C12]; survey data by Windish and Little in 1959 and 1960 (as reported in reference [A14]); and personal exposure data starting in 1960. The data (80 samples collected at 50 different locations) in Windish and Little's survey formed the basis of de Villier's epidemiological studies in 1964 and 1971 [D4, D5]. In the 1964 study, mortality among fluorspar miners was compared with normal mortality in the same geographical region and with that of uranium miners.

394. The WLM method of exposure measurement for individuals was not used until de Villier's 1971 paper [D5]. In the 1971 study, analyses were performed on miners, drillers and muckers. Pre-1960 exposures were included, since the mortality analysis started in 1933; however, no explanation of how they were accounted for is given. The only exposure data mentioned were those given in the Windish survey.

395. The retrospective study of Corkill and Dory [C12] and the study of Morrison et al. [M14] provided more comprehensive data for miners employed during the period

1933–1978, including mortality data from 1933 to 1981. In the analysis by Morrison et al. [M16, M17], the cohort consisted of 1,772 miners employed either by the St. Lawrence Fluorspar Company or Newfoundland Fluorspar Limited. Morrison et al. [M16, M17] described the databases, personal identifying information and occupational histories. The exposure estimates in WLM used in this analysis were calculated on the basis of year, mine and occupation for the period 1933–1960, based on data provided by the AECB [C12].

396. According to Dory and Corkill [D6], from 1961 onward, estimates of RDP concentrations in WL and/or RDP exposures in WLM were available from the miners' individual files. The environmental conditions in the early years of mining were reviewed. Exposure re-evaluations were carried out on the basis of an assessment of the environmental conditions determined by a review of mine maps, inspectors' reports, Royal Commission hearings, anecdotal information from former workers and the authors' own experience. Comparison was also made with the conditions in the mines in later years for which radiation measurements were available. Dory and Corkill [D6] also used computer modelling to simulate expected work environment conditions for each mine for each year of its operation. Most importantly, the authors recognized the uncertainty in the estimation procedure and presented a range of RDP concentrations for various workplaces taking into account the degree of wetness and the degree of ventilation assumed to be present. Corkill and Dory [C12] noted that the ranges they presented do not represent extreme concentrations but are "average workplace concentrations for high, medium and low areas".

397. Corkill and Dory [C12] considered it possible to place job types within certain average concentration ranges. Their exposure re-assessment substantially improved the estimation of RDP concentration (WL) and RDP exposure (WLM) for the Newfoundland fluorspar miners. Nevertheless, as in all attempts at reconstruction, there are large (and probably irreducible) uncertainties in the actual conditions that existed in the workplaces for the period 1933–1960.

398. Detailed occupational histories were obtained from company records. The records for the 1933–1936 period were reconstructed from census data, interviews with company officials and company report reviews [D4].

399. Workers' occupational histories, by accumulated hours of exposure and type and place of work, were prepared for all men on the employee list [D5]. Occupational history was particularly important for fluorspar miners, since epidemiological studies included surface workers as well as underground miners.

400. Corkill and Dory [C12] assumed that the type of job dictated where a man worked, and thus the concentration of RDPs to which a worker was exposed. For example, development miners were assigned the high average concentrations, stope miners the medium average concentrations, and

miners working in an established area near an air circuit the low average concentrations. The high and low averages often differed considerably (by a factor of from 2.5 to 10). To use these averages, good knowledge of each worker's duties was developed.

401. No mention of the effect of job mobility was made in Corkill and Dory's study. This might have been taken into account, at least to a certain degree, since the payroll records used to construct the occupational history were recorded every two weeks.

402. No mention was made of whether previous hard rock mining experience had been taken into account. However, since St. Lawrence was an isolated fishing community, especially in the early years, miners were most likely to have been fishermen before they started work in the St. Lawrence mines.

403. A working month was considered to be 167 hours by de Villiers and Windish [D4] and 170 hours by Morrison et al. [M16, M17] in the WLM calculations. Accumulations of working hours were likely to have been reasonable, since they were compiled from payroll records (on the basis that miners were paid on an hourly basis).

4. Epidemiological studies

404. Early studies of the St. Lawrence fluorspar miners included those of de Villiers and Windish [D4], Parsons et al. [P1] and Wright and Couves [W10]. However, the first attempt to examine the RDP exposure–response relationship was given in a paper published in 1971 by de Villiers et al. [D5]. The number of hours worked underground was used as a surrogate for actual exposure to RDPs. A plot of lung cancer deaths versus number of hours worked revealed an exponential relationship. To adjust for the variable radiation exposure of differing occupations, hours worked were weighted according to occupation. Drifters and stope workers were assigned an RDP concentration of 8 WL, muckers, trammers, and chute operators were assigned a level of 4 WL and shaftmen were assigned a level of 2 WL. With the assignment of these weights, the exposure–response relationship became linear. This procedure was followed by Morrison et al. [M18], who extended the mortality follow-up first to 1978 [M18] and then to 1981 [M14]. In the latter study (published in 1985), the expected number of deaths was calculated from data for an internal control group of unexposed surface workers, with an attempt to account for cigarette smoking and latency period. These analyses, like previous ones, used a modified person-years approach.

405. In 1988, Morrison et al. [M16, M17] modelled the exposure–response relationship using an external control group, and estimated the attributable and additive relative risk coefficients. The radon exposure estimates developed by Corkill and Dory [C12] were used. Attributable and relative risk coefficients were examined by attained age, age when

first exposed and smoking status. In addition, lifetime risk of lung cancer mortality was assessed using both the relative and the attributable risk model. The cohort consisted of 1,772 miners employed by either the St. Lawrence Fluorspar Company or Newfoundland Fluorspar Limited. Morrison et al. [M16, M17] described the databases, personal identifying information and occupational histories. Estimates of exposure in WLM were calculated on the basis of year, mine and occupation for the period 1933–1960, and the calculations provided by the AECB [D6].

406. A 1995 cohort study of the Newfoundland fluorspar miners by Morrison and Villeneuve [M20] and Morrison et al. [M27] examined the mortality experience (1950–1990) of 1,744 underground miners and 321 millers or surface workers. As in the 1988 study, exposure estimates in WLM by year, mine and occupation for 1933–1960 were provided by the AECB [C12]. RDP exposure during the 5 years preceding lung cancer was assumed to be unrelated to lung cancer risk. Overall, 60,000 person-years of follow-up were noted, with a mean cumulative exposure for underground workers of 382.8 WLM over an average of 5.7 years of exposure. Smoking information was available for 65% of the exposed cohort.

407. Values of relative risk (for exposures estimated on the basis of the category of work), adjusted for attained age and period since exposure, increased with cumulative exposure and were statistically significant for cumulative exposures exceeding 200 WLM. On the basis of Poisson regression of the ERRs and the exposure estimates, and through the use of a constrained intercept, the ERR was estimated to be 0.66 per 100 WLM, with a standard error of 0.17%. The effect of cell killing at high WLM was not statistically significant at the $p = 0.05$ level. The attributable risk coefficient for continuous exposure was estimated to be 6.3 deaths (standard error of 0.74) per working level month per 10^6 person-years, with a multiplicative correction estimated from the cohort.

408. The ERR per unit exposure increased with duration of exposure, suggesting that those exposed over longer periods of time had a greater risk than those exposed over a shorter period at the same exposure level.

409. The ERR per unit exposure decreased with increased duration of exposure in a previous 11-cohort study by Lubin et al. [L10]. While this is biologically plausible, these authors presented an alternative explanation for the inverse dose-rate effect, namely that the finding is an artefact resulting from a greater non-differential exposure misclassification at higher exposure rates than at lower exposure rates. Because exposure rates were extrapolated for the period prior to 1960, when they were high, these authors suggested that there may be a much higher degree of miscalculation of these exposures than of exposures from 1960 onward. The effect of non-differential misclassification would be to bias the risk estimates towards lower values, resulting in a greater reduction in risk estimates at high dose rates than at low dose rates. If the inverse exposure-rate effect is indeed the result

of bias, then it follows that the ERR would have been an underestimate, since the significant random exposure misclassification that constituted the basis for the bias should also bias the overall ERR towards lower values at higher exposure rates.

410. The 1995 analysis [M20] found an ERR for lung cancer of 0.66 per 100 WLM, slightly lower than that observed by Lubin et al. [L10] in their analysis of the fluorspar cohort. The difference with the more recent analysis was that it was based on a later follow-up time (1990 versus 1984 for reference [L10]). However, the ERR of 0.66 per 100 WLM was similar to that noted by Lubin for all 11 mining cohort studies combined (0.49 per 100 WLM). Although statistically significant differences in ERR were detected between smokers, non-smokers and former smokers, the joint effects of exposure to RDPs and smoking could not be assessed.

411. A more recent report described an 11-year updated analysis of the mortality experience (1950–2001) of the Newfoundland fluorspar miners [V4]. The new study reports on an analysis of 328 miners who worked exclusively on the surface and 1,742 individuals exposed to RDPs from working underground. When compared with Newfoundland males, the fluorspar miners had significantly increased numbers of deaths for lung cancer, silicosis, and accidents, poisoning and violence. In total, 206 lung cancer deaths were identified, 191 of which occurred among individuals who had at some stage worked underground, the other 15 occurring among miners who had worked only on the surface.

412. Villeneuve et al. [V4] found a strong association between cumulative exposure to RDPs (WLM) and lung cancer risk. Workers with estimated cumulative exposures exceeding 2,100 WLM had relative risks more than 20-fold higher than unexposed miners. After adjusting for age and calendar period, the linear ERR among underground and surface miners (combined) was estimated to be 0.47 (95% CI: 0.28, 0.65) per 100 WLM. The relationship between cumulative exposure in WLM and lung cancer risk was modified by time since last exposure, duration of exposure and exposure rate. In contrast, age at first exposure was not a statistically significant determinant of lung cancer risk. After 35 years since the time of last exposure, lung cancer mortality rates among exposed miners dropped to levels experienced by those who worked exclusively on the surface. Morphology was available for 88 of the 191 lung cancer deaths among those who worked underground. The histology included squamous cell carcinomas (28), adenocarcinomas (8), small cell carcinomas (7) and other carcinomas (45). Owing to the small number of cases, it was not possible to determine the ERR per unit exposure by histological type.

413. Twenty-eight lung cancer deaths occurred among men who started working after 1960 (when ventilation was introduced). There was no significant variation in the ERR per unit exposure between those who started work before and after 1960. However, the evaluation of cancer risk among those who started mining after 1960 is based on younger

men. Thus few cancer deaths were identified, so the statistical power to detect an association in these workers was limited [V4].

414. Some data on smoking were available for 1,107 of the 2,070 miners (53%). There was no statistically significant difference in the ERR per unit exposure between those who had smoked and those who had never smoked. However, strong associations between cumulative radon exposure and lung cancer risk were noted among individuals who had smoked different numbers of cigarettes daily ($p < 0.05$). Specifically, the ERR was 0.31, 0.46 and 0.94 per 100 WLM among individuals who reported smoking <15, 15–<30 and 30 or more cigarettes per day, respectively. An evaluation of the joint effect of exposure to RDPs and smoking (as measured by the number of cigarettes smoked daily) could not adequately discriminate between additive and multiplicative models. However, the data were suggestive of an intermediate relationship (between additive and multiplicative). The evaluation of the joint effects of smoking status and radon was severely limited by the small number of lung cancer deaths that occurred among miners who never smoked ($n = 8$) [V4].

5. Evaluation

415. One possible advantage of the Newfoundland fluor-spar cohort over most other studies of radiation-exposed mining populations is that the source of RDP in the fluor-spar mines was from groundwater and not from radioactive ore. Thus it was possible to exclude the effects of gamma radiation, thoron and radioactive dust. The cohort was, however, exposed to silica. Another advantage is that fluor-spar miners were almost without exception local men with no previous mining experience. Upon ceasing to mine fluor-spar, the people went back to non-mining professions. Unfortunately, all the exposures in the period of high exposure rates (1933–1960) were estimates only and are subject to large uncertainty. While the exposure data are weak, the availability of smoking histories and the ability to investigate the effect of changes in individual smoking histories over time are strengths of the cohort. The study of Villeneuve et al. [V4] confirms the strong association between cumulative exposure in WLM and lung cancer incidence.

J. Chinese miners

416. Uranium prospecting and uranium mining started between 1955 and 1958 in China, and routine monitoring of radon was carried out after 1959. The first uranium mine was established in Hunan province in August 1958; very high levels of radon were measured because of poor ventilation. Radon levels decreased rapidly after 1960. Comprehensive studies of radon exposure were completed in 1993 for 11 uranium prospecting teams and four uranium mines. A total of 27,172 and 108,744 person-years were accumulated for the prospecting teams and the miners, respectively, during the

follow-up period 1971–1985. Over this period, the average RDP concentrations were 0.3 WL for the prospecting teams and 1.0 WL for the miners, resulting in average cumulative exposures of about 80 WLM for each group. In total, there were 28 lung cancers. ERRs of 1.19 per 100 WLM and 1.09 per 100 WLM were estimated for the prospecting teams and the miners, respectively [S52].

417. There are some English language papers (e.g. [L11, L27, S26, S27, S50, S51, S52, S53, S54, S58, T32, Z6]) and many Chinese papers that discuss the lung cancer experiences of miners who worked in the Yunnan tin mines in China. Qiao et al. [Q1] reported on an investigation of risk factors and the early detection of lung cancer in a cohort of Chinese tin miners. They described a dynamic cohort using an ongoing lung cancer screening programme among tin miners exposed to arsenic and RDPs. The investigation noted that about 6,000 tin miners are screened per year with sputum cytology, chest X-rays and personal interviews. The authors calculated SMRs and 95% CIs. They also calculated relative risk and 95% CIs for lung cancer risk factors from a proportional hazards model. Exposures to RDPs and arsenic were the predominant risk factors, but silicon and smoking were also lung cancer risk factors in their cohort.

418. Chen and Chen [C36] reported a nested case-control study of 130 male lung cancer cases and 627 controls from a cohort of 7,855 miners employed for at least 1 year between 1972 and 1974 in any of four tin mines in China. The Maentel-Haenzel OR was used to measure the association between lung cancer and various risk factors. Unlike Qiao et al. [Q1], these authors did not find that silica exposure was related to the risk of lung cancer. However, the authors did find a strong association between risk of lung cancer and cumulative exposure to dust, cumulative exposure to arsenic and duration of dust exposure. The most recent English language summary of this cohort was provided in a paper by Shiquan et al. [S54], which indicated that the high incidence of lung cancer in miners of the Yunnan Tin Corporation (YTC) in Gejiu, South China, had attracted attention since the early 1970s. Underground monitoring of dust started in 1955 and of RDPs in 1972. Data collection from medical examinations, chemical analysis of pulmonary tissues and animal experiments started in 1975. The database for epidemiological studies was established in 1976, and reports from cooperative studies with the National Cancer Institute (NCI) in the United States began in 1998. Some of these epidemiological studies and the database provided by YTC were used by the National Institutes of Health [L10] and BEIR VI [C20] as the basis of data for the Chinese miners in the joint analysis of 11 underground miner cohorts. Lung cancer cases from the YTC, one of the largest of the 11 cohorts, made up 36% of the total cases in the NIH and BEIR VI combined analyses.

419. About 90% of the lung cancer cases at YTC had a history of working underground [S54]. Prior to 1950, the principal work involved men carrying ore on their backs in small tunnels. That working style was gradually abolished after 1953 as mechanization started to be introduced.

YTC miners represented a stable population without significant loss of follow-up. Most miners started work before 1950, some before 1920. Most miners with lung cancer began mining as children under the age of 14, but this group's age of death and risk of lung cancer showed no prominent differences from those of miners who started mining after the age of 15 or 20.

420. The first systematic monitoring for radon was done in four YTC mines in 1972. The highest level was at the L-mine, at 28.6 Bq/L on the average. Radon concentrations decreased after the improvement of underground ventilation in 1974 [S54]. To estimate RDP exposure before 1950, 13 existing small tunnels were measured in around 1980, and showed an average RDP concentration of 2.3 WL. Measurements of ^{210}Pb in rib bones obtained after operations on miners with lung cancer provided additional data for retrospective dose assessment [L27]. Estimates of cumulative exposure to RDPs could be divided into three stages: 1950–1953, when the miners carried ore on their backs; 1953–1972, a period of modern mining but lacking radon monitoring; and after 1972, with radon monitoring.

421. Before 1965, airborne dust concentrations underground were very high at about 27–60 mg/m³, because of dry drilling. In 1965, the dust levels decreased to 6 mg/m³ after the introduction of wet drilling, [L27, S58]. Arsenic and iron were present as relatively insoluble compounds in YTC mines; the arsenic concentration in the rock was about 0.5–1%. Airborne arsenic concentrations measured in the 1970s were about 0.01 mg/m³ and were thought to have been more than 10 times higher in earlier years.

422. A combined effect of exposure to RDPs and arsenic on the aetiology of lung cancer among the YTC miners was reported previously [S51, X1, Y1]. The data for arsenic (dust containing arsenic) and RDPs, the only occupational carcinogens underground, were compared to identify their relative contribution in the aetiology of lung cancer. Since cumulative exposures to RDPs and arsenic were highly correlated, both being related to the duration of underground work, comparison of the relative contributions to risk from RDPs and arsenic had to be approached from differences in lung cancer risks in miners working at different jobs (mining, tunnelling, auxiliary) and different mines (L, M and S, the three largest YTC mines).

423. Sun et al. [S53] suggested that the arsenic adjustment used by the NIH [L4] was unsuitable for use in risk projection. BEIR VI [C20] noted that adjustment for arsenic exposure was difficult because of the strong correlation between RDP and arsenic exposures. Hazelton et al. [H7] noted a high risk from arsenic exposure and an interaction of arsenic with other sources of exposure in the study of the YTC miners. These authors analysed the arsenic, radon, cigarette smoke and pipe smoke exposures using the biologically based two-stage clonal expansion model. They concluded that, of 842 lung cancer deaths among YTC miners in Gejiu, 21.4% were attributable to tobacco smoke alone, 19.7% to a

combination of tobacco smoke and arsenic, 15.8% to arsenic alone, 11% to a combination of arsenic and RDPs, 9.2% to a combination of tobacco smoke and RDPs, 8.7% to a combination of arsenic, tobacco smoke and RDPs, 5.5% to RDPs alone and 8.7% to background gamma radiation.

K. Australia: Radium Hill uranium miners

424. In addition to the studies discussed earlier in this section, other miner groups exposed to radon have been discussed in the literature. For example, the BEIR VI report [C20] also discussed lung cancer in workers in the Radium Hill uranium mine in Australia [W15]. Exposures in this mine were estimated on the basis of 721 measurements of radon concentrations; however, no data on RDP concentrations were reported. Ventilation data were used to estimate mean residence time in the mine and subsequently the exposure of miners in WLM. Overall exposures of this cohort were very low, with a mean exposure of 7 WLM. The authors reported an excess of lung cancer (4 lung cancer deaths) in miners whose estimated cumulative exposures exceeded 40 WLM. Thirty-six per cent of the cohort could not be traced beyond employment at the mine. Overall, while supporting the findings of an association between RDP exposure and increased lung cancer, this study provides no useable information for the evaluation of an exposure–response relationship.

L. Overall evaluation of miner studies

425. All of the miner studies reviewed in this section involve retrospective evaluation of exposures to RDPs. In some cases, such as the Newfoundland fluor spar miners and the Port Radium miners, almost all of the exposures were estimated, whereas for the Czech and Wismut cohorts (starting after about 1971), relatively less speculation was needed. Studies of miners also differ by type of mine (e.g. uranium, iron, tin, fluor spar), exposure rate (i.e. WL in the workplace), size of study (e.g. number of subjects, number of lung cancers) and other factors. All of the miner studies described in previous sections confirm the risk of lung cancer from exposure to RDPs. However, not all of the studies are of the same “quality”. It is evident that the studies summarized in table 21 vary widely with respect to factors that affect the determination of the exposure–response relationship and factors that modify that relationship, including, for example, the number of excess lung cancers, the quality of the exposure data (both the range of exposures and the uncertainty in exposures) and confounders such as smoking and exposure to arsenic. A qualitative overall evaluation of such considerations is provided in table 21.

426. Table 21 is a summary of some of the key features of the various miner studies discussed in this section. The table also provides the average ERR per unit exposure estimated for a simple linear ERR model for each of the studies. The ERR per unit exposure ranges over approximately a factor of 5. On the basis of an analysis of 11 miner cohorts, Lubin

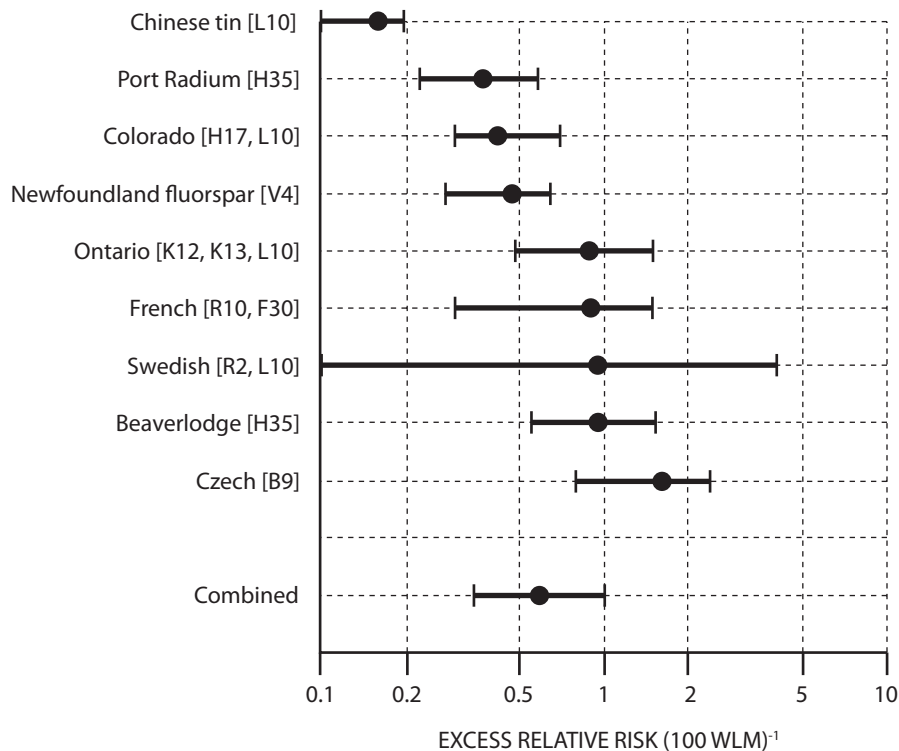
et al. [L10] reported an ERR of 0.49 (95% CI: 0.2, 1.0) per 100 WLM.

427. In a manner similar to that adopted by Lubin et al. [L10], the estimated ERRs reported in table 21 were combined

using a generic inverse variance method and assuming that random effects were present between studies. The results are illustrated in figure XIV, which shows a combined ERR of 0.59 (95% CI: 0.35, 1.0) per 100 WLM, comparable to that reported by Lubin et al. [L10].

Figure XIV. Estimates of ERR per unit exposure from studies of miners.

Combined ERR of 0.59 (95% CI: 0.35, 1.0) per 100 WLM; 95% CI developed with random effects model.



428. It is of interest to compare the risks based on miner studies with those from the more recent pooled residential radon studies. Assuming for purposes of illustration a nominal indoor equilibrium factor of 0.4, 35 years of exposure to indoor radon at 100 Bq/m³ for 7,000 h/a, an exposure of about 19.5 WLM can be estimated. If an ERR of 0.59 (95% CI: 0.35, 1.0) per 100 WLM based on miner studies is assumed (see para. 427), an ERR of about 0.12 (95% CI: 0.04, 0.2) per 100 Bq/m³ can be estimated, a value which

compares remarkably well with values from pooled residential radon studies, as discussed in the next section. The miner studies show that the ERR decreases with increasing time since exposure. This is an important consideration in evaluating potential risks from lifetime exposure. Finally, as evidenced by the updates of the Port Radium and Beaverlodge cohorts [H35], continued follow-up of miner studies is important, because the ERR and other outcomes of the epidemiological analyses may change as the cohort ages.

Table 21 Excess relative risk of lung cancer for exposure to radon in mines

Study	Observed cases	Expected cases	Mean cumulative exposure (WLM)	Mean follow-up time (years)	Person-years	Main potential confounders	Average ERR per unit exposure (per 100 WLM) (95% CI in brackets)	Overall evaluation (subjective)
Colorado Plateau uranium miners [H17, L10]	327	74	807.2	24.6	75 032	Exposure uncertainty; other hard rock mining; smoking	0.42 (0.3, 0.7)	Medium
Ontario uranium miners ^c [K12, K13, L10]	282	221	30.8	17.8	319 701	Other hard rock mining; smoking	0.89 (0.5, 1.5)	High
Czech uranium miners [T39]	915	240.8	70	23.2	261 428	Exposure uncertainty in early years; smoking	1.6 (1.2, 2.2)	High
Swedish iron miners [L10, R2] ^a	79	44.7	80.6	25.7	32 452	Exposure uncertainty ^b ; smoking	0.95 (0.1, 4.1)	Low ^a
Beaverlodge uranium miners [H35]	279	217.8	23.2	33	285 964	Exposure uncertainty ^b ; smoking	0.96 (0.56, 1.56)	High
Wismut uranium miners [K27, K28, K29]	2 328	Not available	242	30.5	1 801 626	Exposure uncertainty (especially prior to 1966); smoking; arsenic; asbestos	0.21 (0.18, 0.24)	Potentially high
Port Radium uranium miners [H35]	230	142.7	174.2	>44	111 222	Exposure uncertainty ^b ; smoking	0.37 (0.23, 0.59)	Low
French miners [R10, T30]	125	83.1	36.5	26	133 521	Exposure uncertainty prior to 1956; smoking	0.8 (0.3, 1.4)	High
Newfoundland fluorspar miners [V41]	206	–	378	>35	70 894	Exposure uncertainty; smoking; high dust levels	0.47(0.28, 0.65)	Low to medium
Chinese tin miners [L10]	936	649	277.4	10	135 357	Exposure uncertainty; smoking; arsenic; age at exposure	0.16 (0.1, 0.2)	Low to medium

^a Exposures in the Malmberget iron ore miner study have been updated, and miner doses have increased by about 50% from those used in reference [R2]; however, at this time the epidemiological study has not been updated.

^b Uncertainty remaining after dose re-evaluation.

^c The Ontario miner study is being updated and will more than double the number of person-years.

V. EPIDEMIOLOGICAL STUDIES OF RESIDENTIAL EXPOSURES

A. Introduction

429. Over the past twenty years or so, there has been a great deal of interest in the risks arising from exposure to radon and its decay products. It is clear from studies of miners that exposure to radon and radon decay products causes lung cancer, as described in section IV (e.g. [C20, D14, D16, I2, I5, L10, N11, U2, U5]). Data from animal experiments such as described in section III also demonstrate that exposure to radon and its decay products causes lung cancer. Until recently, data from studies of underground miners, in uranium mines and other mines, formed the basis for estimating risks from exposure to RDPs and for investigating the exposure–response relationship, as, for example, was carried out by BEIR VI in their pooled analysis of 11 miner cohorts [C20]. Risks from residential RDP exposure were estimated by extrapolation from miner studies. Now, however, there are more than 20 case–control studies of residential radon exposure and lung cancer. While individual studies have limited power, pooled analyses of European [D17, D21], North American [K1, K26] and Chinese [L26] residential radon exposure studies provide a clear demonstration of the risks of lung cancer from residential radon exposure and a direct basis for estimating risk in dwellings from such exposure. This section provides an overview of the case–control residential radon exposure studies and, in addition, a short commentary on the relevance of geographical correlation (“ecological”) studies. Further information on the epidemiology of radon exposure and on “ecological” studies is provided in annex A, “Epidemiological studies of radiation and cancer”.

B. Case–control studies of residential radon

430. Many case–control studies published prior to about 2000 are well described by Lubin and Boice [L4], BEIR VI [C20], NCRP SC65 [N11] and the UNSCEAR 2000 Report [U2]. Lubin et al. [L8] discussed how errors in exposure assessment can affect the interpretation of results. These authors showed that information from seven case–control residential radon studies supported a wide range of risks, ranging from no excess risk to excess risks larger than those predicted using data from miner studies. These authors discussed various sources of error in the estimation of residential exposure, including various potential sources of measurement error. In addition, a number of authors (e.g. [L4, P17]) have reported meta-analyses that are based on published relative risks. However, such studies are not able to correct for smoking, a key determinant of lung cancer and

therefore a matter that requires careful consideration, ideally on a subject-by-subject basis. The pooled studies [D17, D21, K1, K26, L26], based on individual data, are more informative than the previous meta-analyses.

431. Several ways to address the problems resulting from such exposure assessment errors are discussed in the literature, including the use of special films placed on glass artefacts to measure the long-lived RDPs directly [L31]. One of the primary motivations for the glass-based measurements was to take account of systematic increases in residential radon concentration due, for example, to efforts to increase the energy efficiency of homes by providing better insulation and decreasing air leakage. However, United Kingdom data [L45] suggest that, in the United Kingdom at least, any increase has not been substantial. Another motivation for the glass-based measurements was to eliminate the difficulties caused by missing measurements in the residential studies, for example in cases where a house was demolished. Much work in improving the retrospective assessment of radon exposure was conducted using glass-based detectors and other approaches (e.g. [F9, L31, M33, P15]). Bochicchio reviewed the use of nuclear track detectors in the context of residential radon epidemiology and also discussed the various sources of uncertainty in the retrospective estimation of residential exposures. Bochicchio suggests that ^{210}Po alpha activity on the surfaces of glass objects might be a better surrogate for past exposures than contemporary radon measurements. However, he noted that such data are confounded by aerosols from cigarettes, which reduce the ratio of ^{210}Po surface activity to radon concentration [B42].

432. An early case–control study of domestic exposure to RDPs and lung cancer in Port Hope, Ontario, was carried out to determine if there was an excess of lung cancer in Port Hope residents attributable to exposure to elevated RDP levels [L41]. Since smoking is the major cause of lung cancer, the study controlled for smoking. “Cases” were defined as any person who developed or died from lung cancer in the period 1969–1979 and who had lived at least 7 years in Port Hope. The 7-year residence period was selected because this was the shortest possible time between exposure and the occurrence of lung cancer. Twenty-seven cases met the criteria. There were two controls for each case. Estimates of exposure were developed by adding the cumulative exposures estimated for each house occupied by a case or control since 1933. The statistical analysis failed to demonstrate an increased risk of lung cancer from elevated domestic radon exposure, but did identify a very strong risk of lung cancer from cigarette smoking.

433. High concentrations of indoor thoron were observed in the Loess Plateau region of China (e.g. [S67, T35, W13, W20, Y8]). The assessment of the risk from RDPs is known to be affected by the presence of thoron and its decay products [P19, T17]. It was thought that the detectors used in the study by Wang et al. [W13] might have been affected by the presence of thoron [S10, T17] and thus that the measured radon concentrations might have been overestimated. A re-assessment of the exposures from radon and thoron decay products is currently under way. Moreover, the screen-type diffusion battery (SDB) measurements in underground dwellings indicated the presence of ultrafine particles of around 10 nm [Y8]. Current evaluations are based on exposures; however, since the dose conversion factor for these small particles is high, a modified contribution to dose might need to be considered in the future.

434. Several studies investigated residential radon exposure and lung cancer in China. A study carried out by Blot et al. [B25] in Shenyang City, during 1988 and 1989, included 308 females with lung cancer and 356 female controls. The median radon level measured in the homes of both cases and controls was 2.3 pCi/L (85.2 Bq/m³). The median duration of residence was 24 years. No link between radon exposure and an excess risk of lung cancer was found, irrespective of smoking status (other than a non-significant trend in heavy smokers). A study by Wang et al. [W13] included 1,659 cases and 768 controls who lived in an area of Gansu province. Prior to 1976, many of the subjects had lived in underground dwellings (99%), although many had since moved to above-ground houses. The mean radon levels were quite high for both cases and controls, at 230.4 Bq/m³ and 222.2 Bq/m³, respectively. Using a linear model, the authors estimated an excess odds ratio (EOR) of 0.19 (95% CI: 0.05, 0.47) at an exposure of 100 Bq/m³. If adjustments were made for uncertainty in exposure, the EOR increased by about 50%.

435. Tokonami et al. [T35] and Sun et al. [S63] carried out a radon and thoron survey in the Loess Plateau region of China. Their study area was located near Gansu province. Since the geological features seemed to be almost the same as in Gansu province, the characteristics of radon and thoron concentrations were also likely to be similar. The radon concentration was lower than that in the study by Wang et al. [W13], but the thoron concentration was higher. Because thoron was underestimated in the past, reassessment of risks due to radon exposure may need to take the presence of thoron into account.

436. Field et al. described a residential radon study of females in Iowa, United States [F10]. The Iowa radon study included 413 cases and 614 controls. The median residency was 33 years for cases and 31 years for controls. Some 357 cases and 200 controls were “ever smokers” (i.e. people who had at some time smoked); an additional 104 cases and 200 controls were former smokers. The radon exposure assessment involved a number of components, including 1 year of on-site radon measurements, regional outdoor radon measurements and linkage of the subject’s

mobility with exposure to radon indoors and outdoors. Outdoors, radon concentrations varied from 7.4 to 56 Bq/m³, the latter being comparable to the United States national average indoor radon level of 48 Bq/m³ (see figure I). An average of four radon detectors were placed in each home. The measurements exhibited approximately a log-normal distribution. The majority of basement measurements and a significant fraction of the measurements on the ground level and the upper level exceeded 148 Bq/m³ (the United States Environmental Protection Agency action level of 4 pCi/L). The authors calculated cumulative exposures to RDPs that occurred 5–19 years prior to diagnosis for the cases, or prior to time of interview for the controls, as 8.6 WLM and 7.9 WLM, respectively. The odds ratios (ORs) for lung cancer in women who had smoked at least 100 cigarettes or for at least 6 months in their lifetime relative to women who had never smoked was 13.2 (95% CI: 9.5, 18.3). The authors also found a significant positive trend between lung cancer and RDP exposure. The authors estimated risks for a cumulative 15-year radon exposure of 11 WLM (taken by the authors as equivalent to an average radon concentration of 4 pCi/L) for all cases. After adjustment for age, active smoking and education, the authors estimated an EOR of 0.24 (95% CI: –0.05, 0.50) or (95% CI: 0.004, 1.81), when radon exposure was treated as a continuous or a categorical variable, respectively. A subsequent paper by Field et al. [F11] reported a slightly different CI for the EOR of 0.24; this was 95% CI: 0.05, 0.92, calculated treating radon exposure as a continuous variable. Field et al. observed a statistically significant trend for large cell carcinoma and a “suggestive” trend for squamous cell carcinoma (categorical *p* for the trend of 0.06). However, the linear excess odds between different histological types were not significant.

437. Tomasek et al. [T10] reported a residential radon study of 12,000 people living in central Bohemia with a total of 173 lung cancers and a follow-up period of 1961–1995. This follow-up period was later extended to 1999 [T29]. A total of 210 lung cancers were observed. The study area in central Bohemia is mostly granitoid and has radon levels considerably higher than other areas of the Czech Republic. Exposure estimates were based on measurements of the equilibrium-equivalent concentrations (EECs) of radon (i.e. the RDP concentrations) made in most (80%) of the homes in the study area. Typically, two detectors were installed for 1 year in the two most occupied rooms. To compare their results with those of other studies, the authors established a conversion factor on the basis of 652 simultaneous measurements of EEC and radon. Where necessary, mean values for a community were used to replace missing data. The mean radon concentration was estimated at 509 Bq/m³, with 10% of the homes having indoor radon levels in excess of 1,000 Bq/m³. The authors used a linear relative risk model, taking into account the exposures received between 5–34 years previously and estimating expected cases from national mortality data. The authors estimated an ERR of 0.087 (90% CI: 0.017, 0.208) per 100 Bq/m³. The ERR did not change substantially after adjustment for smoking.

438. Barros-Dios et al. [B26] described a population-based case-control study in an area of north-west Spain considered to be radon-prone. The study covered 163 cases (151 men and 12 women) and 241 controls (219 men and 22 women). Radon concentrations were measured using alpha track detectors placed in the homes for a minimum of 90 days and a median of 150 days. The mean radon levels were 141.4 Bq/m³ for the cases and 114.0 Bq/m³ for the controls. Overall, 22% of the homes had radon levels of above 148 Bq/m³. A multiple logistic regression analysis assessed the risk of lung cancer, taking into account a number of possible confounding factors, including smoking, family history of lung cancer, type of dwelling construction and hours per day spent at home. A total of 145 (91.8%) of the cases and 129 (54.7%) of the controls were smokers. The authors reported ORs by quartiles of the radon distribution, and observed a greater than 2-fold increase in the risk of lung cancer for exposures to radon of above 37 Bq/m³. The authors noted that the risk of lung cancer in smokers was 46 times higher than in non-smokers exposed to radon levels of below 37 Bq/m³.

439. Lagarde et al. [L31, L32] reported on a Swedish residential radon study based on an existing database of persons who had never smoked that had been developed to study environmental and occupational exposures to agents other than radon. The database was augmented with measurements of radon concentrations made with alpha track detectors that had been placed in the bedrooms and living rooms of residences for 3 months. The database was also supplemented with data from a nationwide Swedish case-control study for which the radon measurements were similar. On average, about 25 years of the 32-year residential history of subjects was covered by measurements. Covariates included environmental tobacco smoke and history in occupations with a risk of lung cancer. A total of 436 cases and 1,649 controls (all of whom had never smoked) were included in the risk assessment. The excess relative risk per unit of time-weighted residential radon concentration was estimated using conditional logistic regression and a linear relative risk model. The authors found a trend in relative risk with increasing radon exposure, comparable to that found in the nationwide Swedish study. The trend of increased risk with increasing radon levels was limited to subjects exposed to environmental tobacco smoke at home. For those who had never smoked, a relative risk of 1.10 (95% CI: 0.96, 1.38) was estimated, and for those exposed to passive smoking, the relative risk was estimated at 1.29 (95% CI: 0.97, 2.24).

440. Sobue et al. [S34] reported a case-control study of residential radon levels and risk of lung cancer in Misasa, Japan. The case series consisted of 28 lung cancer deaths (26 males and 2 females) between 1976 and 1996, and 36 (33 males and 3 females) controls chosen randomly from residents. Radon levels were measured using alpha track detectors in the most frequented areas of the subjects' homes for a period of 1 year. The average radon level in the study area was about 50 Bq/m³. The residential radon value measured over the year was used as a surrogate for cumulative

radon exposure over the 20 years of the study. None of the ORs calculated using regression was statistically significant. The authors attributed this in part to the small sample size.

441. The risks of lung cancer from residential exposure to radon in Devon and Cornwall in the south-west of the United Kingdom were reported by Darby et al. [D15]. The study looked at 982 cases of lung cancer and 3,185 controls, all under 75 years of age. Detailed information on the demographics of the study population, smoking characteristics and residency was provided. The investigators attempted to measure radon levels at all of the addresses where the subjects had lived in the preceding 30 years. Two alpha track radon detectors produced by the United Kingdom National Radiological Protection Board were installed (one was placed in the living area and the other in the bedroom) for a period of 6 months. For residences in which radon had been measured, the investigators calculated a weighted average radon concentration assuming that residents spent 45% of their time in the living area and 55% of their time in the bedroom. A time-weighted average radon concentration was estimated for the 30-year period of interest using the times spent at each address as the weights. Estimates of the lung cancer ERR per unit concentration in air were obtained using linear logistic regression on the assumption that the subjects' radon exposure was a continuous variable. The regressions also included interactions with the factors noted earlier and other variables. The mean seasonally adjusted radon level in the 9,448 residences was 58 Bq/m³, with a maximum of 3,549 Bq/m³. The ERR for lung cancer risk was 0.08 (95% CI: -0.03, 0.20) per 100 Bq/m³ after adjustments for age, sex, smoking status, county of residence and social class. After further adjustment for the uncertainty in estimates of exposure, the authors reported an ERR of 0.12 (95% CI: -0.05, 0.33) per 100 Bq/m³. The observed variations among different tumour types were no larger than would be expected by chance.

442. A case-control study for the period 1990-1996 in western Germany was carried out by Kreienbrock et al. [K18]. Detailed demographics and information on potential confounding factors (smoking and occupational asbestos exposure) were collected, as was detailed information on residences occupied in the previous 35 years. Radon measurements were made over 1 year using nuclear track detectors exposed in the living rooms and bedrooms of participants' current and previous homes. (Radon exposure was quantified in two ways: a time-weighted average of the living room and bedroom radon concentrations in the last residence; and an estimation of the time-weighted average cumulative radon exposure in the 5-15 years prior to the interview date.) An attempt was made to correct for home alterations and changes to home ventilation. There were 1,449 cases and 2,297 controls for the entire study area, with a subgroup in a radon-prone area consisting of 365 cases and 595 controls. In the overall study area, women constituted 235 of the cases and 432 of the controls. Among the men, 2% of the cases and 23% of the controls had never smoked. Among the women, 31% of the cases and 60% of the controls had never smoked. Occupational exposure to asbestos was identified for men

only (30.6% of the cases and 19.8% of the controls). Rate ratios and 95% CIs were calculated using logistic regression. All ORs were adjusted for age, sex, smoking and occupational asbestos exposure. The authors noted two results. In the entire study area, no rate ratios were significantly different from unity, while in the radon-prone areas, for an increase in radon concentration of 100 Bq/m³, an ERR of 0.13 (95% CI: -0.12, 0.46) was obtained for the exposure assessment based on the last residence only, and an ERR of 0.09 (95% CI: -0.14, 0.38) was obtained for the assessment based on cumulative exposure in the 5–15 years prior to the interview date. After conducting sensitivity analyses, the authors attributed the absence of an observable risk in the radon-prone areas to inaccuracy of radon exposure assessment. The inaccuracy increases if the variation in exposure within the study population is low, which was the case in the entire study area.

443. Kreuzer et al. [K17] reported on a study of residential radon concentrations and lung cancer in Saxony and Thuringia, which are areas in eastern Germany with naturally elevated radon levels. The study included 1,192 cases and 1,640 controls. Alpha track radon detectors were placed for 1 year in the living room and bedrooms of the subjects' homes. Radon exposure was calculated as the time-weighted average of radon concentrations in each room. The authors also calculated a time-weighted average radon concentration for the whole of the study period, which was 5–35 years prior to the interview date. Mean radon concentrations were 76 Bq/m³ among cases and 74 Bq/m³ among controls. Detailed information on demographics and potential confounding factors was obtained using a standardized questionnaire similar to the one used in the study by Kreienbrock et al. [K18]. Approximately 12% of cases and 14% of controls were women. Among the men, only 2% of the cases and 26% of the controls had never smoked. Among the women, 51% of the cases and 77% of the controls had never smoked. ORs and 95% CIs were calculated using conditional logistic regression (for example, smoking and work history with asbestos exposure were quantified). The OR for smokers compared with those who had never smoked was 18 (95% CI: 12, 29) for men and 2.8 (95% CI: 1.70, 4.8) among women. For men, about 30% of cases and 28% of controls had occupational asbestos exposure. Asbestos exposure in women was negligible. ORs adjusted for age, sex, smoking and occupational asbestos exposure, as well as 95% CIs were calculated using conditional logistic regression. Overall, an ERR of 0.08 (95% CI: -0.03, 0.20) per 100 Bq/m³ and 0.09 (95% CI: -0.06, 0.27) per 100 Bq/m³ was found for subjects with complete measurements for all 30 years. Smoking acted as a negative confounder, and there was a moderate increase in lung cancer, which for small cell cancers was pronounced.

444. Conrady et al. [C9, C32] conducted a residential radon study among females living in the Schneeberg and Schlema areas of Saxony in eastern Germany. This study looked at all female lung cancer cases in the study area between 1 January 1952 and 31 December 1989. The final group included

72 cases and 288 controls. About 78% of cases and 94% of controls were non-smokers. Radon measurements were made using alpha track detectors. For 24 houses in Schneeberg, the authors compared the radon data with measurements of ²¹⁰Po in glass samples, which were used for backward extrapolation of the radon concentrations, and concluded that indoor exposure conditions had been "stable" over the study period. They also noted a large variation in indoor radon levels by week and by season, and cautioned that, while 1-year measurements will yield "quite constant" values, shorter-term measurements could be misleading. The authors reported radon levels of 730 Bq/m³ in case homes in Schneeberg, about 540 Bq/m³ in "register" controls and about 290 Bq/m³ in "hospital" controls. Logistic regression was used in the risk analysis. Elevated odds ratios (OR = 4.35; 95% CI: 1.47, 12.90 and OR = 1.94; 95% CI: 0.59, 6.33) were observed in the two highest exposure categories (radon concentrations of 1,000–1,500 Bq/m³ and >1,500 Bq/m³) compared with the reference category of 50 Bq/m³. No elevation in risk was evident in the lower exposure categories, but this might be attributed to the low statistical power. When data were restricted to non-smokers and lung cancer cases with histological confirmation (38 cases, 172 controls), the risk remained elevated in the highest exposure category, with a radon concentration of >1500 Bq/m³, showing a borderline significant trend only among small cell lung cancers.

445. Pisa et al. [P6] carried out a residential radon study in an alpine valley in Italy where residential radon levels averaged 132 Bq/m³, compared with the Italian national average of 77 Bq/m³. "Ecological" evidence suggested the possibility of a weak association between lung cancer and residential radon. Measurements of residential radon levels were carried out over the course of 1 year using alpha track detectors in the bedrooms of the most recent residence for each subject. Between 1 January 1987 and 31 December 1993, 224 residents in the study area died from lung cancer. Of these, interviews were completed for 138 cases (122 men and 16 women) and 291 controls matched for sex and year of birth. Lifetime smoking, dietary variables and occupational history were all considered in the statistical analysis, in which multiple unconditional logistic regression, with radon treated as a continuous variable, was used to estimate the OR and its 95% CI. The OR showed a strong association with smoking in both males and females. No association was observed between lung cancer and exposure to potential occupational carcinogens. An association between radon and lung cancer was seen in men only. Men who lived in homes with radon levels of 40–199 Bq/m³ (mean 80.4 Bq/m³) showed an OR, adjusted for age, sex and smoking, that was approximately a factor of 2 greater than that of men who lived in homes with radon levels of below 40 Bq/m³. The authors reported an OR of 1.4 (95% CI: 0.3, 6.6) per 100 Bq/m³ for the group as a whole.

446. Oberaigner et al. [O2, S59] carried out a residential radon study in the highly radon-prone district of Imst in Tyrol, Austria. Lung cancer deaths and a sample of deaths with causes other than lung cancer during the years 1970–1992

(causes of death highly related to smoking were excluded) were matched by age, sex and year of death. The next of kin of the cases and the controls were interviewed for residential history, smoking and other risk factors. Radon concentrations were measured in the last residence for approximately 1 year by means of alpha track detectors placed in the bedrooms and the living rooms. The study included 194 cases and 198 controls. The percentage of men was 88% among both cases and controls. Measurements covered 68% (cases) and 75% (controls) for the period 5–35 years before death. The mean radon concentrations were 266 Bq/m³ among the cases and 123 Bq/m³ among the controls. ORs were estimated using conditional regression models adjusted for smoking and occupation. The ERR at an increased radon exposure of 100 Bq/m³ was 0.25 (95% CI: 0.08, 0.43).

447. Wichmann et al. [S61, W19] reported the results of a pooled analysis of the two German radon studies. These two case–control studies were performed during 1990–1997 in eastern and in western Germany [K17, K18], with identical study design. The original data were extended and pooled, and included a total of 2,963 incidences of lung cancer and 4,232 population controls. Radon measurements were carried out over the course of 1 year in houses occupied during the 5–35 years prior to the interview date. Conditional logistic and linear relative risk regression was used for the analysis. Measurements showed an average radon exposure of 61 Bq/m³. The smoking- and asbestos-adjusted ORs were 0.97 (95% CI: 0.85, 1.11) for radon concentrations of 50–80 Bq/m³, 1.06 (95% CI: 0.87, 1.30) for radon concentrations of 80–140 Bq/m³ and 1.40 (95% CI: 1.03, 1.89) for radon concentrations of above 140 Bq/m³, compared with the reference category with radon concentrations of <50 Bq/m³. The linear increase in the OR was 0.10 (95% CI: –0.02, 0.30) per 100 Bq/m³ for all subjects and 0.14 (95% CI: –0.03, 0.55) per 100 Bq/m³ for less mobile subjects who had lived in only one home in the previous 5–35 years. The risk coefficients generally were higher when measurement error in the radon concentrations was reduced by restricting the population to those for whom good measurements had been made. With respect to histopathology, the risk for small cell carcinoma was much higher than for other subtypes.

448. Bochicchio et al. [B43] reported on a case–control study of lung cancer and residential radon in Lazio, central Italy, characterized by high levels of indoor radon and by Mediterranean climate and diet. All subjects — 384 cases and 404 controls, aged 35–90 years — were recruited in the hospital. Detailed information regarding smoking, diet and other risk factors were collected by direct interview. Residential history during the 30-year period ending 5 years before enrolment was ascertained. In each dwelling, radon detectors were placed in both the main bedroom and the living room for two consecutive 6-month periods. A quality assurance programme was set up for radon measurements [B41]. ORs and 95% CIs for time-weighted radon concentrations were computed using both categorical and continuous unconditional logistic regression analysis and adjusting for smoking, diet and other variables. Approximately 89% and 91% of the

cases and the controls, respectively, were concluded to have good radon exposure data. The adjusted ORs were 1.30 (1.03, 1.64), 1.48 (1.08, 2.02), 1.49 (0.82, 2.71) and 2.89 (0.45, 18.6) for radon concentrations in the range 50–99, 100–199, 200–399 and above 400 Bq/m³, respectively, compared with the reference category with radon concentrations of 0–49 Bq/m³ (OR = 1; 0.56, 1.79). The adjusted odds ratio risk for 100 Bq/m³ was 0.14 (–0.11, 0.46) for all subjects, 0.24 (–0.09, 0.70) for subjects with complete radon measurements and 0.30 (–0.08, 0.82) for subjects who had lived in no more than one or two dwellings. There was a tendency towards higher risk among subjects with low to medium consumption of dietary antioxidants (EOR = 0.32; –0.19, 1.16). In conclusion, both categorical and continuous analyses clearly support an association between residential radon concentration and lung cancer. Moreover, subjects with a presumed lower uncertainty in the concentration assessment showed a higher risk. Finally, this is the first study indicating that dietary antioxidants may act as an effect modifier for radon.

449. Baysson et al. reported on indoor radon concentration and lung cancer in France [B33]. The study took place from 1992 to 1998 in French districts with elevated concentrations of radon. The study included 486 lung cancers cases recruited from university hospitals, and 984 controls. Subjects were eligible if they had lived in the study area for at least 25 of the previous 35 years. Full residential histories covering the previous 30 years were obtained, with specific information collected for the dwellings that had been occupied for more than 1 year. Radon concentrations were measured using track etch detectors placed for a 6-month period in the current and the former residences. The authors estimated a time-weighted average radon concentration for each subject for the period of 5–30 years prior to the interviews. Finally, the authors considered the effect of smoking and occupational exposure to carcinogens. The ORs and 95% CIs were calculated using logistic regression with adjustments for sex, age, region, smoking and occupational exposure. Adjusted odds ratio risks for 100 Bq/m³ were estimated for all subjects as OR = 1.04 (95% CI: 0.99, 1.11) and for subjects with complete measurements (850 subjects) as OR = 1.07 (95% CI: 1.0, 1.14). The French study supports a small excess risk of lung cancer arising from exposure to residential radon.

C. Ecological studies of residential radon

450. The limitations of ecological epidemiological analyses are well discussed in references [C20, L8, L28, U2, U5], as well as in annex A, “Epidemiological studies of radiation and cancer”. Nonetheless, a few short comments on ecological versus case–control studies are appropriate here. Radon studies that rely on averages over geographical areas are especially vulnerable to biases that are not present in results based on individual-level data such as those used in case–control or cohort studies. This is because radon levels are highly variable even within limited geographical areas. In addition, smoking is the major cause of lung cancer, and risk is highly dependent on individual smoking habits. The

distribution of smoking habits across the population in a particular geographical area is an important confounding factor.

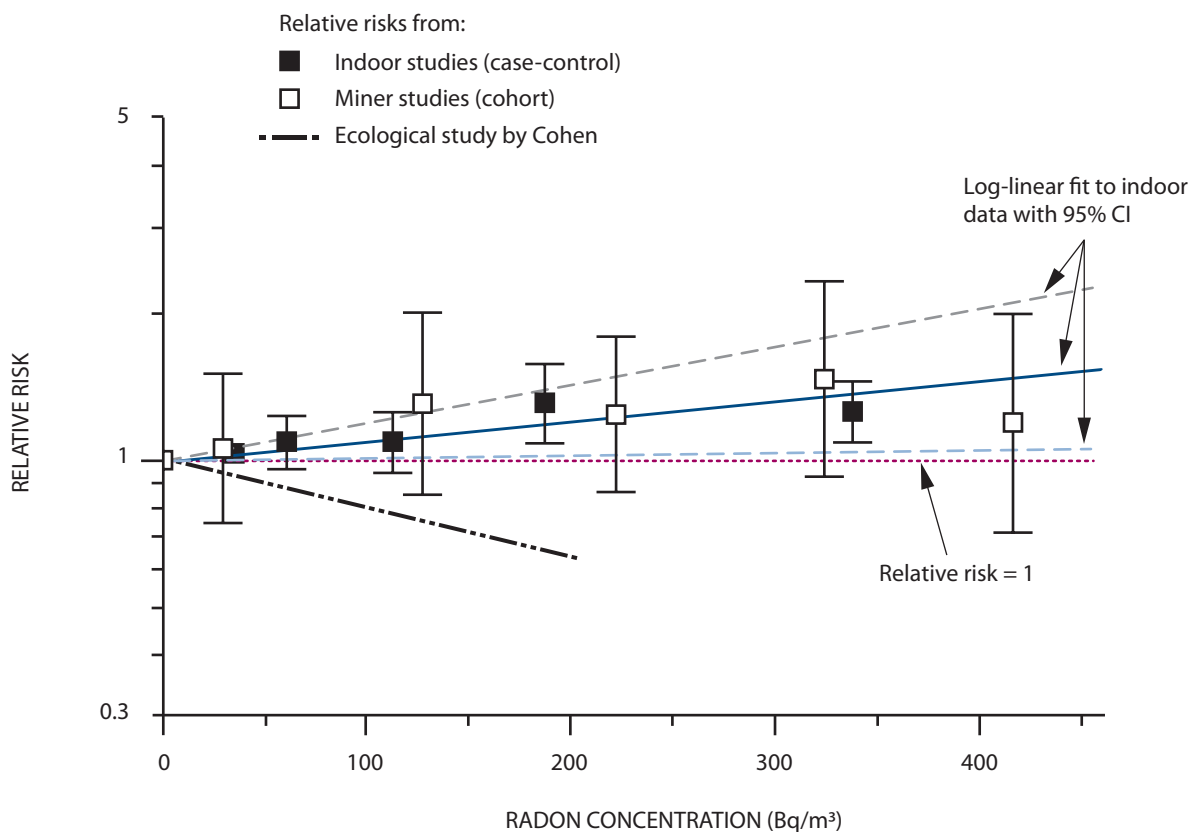
451. Stidley and Samet [S49] reviewed 15 ecological studies of residential radon exposure from a number of countries. A positive association between radon concentration and lung cancer was observed in 7 of the studies, no association was observed in 6 and statistically significant inverse relationships were observed in 2. The authors discussed methodological issues associated with the ecological studies and suggested that ecological studies should be given little weight in assessing the potential risks of residential radon. In a subsequent paper [S3], the authors showed by simulation that even modest levels of error in exposure or misspecification of the risk model could introduce significant biases into the results of ecological studies. As discussed in section B, the pooled analysis of case-control studies in Europe [D17] and North America [K1] have gone to considerable effort to correct for uncertainty in radon concentrations. Further discussion of this limitation of ecological studies is provided in annex A.

452. Cohen used ecological epidemiological studies to investigate the linear no-threshold theory (LNT) by looking

at whether (county average) lung cancer rates in United States counties decreased with increasing (county average) radon concentration. Cohen's work, involving 275,000 measurements in all 50 states [C23, C24, C25, C26, C27, C28], generated a great deal of discussion, including references [A21, F8, F12, G15, G16, L24, L28, L29, L30, S48]. The focus of the discussion was the use of ecological rather than analytical studies to investigate the potential risks from residential radon. Puskin [P10] provided a plausible explanation of Cohen's observation of a negative association (see figure XV) between lung cancer and residential radon concentration. However, Cohen points out that, while an ecological study such as that given in reference [C24] cannot determine a risk versus exposure relationship, it can test the LNT theory. Puskin [P10] found that the inverse association between lung cancer and residential radon was also seen in other smoking-related cancers not related to radon exposure. The result suggested that Cohen's observations could largely be explained by a negative correlation between smoking and radon exposure [P10]. On the other hand, Cohen [C37] argued that Puskin's observation of similar dependence on radon exposure for lung cancer and for other smoking-related cancers is not affected by data on smoking prevalences.

Figure XV. Risk estimates of lung cancer from exposure to radon (adapted from reference [L4]).

Shown are the summary relative risks from meta-analysis of eight indoor radon studies and from the pooled analysis of underground miner studies, restricted to RDP exposures of less than 50 WLM [L19], together with the estimated linear relative risk from the correlation study by Cohen [C24]. Note that references [L4] and [C20] both show Cohen's data extrapolated to beyond where they were actually analysed, about 200 Bq/m³. The figure has been adjusted to reflect this.

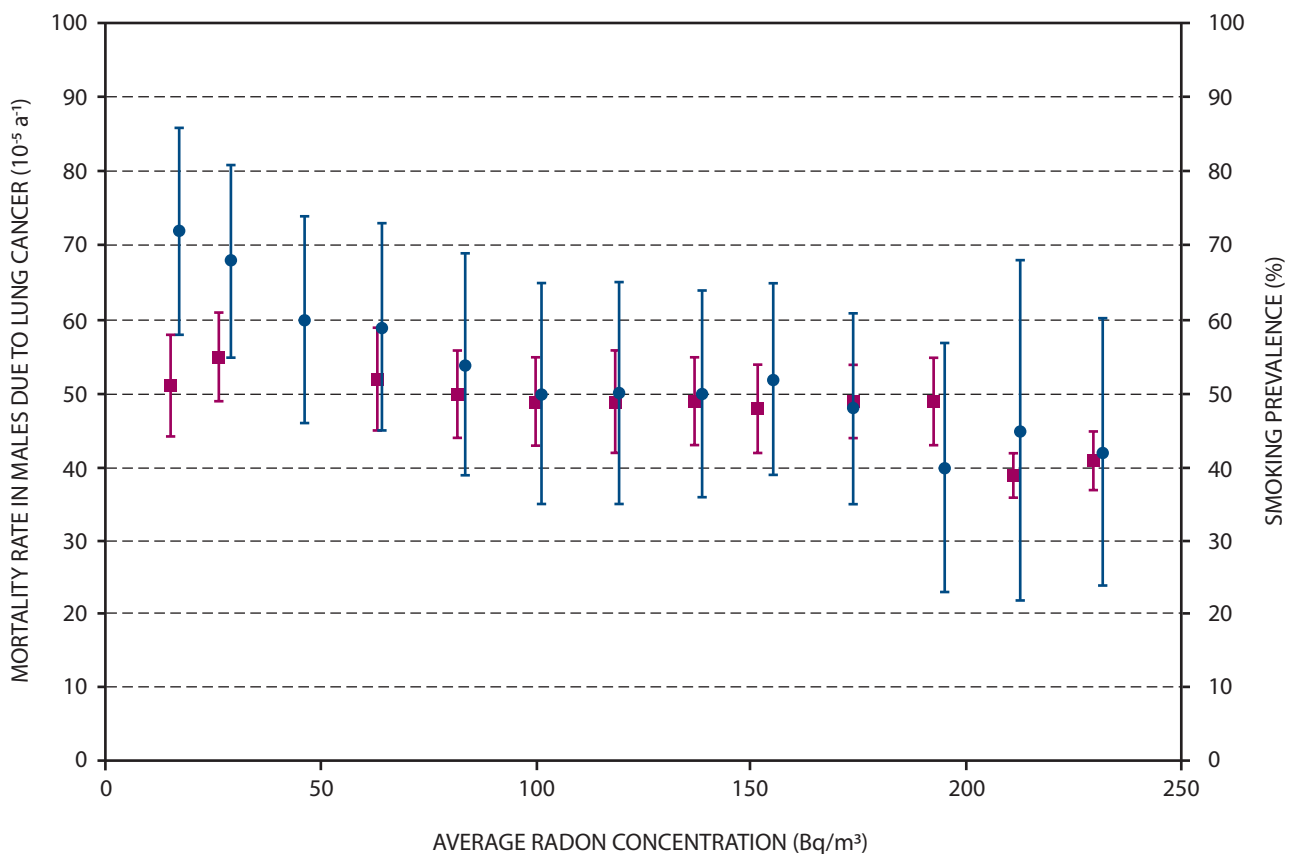


453. Heath et al. [H29] discussed exposure to residential radon and lung cancer risk, as well as providing commentary on Cohen's county-based studies [C24, C25, C35]. To understand Cohen's work, the authors obtained the data sets on which his study was based and carried out an independent analysis of lung cancer mortality on a subset of the data (lung cancer mortality for 1970–1979). Figure XVI (adapted from reference [H29]) shows the results of the analysis of lung cancer mortality in relation to (ecological) radon levels but uncorrected for smoking (solid circles). The same figure shows the pattern of smoking frequencies superimposed (solid squares). The smoking frequencies shown in figure XVI paralleled the pattern of lung cancer except at the lowest radon levels, and

this led the authors to suggest that confounding by smoking is particularly important at low levels of radon. Cohen argued that figure XVI is meaningless because the effects of smoking prevalences were fully taken into account in his analyses, and these smoking prevalences were derived from three independent sources, all giving the same results. He also argued that no remotely plausible correlations between radon concentrations and smoking prevalences or intensities of smoking could substantially change his results. Nonetheless, Heath et al. [H29] suggested that Cohen's ecological studies are of limited value in assessing the risk from residential radon exposure because of their reliance on grouped data, coupled with confounding by cigarette smoking.

Figure XVI. Average annual lung cancer mortality per 100,000 males, 1970–1979 (●), and average percentage of cigarette smokers among males (■) within counties, grouped by average county radon level.

Confidence intervals are expressed as the standard deviation of the distribution for each county group (adapted from [H29]).



454. Large ecological studies such as Cohen's [C24, C25], which show an inverse relationship between county averages of radon exposure and lung cancer mortality, continue to generate controversy, despite the results of the numerous analytical (case-control) studies such as those discussed in the preceding section. As discussed in references [S3, S49] and in annex A, ecological studies have a number of methodological challenges, notably their inability to adjust for mobility and smoking habits. The pooled studies involved control of both individual exposures and smoking habits,

and provide a methodologically sound basis for estimating the risks from residential radon.

D. Overall evaluation of residential radon studies

455. Baysson and Tirmarche [B29] provided an overview of the case-control studies of residential radon concentration and lung cancer carried out since 1990. They suggested that the results of these studies indicated a positive association

between lung cancer risk and residential radon concentration with, an EOR of 0.06–0.09 per 100 Bq/m³. Table 22 and figure XVII summarize much of the currently available data on the risks per unit concentration in air from residential case-control studies. In considering the residential case-control studies summarized above, it is important to understand that some studies have greater statistical power than others, owing to factors such as study size and data quality. The key residential case-control studies were included in the pooled analyses of Darby et al. [D17, D21] and Krewski et al. [K1, K26], as previously discussed. Uncertainty in exposure assessment is a factor in residential radon studies, as are the effects of smoking (both active and passive). However, when the results of individual studies are pooled and analysed in a consistent format, there is a coherent trend of increasing risk of lung cancer with increased radon exposure.

456. Bochicchio [B42] reviewed the application of solid-state nuclear detectors in residential radon studies. He argued that exposure uncertainty is generally non-differential and therefore not only adds to the uncertainty in estimated risks but also introduces a bias towards underestimation of risks. Among the factors discussed by Bochicchio are: seasonal variability, which can vary from house to house depending on the source of the radon; the characteristics of the house and of people's living habits; and the difficulties in retrospective dosimetry. Most of the radon studies reviewed by Bochicchio covered periods of up to 35 years, during which there may have been changes to the house,

the house ventilation system and residents' habits, all of which can affect both the levels of radon and the levels of RDP exposure. Further discussion of the effects of measurement error in assessing radon exposure is provided in references [H37, H41]. As noted previously, the pooled studies in Europe [D17, D21], North America [K1, K26] and China [L26] have attempted to account for measurement error uncertainty (uncertainty in the measurement of radon concentration and exposure) by restricting the assessment to individuals who had lived in only one or two homes. In addition, as noted earlier, Darby et al. attempted a statistical correction for measurement uncertainty [D21].

457. Bochicchio et al. also provided a convenient summary of residential case-control studies [B44]. The OR per unit concentration in air and 95% CIs for each study are shown in figure XVII. Also shown in figure XVII are the results of several meta-analyses and three pooled analyses in Europe, North America and China. These include the Lubin and Boice meta-analysis of the residential studies [L4], Lubin's analysis of North American and Chinese residential studies [L36], the meta-analysis of 17 case-control studies by Pavia et al. [P17], the Wichmann et al. pooled study of residential exposure in Germany [W19], two pooled studies of 13 case-control studies in Europe by Darby et al. [D17, D21], and the pooled study by Krewski et al. [K1, K26] of 47 case-control studies in North America. All of the studies included in the meta-analyses by Wichmann et al. [W19] and Lubin and Boice [L4] are also included in the pooled analyses [D17, K1].

Table 22 Indoor radon case-control studies (adapted from reference [B29])

Reference		Region	Population	Number of cases (controls)	Radon measurements	OR ^a	95% CI
Year	Author						
1990	Schoenberg et al. [S42]	New Jersey, United States	Women	480 (442)	1 year	1.49	0.89, 1.89
1990	Blot et al. [B25]	Shenyang, China	Women	308 (356)	1 year	0.95	Undefined 1.08 ^g
1992	Pershagen et al. [P16]	Stockholm, Sweden	Women	201 (378)	1 year	1.16	0.89, 1.92
1994	Pershagen et al. [P11]	Sweden	Both sexes	1281 (2576)	3 months	1.10	1.01, 1.22
1994	Letourneau et al. [L25]	Winnipeg, Canada	Both sexes	738 (738)	1 year	0.98	0.87, 1.27
1994	Alavanja et al. [A19]	Missouri, United States	Women, non-smokers	538 (1183)	1 year	1.08	0.95, 1.24
1996	Auvinen et al. [A5]	Finland	Both sexes	517 (517)	1 year	1.11	0.94, 1.31
1996	Ruosteenoja et al. [R3]	Southern Finland	Men	164 (331)	2 months	1.80	0.90, 3.50
1997	Lagarde et al. [L1]	Sweden	Both sexes	1281 (2576)		1.17 ^b	1.03, 1.37
1998	Darby et al. [D15]	South-west United Kingdom	Both sexes	982 (3185)	6 months	1.08 1.12 ^b	0.97, 1.20 0.95, 1.33
1999	Alavanja et al. [A18]	Missouri, United States	Women	247 (299) 372 (471)	1 year	0.85 ^c 1.63 ^d	0.73, 1.00 1.07, 2.93

Reference		Region	Population	Number of cases (controls)	Radon measurements	OR ^a	95% CI
Year	Author						
2000	Field et al. [F11]	Iowa, United States	Women	413 (614)	1 year	1.24	0.95, 1.92
2001	Kreienbrock et al. [K18]	Western Germany	Both sexes	1 449 (2 297)	1 year	0.97 ^e 1.09 ^f	0.82, 1.14 0.86, 1.38
2001	Pisa et al. [P6]	Trentino, Italy	–	138 (291)	1 year	1.40	0.3, 6.6
2001	Lagarde et al. [L32]	Sweden	Non-smokers	436 (1649)	3 months	1.10	0.96, 1.38
2001	Tomasek et al. [T10, T29]	Pluton, Czech Republic	–	210	1 year	1.087	1.017, 1.208
2002	Wang et al. [W13]	Gansu, China	Both sexes	768 (1 659)	1 year	1.19	1.05, 1.47
2002	Barros-Dios et al. [B26]	Spain	–	163 (241)	150 days	2.48	1.29, 6.79
2002	Lagarde et al. [L31]	Sweden	Non-smokers	110 (231)	3 months	1.33 ^c 1.75 ^d	0.88, 3.0 0.96, 5.30
2002	Oberaigner et al. [O2]; Schaffrath et al. [S59]	Tyrol, Austria	Both sexes	194 (198)	1 year	1.25	1.08, 1.43
2003	Kreuzer et al. [K17]	Eastern Germany	Both sexes	1 192 (1 640)	1 year	1.08	0.97, 1.20
2004	Wichmann et al. [S61, W19]	Eastern and western Germany	Both sexes	2 963 (4 232)	1 year	1.1	0.98, 1.3
2004	Baysson et al. [B33]	France	Both sexes	486 (984)	6 months	1.04	0.99, 1.11
2005	Bochiccio et al. [B43]	Lazio, Italy	Both sexes	384 (404)	6 + 6 months	1.14	0.89, 1.46

^a OR at 100 Bq/m³ (calculated).

^b Analysis including measurement error.

^c Analysis based on air monitors.

^d Analysis based on surface monitors.

^e Entire study period, 5–15 years.

^f Radon-prone areas.

^g As reported in reference [B29], Blot et al. [B25] found no association between lung cancer and radon.

458. Figure XVII is a graphical presentation of the data for the individual studies shown in table 22. In addition, the results of pooled analyses are also shown in the figure along with an estimate of the ERR from the combined pooled studies (see table 22 and previous paragraph). As discussed earlier, corrections for measurement uncertainty increase the predicted ERR somewhat, as shown in table 22.

459. Lubin [L36] discussed several case–control studies of residential radon in North America and two in China. He acknowledged the inherent difficulties in establishing an association between lung cancer risks and residential radon concentration as a consequence of the overall expected risk from radon and the large uncertainties. He noted the latest studies in dosimetry, and suggested that pooling may permit more detailed assessment of the risk. This has been demonstrated, for example, in the pooled residential case–control studies in Europe [D17, D21], North America [K1, K26] and China [L26].

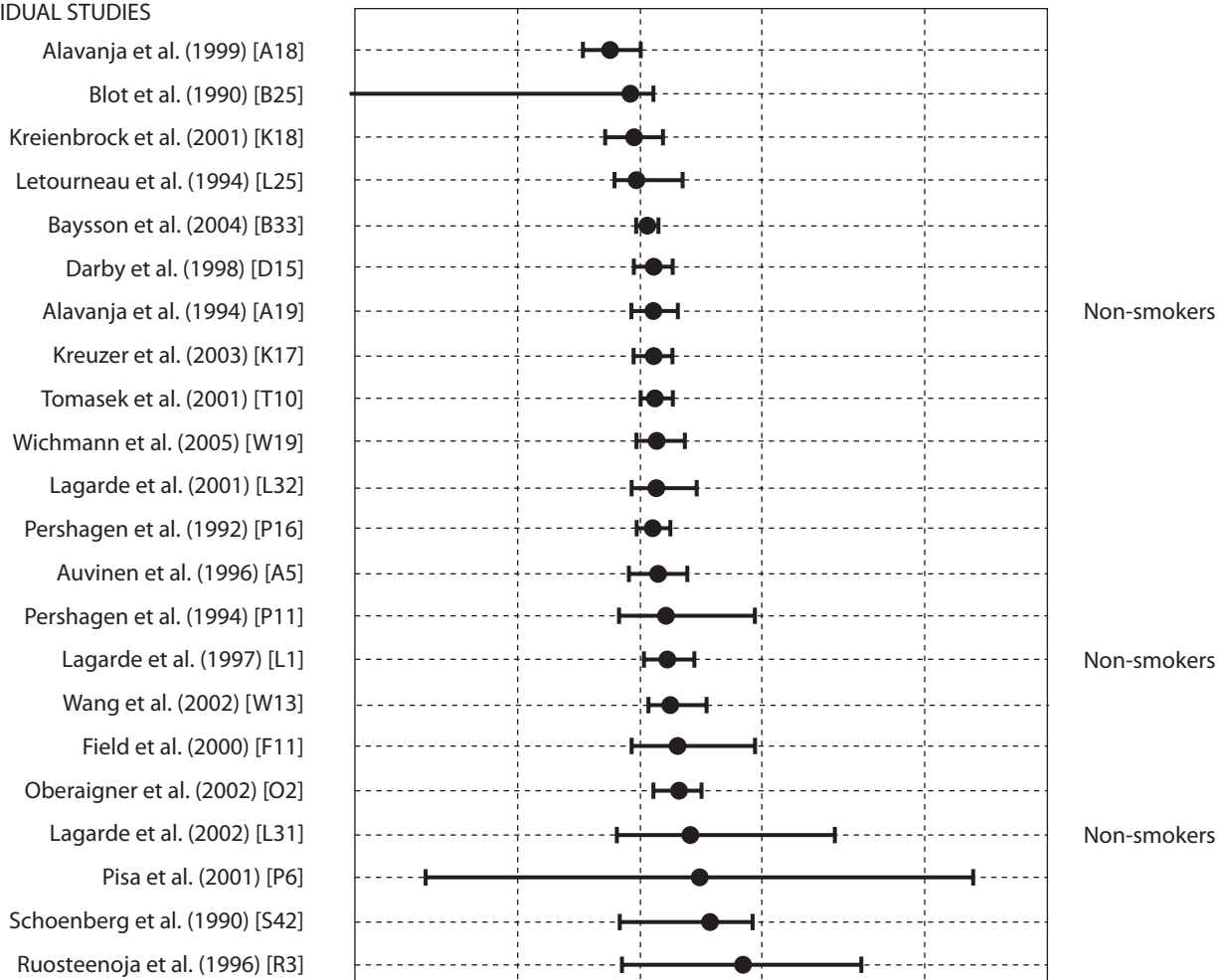
460. Lubin and Boice [L4] undertook a meta-analysis of eight case–control residential radon studies carried out in China, Finland, Sweden, the United Kingdom and the

United States. The meta-analysis included those studies with 200 or more cases and long-term measurements of indoor radon concentrations. The pooled study contained in total 4,263 lung cancer cases and 6,612 controls. Using published data from each of the eight studies, the authors carried out regression analyses. The risks (OR) and 95% CIs for each of the studies are also shown in figure XVII. As was shown also in figure XIV, such results are generally consistent with extrapolations from miner studies based on the results of BEIR IV and BEIR VI [C19, C20]. As noted by Lubin and Boice, the CIs for the individual studies are large and include a relative risk of unity, which is consistent with the possibility that there is no effect from exposure to residential radon. Overall, however, the authors found that the excess risk from the combined data was significantly different from zero. For residential exposure to 150 Bq/m³, the authors estimated a combined OR of 1.1% (95% CI: 1.0, 1.3). Overall, Lubin and Boice concluded that the risk from residential radon is unlikely to be larger than that predicted on the basis of the miner data, that the negative exposure response seen in some studies is likely to have been due to exposure misclassification or uncontrolled confounding factors, and that their results are consistent with a small effect on lung cancer from residential radon [L4].

Figure XVII. Risk estimates from residential radon studies [B29].

Shown are the summary relative risks for exposure at a radon concentration of 100 Bq/m³ (except for references [L4, P17] at 150 Bq/m³) and the corresponding 95% confidence intervals.

INDIVIDUAL STUDIES



POOLED ANALYSES

- Lubin & Boice (1997) [L4]
- Pavia et al. [P17]
- German [W19]
- Chinese¹ [L26]
- North American² [K1, K26]
- European³ [D21]

¹Includes [B25, W13]

²Includes [A18, A19, F11, L25, S42, S76]

³Includes [A5, B26, B33, B43, L32, O2, P11, P16, R3, T29, W19]

461. Pavia et al. [P17] reported the results of a meta-analysis of residential exposures to radon gas and lung cancer from 17 case-control studies. Their analysis suggested an association between residential radon concentration and lung cancer. A weighted log-linear regression analysis was used to develop estimates of pooled ORs for exposure to a radon concentration of 100 Bq/m³, reported as OR = 1.15 (95% CI: 1.07, 1.24). The authors indicated that not all studies were adjusted for smoking and that their results cannot exclude the possibility that their inability to fully adjust for smoking (or other confounders) could account for the increased risk seen in their study.

462. Darby et al. [D17] reported a pooled analysis of 13 European case-control studies of the risk of lung cancer from residential radon. This analysis is of great interest, especially since heterogeneity among the results of the various individual studies disappeared once the data from the 13 studies were put into a common format and analysed in a consistent manner. The study included 7,148 lung cancer cases and 14,208 controls. The mean radon level measured using long-term alpha track-etch detection in the houses of the control group was 97 Bq/m³, and in the houses of lung cancer cases was 104 Bq/m³. Radon exposures during the previous 5–34 years were considered in the analysis. The authors investigated the association between radon concentration and lung cancer using two models. In one model the risk of lung cancer was proportional to $(1 + \beta\chi)$, where χ is the measured radon level and β is the proportional risk factor per unit increase in radon. The second model subdivided cases and controls by categories of radon exposure. The authors noted that the dose-response relationship appeared to be linear with no threshold and did not depend on smoking status. Before correcting for random uncertainties in measuring radon concentrations, the authors reported an increased excess odds ratio (EOR) of about 0.08 (95% CI: 0.03, 0.16) per 100 Bq/m³. Figure XVIII shows the relative

risk of lung cancer according to the time-weighted average observed residential radon concentration, after stratification by study, age, region of residence and smoking habits. The relative risks and 95% CIs for categories of radon concentration are shown in table 23. When the analysis was repeated with only those exposed below a radon concentration of 200 Bq/m³, the dose-response relationship remained statistically significant ($p = 0.04$).

Figure XVIII. Relative risk of lung cancer versus observed residential radon concentration.

The estimated linear relationship $RR = 1 + 0.00084\chi$ (solid line), with 95% confidence limits (dashed lines). The relative risk is equal to 1 at 0 Bq/m³ (adapted from figure 2 of reference [D21]).

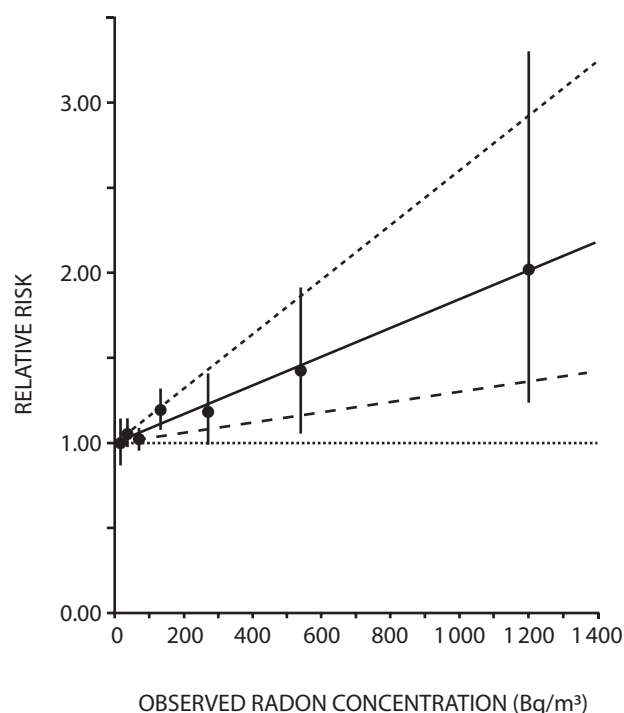


Table 23 Relative risk of lung cancer according to time-weighted average observed residential radon concentration (adapted from table 16 of reference [D21])

Observed radon concentration ^a (Bq/m ³)	Number of cases	Number of controls	Mean observed radon concentration	Relative risk (95% CI)
<25	566	1 474	17	1.00 (0.87, 1.15)
25–49	1 999	3 905	39	1.06 (0.98, 1.15)
50–99	2 618	5 033	71	1.03 (0.96, 1.10)
100–199	1 296	2 247	136	1.20 (1.08, 1.32)
200–299	434	936	273	1.18 (0.99, 1.42)
400–799	169	498	542	1.43 (1.06, 1.92)
≥800	66	115	1 204	2.02 (1.24, 3.31)
Total	7 148	14 208		

^a Observed radon concentration for each address in the 30-year period ending 5 years prior to the index date weighted according to the length of time that the person lived at that address.

463. When the analysis was restricted to people who had lived in at most two residences during the previous 30 years, the excess (EOR) increased to 0.094 (95% CI: 0.034, 0.175) per 100 Bq/m³. Finally, after correction for random uncertainties in the assessment of radon concentrations, the dose-response relationship remained linear, but the relative risk doubled to 0.16 (95% CI: 0.05, 0.31) at 100 Bq/m³. Darby et al. also discussed the combined effect of smoking and residential radon exposure on the absolute risk of lung cancer, and indicated, using the same relative risk factor of 0.16 for lifetime exposure (taken as 75 years) to a radon concentration of 100 Bq/m³, that the risks of lung cancer in lifelong non-smokers and cigarette smokers would be about 0.47% and 11.6%, respectively. Expressed differently, almost all of the risk accrues to the population of smokers.

464. Krewski et al. [K1, K26] reported a pooled analysis of residential radon exposure and lung cancer risk in seven case-control studies in North America. The combined study included some 3,662 cases and 4,966 controls. Residential radon levels were determined using long-term alpha track detection spanning 12 months. The analysis focused on exposures 5–30 years prior to the interview date. Data were analysed using conditional likelihood regression and the linear model $OR(\chi) = 1 + \beta\chi$, where χ is the cumulative radon exposure in the previous 5–30 years. The authors stated that the EORs for individual studies ranged from 0.01 (95% CI: <0.00, 0.42) per 100 Bq/m³ in a Missouri study [A18] to 0.56 (95% CI: 0.22, 2.97) per 100 Bq/m³ in a New Jersey study [S42]. The authors also investigated potential modifying effects of smoking and demographic factors, and noted that, while there was no apparent heterogeneity in EOR by sex or education level, there was some suggestion of a decreasing radon-associated risk with increasing age. The authors also indicated that they had found no significant differences in EOR with measures of smoking status. Overall, an EOR = 0.11 (95% CI: 0.00, 0.28) per 100 Bq/m³ was estimated. The authors also indicated that analyses restricted to subsets of the data with “presumed more accurate radon dosimetry” resulted in increased estimates of the EOR, of 0.18 (95% CI: 0.02, 0.43) per 100 Bq/m³.

465. Smoking is a potential confounding factor in both the residential and the miner studies. However, there is far more information about smoking histories available in the residential studies. Furthermore, several analyses show that

the information collected on smoking habits gives estimates of the risks of lung cancer that are very much in line with those of other studies of lung cancer and smoking [C20, C39, P20].

466. Becker [B34] reviewed the radon experience of miners, residential radon exposure and “the therapeutic use” of radon. Becker noted that BEIR VI [C20] indicated that smoking has a much greater risk of lung cancer than does exposure to radon. He then argued that the correction of the miner data for smoking is complicated by the under-reporting of actual smoking, and hence that the uncertainties associated with retrospective analysis of smoking greatly confound the analysis of radon risk in miners. Similar arguments were made about the residential radon studies, noting that uncertainties in smoking “by far dominate” the uncertainties associated with the retrospective analysis of exposure to radon.

467. **Conclusion.** The main studies of residential radon are the pooled analyses of European [D17, D21], North American [K1, K26] and Chinese [L26] residential case-control studies. These studies indicate a significant association between the risk of lung cancer and exposure to residential radon. The studies also examined the effect of restricting analyses to those who had lived in at most two residences. In addition, Darby et al. [D17, D21] also carried out analyses that adjusted for exposure uncertainty. Both the European and the North American studies have looked at the estimated relative risk from radon for individuals with different smoking habits and demonstrated not only that there is no significant heterogeneity, but that the risk estimates on the relative scale are very similar for individuals in different smoking categories.

468. Table 24 shows the ERR per unit residential radon concentration from three pooled analyses of case-control studies. The pooled analyses also reported the ERR for analyses restricted to individuals who had lived in only one or two residences and hence, whose radon exposures are presumed to be more precisely known than those of individuals who changed residences many times. The ERR estimates from the restricted analyses were higher than the ERR estimates from the primary analyses. The analysis by Darby et al. used a regression model correction for exposure uncertainty, which approximately doubled the ERR obtained from analysis of the primary data [D21].

Table 24 ERR per unit radon concentration in air (per 100 Bq/m³) and 95% confidence intervals from combined residential radon studies

Study	Primary analysis	Restricted analysis	Exposures adjusted for uncertainty
European [D17, D21]	0.084 (0.03, 0.158)	0.094 ^a (0.034, 0.175)	0.16 ^c (0.05, 0.31)
North American [K1, K26]	0.11 (0.00, 0.28)	0.18 ^a (0.02, 0.43)	
Chinese [L26]	0.133 (0.01, 0.36)	0.319 ^b (0.07, 0.91)	
Combined	0.093 (0.04, 0.15)	0.11 (0.05, 0.19)	

^a Only one or two residences and at least 20 years of coverage.

^b Only one residence with complete coverage.

^c Correction for measurement uncertainty.

469. An analysis was conducted to (approximately) combine the risk estimates from these three pooled analyses using weighting by the inverse of the variance in the ERR estimate. The ERR estimate for the combined primary analyses was 0.093 per 100 Bq/m³, and for the restricted analyses, the combined ERR estimate was 0.11 per 100 Bq/m³. The European study [D21] provided 72% of the weight (of information) from the primary analyses and 82% of the weight (of information) from the restricted analyses.

470. The European study provided an ERR estimate of 0.16 per 100 Bq/m³ of residential radon, based on a correction for measurement uncertainty that was about twice as high as the ERR from the primary analyses [D21]. The measurement

error correction of Darby et al. [D21] requires a number of assumptions concerning the distribution describing the long-term average residential radon levels within a geographical area and the variability associated with repeated measurements of radon concentrations in the same dwelling. However, as discussed by Darby et al. [D21], there is also information to support many of the assumptions concerning, for example, the magnitude of year-to-year variability. Nonetheless, at this time it seems reasonable to adopt the estimate corrected for random uncertainties in the assessment of radon concentrations from Darby et al. [D21], namely an ERR of 0.16 (95% CI: 0.05, 0.31) per 100 Bq/m³, as an appropriate, if possibly conservative, estimate of the (lifetime) risk from residential radon.

VI. EFFECTS OF RADON ON ORGANS AND TISSUES OTHER THAN THE LUNG

A. Dosimetric considerations

471. It is generally recognized that the main hazard from inhaled RDPs is from irradiation of the lung. However, in some circumstances, irradiation of the stomach from ingestion of water containing dissolved radon gas may need to be considered. One important factor is the length of time that ingested radon remains in the stomach [I14]. Various estimates of dose to the stomach are within a factor of about 10 [N9], which is quite a small variation considering the uncertainties associated with such estimates. Calculations also suggest that decay products deposited on the skin may be capable of irradiating the sensitive basal cells [S4].

472. In addition to the dose to the lung, Jacobi and Eisfeld [J2] and Harley and Robbins [H38] calculated the dose to organs other than the lung, such as kidney, bone marrow and skin. Kendall and Smith [K21] applied ICRP dose models to estimate effective doses to organs and tissues from radon and its decay products, including doses arising via inhalation, external exposure of the skin and ingestion. The aim was to provide a self-consistent summary that would allow the various hazards to be compared and put into context. The largest dose overall from inhaled radon and its decay products was to the respiratory tract; doses to other organs were usually at least an order of magnitude smaller (see table 25, adapted from reference [K21, table 2]). In particular, doses to tissues with a relatively high fat content (such as red bone marrow), while somewhat higher than those to most other tissues, did not appear to be high enough to present a particular problem. The conclusion was that the conventional focus on risk of lung cancer from inhaled RDPs was appropriate

473. Kendall and Smith [K21] also considered the dose to the foetus. For RDPs, they adopted the foetal discrimination factors of the ICRP. The ICRP makes no recommendation for radon gas. Kendall and Smith noted that, for many radionuclides, the dose to the foetus is similar to that to maternal muscle. Arguing that the fat content of the foetus is low, they assumed that maternal muscle provides a reasonable surrogate. An alternative approach is discussed below.

474. According to Kendall and Smith [K19], the general pattern of doses to different tissues for inhalation and ingestion of radon and its decay products by children is similar to that in adults. Both for inhalation and for ingestion, the organ of intake receives much higher doses than any other organ. In the case of inhalation, the largest doses are to the lung and the extrathoracic part of the respiratory tract (the nose, pharynx and larynx). In the case of ingestion, the stomach receives a

much higher dose than any other organ. Of the other organs and tissues, those with a high fat content receive somewhat higher doses from radon gas. Red bone marrow, thought to be the tissue in which childhood leukaemias originate, does not receive doses that are large compared with those to other tissues. Nevertheless, the calculated doses are high enough to suggest that radon may be responsible for a small proportion of childhood leukaemias [H38]. It is possible that alpha particles from RDPs irradiate the cells in which skin cancers originate and thus induce skin cancer. However, the location of these sensitive cells is not known with certainty, and it is possible that they are too deep to receive a significant dose. If they are irradiated, it is likely that the doses would be larger in the case of children than in adults. However, the evidence so far available is inconclusive.

475. Robbins and Harley [R5] suggested that maternal ingestion of radon in water can deliver a dose to the foetus. Radon is transported by the blood and diffuses throughout the body, including the placenta. Radon and its short-lived decay products can thus reach an embryo/foetus. There is an interval during pregnancy when the foetus is at the highest risk of severe effects from radiation exposure. For the early embryo, there are two important factors to consider: the very small size of the embryo yields a small target for alpha particle hits, but conversely, alpha particle damage to DNA may have major consequences. The dosimetric calculations for the developing embryo and foetus used the maternal and the foetal placental blood supply at different points in time [R5]. These calculations relied on available published data for these, and for foetal weights. The accumulation of radon in various compartments following the ingestion of 100 Bq of radon dissolved in water was estimated, and the dose determined as a function of time, on the basis of the pharmacokinetic model developed. The ratio of the weight of blood in the embryo/newborn infant to its total weight was assumed to be a constant, as was blood flow to the placenta at $115 \text{ mL kg}^{-1} \text{ min}^{-1}$. These parameters need verification, as they are critical not only for such calculations but for other basic toxicological calculations. The clearance half-times of radon for the various tissues in the mother were based on published human data [H24, H25]. For an average intake of 0.6 L of raw tap water per day, containing a radon concentration of 100 Bq/L, the calculated total equivalent dose to the foetus over the term of pregnancy was 250 μSv . The highest calculated equivalent dose of 3 $\mu\text{Sv/week}$ occurred between weeks 6 and 16 [R5]. The foetal doses estimated in reference [R5] are considerably higher than those reported by Kendall and Smith [K19], who have noted that the foetus has little fat until late in gestation. The difference between these estimates originated from the assumptions made in the models.

Table 25 Summary of dose coefficients from inhaled radon decay products together with annual doses from decay products and radon at 200 Bq/m³ (see reference [K21] for discussion)

	Type F					Type M					²²² Rn
	Dose per unit intake (Sv/Bq)					Dose per unit intake (Sv/Bq)					
	²¹⁰ Po	²¹⁴ Pb	²¹⁴ Bi	Mixture	Annual dose (mSv) at 200 Bq/m ³	²¹⁰ Pb	²¹⁴ Pb	²¹⁴ Bi	Mixture	Annual dose (mSv) at 200 Bq/m ³	
Lung	1.1 10 ⁻⁸	1.4 10 ⁻⁹	3.9 10 ⁻⁸	2.5 10 ⁻⁹	35.8	2.7 10 ⁻⁸	1.3 10 ⁻⁷	1.1 10 ⁻⁷	1.1 10 ⁻⁷	159	1.2
Ext.thor.	4.4 10 ⁻⁹	2.7 10 ⁻⁹	6.3 10 ⁻⁸	3.0 10 ⁻⁹	44.5	6.7 10 ⁻⁹	4.8 10 ⁻⁸	9.3 10 ⁻⁸	4.9 10 ⁻⁸	70.9	
Stomach	3.3 10 ⁻¹¹	1.7 10 ⁻¹⁰	9.4 10 ⁻¹¹	1.3 10 ⁻¹⁰	0.19	1.2 10 ⁻¹¹	7.7 10 ⁻¹¹	4.9 10 ⁻¹¹	5.6 10 ⁻¹¹	0.08	0.06
Small intestine	3.0 10 ⁻¹¹	1.7 10 ⁻¹⁰	7.5 10 ⁻¹¹	1.2 10 ⁻¹⁰	0.17	6.9 10 ⁻¹²	4.9 10 ⁻¹¹	1.8 10 ⁻¹¹	3.2 10 ⁻¹¹	0.05	0.06
Colon	2.8 10 ⁻¹¹	1.5 10 ⁻¹⁰	8.2 10 ⁻¹¹	1.1 10 ⁻¹⁰	0.16	3.9 10 ⁻¹²	2.4 10 ⁻¹¹	9.9 10 ⁻¹²	1.7 10 ⁻¹¹	0.02	0.05
RBM	3.9 10 ⁻¹¹	3.2 10 ⁻¹⁰	6.8 10 ⁻¹¹	1.9 10 ⁻¹⁰	0.28	4.2 10 ⁻¹²	3.7 10 ⁻¹¹	7.4 10 ⁻¹²	2.2 10 ⁻¹¹	0.03	0.65
Bone surface	3.4 10 ⁻¹¹	2.1 10 ⁻⁹	6.8 10 ⁻¹¹	1.0 10 ⁻⁹	1.48	3.6 10 ⁻¹²	2.5 10 ⁻¹⁰	7.3 10 ⁻¹²	1.2 10 ⁻¹⁰	0.17	0.03
Liver	5.8 10 ⁻¹¹	5.0 10 ⁻¹⁰	6.8 10 ⁻¹¹	2.9 10 ⁻¹⁰	0.43	6.3 10 ⁻¹²	5.6 10 ⁻¹¹	7.6 10 ⁻¹²	3.2 10 ⁻¹¹	0.05	0.09
Breast	2.8 10 ⁻¹¹	1.4 10 ⁻¹⁰	6.8 10 ⁻¹¹	1.0 10 ⁻¹⁰	0.15	3.0 10 ⁻¹²	1.6 10 ⁻¹¹	7.8 10 ⁻¹²	1.2 10 ⁻¹¹	0.02	0.42
Kidney	8.7 10 ⁻¹¹	4.8 10 ⁻⁹	5.8 10 ⁻⁹	3.6 10 ⁻⁹	5.20	9.5 10 ⁻¹²	5.0 10 ⁻¹⁰	5.9 10 ⁻¹⁰	3.7 10 ⁻¹⁰	0.54	0.05
Gonads	2.8 10 ⁻¹¹	1.4 10 ⁻¹⁰	6.8 10 ⁻¹¹	1.0 10 ⁻¹⁰	0.15	2.9 10 ⁻¹²	1.5 10 ⁻¹¹	7.1 10 ⁻¹²	1.1 10 ⁻¹¹	0.02	0.05
Brain	2.7 10 ⁻¹¹	1.5 10 ⁻¹⁰	6.8 10 ⁻¹¹	1.1 10 ⁻¹⁰	0.15	2.9 10 ⁻¹²	1.5 10 ⁻¹¹	7.1 10 ⁻¹²	1.1 10 ⁻¹²	0.02	0.06
Bladder	2.8 10 ⁻¹¹	2.1 10 ⁻¹⁰	1.0 10 ⁻¹⁰	1.4 10 ⁻¹⁰	0.21	2.9 10 ⁻¹²	2.2 10 ⁻¹¹	1.0 10 ⁻¹¹	1.5 10 ⁻¹¹	0.02	0.05
Muscle	2.8 10 ⁻¹¹	1.4 10 ⁻¹⁰	6.8 10 ⁻¹¹	1.0 10 ⁻¹⁰	0.15	3.0 10 ⁻¹²	1.6 10 ⁻¹¹	7.4 10 ⁻¹²	1.1 10 ⁻¹¹	0.02	0.05
CED	1.4 10 ⁻⁹	2.5 10 ⁻⁹	5.6 10 ⁻⁹	3.6 10 ⁻⁹	5.30	3.3 10 ⁻⁹	1.6 10 ⁻⁹	1.4 10 ⁻⁸	1.3 10 ⁻⁸	19.7	0.28
Foetus	1.3 10 ⁻¹¹	5.3 10 ⁻¹¹	2.6 10 ⁻¹¹	4.1 10 ⁻¹¹	0.06	1.6 10 ⁻¹²	6.2 10 ⁻¹²	2.9 10 ⁻¹²	4.9 10 ⁻¹²	0.01	0.04
Skin					25					25	

Note: The activity of decay products in comparison with radon is taken to be ²¹⁰Po:²¹⁴Pb:²¹⁴Bi in the ratio 0.9:0.45:0.225; F = 0.41. 90% of decay products are taken to be attached to aerosols, 10% unattached. Annual volume breathed taken to be 7,300 m³. Ext.thor. is the extrathoracic part of the respiratory tract. RBM is red bone marrow. CED is committed effective dose. Kidney is shown as the organ receiving the next highest dose after the respiratory tract. The dose to the foetus from radon gas taken to be that to muscle over 9 months. Dose to skin is taken from reference [N9]. It does not depend on type.

476. Kendall and Smith [K19] also investigated differences between doses to adults and those to children aged 1 and 10 years. They calculated both dose coefficients, i.e. the equivalent dose resulting from an intake of unit activity of the material in question, and also annual equivalent doses. The former tend to be higher in children than in adults. The differences between the annual doses to children and adults are smaller than the differences in the dose coefficients, because children breathe less air and ingest less water than adults.

B. Epidemiology of cancers other than lung

477. Annex A, “Epidemiological studies of radiation and cancer”, provides a discussion of the epidemiological methods as well as information complementary to that provided in this annex.

478. The currently available epidemiological evidence indicates that risks other than lung cancer from exposure to radon and its decay products are likely to be small. A number of studies of residential radon and non-lung cancer are available. Laurier et al. [L18] reviewed 19 ecological studies, 8 residential case-control studies and 6 miner cohort studies published between 1997 and 2000 in order to examine a possible association between radon exposure and leukaemia. While the ecological studies suggested a possible positive association, the case-control studies and the miner cohort studies did not. Overall, the authors concluded that the available data (i.e. to 2000) did not provide any evidence of an association between radon exposure and leukaemia [L18].

479. A series of papers [E6, L38, T28] followed the review by Laurier et al. [L18]. Eatough [E6] noted that potential leukaemia risk to miners was investigated by looking for trends in relative risks with increasing exposure to RDPs. However, studies of the dosimetry for radon gas and RDPs suggest that the doses to the red bone marrow are not appreciable [H38]. Tomasek [T28] reported a study of Czech miners in which an excess of leukaemia related to duration of exposure was observed, and went on to suggest that duration of exposure in the mine environment is likely to be a surrogate for exposure to uranium dust, which he considered to be the predominant dose contributor. Laurier et al. [L38] commented that results published since their review [L18] did not modify the conclusions that available epidemiological data do not demonstrate an association between leukaemia and exposure to radon. They also noted that if such a relationship exists, the association would be slight and of little significance for residential exposure.

480. An ecological study of cancer incidence and radon levels in the south-west of the United Kingdom looked at 14 major cancer sites, using data for the South-Western Cancer registry [E5]. Average radon levels for residences were sorted into 10 categories, from low (<40 Bq/m³) to very high (>230 Bq/m³), and age standardized cancer rates were calculated for each category. Incidence rates for lung cancer were

similar across all radon categories. Except for non-melanoma skin cancer, the authors found no significant positive correlation with radon. Overall, the authors found no significant difference in the corrected survival rates for any cancer site between the low- and high-radon areas.

481. A research letter by Law et al. [L20] commented on residential radon exposure and leukaemia, referring to an earlier study [K24] of acute leukaemia in the south-west, north and north-west of England of residents aged 16–69 years. Radon measurements (of about 6 months’ duration) were made in the living rooms and bedrooms of 1,881 houses (about 78% of the homes of the subjects, 76% for the cases and 80% for the controls). No association was found between acute leukaemia and radon concentration; the authors concluded that their study did not support exposure to residential radon as a causal factor in leukaemia in the United Kingdom.

482. Thorne et al. [T24] investigated possible associations between residential radon exposure and paediatric cancers in Devon and Cornwall, United Kingdom. This study compared the incidence of childhood cancers in postal sectors with low radon concentrations (<100 Bq/m³, average 57 Bq/m³), and high radon concentrations (>100 Bq/m³, average 183 Bq/m³) in a total of 238 postal sectors. The authors found no significant difference in cancer incidence rate between low- and high-exposure sectors.

483. Steinbuch et al. [S46] reported an investigation of residential radon exposure and risk of childhood acute myeloid leukaemia (AML). Alpha track detectors were placed in the houses of 173 cases and 254 controls for 1 year. Overall, there was no association between residential radon concentration and the risk of AML. Lubin et al. [L12] reported an age-matched study of childhood acute lymphocytic leukaemia (ALL) and residential radon exposures of children in the United States. Radon levels for the 505 cases and 443 controls were estimated for 97% of the exposure period. The mean radon level was lower for cases (65.4 Bq/m³) than for controls (79.1 Bq/m³). No association between ALL and radon exposure was found.

484. Evrard et al. evaluated the ecological association between indoor radon concentrations and acute leukaemia incidence among children under 15 years of age in France [E7]. The study considered the whole country, divided into 348 geographical units. Incidence data included 4,015 cases of acute leukaemia registered by the French National Registry of Childhood Leukaemia and Lymphoma between 1990 and 1998. Exposure was based on a national campaign of 13,240 indoor radon measurements. A positive ecological association was observed between indoor radon concentration and childhood leukaemia incidence, on the borderline of statistical significance ($p = 0.053$). A significant association was observed for AML ($p = 0.004$) but not for ALL ($p = 0.49$). Consideration of exposure to terrestrial and cosmic radiation did not modify the observed association between radon exposure and incidence of AML [E8].

485. In a 1993 paper, Tomasek et al. [T51] reported on a study of RDP exposure and cancers other than lung cancer in a cohort of uranium miners in western Bohemia. These authors investigated site-specific cancer mortality in 4,320 miners who had been followed-up for an average of 25 years. An average exposure of 219 WLM was reported. Data on smoking habits and alcohol consumption were not available. An analysis of rates of observed to expected numbers of deaths (O/E) showed that overall, the risk of death from non-lung cancers was slightly greater than the natural rates but not significantly so. A significant excess of non-lung cancer mortality in men who started to mine when they were younger than 25 years of age was found, but the increase was not related to cumulative RDP exposure. Overall, the authors concluded that there is no significant risk of any cancer other than lung cancer, although further investigation is needed of the effect of RDP exposure on cancers of the gall bladder and extrahepatic bile duct and on multiple myeloma.

486. Rericha et al. [R13] reported an investigation of the incidence of leukaemia, lymphoma and multiple myeloma, a total of 177 cases, in Czech uranium miners, using a retrospective case-cohort design in a study of 23,043 uranium miners. The authors found no apparent association between RDP exposure and either non-Hodgkin's lymphoma or multiple myeloma, but did find an association between RDP exposure and an increased risk of leukaemia (chronic lymphocytic leukaemia, CLL), which was not previously thought to be radiogenic. The association is based on a comparison of the relative risk (RR) of CLL in miners who had an RDP exposure of 110 WLM with those who had an exposure of 3 WLM. An RR of 1.98 (95% CI: 1.10, 3.59; $p = 0.016$) was reported.

487. Darby et al. [D22] reported on RDP exposure and non-lung cancer in a group of Swedish iron ore miners. These authors observed that, when the mortality from all cancers other than lung in miners was compared with that expected for the northernmost county of Sweden, the O/E ratios were higher in men with exposures of >100 WLM than in men with lower exposures, but the trend was not significant. As in the western Bohemian study [T51], excesses were seen for cancer of the gall bladder and extrahepatic bile duct and for multiple myeloma; however, the increases were not statistically significant.

488. Darby et al. [D12] carried out a collaborative analysis of 11 miner studies to look for risks of cancer other than lung cancer. The miner populations included 10 of the 11 miner cohorts covered by the joint analysis by Lubin et al. [L10] (the Radium Hill study [W15] was excluded since follow-up was incomplete). The 11th miner cohort added in the analyses by Darby et al. was a cohort of Cornish tin miners [H29]. Overall, the study [D12] included some 64,209 men who had worked for an average of 6.4 years, accumulated an average exposure of 155 WLM and been followed for an average of 16.9 years. External mortality data were available for 10 of the studies (but not for the Chinese study of Xuan et al. [X1]). In total, some 1,179 non-lung cancer deaths were

observed, which was close to the expected number (O/E = 1.01; 95% CI: 0.95, 1.07). Among the 28 individual cancer types studied, the only sites of statistically significant excess cancers were the stomach and the liver; however, mortality from these cancers was not related to cumulative RDP exposure in WLM and was thus unlikely to have been caused by radon. Among the leukaemias, the O/E ratio for acute myeloid leukaemia was larger than for other leukaemias, but was not statistically significant. Overall, the authors concluded that exposure to high concentrations of radon in the air is unlikely to result in an increased risk of cancer mortality other than from lung cancer [D12].

489. Möhner et al. [M44] reported on a study of leukaemia in German miners. This case-control study included 377 cases and 980 individually matched controls. Exposures were based on a job-exposure matrix that included exposure to radon and its decay products as well as exposure to external gamma radiation and to long-lived radioactive dust. Using logistic regression methods and taking study power into account, the authors concluded that a "casual relationship between radon progeny and risk of leukaemia can largely be excluded". The study did, however, suggest an elevated risk of leukaemia for cumulative exposures of ≥ 400 WLM.

490. Kreuzer et al. [K29] reported on a study of the risk of lung cancer and other cancers in the German uranium miner cohort. The study, based on external comparisons, showed a statistically significant excess of lung cancer risk and a trend of increasing risk with increasing cumulative RDP exposure. The study also found an excess, although not statistically significant, mortality from liver and lung cancer. The authors indicated that the excess is unrelated to cumulative RDP exposure and noted that, in the early years of mining, the Wismut mining company provided miners with alcohol and cigarettes free of charge. Tirmarche et al. [T9] reported an excess of larynx cancer in French miners; however, Villeneuve et al. [V4] found no significant relationship between RDP exposure and other cancer sites.

491. The update of data for the Eldorado miners [H35] involved 17,660 workers. The update extended the mortality analysis by almost 20 years and added 30 years of new information on cancer incidence. A total of 5,332 deaths occurred between 1950 and 1999, and 2,355 workers developed at least one cancer between 1969 and 1999. Mortality and cancer incidence were compared with those of the general Canadian population. Lung cancer was the only cancer site with an excess for both mortality and cancer incidence. In the internal analysis, there was no meaningful evidence of any causal relationship between RDP exposure and increased risk of any cancer other than lung cancer.

C. Effects other than cancer

492. A full discussion of this subject is provided in annex B, "Epidemiological evaluation of cardiovascular disease and

other non-cancer diseases following radiation exposure". This section provides a few additional observations from epidemiological studies of miners.

493. Villeneuve and Morrison [V2] investigated the mortality from coronary heart disease (CHD). In this study, the cohort consisted of 1,743 underground miners and 321 millers or surface workers. Men in this cohort had a mean exposure to RDPs of 378.6 WLM over an average of 5.7 years. As for other analyses of these miners, exposures were based on the analysis of Corkill and Dory [C12]. Smoking data from a 1993 survey were used to update data from previous surveys in 1960, 1966, 1970 and 1978. Smoking status (current, former or never smokers) was determined for 59% of the cohort. Finally, data on the mortality experience of the cohort were available up to 1990. Multivariate Poisson regression analysis was used to estimate the relative risk of CHD from RDP exposure, with adjustments for attained age, duration of exposure and smoking status. Unlike previous studies of this cohort, the analyses [V2] used internal comparisons to control for bias potentially introduced through the healthy worker effect. This study found that workers with high cumulative exposures to RDPs (over 1,000 WLM) had an elevated risk of CHD (RR = 1.5; 95% CI: 0.77, 2.75) compared with those with no exposure. However, no statistically significant trend of increasing risk with increasing exposure was found, nor was there a statistically significant interaction between cumulative RDP exposure and smoking status. The authors found that smoking status was a significant predictor of CHD.

494. Villeneuve et al. [V6] further explored the relationship between mortality from CHD and RDP exposure using both external and internal cohort comparisons in their most recent update of this cohort. There was no association between cumulative exposure in WLM and CHD mortality. A reduced CHD mortality rate was observed relative to the population of Newfoundland males (SMR = 0.86; 95% CI: 0.74, 0.98); the authors attributed this reduction to a "healthy worker effect".

495. Kreuzer et al. [K30] reported an investigation of the mortality from cardiovascular diseases in a cohort of German

uranium miners. The cohort included 590,001 male subjects who were employed for at least 6 months between 1946 and 1989 at the former Wismut uranium company in eastern Germany. As for other studies of Wismut miners, exposures to RDPs, long-lived radionuclides and external gamma radiation were estimated using a detailed job-exposure matrix. As of 31 December 1998, the cohort included (about) 16,598 deceased miners, with 5,417 deaths from cardiovascular diseases. Linear Poisson regression models were used to estimate the ERR per unit of cumulative radiation exposure after adjusting for attained age and calendar period. The study found no trend in risk of circulatory diseases with increasing cumulative exposure to RDPs (ERR was 0.0006 (95% CI: -0.004, 0.006) per 100 WLM), external gamma radiation (ERR was -0.26 (95% CI: -0.6, 0.05) per Sv) or long-lived radionuclides (ERR was -0.2 (95% CI: -0.5, 0.06) per 100 kBq h m⁻³). The authors concluded that their results did not support an association between cardiovascular disease mortality and exposure to radiation among uranium miners.

496. More recent data are also available from Canada. Howe [H35], in the update of the Eldorado cohort study, reported that for most of the causes of death, the cohort as a whole as well as various subcohorts had reduced risks relative to the population. In particular, the authors stated that, although an increase of hypertensive disease was observed (significant in only one of the subcohorts examined), the study indicated a statistically significant deficit in males of cardiovascular diseases such as stroke and ischaemic heart disease. The authors go on to suggest that this is probably a consequence of the healthy worker effect, since heart disease would be likely to prevent people working in a strenuous physical occupation such as mining. Villeneuve et al. [V6] found that there was no apparent trend between cumulative exposure to radon and the relative risk of death from CHD ($p = 0.63$). Furthermore, this finding was unchanged after adjusting for lifetime smoking status, which was available for approximately 54% of the cohort. Additionally, cumulative exposure to radon was found to be unrelated to diseases of the circulatory system, acute myocardial infarction and cerebrovascular disease. All of these findings are consistent with those of Kreuzer et al. [K30].

VII. IMPLICATIONS FOR RISK ASSESSMENT

497. Epidemiological studies of underground miners provide the current basis for estimating the risk of exposure to radon and its decay products. Studies of smoking and non-smoking miners show that exposure to RDPs carries an enhanced risk of lung cancer. However, miners were not exposed to RDPs alone but rather to RDPs in association with many other agents, among them silica (quartz), various metals, including arsenic, and diesel exhaust. Traditionally, the risks from domestic radon exposure have been developed by extrapolation from the data for miners; however, the risks from residential radon can now be estimated directly using the results of pooled residential case-control studies (see section V).

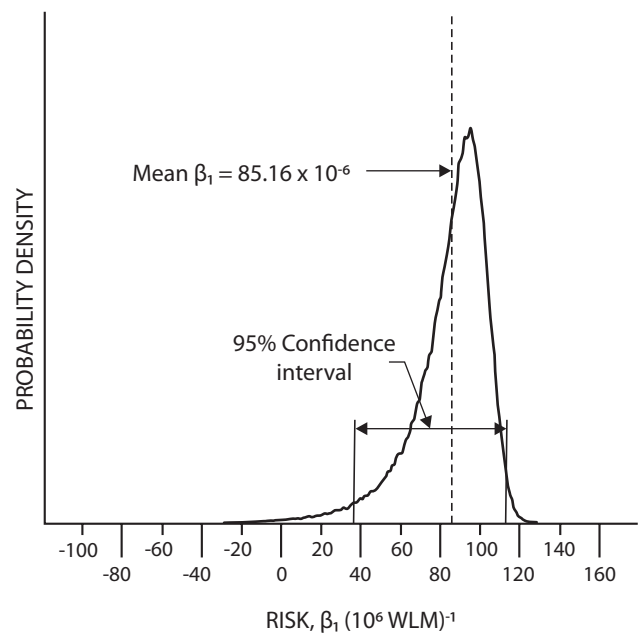
A. General studies of exposure uncertainty

498. There is a great deal of interest in dosimetry-based estimates of risk from exposure to RDPs (see section II). There is a discrepancy between the dosimetry-based and the miner-epidemiology-based approaches, with differences in estimates of a factor of 2–3. The miner epidemiology data provide the basis for investigating biological mechanisms and modifiers such as time since exposure and age at exposure. As discussed in section IV, there is considerable uncertainty in the exposures of miners, with uncertainty increasing the further we go back in time. In view of the ongoing importance of miner studies in understanding the risk from exposure to radon and RDPs at work and in the home, it is useful to consider the effects of uncertainty in miner exposures on the epidemiological analyses performed using miner data.

499. A 1989 report considered the effects of uncertainty in exposure estimates for the studies then available of miners in Ontario, Beaverlodge and Port Radium (Canada), Czechoslovakia, Sweden, and the Colorado Plateau (United States) [S10]. Several subgroups of the Colorado Plateau cohort were also studied. The focus of the study was on adjusting the exposure-response parameter in the simple linear model for excess relative and excess absolute risk. The authors used a Bayesian error-in-variable relative risk approach to analytically assess the effect of uncertainty on the exposure-response relationship. Figure XIX shows the posterior probability density factor for the absolute risk coefficient for Colorado miners with no mining exposure prior to 1950. At that time (i.e. follow-up to 31 December 1982), the cohort with no exposure before 1950 included 168 miners with lung cancer. The mean lifetime risk was estimated at about 85 cases per 10^6 person-years per working level month, with a range of about 60–150 cases per 10^6 person-years per working level month [S10]. The estimate may be compared

with the BEIR IV [C19] estimate of 140 per 10^6 person-years per working level month for male non-smokers. The approach in reference [S10] was necessarily limited because the authors did not have access to data on individual miners and could only utilize published relative risks within categories of cumulative exposure ($J h m^{-3}$ or WLM); similarly, no attempt could be made to evaluate patterns of error for different mining periods within a study. For the ERR model, the authors estimated that the most likely range for the ERR per unit exposure parameter for this group of studies was 0.009 – 0.00005 ($J h m^{-3})^{-1}$ (0.5 – 1.5% WLM $^{-1}$).

Figure XIX. Distribution of absolute risk for Colorado miners with no exposure prior to 1950 [S10].



500. A subsequent paper by Chambers et al. [C15] reviewed the factors affecting exposure estimation for seven groups of uranium miners. These included the temporal and spatial variability of radon and RDP levels in the workplace, changing mining methods and ventilation practices, and uncertainties about the miners' work histories. Bayesian methods were used to develop posterior probability density functions of absolute and excess relative risk coefficients for each of the cohorts. Figure XX shows the posterior probability density function for the ERR. The authors noted that the estimated RDP exposures in WLM are uncertain in all the miner studies. For the reasons given in reference [S10], the studies of United States miners [H17, P14, S10], Ontario miners [M19, M23], and

Czechoslovak miners [S25] were considered at the time to provide the strongest basis for risk estimation. The strengths of these groups are that they are all large, well-traced cohorts for which considerable information existed on which to base risk estimates for the period of interest. To the best of the authors' knowledge, there was no systematic bias with regard to the magnitude of the group exposures estimated in these studies. However, the uncertainties in exposures of individual miners could be very large, particularly for exposures associated with the early years of mining. Of these three groups, the United States miners were exposed to the highest RDP concentrations, the Czechoslovak miners were exposed to intermediate levels, and the Ontario miners were exposed to the lowest levels. While figure XX showed the posterior probability density function for all cohorts, figure XXI illustrates the posterior probability density function developed by combining the three studies considered "best", together with that from combining all the studies (with and without a notional correction for bias in the Swedish and the Beaverlodge

studies). Overall, Chambers et al. [C15] estimated the ERR to have a 95% uncertainty range of 0.5–1.5 per 100 WLM, comparable to the estimates of Lubin et al. [L10].

501. The pooled analysis by Lubin et al. [L10] considered the impact of uncertainties in the estimates of RDP exposure only in the most general way and only within the context of modification of the exposure–response relationship by exposure rate and exposure duration. Exposure uncertainty was considered greatest in the earliest years of mining, i.e. those years in which exposure rates were highest. According to Lubin et al. [L10], exposure uncertainty would therefore have tended to attenuate the effects of high exposures and potentially induce an inverse exposure-rate pattern [L22]. They estimated an ERR and associated 95% CIs as depicted graphically in figure XXII. Lubin et al. [L22] report a combined estimate of ERR of 0.49 (95% CI: 0.2, 1.0) per 100 WLM, where the joint 95% CI is based on a random-effects model.

Figure XX. Excess relative risk (posterior) probability density functions with unadjusted variances [C15].

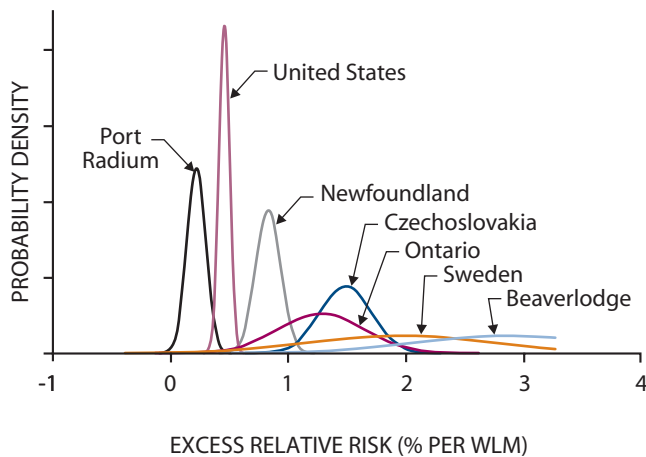
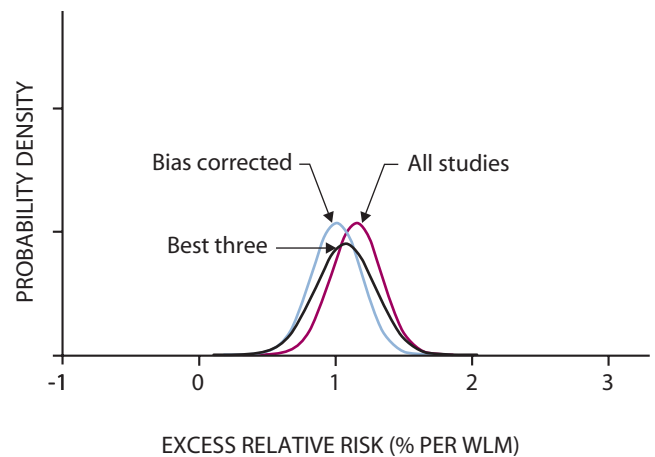


Figure XXI. Combined excess relative risk (posterior) probability density functions [C15].



502. BEIR VI [C20] also investigated the role of uncertainty in estimating lung cancer risk, and developed an extensive table summarizing the sources of uncertainty in estimating lifetime risk from residential exposure to radon. The BEIR VI estimates of lung cancer risk were based on analyses of the data from miner epidemiological studies. The BEIR VI Committee acknowledged that there were uncertainties in exposures to RDPs and other potential factors, such as exposure to cigarette smoke and arsenic. No systematic bias in the estimates of miner exposure to RDPs was identified. BEIR VI also suggested that random errors might result in an underestimate of the slope (risk) of the exposure–response relationship. In addition to extensive qualitative discussion, the BEIR VI Committee applied quantitative methods for uncertainty analysis, acknowledging that their analysis should be considered illustrative, “not to replace the Committee’s new comprehensive qualitative analysis”, since not all sources of uncertainty could be identified and characterized. The BEIR VI

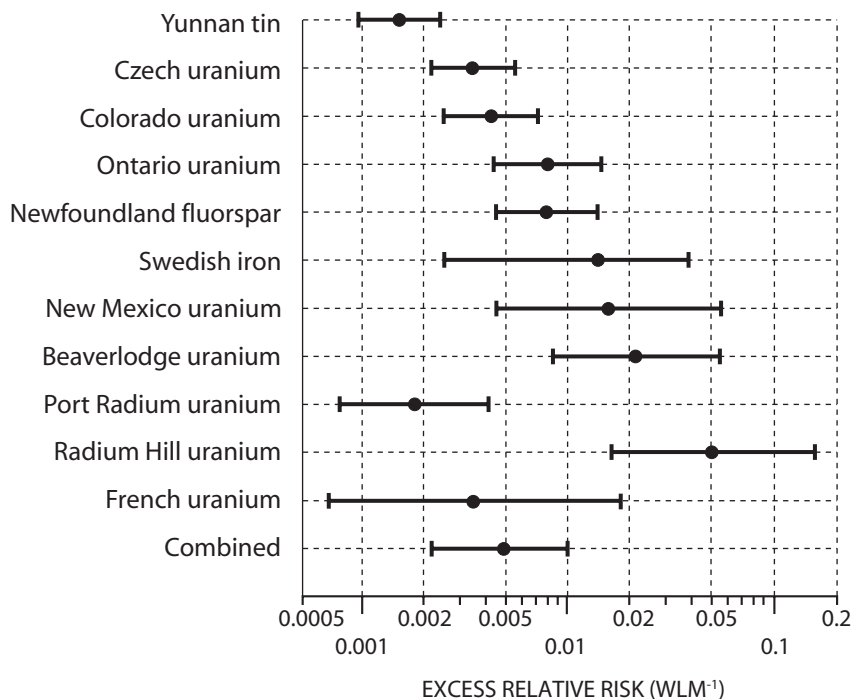
Committee’s “preferred” uncertainty limit was derived using a simple constant relative risk model fitted to the miner data at cumulative exposures of below 50 WLM. On this basis, BEIR VI estimated an ERR of 1.17 per 100 WLM, with a 95% uncertainty interval from 0.2 to 22.5 per 100 WLM.

503. Notwithstanding the different data and methods that have been used to develop combined estimates of ERR from the studies of miners, there is a remarkable consistency in the ranges of ERR per 100 WLM that have been estimated. As discussed in the preceding paragraphs, the 95% CI for the ERR was estimated by Chambers et al. [C15] as 0.5–1.5 per 100 WLM; by Lubin et al. [L10] as 0.2–1.0 per 100 WLM; and by BEIR VI [C20] as 0.2–22.5 per 100 WLM, with central estimates of ERR ranging from 0.49 to 1.17 per 100 WLM. On the basis of the most recent results of miner studies as described earlier in this annex (see para. 427), an estimate of a combined ERR of 0.59 (95% CI: 0.35, 1.0)

per 100 WLM is consistent with previous estimates but with somewhat narrow 95% CIs. By multiplying a notional lifetime risk of lung cancer of, say, 8%, and an average ERR of

0.59 (95% CI: 0.25, 1.0) per 100 WLM, a nominal absolute lifetime risk of about 4.7×10^{-4} (95% CI: 2.8×10^{-4} , 8×10^{-4}) for 100 WLM can be calculated.

Figure XXII. Estimates of excess relative risk of lung cancer per WLM (adapted from [L10]).



B. Biologically based models

504. Several investigators reported analyses of experimental animal data and epidemiological data with multistage models to study mechanisms of carcinogenesis and assess risks from the inhalation of RDPs (e.g. [B20, B28, B29, C20, C29, C30, C31, C33, H26, K22, L16, L23, L33, L34]). The main difference between the approaches was in the choice of exposure–response model to be used for each of the cellular processes in the biologically based model. Age-dependent cancer incidence data do not yet provide the basis for determining the most appropriate model, as evidenced by results of a model comparison using lung cancer data for radon-exposed rats [H26]. The BEIR VI Committee [C20], as noted earlier, acknowledged the importance of biologically based models and indicated that, when biological mechanisms are better understood, such models might become the preferred approach to assessing the risks. Similar views are expressed by Krewski et al. [K22], who applied the two-stage clonal expansion (TSCE) model to two cohorts important to assessing the risks from RDP exposure, namely the Colorado Plateau miners and the Chinese tin miners.

505. An important project, within the framework of the European Union project FIGH-CT1999-00013, looked at a multistage model analysis of the data from the French and Czech uranium miner cohorts [T30]. One of the issues being investigated was how risks can be transferred between

populations with different baseline lung cancer incidence rates. The combined cohort includes 5,098 miners and 125 lung cancer deaths in the French cohort and 5,002 miners and 449 lung cancer deaths in the Czech cohort. The corresponding mean total cumulative exposure levels were 37 and 57 WLM, respectively, for the two cohorts. A two-mutation carcinogenesis model with clonal expansion of cells in the intermediate stage was fitted to the individual miner data. Linear exposure–effect relationships were used for the two mutational steps. The authors found that the fitted linear effect of radon on the first mutational step was an order of magnitude larger than for the second mutational step. Although the baseline lung cancer risk in the Czech miner cohort was considerably higher than that for the French miners, both data sets could be described with the same parameter values for the relative effect of exposure to RDPs on the mutation rates. The authors argued that the uniform description of the effect of RDP exposure for two miner cohorts with distinctly different baseline lung cancer risks (0.09 for the French and 0.23 for the Czech miners) demonstrated the possibility of using the model for risk transfer across populations. In addition, the biologically based model implicitly describes age and exposure–rate effects, and thereby allows for extrapolation to lifetime exposures to low radon concentrations. Lifetime risks were calculated for a 75-year continuous exposure to 1 WLM/a (~ 256 Bq/m³). The lifetime ERR calculated from the model solution in references [B27, B28] was 1.1 for the combined cohorts.

506. Heidenreich et al. [H32] provided further analysis of the French and Czech cohorts using the biologically based TSCE model together with an analysis of the Chinese and Colorado cohorts. The model allows an action of radiation on initiation, promotion and transformation in cancer induction. While all four studies indicate a highly significant action of radiation on promotion, the action on initiation is not significant in the French cohort and is barely significant in the Colorado miner cohort. No action on transformation is found in the Colorado miners, while the other data sets indicate a borderline significance. The doubling exposure rate for initiation is about 3.5 WLM/a in the new data sets, while it is higher in the historic data sets. For transformation, the doubling rate is about 20 WLM/a for the new data sets, while again the historic data give higher estimates. The action of radiation on promotion is different in the four data sets. The larger power of the French and Czech cohorts requires less extrapolation when the risk at very low exposures is estimated.

507. A 1990 study [M11] reanalysed the data for the Colorado Plateau uranium miners in order to investigate RDP exposure, cigarette smoking and lung cancer using a two-mutation model as the biological basis for their assessment. The authors concluded that exposures to both RDPs and cigarette smoke affect the first mutational step and the rate of cell division but that the second mutational step was independent of RDP and cigarette smoke exposures. The authors also concluded that the age-specific risks arising from joint exposure to RDPs and cigarette smoke are more than additive but less than multiplicative. An inverse dose-rate effect was observed, in that fractionally lower RDP exposure resulted in higher lung cancer risks. The authors also obtained the same estimates of lung cancer risk from exposure to residential radon for both smokers and non-smokers. This observation is consistent with observations from the European [D17] and North American [K1] pooled residential radon studies of no significant heterogeneity across categories of smoking. These studies showed an ERR of 0.2 per 100 WLM for non-smokers at age 70 exposed at a rate of 0.2 WLM/a.

508. Luebeck et al. [L16] provided further discussion based on an analysis of the data for the Colorado Plateau uranium miner cohort using a TSCE model. Exposure to RDPs was suggested to affect both the rate of initiation of intermediate cells in the pathway to cancer and their rate of proliferation. However, the effect of radon on the rate of initiation was not statistically significant. The results of reference [L16] showed that, depending on total radon exposure, the lifetime ERR per unit exposure first increased with duration of exposure, reached a maximum and then declined. Non-smoking miners who were exposed to RDPs for 10 years were found to have approximately the same risk (as measured by lifetime ERR per unit exposure) as a non-smoking individual who spent 10–20 years in a residence with very low levels of radon. These authors stated that if the inverse dose-rate effect observed in “miners at much higher total doses were extrapolated naively to durations (and exposure rates) more typical for homes, the risk (lifetime ERR per unit exposure) would be grossly overestimated.”

509. More recent studies such as those reported in references [B27, B28, H32, H36, K22, M11, T30] suggest that mechanistic or biologically based models, which allow the opportunity to investigate mechanisms of carcinogenesis, are likely to find increasing application. Tirmarche et al. [T30], for example, reported estimates of final and second mutation rates based on an analysis of the French and Czech miner cohorts. One observation from their analysis is that an inverse dose-rate effect cannot be excluded for exposure rates of >30 WLM/a.

510. Harley et al. [H4, H36] applied a biologically based model derived from an evaluation of the number of nuclei traversed by an alpha particle that will cycle as a function of time following unit exposure to RDPs. The model was developed in two steps. First, the number of basal cell nuclei traversed by an alpha particle following an exposure of 1 WLM was determined from biological data. Secondly, the number of nuclei traversed and the measured cycling rates in normal bronchial epithelium were used to calculate the dividing population as a function of time after exposure. The cycling rates decreased with a half-time of about 15 years. The authors compared their model fit with the combined excess risk data from the joint analysis of 11 underground mining cohorts [L10], and suggested that their model may explain why the tumour risk decreases with time since exposure and with attained age, in that both could reflect the reduction in cycling frequency of cell nuclei repopulating the basal stem cells.

511. Little [L21] fitted multistage cancer models with clonal expansion to the Colorado miner data, allowing for up to three mutational steps. Both radiation and smoking were allowed to affect the mutation rates as well as cell proliferation in the intermediate stages. The best fit of the data was obtained for a three-mutation model in which the first and second mutation rates increased with RDP exposure, and the first mutation rate increased with smoking rate, in a strongly non-linear way. This three-mutation model was slightly superior to a two-stage model in which the first mutation rate depended in a non-linear fashion on radon and smoking, in combination with a reduction of intermediate cell death/differentiation rate with radon exposure.

512. The preceding examples show that current multistage cancer models with clonal expansion are not specific enough to determine the dose–response relationships for the cellular processes in the model from a fit to cancer incidence data. This was shown in model intercomparison on a large data set of radon-exposed rats [H21]. An alternative approach to this problem of specificity was applied in the two-mutation model fit of Leenhouts to the Colorado uranium miner data [L23]. He used a stepwise fitting approach in combination with biologically motivated mutation equations. The radiation effect in this model was limited to the mutation rates, and no effect of radiation on proliferation rates was assumed. Using a stepwise approach, Leenhouts fitted the background parameters to the lung cancer incidence of non-smokers, the smoking coefficients to the lung cancer incidence of smokers, and the radiation parameters to the miner data. From

the model solution, the following risks were calculated at age 75 for a lifelong exposure to 0.1 WLM/a: EAR of 0.008 for non-smokers, increasing to 0.0014 for smokers, which corresponds to ERR values of 0.19 and 0.009, respectively.

C. Risk projection

513. The ICRP [I3] recommends that radon risk assessment be based on epidemiological studies of miners. The BEIR VI Committee also base their risk estimates on an analysis of pooled miner data [C20]. Both reports predate the pooled analyses of residential case-control radon studies. As discussed below, the present report suggests that the risk of lung cancer from domestic exposure to radon can be estimated using the results of the pooled residential case-control radon studies [D17, D21, K1, K26, L26].

514. It is important to develop reliable risk estimates of lung cancer due to radon exposure in the workplace or in the home. Many published evaluations (e.g. [C20, U2, U17, W18]) are available, as described in previous sections. Whether there is a risk from residential radon exposure has been widely debated in the literature (e.g. [B34, C24, C25, C34, F8, F12, H29, P10]). However, notwithstanding the wide range of results from residential case-control studies and the important effects of confounding by smoking and other factors, overall the pooled European [D17, D21], North American [K1, K26] and Chinese [L26] case-control residential radon studies clearly demonstrate an association between risk of lung cancer and residential radon exposure. There is a remarkable coherence both among the pooled residential studies and the downward extrapolation of radon risk estimates from miner studies.

515. Uncertainties associated with downward extrapolation from miner studies include, but are not limited to: uncertainties in the reconstruction of miner exposures; possible exposure to other carcinogens; the high but uncertain level of smoking among miners; and the fact that exposures in mines historically were at relatively high levels compared with levels in homes and in present-day mines. Uncertainties associated with residential radon studies include, among other factors: lack of contemporary radon measurements in residences (e.g. while it may be possible to measure radon levels today in homes previously occupied by subjects in residential studies, there will be large uncertainties associated with assumptions about changes to home ventilation and in the habits of subjects over time); uncertainties about smoking; and low statistical power.

516. Many studies of lung cancer risks in miners exposed to RDPs (see section IV) and several joint analyses of 11 miner cohorts have been published [C20, L4, L10]. Tirmarche et al. [T30] discussed risk estimates among the general population and reported a comparison of different models used to estimate lifetime risk. These authors estimated a lifetime ERR in the range 0.08–2.31, according to the exposure scenario. These results are in the envelope of

the results in BEIR VI. However, there are wide variations in these studies in size, duration of follow-up, reliability of exposure estimation, availability of smoking data and other factors. For example, it is questionable whether the following cohorts should be used for risk assessment: (a) Radium Hill cohort, because of limited follow-up and the low quality of the exposure data; (b) Port Radium, Newfoundland fluorspar and (without further re-evaluation using the revised dosimetry) Swedish iron miner cohorts, because of the low quality of the exposure data; and (c) Chinese tin mining cohort, because of extreme confounding from arsenic exposure. In addition, the French cohort data [C20, L4, L10] are being updated, and the new Wismut cohort described in section V, is being developed. The current European project in which a joint analysis is being performed of the extended French cohort and the Czech cohort is an important step, as these two cohorts are both of high quality. In addition, the Eldorado cohort, consisting of miners at the Port Radium and Beaverlodge uranium mines and the Port Hope uranium processing facility, has been updated [H35]. One important aspect of this update is the extensive review of work histories and the use of updated exposure algorithms, both of which significantly improve the quality of the epidemiological studies carried out on this cohort.

517. The BEIR VI model was developed from a pooled analysis of 11 underground mining cohorts and takes account of the reduction of relative risk with increasing time since exposure, adjusting for attained age and exposure rate. Estimates of lifetime risks developed with the model also incorporated sex and smoking status. BEIR VI developed two models, an exposure-age concentration model and an exposure-age duration model. The general structure of the BEIR VI model is illustrated below:

$$ERR = \beta [\theta_{5-14} w_{5-14} + \theta_{15-24} w_{15-24} + \theta_{25+} w_{25+}] \theta_{age} \gamma_z$$

where

β	= slope of the exposure-risk relationship for the assumed reference categories of the modifying factors.
$\theta_{5-14}, \theta_{15-24}, \theta_{25+}$	= weighting factor for time periods 5–14, 15–24 and >25 years post-exposure (values of θ_{5-14} , etc., are provided in reference [C20] and range from 0.31 to 0.81).
$w_{5-14}, w_{15-24}, w_{25+}$	= radon exposures in the time windows 5–14, 15–24 and >25 years.
θ_{age}	= parameter to describe the decline in ERR with increasing age. BEIR VI [C20] reported values of 0.13–1.00.
γ_z	= parameter to describe the exposure-rate effect. BEIR VI [C20] reported values of 1.0–10.2.

518. For exposure conditions in modern Saskatchewan uranium mines, the power to detect any excess risk arising from workplace exposure to radon is likely to be very small. A study carried out for the Canadian Nuclear Safety Commission [S55] investigated the feasibility of conducting epidemiological studies of underground miners working under today's exposure conditions. To carry out this assessment, two hypothetical cohorts were considered: a retrospective cohort of miners employed in Saskatchewan uranium mines between 1975 and 2000, and a prospective cohort that included miners employed from 2000 to 2030. The cohorts were developed using demographic data provided by the mining companies, dose data from the National Dose Registry, and reference baseline cancer and mortality rates from all of Canada and Saskatchewan. More than 50% of modern Saskatchewan uranium miners smoke. Adjustments were made for different smoking prevalences in miners and the reference population. In 2000, the average underground miner in northern Saskatchewan was exposed at work to about 0.11 WLM from RDPs (with an upper 95% CI of about 0.43 WLM). At the same time, annual exposure to RDPs at home was estimated to be about 0.4 WLM/a (ranging upward to about 10 WLM/a). The feasibility analysis simulated incremental risks of lung cancer from radon exposure using a relative risk model and an ERR of 0.89% WLM⁻¹ based on the Ontario cohort, since it has provided the largest and best exposure data of the Canadian uranium miner studies. Sensitivity analyses were carried out with respect to both reference risk and exposures. Two statistical measures were estimated using probabilistic simulation, standardized mortality ratio (SMR) and regression analysis, which produced an estimate of excess lung cancer risks for a working level month. Both cohorts were modelled to 2030. SMRs were distributed on an average of 1.01, with most (80%) falling in the range 0.93–1.08. Similarly, the slope estimated from the regression analysis had a mean of 1.01, with 80% of the slope estimated to be in the range 0.083–0.105 WLM⁻¹. This quite large range reflected the statistical uncertainty estimated in the cohorts. The results of the regression analyses showed little probability (power) of detecting the predicted excess risks in the cohorts, because the probability of the lower confidence level on the slopes exceeding zero is only about 3% (i.e. much less than the objective of 80%) for most combinations of scenario, cohort and follow-up period. Moreover, exposure at home accounted for about 98% of the total for modern Saskatchewan miners.

519. There is now a great deal of information available concerning the risks from exposure to radon and its decay products. The studies of underground miners exposed to high levels of radon in the past have formed the principal source of information on the risks, and serve as the basis for developing exposure–response models and evaluating modifiers of effects such as time since exposure and age at exposure. A great deal of study has also been given to dosimetric evaluations, both as a means for transferring risk estimates from a miner population to other circumstances and also in their role as a source of *ab initio* risk estimates. While dosimetric evaluations remain important for understanding the

biological and physical mechanisms of carcinogenesis, given the new information that has emerged from the pooling of residential radon case–control studies, the reconciliation of the dosimetric and epidemiological evaluations is now much less important for estimating risks from residential radon. Experimental studies will continue to play an important role in understanding the mechanisms and risks arising from exposure to radon at work and at home, and biologically based models will play an increasingly important role in the future; however, at this time, the epidemiological studies of miners and the residential case–control studies provide the strongest basis for risk assessment.

520. In the past, radon risk estimates for residential exposures were based on downward extrapolation of evidence from studies of miners who were exposed at higher exposures for shorter times. Pooled analyses of European, North American and Chinese residential case–control studies provide strong evidence supporting the evidence from miner studies that exposure to high levels of radon and its decay products in homes leads to an increased incidence of lung cancer.

521. Both the miner and the residential studies have advantages and disadvantages, some of which are briefly summarized below. The advantages of miner studies discussed in section IV include:

- Relatively high (relative to domestic) cumulative exposures and exposure rates, which allowed the development of dose (exposure)–response relationships, at least at high cumulative exposures.
- The ability to examine factors that modify the simple linear dose–effect relationship (time since exposure, age at exposure, exposure rate).
- Information on risks over a lifetime, not just a window of 30 years or so. Miner studies will require continued follow-up to realize this potential to the full.
- Exposure estimates based on contemporary measurements (albeit incomplete, most often area measurements rather than individual measurements and subject to uncertainty). While some evaluations of the effects of the uncertainties in these measurements on the resulting risk estimates were carried out, more remains to be done in this area.
- Information on all causes of death and cancer incidence other than lung cancer.

522. Many residential case–control studies (section V), have been published. Individually, they have limited statistical power, and meta-analysis has suggested that the results of the studies are inconsistent. However, more recent pooled analyses, especially those of European [D17, D21] and North American [K1, K26] studies, combined the data on all the individuals in a number of residential studies and have greater power than the individual component studies. The pooled residential radon studies provide strong, direct

evidence of risk from residential radon. The pooled residential studies have certain advantages over the miner studies:

- Exposures received under aerosol conditions similar to those of interest.
- Exposures received at concentrations similar to those of interest (thus reducing the need to extrapolate from the relatively high rates of exposure in mines).
- Reduced confounding from possible exposure to occupational carcinogens such as arsenic.
- Detailed individual smoking histories for study participants.
- Detailed individual exposure data based on measurements in the homes where the individuals had lived. Nonetheless, exposure uncertainties are also part of residential studies. For example, the measurements were usually made some time after the period over which the risk was to be assessed, and in some instances, there were alterations to the home between the time when the resident lived there and the time when the measurement was made. Data are available regarding the uncertainties in the assessment of residential radon

exposures, and the European pooled analysis, in particular, carried out calculations quantifying the effect of this uncertainty on the risk estimates.

- Data for both men and women for a wide variety of ages.

523. Notwithstanding the strengths and weaknesses of risk estimates from studies of miners and of residential radon, there is now a remarkable coherence between the risk estimates developed from epidemiological studies of miners and pooled residential case-control radon studies. While both the miner studies and the residential case-control radon studies are subject to various limitations arising from exposure uncertainty and confounding by smoking, for example, both types of study are suitable for risk estimation. The miner studies provide a strong basis for evaluating risks from RDP exposure to people at work and at home, and for investigating the effects of modifiers to the exposure-response relationship [S2]. However, the results of the pooled residential studies now provide a direct method of estimating risks to people at home without the need for (downward) extrapolation from miner studies. The measurement-adjusted risk coefficients reported, for example from the European pooling study, provide an appropriate basis for estimating risks to people at home.

VIII. OVERALL CONCLUSIONS

524. This annex, “Sources-to-effects assessment for radon in homes and workplaces”, discusses: potential sources of exposure of workers and the public to radon; issues of current interest in the dosimetry of radon and its decay products; information from animal experiments and experiments at the cellular and subcellular levels that are important in understanding the mechanisms of carcinogenesis; epidemiological studies of miner and residential exposure to radon; and approaches to risk projection.

525. During daily life, everyone is exposed to radon, an inert radioactive gas that occurs naturally and is present everywhere in the atmosphere. The levels of radon indoors vary widely both within countries and between countries, with (nominal) geometric mean concentrations of radon in air indoors ranging from less than 10 Bq/m³ in the Middle East to up to around 100 Bq/m³ in a number of European countries.

526. The annual per caput dose from inhalation of radon gas and its decay products represents typically about one-half of the effective dose received by members of the public from all natural sources of ionizing radiation. For certain occupations, radon gas is the predominant source of occupational radiation exposure.

527. Radon decay products are well established as lung carcinogens. However, the doses to other organs and tissues arising from the inhalation of radon and its decay products are quite small, usually at least an order of magnitude smaller than those to the lung. Moreover, epidemiological data provide little support for increased risks of mortality other than from lung cancer.

528. A factor for calculating the dose from a given exposure to radon and its decay products is needed for risk management, including regulatory purposes, and to allow comparison with other sources of radiation exposure. There are two approaches for deriving a dose conversion factor. A “dosimetric approach” derives the dose from a given exposure based on the deposition characteristics of radon decay products in the respiratory tract. An “epidemiological approach” was used by the International Commission on Radiological Protection (ICRP) to derive the dose conversion factor from epidemiological studies using the ratio of the risk of lung cancer in miners to the overall risk of cancer in the survivors of the atomic bombings in Japan. In the UNSCEAR 2000 Report, there was a difference of about a factor of 2 between the two approaches. However, the most recent data that have been published on the risks

to underground miners (derived from updated studies of cohorts of uranium miners) suggest that the two approaches are less different than initially thought. The Committee therefore continues to recommend a radon dose conversion factor of 9 nSv (Bq h m⁻³)⁻¹ to evaluate the effective dose from radon inhalation. The dose conversion convention recommended in ICRP Publication 65 [12] is approximately 30% lower than this factor but the difference is not considered significant.

529. Studies of miners exposed to radon and its decay products provide a direct basis for assessing lung cancer risk. The United States National Research Council’s 6th Committee on the Biological Effects of Ionizing Radiation (BEIR VI) [C20] reported an excess relative risk from exposure to radon that was equivalent to 1.8% (95% CI: 0.3%, 35%) (MBq h m⁻³)⁻¹ for miners with cumulative exposures of below 30 MBq h m⁻³. There are various sources of error in the exposure assessment of miners, especially in the earliest years of mining, when exposures were at their highest. Other factors that complicate the analysis of data on miners include: the high percentage of miners who smoke; workplace exposure to dust contaminants, such as arsenic, diesel exhaust in the dust and other pollutants; and periods spent working in non-uranium mines. The power to detect any excess risks due to the exposures that miners nowadays receive is likely to be small, in part because the exposures are much smaller than those in the early years of mining. Because of the high exposures in the early days of mining, it is possible to detect trends in lung cancer incidence and to investigate factors that affect the exposure–response relationship, such as the age at exposure, the effect of exposure rate and the reduction of risk with increasing time since exposure, as well as the effect of confounding factors such as smoking.

530. The BEIR VI model developed from the pooled analysis of 11 cohorts of underground miners provides a well-established basis for estimating risks from occupational exposures to radon, and accounts for factors such as the reduced risk with increasing time since exposure. Since the BEIR VI report was published, studies of various miner cohorts have been updated, and confirm the general patterns of risk with dose and with time since exposure that were reported by BEIR VI. They also provide updated coefficients to take account of the effects of time since exposure on ageing populations. Miner studies therefore provide a strong basis for evaluating risks from exposure to radon and for investigating the effects of modifiers to the dose–response relationship.

531. Biological and cellular models of the multistage process of carcinogenesis are used to analyse the data from studies on miners, and offer the possibility to assess uncertainties in our understanding of the mechanisms for the development of cancer and their modelling for the purposes of risk estimation.

532. The extrapolation of radon concentrations in the air in mines to those in homes provides an indirect basis for assessing the risks from residential exposure to radon. However, there are now over 20 analytical studies of residential radon exposure and lung cancer. These studies typically assess the relative risk from exposure to radon on the basis of estimates of residential exposure over a period of 25–30 years prior to the diagnosis of lung cancer. More recent pooled analyses of residential case–control studies support a small but detectable lung cancer risk from residential exposure, and this risk increases with increasing radon concentrations. The excess relative risk of lung cancer from long-term residential exposure to a radon concentration of 100 Bq/m³ is established with reasonably good precision and is considered to be about 16% for both smokers and non-smokers (after correction for uncertainties in the exposure assessment), with an uncertainty of about a factor of 3 higher or lower than this value. Because the baseline lung cancer rate for smokers is

much higher than that for non-smokers, smokers account for nearly 90% of the population risk of lung cancer.

533. Although there are major uncertainties in extrapolating the risks of exposure to radon from the miner studies in order to assess the risks in the home, there is nevertheless remarkably good agreement between the risk factors derived from the miner studies and from the pooled residential case–control studies. The ERR per unit radon concentration in air estimated in this annex from miner studies is 0.12 (95% CI: 0.04, 0.2) per 100 Bq/m³ (see para. 424); that from the pooled residential case–control studies (based on the restricted analysis) for Europe is 0.094 (95% CI: 0.034, 0.175) per 100 Bq/m³ [D17, D21] and for North America is 0.18 (95% CI: 0.02, 0.43) [K1, K26] per 100 Bq/m³ (see table 24). The studies of uranium miners also provide important information on the effects of modifiers to the exposure–response relationship, and further follow-up is encouraged. The pooling of residential case–control studies in Europe, North America and China now provides an appropriate basis for estimating the risks from long-term residential exposure to radon. On the basis of current information, the Committee considers the use of measurement-adjusted risk coefficients from pooled studies as an appropriate basis for estimating the risks to people at home.

Appendix

Quantities, units and conversion factors relevant to radon and its decay products

<i>Term</i>	<i>Definition</i>	<i>Unit and Selected Conversions</i>
Absorbed dose	The energy absorbed through exposure to radiation divided by the mass of the body or by the mass of the part of the body that absorbs the radiation.	Gy 1 Gy = 1 J/kg = 100 rad
Effective dose	The sum over all tissues and organs of the equivalent doses weighted by the tissue weighting factor, which represents the contribution of that organ or tissue to the total detriment resulting from uniform irradiation of the whole body.	Sv 1 Sv = 100 rem
Equilibrium equivalent concentration	The concentration of radon in air, in equilibrium with its short-lived decay products, which would have the same potential alpha energy concentration as the existing non-equilibrium mixture.	Bq/m ³ 1 Bq/m ³ = 5.56 × 10 ⁻⁹ J/m ³
Equilibrium equivalent exposure	Time integral of the corresponding equilibrium equivalent concentration of radon to which the individual is exposed over a given time period.	Bq h m ⁻³ 1 Bq h m ⁻³ = 5.56 × 10 ⁻⁹ J h m ⁻³ 1 Bq h m ⁻³ = 1.57 × 10 ⁻⁶ WLM
Mache unit	A measure of radon concentration (historically radium emanation), where 1000 Mache units equals the amount in equilibrium with 1/2000 mg of radium.	Mache unit 1 Mache unit = 275 pCi/L
Potential alpha energy concentration	The concentration of short-lived radon decay products in air in terms of the alpha energy released during complete decay through polonium-214.	J/m ³ 1 J/m ³ = 1.80 × 10 ⁸ Bq/m ³
Potential alpha energy exposure	Time integral of the potential alpha energy concentration in air to which the individual is exposed over a given time period.	J h m ⁻³ 1 J h m ⁻³ = 1.80 × 10 ⁸ Bq h m ⁻³ 1 J h m ⁻³ = 282 WLM
Radon levels	Radon concentration in air.	pCi/L 1 pCi/L = 0.037 Bq/L 1 pCi/L = 37 Bq/m ³
Relative risk coefficient	The ratio of the risk in an exposed population to that in a similar unexposed population per unit exposure.	—
Working level	Any combination of the short-lived decay products of radon in one litre of air that will result in the emission of 1.3 × 10 ⁵ MeV of potential alpha energy.	WL 1 WL = 100 pCi/L (assuming 100% equilibrium, i.e. F = 1) 1 WL = 250 pCi/L (assuming 40% equilibrium, i.e. F = 0.4)
Working level month	The cumulative exposure from breathing an atmosphere at a concentration of 1 WL for a working month of 170 hours.	WLM 1 WLM = 3.54 × 10 ⁻³ J h m ⁻³ 1 WLM = 6.38 × 10 ⁵ Bq h m ⁻³ 1 WLM = WL × exposure time (h/a)/(170 h/WLM)

References

- A1 Abbatt, J.D. and H.B. Newcombe. The Eldorado Epidemiology Project. Health Follow-Up of Eldorado Uranium Workers. Eldorado Nuclear Ltd., 1987.
- A2 Albertini, R.J., L.S. Clark, J.A. Nicklas et al. Radiation quality affects the efficiency of induction and the molecular spectrum of HPRT mutations in human T cells. *Radiat. Res.* 148(5): S76-S86 (1997).
- A3 Altshuler, B., N. Nelson and M. Kuschner. Estimation of lung tissue dose from the inhalation of radon and daughters. *Health Phys.* 10(12): 1137-1161 (1964).
- A4 Archer, V.E., J.K. Wagoner and F.E. Lundin. Lung cancer among uranium miners in the United States. *Health Phys.* 25(4): 351-371 (1973).
- A5 Auvinen, A., I. Makelainen, M. Hakama et al. Indoor radon exposure and risk of lung cancer: A nested case-control study in Finland. *J. Natl. Cancer Inst.* 88(14): 966-972 (1996).
- A6 Axelson, O. and M. Rehn. Lung cancer in miners. *Lancet* 2(7726): 706-707 (1971).
- A7 Axelson, O. and L. Sundell. Mining, lung cancer and smoking. *Scand. J. Work Environ. Health* 4(1): 46-52 (1978).
- A8 Axelson, O. Room for a role for radon in lung cancer causation? *Med. Hypotheses* 13(1): 51-61 (1984).
- A9 Axelson, O. The case-referent (case-control) study in occupational health epidemiology. *Scand. J. Work Environ. Health* 5(2): 91-99 (1979).
- A10 Archer, V.E., E.P. Radford and O. Axelson. Factors in exposure-response relationships of radon daughter injury. Presented at the Conference/Workshop on Lung Cancer Epidemiology and Industrial Applications of Sputum Cytology, 14-16 November 1978. Colorado School of Mines Press, Golden, Colorado, 1979.
- A11 Axelson, O. Lung cancer among Swedish miners and exposure to radon and radon daughter. Statement of Evidence Presented to the British Columbia Royal Commission of Inquiry into Uranium Mining (1980).
- A12 Archer, V.E., H.J. Magnuson, D.A. Holaday et al. Hazards to health in uranium mining and milling. *J. Occup. Med.* 4(2): 55-60 (1962).
- A13 Axelson, O. Epidemiology of occupational cancer: mining and ore processing. p. 135-149 in: *Prevention of Occupational Cancer — International Symposium. Occupational Safety and Health Series No. 46, 1982.*
- A14 Aylward et al. Report of Royal Commission respecting radiation, compensation and safety at the Fluorspar mines, St. Lawrence, Nfld. (1969).
- A15 Azzam, E.I., S.M. de Toledo and J.B. Little. Direct evidence for the participation of gap junction-mediated intercellular communication in the transmission of damage signals from alpha-particle irradiated to nonirradiated cells. *Proc. Natl. Acad. Sci. U.S.A.* 98(2): 473-478 (2001).
- A16 Archer, V.E. Radon daughter risks to miners and others. *Health Phys.* 45(1): 169-173 (1983).
- A17 Archer, V.E., J.K. Wagoner and F.E. Lundin Jr. Uranium mining and cigarette smoking effects on man. *J. Occup. Med.* 15(3): 204-211 (1973).
- A18 Alavanja, M.C., J.H. Lubin, J.A. Mahaffey et al. Residential radon exposure and risk of lung cancer in Missouri. *Am. J. Public Health* 89(7): 1042-1048 (1999).
- A19 Alavanja, M.C., R.C. Brownson, J.H. Lubin et al. Residential radon exposure and lung cancer among nonsmoking women. *J. Natl. Cancer Inst.* 86(24): 1829-1837 (1994).
- A20 Austin, S.R. and R.F. Drouillard. Radon emanation from domestic uranium ores determined by modifications of the closed-can, gamma-only assay method. Report of investigations 1977. Technical Report, PB-279647, BM-RI-8264. Bureau of Mines, Denver, CO, U.S.A. (1978).
- A21 Archer, V.E. Errors in Cohen's home radon-lung cancer analyses. *Health Phys.* 75(6): 652-654 (1998).
- A22 American National Standards Institute, Inc. (ANSI). Radiation Protection in Uranium Mines. ANSI N13.8-1973, New York, 1973.
- A23 Alavanja, M.C.R. Biologic damage resulting from exposure to tobacco smoke and from radon: implication for preventive interventions. *Oncogene* 21(48): 7365-7375 (2002).
- A24 Alberg, A.J. and J.M. Samet. Epidemiology of lung cancer. *Chest* 123 (1 Suppl.): 21S-49S (2003).
- A25 Abu-Jarad, F. and M.I. Al-Jarallah. Radon in Saudi houses. *Radiat. Prot. Dosim.* 14(3): 243-249 (1986).
- A26 Anderson, R.M., V.V. Tsepenco, G.N. Gasteva et al. mFISH analysis reveals complexity of chromosome aberrations in individuals occupationally exposed to internal plutonium: a pilot study to assess the relevance of complex aberrations as biomarkers of exposure to high-LET alpha particles. *Radiat. Res.* 163(1): 26-35 (2005).
- A27 Anderson, R.M., S.J. Marsden, S.J. Paice et al. Transmissible and nontransmissible complex chromosome aberrations characterized by three-color and mFISH define a biomarker of exposure to high-LET alpha particles. *Radiat. Res.* 159(1): 40-48 (2003). *Erratum in: Radiat. Res.* 159(3): 437 (2003).
- A28 Anderson, R.M., D.L. Stevens and D.T. Goodhead. M-FISH analysis shows that complex chromosome aberrations induced by alpha-particle tracks are cumulative products of localized rearrangements. *Proc. Natl. Acad. Sci. U.S.A.* 99(19): 12167-12172 (2002).
- A29 Anderson, R.M., S.J. Marsden, E.G. Wright et al. Complex chromosome aberrations in peripheral blood lymphocytes as a potential biomarker of exposure to high-LET alpha-particles. *Int. J. Radiat. Biol.* 76(1): 31-42 (2000).

- A30 Australian Radiation Protection and Nuclear Safety Agency (ARPANSA). Naturally-occurring radioactive material (NORM) in Australia: issues for discussion. Prepared by the Radiation Health & Safety Advisory Council for the CEO of ARPANSA (30 June 2004).
- B1 Bale, W.F. Memorandum to the files, March 14, 1951: Hazards Associated with Radon and Thoron. Division of Biology and Medicine, Atomic Energy Commission, Washington, D.C., 1951. *Also found in Health Phys.* 38(6): 1062-1066 (1980).
- B2 Brooks, A.L. Biomarkers of exposure, sensitivity and disease. *Int. J. Radiat. Biol.* 75(12): 1481-1503 (1999).
- B3 Behounek, F. Distribution of radioactivity in the pitchblende mining district of St. Joachimsthal in Bohemia. *Ger. Phys. Z.* 28: 333-342 (1927). (In German).
- B4 Brooks, A.L., G.J. Newton, L.J. Shyr et al. The combined effects of alpha-particles and X-rays on cell killing and micronuclei induction in lung epithelial cells. *Int. J. Radiat. Biol.* 58(5): 799-811 (1990).
- B5 Brüske-Hohlfeld, I., M. Möhner and H.E. Wichmann. Predicted number of lung cancer cases in Germany among former uranium miners of the Wismut. *Health Phys.* 72(1): 3-9 (1997).
- B6 Busigin, A., A. Van der Vooren and C.R. Phillips. Measurement of the total and radioactive aerosol size distributions in a Canadian uranium mine. *Am. Ind. Hyg. Assoc. J.* 42(4): 310-314 (1981).
- B7 Bigu, J. and P. Duport. Characterization of long-lived radioactive dust levels generated in uranium mill operations. CANMET Report 87-105 (1987).
- B8 Bigu, J. and M.G. Grenier. Studies of radioactive dust in Canadian uranium mines. *CIM Bull.* 77(869): 62-68 (1984).
- B9 Bigu, J. and J. Kirk. Experimental determination of the unattached radon daughter fraction and dust size distribution in some Canadian uranium mines. *Can. Min. J.* (August): 39-45 (1982).
- B10 Brenner, D.J. and E.J. Hall. Radiation-induced oncogenic transformation: the interplay between dose, dose protraction, and radiation quality. *Adv. Radiat. Biol.* 16: 167-179 (1992).
- B11 Bloy, H. Pioneering uranium mining and radon testing in Canada. Presented at the International Conference on Radiation Safety in Mining, Saskatoon, Saskatchewan, 25-28 May 1992.
- B12 Bloy, H. Plastics in mine ventilation. Presented at the Seventh Conference on Dust Ventilation of the Mines Accident Prevention Association of Ontario, University of Toronto, May 1958.
- B13 Bloy, H. Second survey of radioactivity, dust, ventilation at Eldorado Mining & Refining Port Radium operation — July and August 1957. Eldorado Nuclear Limited (1957).
- B14 Bloy, H. A survey of radiation and ventilation and dust at the Port Radium operation — September 1956. Eldorado Nuclear Limited (1956).
- B15 Bochicchio, F., G. Campos Venuti, C. Nuccetelli et al. Results of the representative Italian national survey on radon indoors. *Health Phys.* 71(5): 741-748 (1996).
- B16 Bochicchio, F., G. Campos Venuti, C. Nuccetelli et al. Indoor measurements of ^{220}Rn and ^{222}Rn and their decay products in a mediterranean climate area. *Environ. Int.* 22 (Suppl. 1): 633-639 (1996).
- B17 Bernhard, S., J. Pineau, J. Skawnek et al. The radon hazard in non-uranium European mines: retrospective of a survey conducted between 1978 and 1982 in different mines across Europe and new results in French mines. Presented at the International Conference on Technologically Enhanced Natural Radiation Caused by Non-Uranium Mining, Poland, 1996.
- B18 Birchall, A. and A.C. James. Uncertainty analysis of the effective dose per unit exposure from radon progeny and implications for ICRP risk-weighting factors. *Radiat. Prot. Dosim.* 53(1): 133-140 (1994).
- B19 Brenner, D.J., R.C. Miller, Y. Huang et al. The biological effectiveness of radon-progeny alpha particles. III. Quality factors. *Radiat. Res.* 142(1): 61-69 (1995).
- B20 Bijwaard, H., M.J. Brugmans and H.P. Leenhouts. A consistent two-mutation model of lung cancer for different data sets of radon-exposed rats. *Radiat. Environ. Biophys.* 40(4): 269-277 (2001).
- B21 Brenner, D.J., J.B. Little and R.K. Sachs. The bystander effect in radiation oncogenesis: II. A quantitative model. *Radiat. Res.* 155(3): 402-408 (2001).
- B22 Bauer, H.D. Studie zur retrospektiven Analyse der Belastungssituation im Uranerzbergbau der ehemaligen SDAG Wismut mit Ausnahme der Strahlenbelastung für die Zeit von 1946 bis 1990. Hauptverband der gewerblichen Berufsgenossenschaften (HVBG), 2000. ISBN 3-88383-566-8.
- B23 Brüske-Hohlfeld, I., A. Schaffrath Rosario and H.E. Wichmann. Lungenkrebsrisiko bei ehemaligen Uranerzbergarbeitern der Wismut. *Zentralbl. Arbeitsmed. Arbeitsschutz, Prophyl. Ergon.* 52(4): 169 (2002).
- B24 Brüske-Hohlfeld, I., A. Schaffrath Rosario, G. Wölke et al. Fall-Kontroll-Studie zum Lungenkrebs bei Wismut-Beschäftigten. Abschlussbericht an das Bundesamt für Strahlenschutz und den Bundesminister für Umwelt, Naturschutz und Reaktorsicherheit im Vorhaben StSch 4263, Neuherberg/Hannover 2004.
- B25 Blot, W.J., Z.Y. Xu, J.D. Boice Jr. et al. Indoor radon and lung cancer in China. *J. Natl. Cancer Inst.* 82(12): 1025-1030 (1990).
- B26 Barros-Dios, J.M., M.A. Barreiro, A. Ruano-Ravina et al. Exposure to residential radon and lung cancer in Spain: a population-based case-control study. *Am. J. Epidemiol.* 156(6): 548-555 (2002).
- B27 Brugmans, M.J., S.M. Rispens, H. Bijwaard et al. Radon-induced lung cancer in French and Czech miner cohorts described with a two-mutation cancer model. *Radiat. Environ. Biophys.* 43(3): 153-163 (2004).

- B28 Brugmans, M.J.P., S.M. Rispens, H. Bijwaard et al. Multistage model description of French and Czech miner data: implications for radon-induced lung cancer risks. Paper 1b5 presented in Session 1, Radiation Effects of the 11th International Congress of the International Radiation Protection Association, Madrid, Spain, 23-28 May 2004.
- B29 Baysson, H. and M. Tirmarche. Indoor radon exposure and lung cancer risk: a review of case-control studies. *Rev. Epidemiol. Santé Publique* 52(2): 161-171 (2004). (In French).
- B30 Bandom, W.F., G. Saccomanno, V.E. Archer et al. Chromosome aberrations in uranium miners occupationally exposed to 222 radon. *Radiat. Res.* 52(1): 204-215 (1972).
- B31 Bennett, W.P., M.C. Alavanja, B. Blomeke et al. Environmental tobacco smoke, genetic susceptibility, and risk of lung cancer in never-smoking women. *J. Natl. Cancer Inst.* 91(23): 2009-2014 (1999).
- B32 Brenner, D.J., N. Okladnikova, P. Hande et al. Biomarkers specific to densely-ionising (high LET) radiations. *Radiat. Prot. Dosim.* 97(1): 69-73 (2001).
- B33 Baysson, H., M. Tirmarche, G. Tymen et al. Indoor radon and lung cancer in France. *Epidemiology* 15(6): 709-716 (2004).
- B34 Becker, K. Health effects of high radon environments in Central Europe: another test for the LNT hypothesis? *Nonlinearity Biol. Toxicol. Med.* 1(1): 3-35 (2003).
- B35 Balashazy, I., W. Hofmann, A. Farkas et al. Modelling carcinogenic effects of low doses of inhaled radon progenies. *J. Radiol. Prot.* 22(3A): A89-A93 (2002).
- B36 Baysson, H., S. Billon, D. Laurier et al. Seasonal correction factors for estimating radon exposure in dwellings. *Radiat. Prot. Dosim.* 104(3): 245-252 (2003).
- B37 Bijwaard, H., M.J. Brugmans and S.M. Rispens. Comment on "Studies of radon-exposed miner cohorts using a biologically based model: comparison of current Czech and French data with historic data from China and Colorado" by W.F. Heidenreich, L. Tomasek, A. Rogel, D. Laurier and M. Tirmarche (2004) *Radiat. Environ. Biophys.* 43:247-256. *Radiat. Environ. Biophys.* 44(2): 149-151; *author reply*: 153-154 (2005).
- B38 Billon, S., A. Morin, S. Caër et al. French population exposure to radon, terrestrial gamma and cosmic rays. *Radiat. Prot. Dosim.* 113(3): 314-320 (2005).
- B39 Brüske-Hohlfeld, I., A.S. Rosario, G. Wölke et al. Lung cancer risk among former uranium miners of the WISMUT Company in Germany. *Health Phys.* 90(3): 208-216 (2006).
- B40 Bochicchio, F., G. Campos-Venuti, S. Piermattei et al. Annual average and seasonal variations of residential radon concentration for all the Italian regions. *Radiat. Meas.* 40(2-6): 686-694 (2005).
- B41 Bochicchio, F., F. Forastiere, S. Farchi et al. Quality assurance program for LR 115 based radon concentration measurements in a case-control study: description and results. *Radiat. Meas.* 36(1): 205-210 (2003).
- B42 Bochicchio, F. Radon epidemiology and nuclear track detectors: Methods, results and perspectives. *Radiat. Meas.* 40(2-6): 177-190 (2005).
- B43 Bochicchio, F., F. Forastiere, S. Farchi et al. Residential radon exposure, diet and lung cancer: a case-control study in a Mediterranean region. *Int. J. Cancer* 114(6): 983-991 (2005).
- B44 Bochicchio, F., F. Forastiere, D. Abeni et al. Epidemiologic studies on lung cancer and residential exposure to radon in Italy and other countries. *Radiat. Prot. Dosim.* 78(1): 33-38 (1998).
- B45 Bergdahl, I.A., G. Akerblom, K. Andersson et al. Radonexponering i Kirunavaara och Malmbergets järnmalmgruvor under 1900-talet. *Health Effects Among Miners in Northern Sweden*, HEM, ISBN91-89048-16-4 (2005). (In Swedish).
- B46 Bernhard, S., J. Pradel, M. Tirmarche et al. Bilan et enseignement de la radioprotection dans les mines d'uranium depuis 45 ans (1948-1992). *Rev. Gen. Nucl.* 6: 491-497 (1992). Attention, le texte doit être revu en conséquence mais seulement sur la forme (1992).
- B47 Butler, C., J.M. Samet, W.C. Black et al. Histopathologic findings of lung cancer in Navajo men: relationship to U mining. *Health Phys.* 51(3): 365-368 (1986).
- C1 Chamberlain, A.C. and E.D. Dyson. The dose to the trachea and bronchi from the decay products of radon and thoron. *Br. J. Radiol.* 29(342): 317-325 (1956).
- C2 Chambers, D.B., P. Piersol, G.G. Case et al. Industrial Hygiene Survey of the Uranium Mining and Milling Industry. Prepared for the Canada Centre for Mineral and Energy Technology by James F. MacLaren Ltd., Toronto, Ontario, 1977.
- C3 Chambers, D.B. and H.A. Phillips. The current status of biological dosimeters. p. 507-516 in: *Medical Management of Radiation Accidents*, Second Edition (I.A. Gusev, A.K. Guskova and F.A. Mettler, eds.). CRC Press, Boca Raton, Florida, 2001.
- C4 Chameaud, J., R. Perraud, J. Chretien et al. Combined effects of inhalation of radon daughter products and tobacco smoke. p. 551-557 in: *Pulmonary Toxicology of Respirable Particles* (C.L. Sanders, F.T. Cross, G.E. Dagle et al., eds.). CONF-791002 (1980).
- C5 Ching, S.H., D. Corkill and K.P. Ho. A review of gamma doses and radon daughter exposures for workers in some Canadian underground uranium mines from 1984 to 1987. Presented at 10th Annual Conference of the Canadian Radiation Protection Association, Victoria, B.C., 1989.
- C6 Ching, S.H., K.P. Ho and H. Bayne. Gamma doses and radon daughter exposures in some Canadian uranium mines and implications with respect to the proposed regulatory amendments. p. 567-577 in: *Proceedings of an International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.

- C7 Cohen, B.L. Radon and lung cancer in Swedish miners. *N. Engl. J. Med.* 313(18): 1158-1159 (1985).
- C8 Cohn, S.H., R.K. Skow and J.K. Gong. Radon inhalation studies in rats. *A.M.A. Arch. Ind. Hyg. Occup. Med.* 7(6): 508-515 (1953).
- C9 Conrady, J., K. Martin, A. Poffijn et al. High residential radon health effects in Saxony (Schneeberg study). Contract No. F14P-CT95-0027. European Commission, DG XII, Nuclear Fission Safety Programme (August 1999).
- C10 Cooper, J.A., P.O. Jackson, J.C. Langford et al. Characteristics of attached radon-222 daughters under both laboratory and field conditions with particular emphasis upon underground mine environments. Report to the U.S. Bureau of Mines, Contract H0220029. Battelle Pacific Northwest Laboratories, Richland, Washington (1973).
- C11 Cooper, W.C. Proceedings of the Conference on Epidemiologic Research in Occupational Health. Design of field studies. Case study No. 1: Uranium milling and mining. *J. Occup. Med.* 4(Suppl.): 614-627 (1962).
- C12 Corkill, D.A. and A.B. Dory. A retrospective study of radon daughter concentrations in the workplace in the fluorspar mines of St. Lawrence, Nfld. AECB INFO-0127 (1984).
- C13 Cross, F.T., R.F. Palmer, R.E. Filipy et al. Study of the combined effects of smoking and inhalation of uranium ore dust, radon daughters and diesel oil exhaust fumes in hamsters and dogs. PNL-2744 (1978).
- C14 Cross, F.T., R.F. Palmer, R.E. Filipy et al. Carcinogenic effects of radon daughters, uranium ore dust and cigarette smoke in beagle dogs. *Health Phys.* 42(1): 33-52 (1982).
- C15 Chambers, D.B., P.M. Reilly, L.M. Lowe et al. Effects of exposure uncertainty on estimation of radon risks. p. 987-1011 in: *Indoor Radon and Lung Cancer: Reality or Myth?* (F.T. Cross, ed.). 29th Hanford Symposium on Health and the Environment. Battelle Press, Columbus-Richland, Washington, 1990.
- C16 Collier, C.G., J.C. Strong, S.T. Baker et al. Effects of continuous inhalation exposure of rats to radon and its progeny at various levels of dose and dose rate: interim results. *Radiat. Res.* 152(6): S141-S144 (1999).
- C17 Chambers, D.B., R.H. Stager and S.E. Frost. How uncertainty could affect dose-response in the Beaverlodge, Canada miners studies. In: *Proceedings of a Workshop on "Uncertainties in Radiation Dosimetry and Their Impact on Dose-Response Analysis"*, 3-5 September, 1997, Bethesda, Maryland. National Cancer Institute, National Institutes of Health, 1997.
- C18 Committee on the Biological Effects of Ionizing Radiations (BEIR III). *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation: 1980*. National Academy of Sciences, National Research Council. National Academy Press, Washington, 1980.
- C19 Committee on the Biological Effects of Ionizing Radiations (BEIR IV). *Health Risks of Radon and Other Internally Deposited Alpha-Emitters*. National Academy of Sciences, National Research Council. National Academy Press, Washington, 1988.
- C20 Committee on the Biological Effects of Ionizing Radiations (BEIR VI). *The Health Effects of Exposure to Indoor Radon*. National Academy of Sciences, National Research Council. National Academy Press, Washington, 1999.
- C21 Chittaporn, P., N.H. Harley, R. Medora et al. Measurements of outdoor radon and thoron at Fernald, OH, New York City and New Jersey. *Health Phys.* 80 (Suppl. 6): S171 (2001).
- C22 Chittaporn, P., N.H. Harley, R. Medora et al. Indoor and outdoor thoron decay product equilibrium at Fernald, OH, New York City, and New Jersey. *Health Phys.* 84(6): S199 (2003).
- C23 Cohen, B.L. A test of the linear-no threshold theory of radiation carcinogenesis. *Environ. Res.* 53(2): 193-220 (1990).
- C24 Cohen, B.L. Test of the linear-no threshold theory of radiation carcinogenesis for inhaled radon decay products. *Health Phys.* 68(2): 157-174 (1995).
- C25 Cohen, B.L. Lung cancer rate vs. mean radon level in U.S. counties of various cancer characteristics. *Health Phys.* 72(1): 114-119 (1997).
- C26 Cohen, B.L. Problems in the radon vs. lung cancer test of the linear no-threshold theory and a procedure for resolving them. *Health Phys.* 72(4): 623-628 (1997).
- C27 Cohen, B.L. Response to criticisms of Smith et al. *Health Phys.* 75(1): 23-33 (1998).
- C28 Cohen, B.L. Testing a BEIR-VI suggestion for explaining the lung cancer vs. radon relationship for U.S. counties. *Health Phys.* 78(5): 522-527 (2000).
- C29 Castrén, O. Implications of a two-stage clonal expansion model to indoor radon risk assessment. *Health Phys.* 76(4): 393-397 (1999).
- C30 Crawford-Brown, D.J. and W. Hofmann. Analysis of radon-induced lung cancer risk by a stochastic state-vector model of radiation carcinogenesis. *J. Radiol. Prot.* 22(3A): A61-A65 (2002).
- C31 Curtis, S.B., E.G. Luebeck, W.D. Hazelton et al. A new perspective of carcinogenesis from protracted high-LET radiation arises from the two-stage clonal expansion model. *Adv. Space Res.* 30(4): 937-944 (2002).
- C32 Conrady, J., K. Martin, J. Lembecke et al. The true size of the lung cancer risk from indoor radon: hidden behind a smoke screen? *Int. Congress Series* 1225: 253-258 (2002).
- C33 Chung, W.H., S. Tokonami and M. Furukawa. Preliminary survey on radon and thoron concentrations in Korea. *Radiat. Prot. Dosim.* 80(4): 423-426 (1998).
- C34 Collier, C.G., J.C. Strong, S.T. Baker et al. Update on the progress of a lifespan study in animals to investigate the effect of dose and dose rate on lung tumour

- induction by radon/radon progeny. Presented at the Eurosymposium on Protection Against Radon, Liege, Belgium, 10-11 May 2001.
- C35 Cohen, B.L. Response to “residential radon exposure and lung cancer risk: commentary on Cohen’s county-based study”. *Health Phys.* 87(6): 656-658 (2004).
- C36 Chen, W. and J. Chen. Nested case-control study of lung cancer in four Chinese tin mines. *Occup. Environ. Med.* 59(2): 113-118 (2002).
- C37 Cohen, B.L. The Puskin observation on smoking as a confounder in ecologic correlations of cancer mortality rates with average county radon levels. *Health Phys.* 86(2): 203-204 (2004).
- C38 Collier, C.G., J.C. Strong, J.A. Humphreys et al. Carcinogenicity of radon/radon decay product inhalation in rats — effect of dose, dose rate and unattached fraction. *Int. J. Radiat. Biol.* 81(9): 631-647 (2005).
- C39 Crispo, A., P. Brennan, K.H. Jöckel et al. The cumulative risk of lung cancer among current, ex- and never-smokers in European men. *Br. J. Cancer* 91(7): 1280-1286 (2004).
- C40 Cross, F.T. and G. Monchaux. Risk assessment of radon health effects from experimental animal studies. A joint review of PNNL (USA) and CEA-COGEMA (France) data. p. 85-105 in: *Indoor Radon Exposure and its Health Consequences. Quest for the True Story of Environmental Radon and Lung Cancer* (J. Inaba, H. Yonehara, M. Doi, eds.). Kodansha Scientific Limited, Tokyo, 1999.
- C41 Canoba, A., F.O. Lopez, M.I. Arnaud et al. Indoor radon measurements in six Latin American countries. *Geofis. Int.* 41(4): 453-457 (2002).
- C42 Cooper, M. Naturally occurring radioactive materials (NORM) in Australian industries — Review of current inventories and future generation. A report prepared for the Radiation Health and Safety Advisory Council. ERS-006, EnviroRad Services Pty. Ltd. (December 2003).
- C43 Chittaporn, P. and N.H. Harley. Indoor and outdoor ²²²Rn measurements in Bangkok and Chiang Mai, Thailand. *Technology* 7: 491-495 (2000).
- C44 Cross, F.T. Invited commentary: residential radon risks from the perspective of experimental animal studies. *Am. J. Epidemiol.* 140(4): 333-339 (1994).
- D1 Damber, L. and L.G. Larsson. Combined effects of mining and smoking in the causation of lung carcinoma — a case-control study in northern Sweden. *Acta Radiol. Oncol.* 21(5): 305-313 (1982).
- D2 Damber, L. and L.G. Larsson. Underground mining, smoking, and lung cancer: a case-control study in the iron ore municipalities in northern Sweden. *J. Natl. Cancer Inst.* 74(6): 1207-1213 (1985).
- D3 Deshpande, A., E.H. Goodwin, S.M. Bailey et al. Alpha-particle-induced sister chromatid exchange in normal human lung fibroblasts: evidence for an extranuclear target. *Radiat. Res.* 145(3): 260-267 (1996).
- D4 De Villiers, A.J. and J.P. Windish. Lung cancer in a fluorspar mining community. I. Radiation, dust, and mortality experience. *Br. J. Ind. Med.* 21: 94-109 (1964).
- D5 De Villiers, A.J., J.P. Windish, F. Brent et al. Mortality experience of the community and of the fluorspar mining employees at St. Lawrence, Newfoundland. *Occup. Health Rev.* 22(1): 1-15 (1971).
- D6 Dory, A.B. and D.A. Corkill. Practical approach to retrospective estimation of radon daughters concentration in the underground mining environment. p. 182-188 in: *Proceedings of an International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.
- D7 DSMA Atcon Ltd. Elliot Lake Study: Factors affecting the uranium mine working environment prior to introduction of current ventilation practices. AECB INFO-0154 (1985).
- D8 DSMA Atcon Ltd. Comparison of radon and thoron daughter behaviour in two underground uranium mine environments. AECB INFO-0164 (1985).
- D9 Duport, P. Annual number of measurements realized in French uranium mines during the period 1953-1982 (previous to the use of individual dosimeters). Communication to the UNSCEAR Secretariat (1994).
- D10 Duport, P. and E. Edwardson. Determination of the contribution of long-lived dust to the committed dose equivalent received by uranium mine and mill workers in the Elliot Lake area. AECB INFO-0167-1 and INFO-0167-2 (1985).
- D11 Doi, M., K. Fujimoto, S. Kobayashi et al. Spatial distribution of thoron and radon concentrations in the indoor air of a traditional Japanese wooden house. *Health Phys.* 66(1): 43-49 (1994).
- D12 Darby, S.C., E. Whitley, G.R. Howe et al. Radon and cancers other than lung cancer in underground miners: a collaborative analysis of 11 studies. *J. Natl. Cancer Inst.* 87(5): 378-384 (1995).
- D13 Dano, L., M.N. Guilly, M. Muleris et al. CGH analysis of radon-induced rat lung tumors indicates similarities with human lung cancers. *Genes Chromosomes Cancer* 29(1): 1-8 (2000).
- D14 Darby, S., D. Hill and R. Doll. Radon: a likely carcinogen at all exposures. *Ann. Oncol.* 12(10): 1341-1351 (2001).
- D15 Darby, S., E. Whitley, P. Silcocks et al. Risk of lung cancer associated with residential radon exposure in south-west England: a case-control study. *Br. J. Cancer* 78(3): 394-408 (1998).
- D16 Darby, S.C. and D.C. Hill. Health effects of residential radon: a European perspective at the end of 2002. *Radiat. Prot. Dosim.* 104(4): 321-329 (2003).
- D17 Darby, S., D. Hill, A. Auvinen et al. Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case-control studies. *Br. Med. J.* 330(7485): 223 (2005).
- D18 Doi, M. and S. Kobayashi. Vertical distribution of outdoor radon and thoron in Japan using a new discriminative dosimeter. *Health Phys.* 67(4): 385-392 (1994).

- D19 Dano, L., M.N. Guilly, B. Dutrillaux et al. Clonal evolution of a radon-induced rat lung tumor. *Cancer Genet. Cytogenet.* 125(1): 52-58 (2001).
- D20 Dubois, G. An overview of radon surveys in Europe. EUR 21892 EN, European Commission (2005).
- D21 Darby, S., D. Hill, H. Deo et al. Residential radon and lung cancer — detailed results of a collaborative analysis of individual data on 7148 persons with lung cancer and 14 208 persons without lung cancer from 13 epidemiologic studies in Europe. *Scand. J. Work Environ. Health* 32 (Suppl. 1): 1-84 (2006).
- D22 Darby, S.C., E.P. Radford and E. Whitley. Radon exposure and cancers other than lung cancer in Swedish iron miners. *Environ. Health Perspect.* 103 (Suppl. 2): 45-47 (1995).
- D23 Desrosiers, A., A. Kennedy and J.B. Little. ²²²Rn daughter dosimetry in the Syrian golden hamster lung. *Health Phys.* 35(5): 607-623 (1978).
- E1 Edling, C. Lung cancer and smoking in a group of iron ore miners. *Am. J. Ind. Med.* 3(2): 191-199 (1982).
- E2 Edling, C. and O. Axelson. Quantitative aspects of radon daughter exposure and lung cancer in underground miners. *Br. J. Ind. Med.* 40(2): 182-187 (1983).
- E3 Eigenwillig, G.G. and E. Ettenhuber. Radiation Exposure and Radiation Induced Occupational Diseases in Uranium Mining Using Wismut as an Example. Publication Series, Progress in Radiation Protection: ISSN 1013-4506. Verlag TÜV Rheinland, Köln, 2000.
- E4 Enderle, G.J. and K. Friedrich. East German uranium miners (Wismut) — exposure conditions and health consequences. *Stem Cells* 13 (Suppl. 1): 78-89 (1995).
- E5 Etherington, D.J., D.F. Pheby and F.I. Bray. An ecological study of cancer incidence and radon levels in South West England. *Eur. J. Cancer* 32A(7): 1189-1197 (1996).
- E6 Eatough, J.P. Radon and leukemia risk in underground miners: are working level months the most appropriate exposure parameter? *Health Phys.* 86(4): 425-426 (2004).
- E7 Evrard, A.S., D. Hémon, S. Billon et al. Ecological association between indoor radon concentration and childhood leukaemia incidence in France, 1990-1998. *Eur. J. Cancer Prev.* 14(2): 147-157 (2005).
- E8 Evrard, A.S., D. Hémon, S. Billon et al. Childhood leukemia incidence and exposure to indoor radon, terrestrial and cosmic gamma radiation. *Health Phys.* 90(6): 569-579 (2006).
- F1 Falk, R., K. Almrén and I. Östergren. Experience from retrospective radon exposure estimations for individuals in a radon epidemiological study using solid-state nuclear track detectors. *Sci. Total Environ.* 272(1-3): 61-66 (2001).
- F2 Federal Radiation Council. Guidance for the control of radiation hazards in uranium mining. Staff Report No. 8 (Revised). U.S. Government Printing Office, Washington (1967).
- F3 Fisher, E.L., R.W. Field, B.J. Smith et al. Spatial variation of residential radon concentrations: the Iowa Radon Lung Cancer Study. *Health Phys.* 75(5): 506-513 (1998).
- F4 Forastiere, F., A. Sperati, G. Cherubini et al. Adult myeloid leukaemia, geology, and domestic exposure to radon and gamma radiation: a case control study in central Italy. *Occup. Environ. Med.* 55(2): 106-110 (1998).
- F5 Frost, S.E. Port Radium working level month calculations. Draft report. Cameco Limited, Saskatoon (1995).
- F6 Frost, S.E. Port Radium working level month calculations (Draft #1). Eldorado Resources Limited, Ottawa (1983). *As cited in [H18]*.
- F7 Frost, S.E. Beaverlodge working level month calculations (Draft #4). Eldorado Resources Limited, Ottawa (1983).
- F8 Field, R.W., P.J. Duport and W.R. Hendee. Point/Counterpoint: Exposure to residential radon causes lung cancer. *Med. Phys.* 30(4): 485-488 (2003).
- F9 Field, R.W., D.J. Steck, M.A. Parkhurst et al. Inter-comparison of retrospective radon detectors. *Environ. Health Perspect.* 107(11): 905-910 (1999).
- F10 Field, R.W., D.J. Steck, B.J. Smith et al. The Iowa radon lung cancer study — phase I: Residential radon gas exposure and lung cancer. *Sci. Total Environ.* 272(1-3): 67-72 (2001).
- F11 Field, R.W., D.J. Steck, B.J. Smith et al. Residential radon gas exposure and lung cancer. *The Iowa Radon Lung Cancer Study. Am. J. Epidemiol.* 151(11): 1091-1102 (2000).
- F12 Field, R.W., K. Becker and J.C. McDonald. Topics under debate — Does exposure to residential radon increase the risk of lung cancer? *Radiat. Prot. Dosim.* 95(1): 75-81 (2001).
- F13 Finkelstein, M.M. Silicosis, radon, and lung cancer risk in Ontario miners. *Health Phys.* 69(3): 396-399 (1995).
- F14 Fujimoto, K. Mapping of nationwide indoor radon survey in Japan. NIRS-M-171 (2004).
- F15 Fujimoto, K., S. Kobayashi, M. Uchiyama et al. Nationwide indoor radon survey in Japan. *Jpn. J. Health Phys.* 32(1): 41-51 (1997).
- F16 Fukutsu, K., Y. Yamada, S. Tokonami et al. A new graded screen array for radon progeny size measurements and its numerical verification. *J. Atmos. Electr.* 23: 49-56 (2003).
- F17 Fukutsu, K., Y. Yamada, S. Tokonami et al. Newly designed graded screen array for particle size measurements of unattached radon decay products. *Rev. Sci. Instrum.* 75(3): 783-787 (2004).
- F18 Francis, M., S. Selvin, R. Spear et al. The effect of autocorrelation on the estimation of worker's daily exposures. *Am. Ind. Hyg. Assoc. J.* 50(1): 37-43 (1989).
- F19 Frost, S. Eldorado epidemiology update. Final report: Preparation of the cohort, work histories and dosimetry. Saskatchewan Uranium Miners Study. Prepared

- by Frost & Frost, Saskatoon, Saskatchewan (March 31, 2005).
- F20 Fan, X., Y. Zhang, H. Yu et al. Level of radon and its daughters, and internal exposure doses in Shaanxi province. *Chin. J. Radiol. Med. Prot.* 12(3): 175-181 (1992). (In Chinese).
- G1 George, A.C., L. Hinchliffe and R. Sladowski. Size distribution of radon daughter particles in uranium mine atmospheres. *Am. Ind. Hyg. Assoc. J.* 36(6): 484-490 (1975).
- G2 George, A.C. and A.J. Breslin. The distribution of ambient radon and radon daughters in residential buildings in the New Jersey–New York area. p. 1272-1292 in: *Natural Radiation Environment III, Volume 2* (T.F. Gesell and W.M. Lowder, eds.). CONF-780422 (1980).
- G3 Gilbert, E.S., F.T. Cross and G.E. Dagle. Analysis of lung tumor risks in rats exposed to radon. *Radiat. Res.* 145(3): 350-360 (1996).
- G4 Gilliland, F.D., W.C. Hunt, V.E. Archer et al. Radon progeny exposure and lung cancer risk among non-smoking uranium miners. *Health Phys.* 79(4): 365-372 (2000).
- G5 Grace, M., M. Larson and J. Hanson. Bronchogenic carcinoma among former uranium mine workers at Port Radium, Canada — a pilot study. *Health Phys.* 38(4): 657-661 (1980).
- G6 Grainger, P., S.H. Shalla, A.W. Preece et al. Home radon levels and seasonal correction factors for the Isle of Man. *Phys. Med. Biol.* 45(8): 2247-2252 (2000).
- G7 Greenberg, M. and I.J. Selikoff. Lung cancer in the Schneeberg mines: a reappraisal of the data reported by Harting and Hesse in 1879. *Ann. Occup. Hyg.* 37(1): 5-14 (1993).
- G8 Goodwin, E. and B.E. Lehnert. Bystander effects of radiation: mechanisms of action and significance in risk assessment. *Radiat. Res.* 151(1): 114-116 (1999).
- G9 Garbutt, G.C. (ed.). *Uranium in Canada. Eldorado Mining and Refining Ltd., Canada, 1964.*
- G10 Green, B.M.R., J.C.H. Miles, E.J. Bradley et al. Radon atlas of England and Wales. NRPB-W26 (2002).
- G11 Green, B.M.R., P.R. Lomas and G.M. Kendall. Memorandum. Radon in dwellings in Scotland: 1996 review. NRPB-M569 (1996).
- G12 Green, B.M.R., P.R. Lomas, J.C.H. Miles et al. Radon in dwellings in Northern Ireland: atlas and 1999 review. NRPB-R308 (1999).
- G13 Guo, Q., T. Iida, K. Okamoto et al. Measurements of thoron concentration by passive cup method and its application to dose assessment. *J. Nucl. Sci. Technol.* 32(8): 794-803 (1995).
- G14 Grosche, B., M. Kreuzer, A. Brachner et al. Investigation of health effects among German uranium miners: the design of three studies. *Epidemiology* 12 (Suppl. 4): S74 (2001).
- G15 Goldsmith, J.R. The residential radon-lung cancer association in U.S. counties: a commentary. *Health Phys.* 76(5): 553-557 (1999).
- G16 Greenland, S. and J. Robins. Invited commentary: ecologic studies — biases, misconceptions, and counterexamples. *Am. J. Epidemiol.* 139(8): 747-760 (1994).
- G17 Grosche, B., M. Kreuzer, M. Kreisheimer et al. The risk of lung cancer in the German uranium miners cohort — a first analysis and comparison with results from BEIR VI. p. 136 in: *HEIR 2004. Proceedings of the Ninth International Conference on Health Effects of Incorporated Radionuclides. Emphasis on Radium, Thorium, Uranium and their Daughter Products* (U. Oeh, P. Roth and H.G. Paretzke, eds.), Neuherberg, Germany, 2004. GSF-Bericht 06/05 (2005).
- H1 Ham, J.M. Report of the Royal Commission on the health and safety of workers in mines. Ministry of the Attorney General of the Province of Ontario, Toronto, Canada (1976).
- H2 Hamilton, L.D., L.W. Swent and D.B. Chambers. Visit to the Centre of Radiation Hygiene, Institute of Hygiene and Epidemiology, Prague, Czechoslovakia. Trip Report to Division of Environmental Health, World Health Organization, Geneva, Switzerland. BNL-49734 (1990).
- H3 Haque, A.K. and A.J. Collinson. Radiation dose to the respiratory system due to radon and its daughter products. *Health Phys.* 13(5): 431-443 (1967).
- H4 Harley, N.H., B.S. Cohen and E.S. Robbins. The variability in radon decay product bronchial dose. *Environ. Int.* 22 (Suppl. 1): S959-S964 (1996).
- H5 Harley, N.H. and B.S. Pasternack. Alpha absorption measurements applied to lung dose from radon daughters. *Health Phys.* 23(6): 771-782 (1972).
- H6 Harley, N.H. and B.S. Pasternack. Environmental radon daughter alpha dose factors in a five-lobed human lung. *Health Phys.* 42(6): 789-799 (1982).
- H7 Hazelton, W.D., E.G. Luebeck, W.F. Heidenreich et al. Analysis of a historical cohort of Chinese tin miners with arsenic, radon, cigarette smoke, and pipe smoke exposures using the biologically based two-stage clonal expansion model. *Radiat. Res.* 156(1): 78-94 (2001).
- H8 Heidenreich, W.F., P. Jacob, H.G. Paretzke et al. Two-step model for the risk of fatal and incidental lung tumors in rats exposed to radon. *Radiat. Res.* 151(2): 209-219 (1999).
- H9 Hornung, R.W., J.A. Deddens and R.J. Roscoe. Modifiers of lung cancer risk in uranium miners from the Colorado plateau. *Health Phys.* 74(1): 12-21 (1998).
- H10 Hofmann, W. Cellular lung dosimetry for inhaled radon decay products as a base for radiation-induced lung cancer risk assessment. I. Calculation of mean cellular doses. *Radiat. Environ. Biophys.* 20(2): 95-112 (1982).
- H11 Holaday, D.A. History of the exposure of miners to radon. *Health Phys.* 16(5): 547-552 (1969).
- H12 Holaday, D.A. Radiation hazards in uranium mines. *Radiol. Health Data Rep.* 8(3): 135-138 (1967).

- H13 Holaday, D.A. and H.N. Doyle. Environmental studies in the uranium mines. p. 9-20 in: Radiological Health and Safety in Mining and Milling of Nuclear Materials, Vol. 1. IAEA, Vienna (1964).
- H14 Holaday, D.A. The radon problem in deep-level mining. *AMA Arch. Ind. Health* 12(2): 163-166 (1955).
- H15 Howe, G. Computer files containing work histories and exposure estimates for the Port Radium case-control group previously used in epidemiological studies. Communication to the UNSCEAR Secretariat (1994).
- H16 Howe, G.R. and R.H. Stager. Risk of lung cancer mortality after exposure to radon decay products in the Beaverlodge cohort based on revised exposure estimates. *Radiat. Res.* 146(1): 37-42 (1996).
- H17 Hornung, R.W. and T.J. Meinhardt. Quantitative risk assessment of lung cancer in U.S. uranium miners. *Health Phys.* 52(4): 417-430 (1987).
- H18 Howe, G.R., R.C. Nair, H.B. Newcombe et al. Lung cancer mortality (1950-80) in relation to radon daughter exposure in a cohort of workers at the Eldorado Port Radium uranium mine: possible modification of risk by exposure rate. *J. Natl. Cancer Inst.* 79(6): 1255-1260 (1987).
- H19 Howe, G.R., R.C. Nair, H.B. Newcombe et al. Lung cancer mortality (1950-80) in relation to radon daughter exposure in a cohort of workers at the Eldorado Beaverlodge uranium mine. *J. Natl. Cancer Inst.* 77(2): 357-362 (1986).
- H20 Harley, N.H. and P. Chittaporn. Long term measurement of indoor and outdoor ^{212}Pb decay products, with estimates of aerosol particle size. *Technology* 7(2/4): 407-414 (2000).
- H21 Harley, N.H., P. Chittaporn, R. Medora et al. Indoor thoron profiles at Fernald, OH and a New Jersey home. *Health Phys.* 84(6): S199 (2003).
- H22 Hopke, P.K., B. Jensen, C.S. Li et al. Assessment of the exposure to and dose from radon decay products in normally occupied homes. *Environ. Sci. Technol.* 29(5): 1359-1364 (1995).
- H23 Hopke, P.K. A critical review of measurements of the "unattached" fraction of the radon decay products. DOE/ER-045IP (1990).
- H24 Harley, J.H., E.S. Jetter and N. Nelson. Elimination of radon from the body. *Environ. Int.* 20(5): 573-584 (1994). Reprinted from USAEC, Health and Safety Laboratory Report 32 (1958).
- H25 Hursh, J.B., D.A. Morken, T.P. Davis et al. The fate of radon ingested by man. *Health Phys.* 11(6): 465-476 (1965).
- H26 Heidenreich, W.F., M.J. Brugmans, M.P. Little et al. Analysis of lung tumour risk in radon-exposed rats: an intercomparison of multi-step modelling. *Radiat. Environ. Biophys.* 39(4): 253-264 (2000).
- H27 Holaday, D.A., D.E. Rushing, R.D. Coleman et al. Control of radon and daughters in uranium mines and calculations on biological effects. PHS Publication No. 494. U.S. Government Printing Office, Washington, D.C., 1957.
- H28 Hussain, S.P., C.H. Kennedy, P. Amstad et al. Radon and lung carcinogenesis: mutability of *p53* codons 249 and 250 to ^{238}Pu alpha-particles in human bronchial epithelial cells. *Carcinogenesis* 18(1): 121-125 (1997).
- H29 Heath, C.W. Jr., P.D. Bond, D.G. Hoel et al. Residential radon exposure and lung cancer risk: commentary on Cohen's county-based study. *Health Phys.* 87(6): 647-655 (2004).
- H30 Hauptmann, M., K. Berhane, B. Langholz et al. Using splines to analyse latency in the Colorado Plateau uranium miners cohort. *J. Epidemiol. Biostat.* 6(6): 417-424 (2001).
- H31 Harley, N.H. and B.S. Pasternack. Experimental absorption applied to lung dose from thoron daughters. *Health Phys.* 24(4): 379-386 (1973).
- H32 Heidenreich, W.F., L. Tomasek, A. Rogel et al. Studies of radon-exposed miner cohorts using a biologically based model: comparison of current Czech and French data with historic data from China and Colorado. *Radiat. Environ. Biophys.* 43(4): 247-256 (2004).
- H33 Heidenreich, W.F. Response to the comment on "Studies of radon-exposed miner cohorts using a biologically based model: comparison of current Czech and French data with historic data from China and Colorado" by W.F. Heidenreich, L. Tomasek, A. Rogel, D. Laurier, M. Tirmarche (2004) *Radiat. Environ. Biophys.* 43:247-256. *Radiat. Environ. Biophys.* 44(2): 153-154 (2005).
- H34 Heidenreich, W.F., C. Collier, J.P. Morlier et al. Age-adjustment in experimental animal data and its application to lung cancer in radon-exposed rats. *Radiat. Environ. Biophys.* 43(3): 183-188 (2004).
- H35 Howe, G. Final Report. Eldorado Nuclear Epidemiology Study Update. Eldorado Uranium Miners' Cohort: Part I of the Saskatchewan Uranium Miners' Cohort Study. RSP-0205. Prepared for the Canadian Nuclear Safety Commission (March 16, 2006).
- H36 Harley, N.H., P. Chittaporn, O.A. Meyers et al. A biological model for lung cancer risk from ^{222}Rn exposure. *Environ. Int.* 22 (Suppl. 1): 977-984 (1996).
- H37 Heid, I.M., H. Küchenhoff, J. Miles et al. Two dimensions of measurement error: classical and Berkson error in residential radon exposure assessment. *J. Expo. Anal. Environ. Epidemiol.* 14(5): 365-377 (2004).
- H38 Harley, N.H. and E.S. Robbins. ^{222}Rn alpha dose to organs other than lung. *Radiat. Prot. Dosim.* 45(1): 619-622 (1992).
- H39 Harley, N.H. Radon daughter dosimetry in the rat tracheobronchial tree. *Radiat. Prot. Dosim.* 24(1): 457-461 (1988).
- H40 Harley, N.H., O.A. Meyers and E.S. Robbins. ^{222}Rn dosimetry in the dog lung. *Radiat. Prot. Dosim.* 45(1): 611-617 (1992).
- H41 Heid, M., H. Küchenhoff, J. Wellmann et al. On the potential of measurement error to induce differential bias on odds ratio estimates: an example from radon epidemiology. *Stat. Med.* 21(21): 3261-3278 (2002).

- I1 International Commission on Radiological Protection. Biological Effects of Inhaled Radionuclides. ICRP Publication 31. Pergamon Press, Oxford, 1980.
- I2 International Commission on Radiological Protection. Protection Against Radon-222 at Home and at Work. Annals of the ICRP 23(2). ICRP Publication 65. Pergamon Press, Oxford, 1993.
- I3 International Commission on Radiological Protection. Human Respiratory Tract Model for Radiological Protection. Annals of the ICRP 24(1-3). ICRP Publication 66. Pergamon Press, Oxford, 1994.
- I4 International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection. Report of Committee II on Permissible Dose for Internal Radiation. ICRP Publication 2. Pergamon Press, London, 1959.
- I5 International Commission on Radiological Protection. Lung Cancer Risk from Indoor Exposures to Radon Daughters. Annals of the ICRP 17(1). ICRP Publication 50. Pergamon Press, Oxford, 1987.
- I6 Ishikawa, T., S. Tokonami, S. Yoshinaga et al. Airborne and waterborne radon concentrations in areas with use of groundwater supplies. *J. Radioanal. Nucl. Chem.* 267(1): 85-88 (2006).
- I7 Ishikawa, T., S. Tokonami, H. Yonehara et al. Effects of activity size distribution on dose conversion factor for radon progeny. *J. Health Phys.* 36(4): 329-338 (2001). (In Japanese).
- I8 Israël, H. The radon-220 content of the atmosphere. p. 313-314 in: *The Natural Radiation Environment* (J.A.S. Adams and W.M. Lowder, eds.). The University of Chicago Press, Chicago and London, 1964.
- I9 Iida, T., Q. Guo and Y. Ikebe. Some problems on the measurement of ^{222}Rn concentrations by passive cup method. *Health Phys.* 69(4): 508-512 (1995).
- I10 International Commission on Radiological Protection. Limits for Inhalation of Radon Daughters by Workers. ICRP Publication 32. Annals of the ICRP 6(1). Pergamon Press, Oxford, 1981.
- I11 International Commission on Radiological Protection. Relative Biological Effectiveness (RBE), Quality Factor (Q) and Radiation Weighting Factor (W_R). ICRP Publication 92. Pergamon Press, Oxford, 2004.
- I12 Iimoto, T., Y. Shirakata, S. Tokonami et al. Continuous ^{220}Rn concentration monitor using an electrostatic collection method. *Radiat. Prot. Dosim.* 77(3): 185-189 (1998).
- I13 Ishikawa, T., Y. Yamada, K. Fukutsu et al. Deposition and clearance for radon progeny in the human respiratory tract. *Radiat. Prot. Dosim.* 105(1-4): 143-148 (2003).
- I14 Ishikawa, T., Y. Narazaki, Y. Yasuoka et al. Biokinetics of radon ingested from drinking water. *Radiat. Prot. Dosim.* 105(1-4): 65-70 (2003).
- I15 Ishikawa, T. Effects of thoron on a radon detector of pulse-ionization chamber type. *Radiat. Prot. Dosim.* 108(4): 327-330 (2004).
- J1 Jacobi, W. The dose to the human respiratory tract by inhalation of short-lived ^{222}Rn - and ^{220}Rn -decay products. *Health Phys.* 10(12): 1163-1175 (1964).
- J2 Jacobi, W. and K. Eisfeld. Dose to tissues and effective dose equivalent by inhalation of ^{222}Rn , ^{220}Rn and their short-lived daughters. GSF-Report-S626 (1980).
- J3 Jacobi, W. Radiation exposure and attributable cancer risk in former miners of the Wismut uranium mining company. *Kerntechnik* 64(1-2): 39-43 (1999).
- J4 Jacobi, W. and P. Roth. Risiko und Verursachungswahrscheinlichkeit von extra-pulmonalen Krebserkrankungen durch die berufliche Strahlenexposition von Beschäftigten der ehemaligen Wismut AG. GSF-Bericht 4/95 (1995).
- J5 James, A.C. Dosimetry of inhaled radon and thoron progeny. p. 161-180 in: *Internal Radiation Dosimetry* (O.G. Raabe, ed.). Medical Physics Publishing, Madison Wisconsin, 1993.
- J6 James, A.C., J.R. Greenhalgh and A. Birchall. A dosimetric model for tissues of the human respiratory tract at risk from inhaled radon and thoron daughters. p. 1045-1048 in: *Radiation Protection: A Systematic Approach to Safety*. Vol. 2. Proceedings of the Fifth Congress of the International Radiation Protection Association Society, Jerusalem. Pergamon Press, Oxford, 1980.
- J7 Jammet, H. and J. Pradel. Le Problème du Radon dans les Mines d'Uranium. in: *Conférence Internationale sur l'Utilisation de l'Énergie Atomique à des Fins Pacifiques* France, June 1955.
- J8 Jamil, K. and S. Ali. Estimation of radon concentrations in coal mines using a hybrid technique calibration curve. *J. Environ. Radioact.* 54(3): 415-422 (2001).
- J9 Johnson, J.R. A review of the dosimetry from inhalation of long lived alpha activity in ore dust. p. 495-502 in: *Proceedings of an International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.
- J10 Joint Committee on Atomic Energy (JCAE), Congress of the United States. Radiation Exposure of Uranium Miners. Hearings before the Subcommittee on Research, Development and Radiation, Part 2, 1967.
- J11 Jorgensen, H. and A. Svensson. Studies on pulmonary function and respiratory tract symptoms of workers in an iron ore mine where diesel trucks are used underground. *J. Occup. Med.* 12(9): 348-354 (1970).
- J12 Jorgensen, H.S. A study of mortality from lung cancer among miners in Kiruna 1950-1970. *Work Environ. Health* 10: 126-133 (1973).
- J13 Jorgensen, H.S. Lung cancer among underground workers in the iron ore mine of Kiruna based on thirty years of observation. *Ann. Acad. Med. Singapore* 13 (2 Suppl.): 371-377 (1984).

- J14 Jostes, R.F. Genetic, cytogenetic, and carcinogenic effects of radon: a review. *Mutat. Res.* 340(2-3): 125-139 (1996).
- J15 James, A.C., A. Birchall and G. Akabani. Comparative dosimetry of BEIR VI revisited. *Radiat. Prot. Dosim.* 108(1): 3-26 (2004).
- J16 James, A.C., W. Jacobi and F. Steinhäusler. Respiratory tract dosimetry of radon and thoron daughters: the state-of-the-art and implications for epidemiology and radiobiology. p. 42-54 in: *Radiation Hazards in Mining: Control, Measurement and Medical Aspects* (M. Gomez, ed.). Society of Mining Engineers, New York, 1982.
- K1 Krewski, D., J. Lubin, J.M. Zielinski et al. Risk of lung cancer in North America associated with residential radon. *Epidemiology* 16(2): 137-145 (2005).
- K2 Knight, G. Airborne contaminants and ventilation in mines. Report MRP/MRL 76-5 (OP). Canada Centre for Mineral and Energy Technology, Department of Energy, Mines and Resources, Canada (1975).
- K3 Knight, G. and R.A. Washington. Dust and radon production in mines: First survey in a mine with a high quartz content in the ore. Internal Report 74/41. Mines Branch, Department of Energy, Mines and Resources, Canada (1974).
- K4 Knight, G. and T.S. Cochrane. Gravimetric dust sampling with quartz analysis and its use in metal and mineral mines. Divisional Report 74/131. Mines Branch, Department of Energy, Mines and Resources, Canada (1974).
- K5 Kreuzer, M., B. Grosche, A. Brachner et al. The German uranium miners cohort study: feasibility and first results. *Radiat. Res.* 152(6): S56-S58 (1999).
- K6 Kupsch, W.O. From Erzgebirge to Cluff Lake — A scientific journey through time. p. 66-74 in: *Musk Ox, Vol. 23*. Institute of Northern Studies, University of Saskatchewan, Saskatoon, Canada, 1978.
- K7 Kunz, E. and J. Sevc. Radiation risks to underground miners in light of Czechoslovakian epidemiological studies. Presented at the International Workshop on Radiological Protection in Mining, Darwin, Australia, April 1988.
- K8 Kunz, E., J. Sevc and V. Placek. Lung cancer mortality in uranium miners (methodological aspects). *Health Phys.* 35(4): 579-580 (1978).
- K9 Kunz, E., J. Sevc, V. Placek et al. Lung cancer in man in relation to different time distribution of radiation exposure. *Health Phys.* 36(6): 699-706 (1979).
- K10 Kushneva, V.S. On the problem of the long-term effects of combined injury to animals of silicon dioxide and radon. p. 21. TR-4473. USAEC, Division of Technical Information, Washington (1959).
- K11 Kusiak, R.A., J. Springer, A.C. Ritchie et al. Lung cancer mortality in Ontario gold miners. *Chronic Diseases (Canada)* 13 (6 Suppl.): S23-S26 (1992).
- K12 Kusiak, R.A., J. Springer, A.C. Ritchie et al. Carcinoma of the lung in Ontario gold miners: possible aetiological factors. *Br. J. Ind. Med.* 48(12): 808-817 (1991).
- K13 Kusiak, R.A., A.C. Ritchie, J. Muller et al. Mortality from lung cancer in Ontario uranium miners. *Br. J. Ind. Med.* 50(10): 920-928 (1993).
- K14 Kreuzer, M., K.M. Müller, A. Brachner et al. Histopathologic findings of lung carcinoma in German uranium miners. *Cancer* 89(12): 2613-2621 (2000).
- K15 Kreuzer, M., B. Grosche, A. Brachner et al. The German uranium miners cohort study — First results. Presented at the IRPA-10, Scientific topics — 2. Health Effects of Ionizing Radiation, Hiroshima, Japan, 14-19 May 2000.
- K16 Kreuzer, M., A. Brachner, F. Lehmann et al. Characteristics of the German uranium miners cohort study. *Health Phys.* 83(1): 26-34 (2002).
- K17 Kreuzer, M., J. Heinrich, G. Wolke et al. Residential radon and risk of lung cancer in eastern Germany. *Epidemiology* 14(5): 559-568 (2003).
- K18 Kreienbrock, L., M. Kreuzer, M. Gerken et al. Case-control study on lung cancer and residential radon in western Germany. *Am. J. Epidemiol.* 153(1): 42-52 (2001).
- K19 Kendall, G.M. and T.J. Smith. Doses from radon and its decay products to children. *J. Radiol. Prot.* 25(3): 241-256 (2005).
- K20 Kobayashi, T. and Y. Takaku. Intermittent measurements of ^{222}Rn and ^{220}Rn progeny in air for four years. *Radioisotopes* 46(9): 603-614 (1997).
- K21 Kendall, G.M. and T.J. Smith. Doses to organs and tissues from radon and its decay products. *J. Radiol. Prot.* 22(4): 389-406 (2002).
- K22 Krewski, D., J.M. Zielinski, W.D. Hazelton et al. The use of biologically based cancer risk models in radiation epidemiology. *Radiat. Prot. Dosim.* 104(4): 367-376 (2003).
- K23 Kusnetz, H.L. Radon daughters in mine atmospheres; a field method for determining concentrations. *Am. Ind. Hyg. Assoc. Q.* 17(1): 85-88 (1956).
- K24 Kane, E.V., E. Roman, R. Cartwright et al. Tobacco and the risk of acute leukaemia in adults. *Br. J. Cancer* 81(7): 1228-1233 (1999).
- K25 Kaiser, J.C., W.F. Heidenreich, G. Monchaux et al. Lung tumour risk in radon-exposed rats from different experiments: comparative analysis with biologically based models. *Radiat. Environ. Biophys.* 43(3): 189-201 (2004).
- K26 Krewski, D., J.H. Lubin, J.M. Zielinski et al. A combined analysis of North American case-control studies of residential radon and lung cancer. *J. Toxicol. Environ. Health Part A* 69(7-8): 533-598 (2006).
- K27 Kobayashi, Y., S. Tokonami, Y. Narazaki et al. Enhanced indoor radon concentration by using radon-rich well water in a Japanese wooden house in Fukuoka, Japan. *J. Radioanal. Nucl. Chem.* 266(3): 389-396 (2005).
- K28 Kobayashi, Y., S. Tokonami, H. Takahashi et al. Practicality of the thoron calibration chamber system at NIRS, Japan. p. 281-282 in: *High Levels of Natural Radiation and Radon Areas: Radiation Dose and*

- Health Effects (T. Sugahara et al., eds.). International Congress Series 1276. Elsevier, 2005.
- K29 Kreuzer, M., M. Schnelzer, A. Tschense et al. Risk of lung cancer and other cancers in the German uranium miners cohort study. Presented at the 11th International Congress of the International Radiation Protection Association, Madrid, Spain, 23-28 May 2004.
- K30 Kreuzer, M., M. Kreisheimer, M. Kandel et al. Mortality from cardiovascular diseases in the German uranium miners cohort study, 1946-1998. *Radiat. Environ. Biophys.* 45(3): 159-166 (2006).
- K31 Kreuzer, M., M. Kreisheimer, M. Kandel et al. Radiation and risk of circulatory diseases in the German uranium miners cohort study. Presented at the Second European IRPA Congress on Radiation Protection, Paris, France, 15-19 May 2006.
- L1 Lagarde, F., G. Pershagen, G. Åkerblom et al. Residential radon and lung cancer in Sweden: risk analysis accounting for random error in the exposure assessment. *Health Phys.* 72(2): 269-276 (1997).
- L2 Larsson, L.G. and L. Damber. Interaction between underground mining and smoking in the causation of lung cancer: a study of non-uranium miners in northern Sweden. *Cancer Detect. Prev.* 5(4): 385-389 (1982).
- L3 Lehmann, F. Belastung durch ionisierende Strahlung im Uranerzbergbau der ehemaligen DDR. Abschlussbericht zu einem Forschungs-vorhaben. Hauptverband der gewerblichen Berufsgenossenschaft (HVBG) Bergbau-Berufsgenossenschaft — BBG (1998).
- L4 Lubin, J.H. and J.D. Boice Jr. Lung cancer risk from residential radon: meta-analysis of eight epidemiologic studies. *J. Natl. Cancer Inst.* 89(1): 49-57 (1997).
- L5 Little, J.B., H. Nagasawa and E. Azzam. Effect of low-dose alpha irradiation in human cells: the role of induced genes and the bystander effect. In: DOE Low Dose Radiation Research Program Workshop I: Abstracts, Washington, D.C., 10-12 November 1999. Toxicology and Risk Analysis Section Life Sciences Division, Oak Ridge National Laboratory, 1999.
- L6 Lorenz, E. Radioactivity and lung cancer: a critical review of lung cancer in the miners of Schneeberg and Joachimsthal. *J. Natl. Cancer Inst.* 5: 1-15 (1944).
- L7 Lowe, L.M. and D.B. Chambers. Comment on ICRP recommendations on radon and revised background doses from radon. *Environ. Int.* 22 (Suppl. 1): 1037-1044 (1996).
- L8 Lubin, J.H., J.D. Boice Jr. and J.M. Samet. Errors in exposure assessment, statistical power and the interpretation of residential radon studies. *Radiat. Res.* 144(3): 329-341 (1995).
- L9 Luebeck, E.G., S.B. Curtis, F.T. Cross et al. Two-stage model of radon-induced malignant lung tumors in rats: effects of cell killing. *Radiat. Res.* 145(2): 163-173 (1996).
- L10 Lubin, J.H., J.D. Boice, C. Edling et al. Radon and Lung Cancer Risk: A Joint Analysis of 11 Underground Miner Studies. NIH Publication No. 94-3644 (1994).
- L11 Lubin, J.H., Y.L. Qiao, P.R. Taylor et al. Quantitative evaluation of the radon and lung cancer association in a case control study of Chinese tin miners. *Cancer Res.* 50(1): 174-180 (1990).
- L12 Lubin, J.H., M.S. Linet, J.D. Boice Jr. et al. Case-control study of childhood acute lymphoblastic leukemia and residential radon exposure. *J. Natl. Cancer Inst.* 90(4): 294-300 (1998).
- L13 Lundin, F.E. Jr., J.K. Wagoner and V.E. Archer. Radon Daughter Exposure and Respiratory Cancer: Quantitative and Temporal Aspects. National Institute for Occupational Safety and Health and National Institute of Environmental Health Sciences, Joint Monograph No. 1. Washington, D.C., 1971.
- L14 Lundin, F.E. Jr., J.W. Lloyd, E.M. Smith et al. Mortality of uranium miners in relation to radiation exposure, hard-rock mining and cigarette smoking — 1950 through September 1967. *Health Phys.* 16(5): 571-578 (1969).
- L15 Lundin, E. Jr. Correspondence concerning calculation of OHR from Dr. Lundin to L.W. Swent, 27 March 1969. Department of Health Education and Welfare (DHEW) (1969).
- L16 Luebeck, E.G., W.F. Heidenreich, W.D. Hazelton et al. Biologically based analysis of the data for the Colorado uranium miners cohort: age, dose and dose-rate effects. *Radiat. Res.* 152(4): 339-351 (1999).
- L17 L'Abbé, K.A., G.R. Howe, J.D. Burch et al. Radon exposure, cigarette smoking, and other mining experience in the Beaverlodge uranium miners cohort. *Health Phys.* 60(4): 489-495 (1991).
- L18 Laurier, D., M. Valenty and M. Tirmarche. Radon exposure and the risk of leukemia: a review of epidemiological studies. *Health Phys.* 81(3): 272-288 (2001).
- L19 Lubin, J.H., L. Tomásek, C. Edling et al. Estimating lung cancer mortality from residential radon using data for low exposures of miners. *Radiat. Res.* 147(2): 126-134 (1997).
- L20 Law, G.R., E.V. Kane, E. Roman et al. Residential radon exposure and adult acute leukaemia. *Lancet* 355(9218): 1888 (2000).
- L21 Little, M.P. Comparisons of lung tumour mortality risk in the Japanese A-bomb survivors and in the Colorado Plateau uranium miners: support for the ICRP lung model. *Int. J. Radiat. Biol.* 78(3): 145-163 (2002).
- L22 Lubin, J.H., J.D. Boice Jr., C. Edling et al. Radon-exposed underground miners and inverse dose-rate (protraction enhancement) effects. *Health Phys.* 69(4): 494-500 (1995).
- L23 Leenhouts, H.P. Radon-induced lung cancer in smokers and non-smokers: risk implications using a two-mutation carcinogenesis model. *Radiat. Environ. Biophys.* 38(1): 57-71 (1999).

- L24 Lagarde, F. and G. Pershagen. Parallel analyses of individual and ecologic data on residential radon, cofactors, and lung cancer in Sweden. *Am. J. Epidemiol.* 149(3): 268-274 (1999).
- L25 Letourneau, E.G., D. Krewski, N.W. Choi et al. Case-control study of residential radon and lung cancer in Winnipeg, Manitoba, Canada. *Am. J. Epidemiol.* 140(4): 310-322 (1994).
- L26 Lubin, J.H., Z.Y. Wang, J.D. Boice Jr. et al. Risk of lung cancer and residential radon in China: pooled results of two studies. *Int. J. Cancer* 109(1): 132-137 (2004).
- L27 Laurer, G.R., Q.T. Gang, J.H. Lubin et al. Skeletal ²¹⁰Pb levels and lung cancer among radon-exposed tin miners in southern China. *Health Phys.* 64(3): 253-259 (1993).
- L28 Lubin, J.H. On the discrepancy between epidemiologic studies in individuals of lung cancer and residential radon and Cohen's ecologic regression. *Health Phys.* 75(1): 4-10 (1998).
- L29 Lubin, J.H. Response to Cohen's comments on the Lubin rejoinder. *Health Phys.* 77(3): 330-332 (1999).
- L30 Lubin, J.H. Invited commentary: lung cancer and exposure to residential radon. *Am. J. Epidemiol.* 140(4): 323-332 (1994).
- L31 Lagarde, F., R. Falk, K. Almren et al. Glass-based radon-exposure assessment and lung cancer risk. *J. Expo. Anal. Environ. Epidemiol.* 12(5): 344-354 (2002).
- L32 Lagarde, F., G. Axelsson, L. Damber et al. Residential radon and lung cancer among never-smokers in Sweden. *Epidemiology* 12(4): 396-404 (2001).
- L33 Leenhouts, H.P. and M.J. Brugmans. Calculation of the 1995 lung cancer incidence in the Netherlands and Sweden caused by smoking and radon: risk implications for radon. *Radiat. Environ. Biophys.* 40(1): 11-21 (2001).
- L34 Little, M.P., R.G. Haylock and C.R. Muirhead. Modelling lung tumour risk in radon-exposed uranium miners using generalizations of the two-mutation model of Moolgavkar, Venzon and Knudson. *Int. J. Radiat. Biol.* 78(1): 49-68 (2002).
- L35 Little, M.P. and R. Wakeford. The bystander effect in C3H 10T cells and radon-induced lung cancer. *Radiat. Res.* 156(6): 695-699 (2001).
- L36 Lubin, J.H. Studies of radon and lung cancer in North America and China. *Radiat. Prot. Dosim.* 104(4): 315-319 (2003).
- L37 Li, Y., S.D. Schery and B. Turk. Soil as a source of indoor ²²⁰Rn. *Health Phys.* 62(5): 453-457 (1992).
- L38 Laurier, D., A. Rogel, M. Valenty et al. Discussion on radon and leukemia risk in underground miners: are working level months the most appropriate exposure parameter? *Health Phys.* 86(4): 427-428 (2004).
- L39 Langholz, B., D. Thomas, A. Xiang et al. Latency analysis in epidemiologic studies of occupational exposures: application to the Colorado Plateau uranium miners cohort. *Am. J. Ind. Med.* 35(3): 246-256 (1999).
- L40 Laurier, D., A. Rogel, M. Tirmarche et al. Lung cancer risk associated with low chronic exposure to radon in the French cohort of uranium miners. Abstract presented at the Seventh International Symposium on the Natural Radiation Environment (NRE-VII), Rhodes, Greece, 20-24 May 2002.
- L41 Lees, R.E., R. Steele and J.H. Roberts. A case-control study of lung cancer relative to domestic radon exposure. *Int. J. Epidemiol.* 16(1): 7-12 (1987).
- L42 Laurier, D., A. Rogel, L. Tomasek et al. Comment on "Studies of radon-exposed miner cohorts using a biologically based model: comparison of current Czech and French data with historic data from China and Colorado" by W.F. Heidenreich, L. Tomasek, A. Rogel, D. Laurier, M. Tirmarche (2004) *Radiat. Environ. Biophys.* 43:247-256, and "Radon-induced lung cancer in French and Czech miner cohorts described with a two-mutation cancer model" by M.J.P. Brugmans, S.M. Rispens, H. Bijwaard, D. Laurier, A. Rogel, L. Tomasek, M. Tirmarche (2004) *Radiat. Environ. Biophys.* 43:153-163. *Radiat. Environ. Biophys.* 44(2): 155-156 (2005).
- L43 Laurier, D., G. Monchaux, A. Rogel et al. Lung cancer risk associated with low chronic radon exposure: results from epidemiology and animal experiments in France. Paper 1f7 presented in Session 1, Radiation Effects of the 11th International Congress of the International Radiation Protection Association, Madrid, Spain, 23-28 May 2004.
- L44 Laurier, D., M. Tirmarche, N. Mitton et al. An update of cancer mortality among the French cohort of uranium miners: extended follow-up and new source of data for causes of death. *Eur. J. Epidemiol.* 19(2): 139-146 (2004).
- L45 Lomas, P.R. and B.M.R. Green. Temporal variations of radon levels in dwellings. *Radiat. Prot. Dosim.* 56(1): 323-325 (1994).
- L46 Liu, F., Z. Pan, S. Liu et al. The estimation of the number of underground coal miners and the annual dose to coal miners in China. *Health Phys.* 93(2): 127-132 (2007).
- L47 Létourneau, E.G., D. Krewski, J.M. Zielinski et al. Cost effectiveness of radon mitigation in Canada. *Radiat. Prot. Dosim.* 45(1): 593-598 (1992).
- L48 Lowy, J. Über die Joachimstaler Bergkrankheit. *Med. Klin.* 25 (4): 1259, 141-142 (1929).
- M1 Marcinowski, F., R.M. Lucas and W.M. Yeager. National and regional distributions of airborne radon concentrations in U.S. homes. *Health Phys.* 66(6): 699-706 (1994).
- M2 Marsh, J.W. and A. Birchall. Sensitivity analysis of the weighted equivalent lung dose per unit exposure from radon progeny. *NRPB-M929* (1998).
- M3 Muller, J. and W.C. Wheeler. Causes of death in Ontario uranium miners. Presented at the ILO Symposium on Radiation Protection in Mining and Milling of Uranium and Thorium, Bordeaux, France, 1974.

- M4 McCrodan, P. Elliot lake — background, current problems and responses. Part VII of brief presented to Royal Commission on the Health and Safety of Miners by the Ontario Ministry of Natural Resources (4 June 1975).
- M5 McNeill, K.G. Radon progeny exposure re-evaluation techniques. Prepared for the Atomic Energy Control Board (September 1995).
- M6 McNiven, J.G. History of the Eldorado mine, Port Radium. Canadian Institute of Mining and Metallurgy, Montreal (November and December 1967).
- M7 Miller, R.C., G. Randers-Pehrson, C.R. Geard et al. The oncogenic transforming potential of the passage of single alpha particles through mammalian cell nuclei. *Proc. Natl. Acad. Sci. U.S.A.* 96(1): 19-22 (1999).
- M8 Muller, J. Uranium Mining and Milling in Ontario: Epidemiological Studies. Statement of Evidence Presented to the British Columbia Royal Commission of Inquiry into Uranium Mining, Canada, 1980.
- M9 Mitchel, R.E.J., J.S. Jackson and B. Heinmiller. Inhaled uranium ore dust and lung cancer risk in rats. *Health Phys.* 76(2): 145-155 (1999).
- M10 Monchaux, G., J.P. Morlier, S. Altmeyer et al. Influence of exposure rate on lung cancer induction in rats exposed to radon progeny. *Radiat. Res.* 152 (6 Suppl.): S137-S140 (1999).
- M11 Moolgavkar, S.H., F.T. Cross, G. Luebeck et al. A two-mutation model for radon-induced lung tumors in rats. *Radiat. Res.* 121(1): 28-37 (1990).
- M12 Morken, D.A. Acute toxicity of radon. *AMA Arch. Ind. Health* 12(4): 435-438 (1955).
- M13 Morken, D.A. and J.K. Scott. The effects on mice of continual exposure to radon and its decay products on dust. University of Rochester Atomic Energy Project Report UR-669 (1966).
- M14 Morrison, H.I., R.M. Semenciw, Y. Mao et al. Lung cancer mortality and radiation exposure among the Newfoundland fluorspar miners. p. 365-368 in: *Proceedings of an International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.
- M15 Muller, J., W.C. Wheeler, J.F. Gentleman et al. The Ontario miners mortality study: general outline and progress report. p. 359-362 in: *Radiation Hazards in Mining: Control, Measurement and Medical Aspects* (M. Gomez, ed.). Society of Mining Engineers of the American Institute of Mining, Metallurgical and Petroleum Engineers Inc., New York, 1981.
- M16 Morrison, H.I., R. Semenciw, Y. Mao et al. The mortality experience of a group of Newfoundland fluorspar miners exposed to Rn progeny. AECB INFO-0280 (1988).
- M17 Morrison, H.I., R.M. Semenciw, Y. Mao et al. Cancer mortality among a group of fluorspar miners exposed to radon progeny. *Am. J. Epidemiol.* 128(6): 1266-1275 (1988).
- M18 Morrison, H.I., D.T. Wigle, H. Stocker et al. Lung cancer mortality and radiation exposure among the Newfoundland fluorspar miners. p. 372-376 in: *Radiation Hazards in Mining: Control, Measurement and Medical Aspects* (M. Gomez, ed.). Society of Mining Engineers of the American Institute of Mining, Metallurgical and Petroleum Engineers Inc., New York, 1981.
- M19 Muller, J., W.C. Wheeler, J.F. Gentleman et al. Study of Mortality of Ontario Miners, 1955-1977. Part 1. Ontario Ministry of Labour, Toronto, 1983.
- M20 Morrison, H.I. and P.J. Villeneuve. Radon progeny exposure and lung cancer risk: Analyses of a cohort of Newfoundland fluorspar miners. AECB Project No. 7.142.1. Research and Support Program, Ottawa, Canada (1995).
- M21 Muller, J. and R.A. Kusiak. Modifying Factors in Lung Cancer Risk of Ontario Uranium Miners 1955-1981. Ontario Ministry of Labour, Toronto, 1989.
- M22 Muller, J. and R.A. Kusiak. Lung Cancer Risk in Uranium Miners. Paper Presented to the Annual Meeting of the NCRP, Washington, 1988.
- M23 Muller, J., R.A. Kusiak, G. Suranyi et al. Modifying Factors in Lung Cancer Risk of Ontario Uranium Miners 1955-1981. Ontario Ministry of Labour, Toronto, 1987.
- M24 Muller, J., R.A. Kusiak and G. Suranyi. Addendum to Study of Mortality of Ontario Gold Miners 1955-1977. Ontario Ministry of Labour, Toronto, 1987.
- M25 Muller, J., R.A. Kusiak et al. Study of Mortality of Ontario Gold Miners 1955-1977. Ontario Ministry of Labour, Toronto, 1986.
- M26 Muller, J., W.C. Wheeler, J.F. Gentleman et al. Study of mortality of Ontario miners. p. 335-343 in: *Proceedings of an International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.
- M27 Morrison, H.I., P.J. Villeneuve, J.H. Lubin et al. Radon-progeny exposure and lung cancer risk in a cohort of Newfoundland fluorspar miners. *Radiat. Res.* 150(1): 58-65 (1998).
- M28 Monchaux, G. and J.P. Morlier. Influence of exposure rate on radon-induced lung cancer in rats. *J. Radiol. Prot.* 22(3A): A81-A87 (2002).
- M29 Monchaux, G. Contribution of animal experimental data for the risk assessment of exposure to radon decay products. p. 66-76 in: *Radioactivity in the Environment, Vol. 7: The Natural Radiation Environment VII. Seventh International Symposium on the Natural Radiation Environment (NRE-VII)* (J.P. McLaughlin, S.E. Simopoulos and F. Steinhäusler, eds.). Elsevier Ltd., London, 2005.
- M30 Morlier, J.P., M. Morin, G. Monchaux et al. Lung cancer incidence after exposure of rats to low doses of radon: influence of dose rate. *Radiat. Prot. Dosim.* 56(1): 93-97 (1994).
- M31 Monchaux, G., J.P. Morlier, M. Morin et al. Carcinogenic and cocarcinogenic effects of radon and radon daughters in rats. *Environ. Health Perspect.* 102(1): 64-73 (1994).

- M32 Marsh, J.W., A. Birchall, G. Butterweck et al. Uncertainty analysis of the weighted equivalent lung dose per unit exposure to radon progeny in the home. *Radiat. Prot. Dosim.* 102(3): 229-248 (2002).
- M33 McLaughlin, J.P. Approaches to the assessment of long term exposure to radon and its progeny. *Sci. Total Environ.* 272(1-3): 53-60 (2001).
- M34 Morgan, M.V. and J.M. Samet. Radon daughter exposures of New Mexico U miners, 1967-1982. *Health Phys.* 50(5): 656-662 (1986).
- M35 Makepeace, C.E. and H. Stocker. Statistical interpretation of a programme of monitoring frequency designed for the protection of underground uranium miners from overexposure to radon daughters. p. 123-164 in: *Occupational Radiation Exposure in Nuclear Fuel Cycle Facilities*. STI/PUB/527. IAEA, Vienna (1980).
- M36 Mothersill, C. and C. Seymour. Radiation-induced bystander effects: past history and future directions. *Radiat. Res.* 155(6): 759-767 (2001).
- M37 Morgan W.F. Non-targeted and delayed effects of exposure to ionizing radiation: I. radiation-induced genomic instability and bystander effects in vitro. *Radiat. Res.* 159(5): 567-580 (2003).
- M38 Morgan W.F. Non-targeted and delayed effects of exposure to ionizing radiation: II. radiation-induced genomic instability and bystander effects in vivo, clastogenic factors and transgenerational effects. *Radiat. Res.* 159(5): 581-596 (2003).
- M39 Martinea, T., L. Cabrera, J.L. Oliveres et al. Radon and thoron levels in Mexico city dwellings. In: *Proceedings of the Fifth Regional Congress on Radiation Protection and Safety*, Recife, Brazil, 2001.
- M40 Ma, J., H. Yonehara, T. Aoyama et al. Influence of air flow on the behavior of thoron and its progeny in a traditional Japanese house. *Health Phys.* 72(1): 86-91 (1997).
- M41 Magalhaes, M.H., E.C. Amaral, I. Sachett et al. Radon-222 in Brazil: an outline of indoor and outdoor measurements. *J. Environ. Radioact.* 67(2): 131-143 (2003).
- M42 Marsh, J.W. and A. Birchall. Letter to the Editor — The thoron issue: monitoring activities, measuring techniques and dose conversion factors. *Radiat. Prot. Dosim.* 81(4): 311-312 (1999).
- M43 Mitchell, S.A., G. Randers-Pehrson, D.J. Brenner et al. The bystander response in C3H 10T1/2 cells: the influence of cell-to-cell contact. *Radiat. Res.* 161(4): 397-401 (2004).
- M44 Möhner, M., M. Lindtner, H. Otten et al. Leukemia and exposure to ionizing radiation among German uranium miners. *Am. J. Ind. Med.* 49(4): 238-248 (2006).
- N1 Narayanan, P.K., E.H. Goodwin and B.E. Lehnert. Alpha particles initiate biological production of superoxide anions and hydrogen peroxide in human cells. *Cancer Res.* 57(18): 3963-3971 (1997).
- N2 Narayanan, P.K., K.E. LaRue, E.H. Goodwin et al. Alpha particles induce the production of interleukin-8 by human cells. *Radiat. Res.* 152(1): 57-63 (1999).
- N3 National Research Council. Report of the oversight Committee. p. 15-36 in: *Biologic Markers in Reproductive Toxicology*. National Academies Press, Washington, D.C., 1989.
- N4 National Academy of Sciences (NAS). Radiation exposure of uranium miners. A report of an Advisory Committee from the Division of Medical Sciences. National Research Council, National Academy of Engineering, Washington, D.C. (1968).
- N5 National Cancer Institute. Uncertainties in Radiation Dosimetry and their Impact on Dose-Response Analyses (E. Ron and F.O. Hoffman, eds.). National Institute of Health, 1999.
- N6 Nair, R.C., J.D. Abbatt, G.R. Howe et al. Mortality experience among workers in the uranium industry. p. 354-364 in: *Proceedings of the International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.
- N7 National Council on Radiation Protection and Measurements. Evaluation of occupational and environmental exposures to radon and radon daughters in the United States. NCRP Report No. 78 (1984).
- N8 Nuclear Energy Agency. Dosimetry aspects of exposure to radon and thoron daughter products. NEA Experts Report. ISBN 92-64-12520-5 (1983).
- N9 National Research Council. Risk Assessment of Radon in Drinking Water. National Academy Press, Washington, D.C., 1999.
- N10 National Research Council. Comparative Dosimetry of Radon in Mines and Homes. National Academy Press, Washington, D.C., 1991.
- N11 National Council on Radiation Protection and Measurements. Evaluation of occupational and environmental radon risk. NCRP SC 65 (2004).
- N12 Nikiforov, A.I. and R.B. Schlesinger. Morpho-metric variability of the human upper bronchial tree. *Respir. Physiol.* 59(3): 289-299 (1985).
- N13 Nikezic, D., K.N. Yu and D. Vucic. Absorbed fraction and dose conversion coefficients of alpha particles for radon dosimetry. *Phys. Med. Biol.* 46(7): 1963-1974 (2001).
- N14 Nikezic, D. and K.N. Yu. Microdosimetric calculation of absorption fraction and the resulting dose conversion factor for radon progeny. *Radiat. Environ. Biophys.* 40(3): 207-211 (2001).
- N15 National Council on Radiation Protection and Measurements. Measurement of radon and radon daughters in air. NCRP Report No. 97 (1988).
- N16 Nuccetelli, C. and F. Boichichio. The thoron issue: monitoring activities, measuring techniques and dose conversion factors. *Radiat. Prot. Dosim.* 78(1): 59-64 (1998).
- N17 Németh, C., S. Tokonami, T. Ishikawa et al. Measurements of radon, thoron and their progeny in Gifu prefecture, Japan. *J. Radioanal. Nucl. Chem.* 267(1): 9-12 (2005).
- O1 O'Heany, J.M., R. Kusiak, R. Willett et al. Arsenic exposure and absorption in underground miners in an

- Ontario gold mine. *Occup. Health Ontario* 9(3): 158-169 (1988).
- O2 Oberaigner, W., L. Kreienbrock, A. Schaffrath Rosario et al. Radon und Lungenkrebs im Bezirk Imst/Österreich (H.E. Wichmann, H.W. Schlipkötter and G. Fülgraff, eds.). *Fortschritte in der Umweltmedizin*. Ecomed Verlag, Landsberg am Lech, 2002.
- O3 Oestreicher, U., H. Braselmann and G. Stephan. Cytogenetic analyses in peripheral lymphocytes of persons living in houses with increased levels of indoor radon concentrations. *Cytogenet. Genome Res.* 104(1-4): 232-236 (2004).
- P1 Parsons, W.D., A.J. de Villiers, L.S. Bartlett et al. Lung cancer in a fluorspar mining community. II. Prevalence of respiratory symptoms and disability. *Br. J. Ind. Med.* 21: 110-116 (1964).
- P2 Pekarek, V., M. Martinec and J. Urbanec. The incidence of lung cancer among miners in the ore mines of northern Bohemia. Battelle Northwest Laboratory, BNWL-TR-84 (1984). (In Czech).
- P3 Popp, W., U. Plappert, W.U. Müller et al. Biomarkers of genetic damage and inflammation in blood and bronchoalveolar lavage fluid among former German uranium miners: a pilot study. *Radiat. Environ. Biophys.* 39(4): 275-282 (2000).
- P4 Perraud, R., J. Chameaud, R. Masse et al. Experimental pulmonary cancer in rats after inhalation of radon associated with nonradioactive dust. *C.R. Acad. Sci. Hebd. Seances Acad. Sci., Ser. D* 270(21): 2594-2595 (1970). (In French).
- P5 Pirchan, A. and H. Sikl. Cancer of the lung in the miners of Jachymov (Joachimsthal). *Am. J. Cancer* 4: 681-722 (1932).
- P6 Pisa, F.E., F. Barbone, A. Betta et al. Residential radon and risk of lung cancer in an Italian alpine area. *Arch. Environ. Health* 56(3): 208-215 (2001).
- P7 Piao, C.Q. and T.K. Hei. The biological effectiveness of radon daughter alpha particles I. Radon, cigarette smoke and oncogenic transformation. *Carcinogenesis* 14(3): 497-501 (1993).
- P8 Pradel, J. and P. Zettwoog. Hier et maintenant: la radioprotection dans les mines d'uranium. *Rev. Gen. Nucl.* 1: 38-57 (1984).
- P9 Pradel, J. and P. Zettwoog. La radioprotection dans l'extraction et le traitement des minerais d'uranium en France. *Occupational Safety and Health Series No. 32*. ILO Publication, 1974.
- P10 Puskin, J.S. Smoking as a confounder in ecologic correlations of cancer mortality rates with average county radon levels. *Health Phys.* 84(4): 526-532 (2003).
- P11 Pershagen, G., G. Akerblom, O. Axelson et al. Residential radon exposure and lung cancer in Sweden. *N. Engl. J. Med.* 330(3): 159-164 (1994).
- P12 Porstendorfer, J. Physical parameters and dose factors of the radon and thoron decay products. *Radiat. Prot. Dosim.* 94(4): 365-373 (2001).
- P13 Pierce, D.A., Y. Shimizu, D.L. Preston et al. Studies of the mortality of atomic bomb survivors. Report 12, Part I. Cancer: 1950-1990. *Radiat. Res.* 146(1): 1-27 (1996).
- P14 Puskin, J.S. An analysis of the uncertainties in estimates of radon-induced lung cancer. *Risk Anal.* 12(2): 277-285 (1992).
- P15 Paridaens, J., H. Vanmarcke, K. Jacobs et al. Retrospective radon assessment by means of ^{210}Po activity measurements. *Appl. Radiat. Isot.* 53(1-2): 361-364 (2000).
- P16 Pershagen, G., Z.H. Liang, Z. Hrubec et al. Residential radon exposure and lung cancer in Swedish women. *Health Phys.* 63(2): 179-186 (1992).
- P17 Pavia, M., A. Bianco, C. Pileggi et al. Meta-analysis of residential exposure to radon gas and lung cancer. *Bull. World Health Organ.* 81(10): 732-738 (2003).
- P18 Petitot, F., J.P. Morlier, M. Debroke et al. A new method specifically designed to expose cells isolated in vitro to radon and its decay products. *Radiat. Res.* 157(6): 693-699 (2002).
- P19 Pearson, M.D. and R.R. Spangler. Calibration of α -track monitors for measurement of thoron (^{220}Rn). *Health Phys.* 60(5): 697-701 (1991).
- P20 Peto, R., S. Darby, H. Deo et al. Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. *Br. Med. J.* 321(7257): 323-329 (2000).
- Q1 Qiao, Y.L., P.R. Taylor, S.X. Yao et al. Risk factors and early detection of lung cancer in a cohort of Chinese tin miners. *Ann. Epidemiol.* 7(8): 533-541 (1997).
- R1 Radford, E.P. Investigation of the status of research on lung cancer in underground miners in Europe, 1976. Report to the National Institute of Occupational Health, Order No. 96-3825 (1976).
- R2 Radford, E.P. and K.G. Renard. Lung cancer in Swedish iron miners exposed to low doses of radon daughters. *N. Engl. J. Med.* 310(23): 1485-1494 (1984).
- R3 Ruostenoja, E., I. Mäkeläinen, T. Rytömaa et al. Radon and lung cancer in Finland. *Health Phys.* 71(2): 185-189 (1996).
- R4 Richmond, C.R. and B.B. Boecker. Experimental Studies. Final Report of Subgroup IB, Interagency Uranium Mining Radiation Review Group (IUMRRG71). EPA, Rockville, MD., 1971.
- R5 Robbins, E.S. and N.H. Harley. Dose to the fetus from ^{222}Rn in maternal drinking water. 749-755 in: *Radioactivity in the Environment, Vol. 7: The Natural Radiation Environment VII. Seventh International Symposium on the Natural Radiation Environment (NRE-VII)* (J.P. McLaughlin, S.E. Simopoulos and F. Steinhäusler, eds.). Elsevier Ltd., London, 2005.
- R6 Ramachandran, T.V. and M.C. Subba Ramu. Variation of equilibrium factor F between radon and its short-lived decay products in an indoor atmosphere. *Nucl. Geophys.* 8(5): 499-503 (1994).
- R7 Reineking, A. and J. Porstendorfer. High-volume screen diffusion batteries and α -spectroscopy for measurement of the radon daughter activity size distributions in the environment. *J. Aerosol. Sci.* 17(5): 873-879 (1986).

- R8 Roscoe, R.J. An update of mortality from all causes among white uranium miners from the Colorado Plateau Study Group. *Am. J. Ind. Med.* 31(2): 211-222 (1997).
- R9 Roscoe, R.J., J.A. Deddens, A. Salvan et al. Mortality among Navajo uranium miners. *Am. J. Public Health* 85(4): 535-540 (1995).
- R10 Rogel, A., D. Laurier, M. Tirmarche et al. Lung cancer risk in the French cohort of uranium miners. *J. Radiol. Prot.* 22(3A): A101-A106 (2002).
- R11 Rutherford, P.M., M.J. Dudas and R.A. Samek. Environmental impacts of phosphogypsum. *Sci. Total Environ.* 149(1-2): 1-38 (1994).
- R12 Ren, T.S. Source, level and control of indoor radon. *Radiat. Prot.* 21(5): 291-299 (2001). (In Chinese).
- R13 Rericha, V., M. Kulich, R. Rericha et al. Incidence of leukemia, lymphoma and multiple myeloma in Czech uranium miners: a case-cohort study. *Environ. Health Perspect.* 114(6): 818-822 (2006).
- S1 Samet, J.M., D.R. Pathak, M.V. Morgan et al. Radon progeny exposure and lung cancer risk in New Mexico U miners: a case-control study. *Health Phys.* 56(4): 415-421 (1989).
- S2 Samet, J.M. Residential radon and lung cancer: end of the story? *J. Toxicol. Environ. Health Part A* 69(7-8): 527-531 (2006).
- S3 Stidley, C.A. and J.M. Samet. Assessment of ecologic regression in the study of lung cancer and indoor radon. *Am. J. Epidemiol.* 139(3): 312-322 (1994).
- S4 Samet, J.M. Lung cancer epidemiology in New Mexico uranium miners. Third annual report for the period 1 April 1985–15 December 1985. University of New Mexico (1985).
- S5 Samet, J.M., M.V. Morgan, R.W. Buechley et al. Study of Grants, New Mexico. p. 680-684 in: *Uranium Miners: Current Status* (M. Gomez, ed.). Society of Mining Engineers, American Institute of Mining, Metallurgical and Petroleum Engineers Inc., New York, 1981.
- S6 Schiager, K.J. and C.W. Hersloff. Appendix F — Review of radon daughter exposure measurements in U.S. uranium miners — past and present. SENES Consultants Limited, Ontario (1984).
- S7 Schneider, J., P. Presek, A. Braun et al. Serum levels of pantropic p53 protein and EGF-receptor, and detection of anti-p53 antibodies in former uranium miners (SDAG Wismut). *Am. J. Ind. Med.* 36(6): 602-609 (1999).
- S8 Schwartz, J.L., J.D. Shadley, R.W. Atcher et al. Comparison of radon-daughter-induced effects in repair-proficient and repair-deficient CHO cell lines. *Environ. Mol. Mutagen.* 16(3): 178-184 (1990).
- S9 SENES Consultants. Exploratory analysis of white male U.S. underground uranium miners with mortality follow-up to 31 December 1985. SENES Consultants Limited, Ontario (1992).
- S10 SENES Consultants. Uncertainty in exposure of underground miners to radon daughters and the effect of uncertainty on risk estimates. Report to the Atomic Energy Control Board of Canada. SENES Consultants Limited, Ontario (1989).
- S11 SENES Consultants. Report on electrostatic purification of uranium mine stope atmospheres. Report to the Atomic Energy Control Board of Canada. SENES Consultants Limited, Ontario (1986).
- S12 SENES Consultants. Detailed reconstruction of radon daughter exposures of Eldorado Beaverlodge uranium mine employees. SENES Consultants Limited, Ontario (1991).
- S13 SENES Consultants. Preliminary feasibility study into the re-evaluation of exposure data for the Colorado Plateau uranium miner cohort study. SENES Consultants Limited, Ontario (1995).
- S14 SENES Consultants. An algorithm for estimating radon decay product exposures from underground employment at the Eldorado Beaverlodge mine. SENES Consultants Limited, Ontario (1996).
- S15 SENES Consultants. A re-evaluation of radon decay product exposures to underground workers at the Port Radium mine. SENES Consultants Limited, Ontario (1996).
- S16 Snihs, J.O. Letter to Dr. Victor Archer, NIOSH, concerning Swedish data on radon daughter levels in Swedish mines. Communication to the UNSCEAR Secretariat (1972).
- S17 Sevc, J., V. Placek and J. Jerabek. Lung cancer risk in relation to long-term radiation exposure in uranium mines. p. 315-326 in: *Proceedings of the Fourth Conference on Radiation Hygiene, Jasna pod Chopkom, Part II*. Purkyne Medical Research and Postgraduate Institute, Hradec Kralove, Czechoslovakia, 1971.
- S18 Sevc, J. and V. Placek. Lung cancer risk in relation to long-term exposure to radon daughters. p. 129-136 in: *Proceedings of the Second European Congress on Radiation Protection of the International Radiation Protection Association, Budapest, 1972*.
- S19 Sevc, J. and V. Placek. Radiation induced lung cancer: relation between lung cancer and long-term exposure to radon daughters. p. 305-310 in: *Proceedings of the Sixth Conference on Radiation Hygiene, Jasna Pod Chopkom, CSSR, 1973*.
- S20 Sevc, J., E. Kunz and V. Placek. Lung cancer in uranium miners and long-term exposure to radon daughter products. *Health Phys.* 30(6): 433-437 (1976).
- S21 St. Clair Renard, K.G. Lung cancer among mine workers in Sweden. *Swedish Mine Owners Associations No. 140/1970* (1972). (Translated from Swedish).
- S22 St. Clair Renard, K.G. Respiratory cancer mortality in an iron ore mine in northern Sweden. *Ambio* 3(2): 67-69 (1974).
- S23 Strandén, E. and L. Berteig. Radon daughter equilibrium and unattached fraction in mine atmospheres. *Health Phys.* 42(4): 479-487 (1982).
- S24 Sevc, J., E. Kunz, V. Placek et al. Comments on lung cancer risk estimates. *Health Phys.* 46(4): 961-965 (1984).

- S25 Sevc, J., E. Kunz, L. Tomasek et al. Cancer in man after exposure to Rn daughters. *Health Phys.* 54(1): 27-46 (1988).
- S26 Sun, Y., B.L. Mao, G.Q. Shi et al. Epidemiological and clinical studies of lung cancer in a tin mine in China. *Proc. Am. Assoc. Cancer Res.* 22: 496 (1981).
- S27 Sun, S., X. Yang, L. Yang et al. Latent period and temporal aspects of lung cancer among miners. *Radiat. Prot.* 4(5): 330-339 (1984).
- S28 Swent, L.W. and D.B. Chambers. Comments submitted to the U.S. Mine Safety and Health Administration for the American Mining Congress by Langan W. Swent and Douglas B. Chambers on their visit to the Malmberget mines of LKAB, in northern Sweden, and their investigation of exposure of miners to radon daughter in these mines (May 1986).
- S29 Swent, L. Communication to the UNSCEAR Secretariat (1984).
- S30 Simpson, S.D., C.G. Stewart, G.R. Yourt et al. Canadian experience in the measurement and control of radiation hazards in uranium mines and mills. p. 73-90 in: *Progress in Nuclear Energy Series XII, Vol. I. Health Physics*, 1959.
- S31 Seven States. Digest of the Proceedings of the Seven State Conference on health hazards in uranium mining. *Arch. Ind. Health* 12: 46-47 (1955).
- S32 Shadley, J.D., J.L. Whitlock, J. Rotmensch et al. The effects of radon daughter alpha-particle irradiation in K1 and xrs-5 CHO cell lines. *Mutat. Res.* 248(1): 73-83 (1991).
- S33 Shapiro, J. An evaluation of the pulmonary radiation dosage from radon and its daughter products. University of Rochester Atomic Energy Project Report UR-298 (1954).
- S34 Sobue, T., V.S. Lee, W. Ye et al. Residential radon exposure and lung cancer risk in Misasa, Japan: a case-control study. *J. Radiat. Res.* 41(2): 81-92 (2000).
- S35 Somosy, Z. Radiation response of cell organelles. *Micron* 31(2): 165-181 (2000).
- S36 Snihs, J.O. The approach to radon problems in non-uranium mines in Sweden. p. 900-911 in: *Third International Congress of the International Radiation Protection Association*, Washington, D.C., 1973.
- S37 Snihs, J.O. and H. Ehdwall. Supervision of radon daughter exposure in mines in Sweden. NEA Specialist Meeting on Personal Dosimetry and Area Monitoring Suitable for Radon and Daughter Products, Elliot Lake, Ontario, 1976.
- S38 Snihs, J.O. Lung cancer among mine workers in Sweden, 1961-1971. in: *Lung Cancer hos Gruvarbetare 1961-1971* (1977). (In Swedish).
- S39 Snihs, J.O. and H. Ehdwall. p. 283-285 in: *Radiation Protection in Swedish Mines. Special Problems* (M. Gomez, ed.). Society of Mining Engineers, American Institute of Mining, Metallurgical and Petroleum Engineers Inc., New York, 1981.
- S40 Steck, D.J., R. William Field and C.F. Lynch. Exposure to atmospheric radon. *Environ. Health Perspect.* 107(2): 123-127 (1999).
- S41 Steinhäusler, F. Environmental ^{220}Rn : a review. *Environ. Int.* 22 (Suppl. 1): 1111-1123 (1996).
- S42 Schoenberg, J.B., J.B. Klotz, H.B. Wilcox et al. Case-control study of residential radon and lung cancer among New Jersey women. *Cancer Res.* 50(20): 6520-6524 (1990).
- S43 Skowronek, J. Radiation exposures to miners in Polish coal mines. *Radiat. Prot. Dosim.* 82(4): 293-300 (1999).
- S44 Strong, J.C. The size of attached and unattached radon daughters in room air. *J. Aerosol Sci.* 19(7): 1327-1330 (1988).
- S45 Solomon, S.B. Assessment of radiation dose from exposure to radon and thoron progeny. p. 356-366 in: *Radon and Thoron in the Human Environment. Proceedings of the Seventh Tohwa University International Symposium* (A. Katase and M. Shimo, eds.). Tohwa, Japan, 1998.
- S46 Steinbuch, M., C.R. Weinberg, J.D. Buckley et al. Indoor residential radon exposure and risk of childhood acute myeloid leukaemia. *Br. J. Cancer* 81(5): 900-906 (1999).
- S47 Sawant, S.G., G. Randers-Pehrson, C.R. Geard et al. The bystander effect in radiation oncogenesis: I. Transformation in C3H 10T $\frac{1}{2}$ cells in vitro can be initiated in the unirradiated neighbors of irradiated cells. *Radiat. Res.* 155(3): 397-401 (2001).
- S48 Smith, B.J., R. William Field and C.F. Lynch. Residential ^{222}Rn exposure and lung cancer: testing the linear no-threshold theory with ecologic data. *Health Phys.* 75(1): 11-17 (1998).
- S49 Stidley, C.A. and J.M. Samet. A review of ecologic studies of lung cancer and indoor radon. *Health Phys.* 65(3): 234-251 (1993).
- S50 Sun, S. Risk coefficient of radon-induced lung cancer and combined effect of arsenic in miners of Yunnan tin mine of China. *Chin. J. Radiol. Med. Prot.* 18(4): 217-224 (1998).
- S51 Sun, S.Q. Etiology of lung cancer in the Gejiu tin mine, China. p. 103-115 in: *Unusual Occurrences as Clues to Cancer Etiology* (R.W. Miller et al., eds.). Japan Scientific Societies Press, Tokyo, 1988.
- S52 Shiquan, S., Li Suyun, Yuan Liyun et al. Radioepidemiological studies on the occupational exposure of workers in nuclear industry of China. China Nuclear Science, Technical Report CNIC-00778 (1993).
- S53 Sun, S.Q., Z.Y. You, S.Y. Tan et al. Patho-histogenetic approach on the etiology of Yunnan tin miner's lung cancer. *Chin. Med. J.* 102(5): 347-355 (1989).
- S54 Shiquan, S., Q. Youlin, Y. Shuziang et al. Effects of radon and arsenic in the etiology of lung cancer in a non-ferrous metal mine in China. Communication to the UNSCEAR Secretariat (2003).
- S55 SENES Consultants. Feasibility Study: Saskatchewan Uranium Miners Cohort Study (Part II). Prepared for the Canadian Nuclear Safety Commission. SENES Consultants Limited, Ontario (2003).

- S56 Schiager, K.J., T.B. Borak and J.A. Johnson. Radiation monitoring for uranium miners: evaluation and optimization. Final report on contract No. J0295026 with U.S. Department of Interior, Bureau of Mines. ALARA Inc. (1981).
- S57 Shimo, M. and H. Saito. Size distribution of radon progeny aerosols in indoor and outdoor air. *J. Environ. Radioact.* 51(1): 49-57 (2000).
- S58 Smerhovsky, Z., K. Landa, P. Rossner et al. Increased risk of cancer in radon-exposed miners with elevated frequency of chromosomal aberrations. *Mutat. Res.* 514(1-2): 165-176 (2002).
- S59 Schaffrath Rosario, A., W. Oberaigner, J. Wellmann et al. Residential radon and lung cancer risk: first analyses of a case-control study in the district of Imst, Tyrol/Austria. p. 629-634 in: *Indoor Air 2002. Proceedings of the Ninth International Conference on Indoor Air Quality and Climate, Monterey, California, 2002.* Vol. 1 (H. Levin, ed.). International Society of Indoor and Climate, Santa Cruz, CA, 2002.
- S60 Stram, D.O., B. Langholz, M. Huberman et al. Correcting for exposure measurement error in a reanalysis of lung cancer mortality for the Colorado Plateau uranium miners cohort. *Health Phys.* 77(3): 265-275 (1999).
- S61 Schaffrath Rosario, A., I.M. Heid, L. Kreienbrock et al. Bewertung des Lungenkrebs-risikos durch Radon in Wohnungen in Deutschland mit Hilfe statistisch-epidemiologischer Modelle. Abschlussbericht an das Bundesamt für Strahlenschutz und den Bundesminister für Umwelt, Naturschutz und Reaktorsicherheit. Forschungsvorhaben StSch 4237. Bundesamt für Strahlenschutz, Neuherberg (2004).
- S62 Stather, J.W. Dosimetric and epidemiological approaches to assessing radon doses — can the differences be reconciled? *Radiat. Prot. Dosim.* 112(4): 487-492 (2004).
- S63 Sun, Q., S. Tokonami, C. Hou et al. Epidemiological potentials of radon- and thoron-prone area in China. *Jpn. J. Health Phys.* 39(3): 258-262 (2004).
- S64 Sanada, T., K. Fujimoto, K. Miyano et al. Measurement of nationwide indoor Rn concentration in Japan. *J. Environ. Radioact.* 45(2): 129-137 (1999).
- S65 Sanchez, L.H., M.T. Quintanilla and A.M. Aguilar. Resultados del programa nacional de monitoreo de gas radon en casas habitación en la Republica Mexicana. Comisión Nacional de Seguridad Nuclear y Salvaguardias. XIII Congreso Anual SNM, XX Reunion Anual SMSR. Ixtapa, Zihuatanejo, Mexico (2002).
- S66 Sun, Q., S. Tokonami, Y. Yamada et al. Main meteorological parameters to influence indoor radon level. *Radioisotopes* 51(3): 120-126 (2002).
- S67 Shang, B., D. Xu, H. Cui et al. Radon Levels Survey in Residences in China. National Institute for Radiological Protection, China, 2006.
- S68 Shang, B., B. Chen, Y. Gao et al. Thoron levels in traditional Chinese residential dwellings. *Radiat. Environ. Biophys.* 44(3): 193-199 (2005).
- S69 Shang, B., T. Iida, Z.Y. Wang et al. Influence of ^{220}Rn on ^{222}Rn measurement in Chinese cave dwellings. p. 379-384 in: *Radon and Thoron in the Human Environment* (A. Katase and M. Shimo, eds.). World Scientific, Singapore, 1998.
- S70 Sugino, M., S. Tokonami and W. Zhuo. Radon and thoron concentrations in offices and dwellings of the Gunma prefecture. *Japan. J. Radioanal. Nucl. Chem.* 266(2): 205-209 (2005).
- S71 Srivastava, G.K., A.H. Khan and M. Raghavayya. Utility of low-level radon detection system. *Bull. Radiat. Prot.* 8(2): April-June (1985).
- S72 Srivastava, G.K., M. Raghavayya, A.H. Khan et al. A low-level radon detection system. *Health Phys.* 46(1): 225-228 (1984).
- S73 Sowa Resat, M.B. and W.F. Morgan. Radiation-induced genomic instability: a role for secreted soluble factors in communicating the radiation response to non-irradiated cells. *J. Cell. Biochem.* 92(5): 1013-1019 (2004).
- S74 Samet, J.M., D.R. Pathak, M.V. Morgan et al. Lung cancer mortality and exposure to radon progeny in a cohort of New Mexico underground uranium miners. *Health Phys.* 61(6): 745-752 (1991).
- S75 Samet, J.M., M.V. Morgan, C.R. Key et al. Studies of uranium miners in New Mexico. *Annals of the American Conference of Governmental Industrial Hygienists* 15: 351-355 (1986).
- S76 Sandler, D.P., C.R. Weinberg, V.E. Archer et al. Indoor radon and lung cancer risk: a case-control study in Connecticut and Utah. *Radiat. Res.* 151(1): 103-104 (1999).
- T1 Taylor, J.A., M.A. Watson, T.R. Devereux et al. *p53* mutation hotspot in radon-associated lung cancer. *Lancet* 343(8889): 86-87 (1994).
- T2 Tomasek, L. and V. Placek. Radon exposure and lung cancer risk: Czech cohort study. *Radiat. Res.* 152 (6): S59-S63 (1999).
- T3 Thompkins, R.W. *The Richest Canadian — The Life and Career of a Canadian Mining Engineer.* p. 80-86. Whistler House, Toronto, 1994.
- T4 Thompkins, R.W. *Ventilation and dust report.* Eldorado Mining & Refining, Port Radium, Northwest Territories (August 1945).
- T5 Tirmarche, M., D. Laurier, L. Tomasek et al. The French cohort of uranium miners: Analysis of lung cancer risk linked to radon exposure in a population exposed to relatively low concentration over a long duration. p. 693-696 in: *Low Doses of Ionizing Radiation: Biological Effects and Regulatory Control.* Contributed Papers. IAEA-TECDOC-976. IAEA, Vienna (1997).
- T6 Tirmarche, M., A. Raphalen, F. Allin et al. Mortality of a cohort of French uranium miners exposed to relatively low radon concentrations. *Br. J. Cancer* 67(5): 1090-1097 (1993).
- T7 Tirmarche, M., A. Raphalen, F. Allin et al. Epidemiological study of the mortality of a group of uranium miners in France. p. 550-554 in: *IRPA 8. Proceedings*

- of the International Radiation Protection Association, Vol. 1, Montreal, May 1992.
- T8 Tirmarche, M., A. Raphalen and J. Chameaud. Epidemiological study of French uranium miners. *Cancer Detect. Prev.* 16(3): 169-172 (1992).
- T9 Tirmarche, M., J. Brenot, J. Piechowski et al. The present state of an epidemiological study of uranium miners in France. p. 344-349 in: *Proceedings of an International Conference on Occupational Radiation Safety in Mining*, Toronto, October 1984 (H. Stocker, ed.). Canadian Nuclear Association, Toronto, 1985.
- T10 Tomasek, L., E. Kunz, T. Muller et al. Radon exposure and lung cancer risk — Czech cohort study on residential radon. *Sci. Total Environ.* 272(1-3): 43-51 (2001).
- T11 Tirmarche, M., B. Grosche, D. Laurier et al. Uranium miners studies in Europe. Final Report, Period 1996-1999, EC Contract F14-CT95-0031. European Community (1999).
- T12 Tokonami, S., H. Yonehara, W. Zhuo et al. Understanding of high radon concentrations observed in a well-ventilated Japanese wooden house. p. 665-669 in: *Indoor Air 2002. Proceedings of the Ninth International Conference on Indoor Air Quality and Climate*, Monterey, California, 2002. Vol. 1 (H. Levin, ed.). International Society of Indoor and Climate, Santa Cruz, CA, 2002.
- T13 Tokonami, S., F. Takahashi, T. Iimoto et al. A new device to measure the activity size distribution of radon progeny in a low level environment. *Health Phys.* 73(3): 494-497 (1997).
- T14 Tokonami, S., M. Yang, H. Yonehara et al. Simple, discriminative measurement technique for radon and thoron concentrations with a single scintillation cell. *Rev. Sci. Instrum.* 73(1): 69-72 (2002).
- T15 Tokonami, S., Q. Sun, H. Yonehara et al. A simple measurement technique of the equilibrium equivalent thoron concentration with a CR-39 detector. *Jpn. J. Health Phys.* 37(1): 59-63 (2002).
- T16 Tokonami, S., M. Yang, T. Sanada et al. Thoron spike test for passive radon detectors. *Sci. Total Environ.* 272(1-3): 247-248 (2001).
- T17 Tokonami, S., M. Yang and T. Sanada. Contribution from thoron on the response of passive radon detectors. *Health Phys.* 80(6): 612-615 (2001).
- T18 Tokonami, S., Q. Sun, S. Akiba et al. Natural radiation exposures for cave residents in China. p. 560-566 in: *Radioactivity in the Environment, Vol. 7: The Natural Radiation Environment VII. Seventh International Symposium on the Natural Radiation Environment (NRE-VII)* (J.P. McLaughlin, S.E. Simopoulos and F. Steinhäusler, eds.). Elsevier Ltd., London, 2005.
- T19 Tokonami, S. Determination of the diffusion coefficient of unattached radon progeny with a graded screen array at the EML radon/aerosol chamber. *Radiat. Prot. Dosim.* 81(4): 285-290 (1999).
- T20 Tokonami, S. Experimental verification of the attachment theory of radon progeny onto ambient aerosols. *Health Phys.* 78(1): 74-79 (2000).
- T21 Tso, M.Y. and C.C. Li. Indoor and outdoor ^{222}Rn and ^{220}Rn daughters in Hong Kong. *Health Phys.* 53(2): 175-180 (1987).
- T22 Trautmannsheimer, M., W. Schindlmeier and K. Börner. Radon concentration measurements and personnel exposure levels in Bavarian water supply facilities. *Health Phys.* 84(1): 100-110 (2003).
- T23 Tu, K.W. and E.O. Knutson. Indoor radon progeny particle size distribution measurements made with two different methods. *Radiat. Prot. Dosim.* 24(1): 251-255 (1988).
- T24 Thorne, R., N.K. Foreman and M.G. Mott. Radon in Devon and Cornwall and paediatric malignancies. *Eur. J. Cancer* 32A(2): 282-285 (1996).
- T25 Tu, K.W., E.O. Knutson and A.C. George. Indoor radon progeny aerosol size measurements in urban, suburban and rural regions. *Aerosol Sci. Technol.* 15(3): 170-178 (1991).
- T26 Tomasek, L. Czech miner studies of lung cancer risk from radon. *J. Radiol. Prot.* 22(3A): A107-A112 (2002).
- T27 Tomasek, L., M. Tirmarche and D. Laurier. Risk of lung cancer death among the Czech and French uranium miners: effect of low radon exposure. Presented at the 13th International Conference of the International Society for Environmental Epidemiology, Garmisch-Partenkirchen, Germany, 2-5 September 2001.
- T28 Tomasek, L. Leukemia among uranium miners — late effects of exposure to uranium dust? *Health Phys.* 86(4): 426-427 (2004).
- T29 Tomasek, L., T. Muller, E. Kunz et al. Study of lung cancer and residential radon in the Czech Republic. *Cent. Eur. J. Public Health* 9(3): 150-153 (2001).
- T30 Tirmarche, M., D. Laurier, D. Bergot et al. Quantification of lung cancer risk after low radon exposure and low exposure rate: synthesis from epidemiological and experimental data. Research Project UMINERS+ANIMAL DATA. Final Technical Report, EC contract No. FIGH-CT1999-00013. European Community (2003).
- T31 Tirmarche, M., D. Laurier, N. Mitton et al. Lung cancer risk associated with low chronic radon exposure: Results from the French uranium miners cohort and the European project. In: *IRPA-10, 10th International Congress of the International Radiation Protection Association*, Hiroshima, Japan, 14-19 May 2000.
- T32 Taylor, P.R., Y.L. Qiao, A. Schatzkin et al. Relation of arsenic exposure to lung cancer among tin miners in Yunnan Province, China. *Br. J. Ind. Med.* 46(12): 881-886 (1989).
- T33 Tokonami, S., H. Yonehara, S. Akiba et al. Natural radiation levels in Tamil Nadu and Kerala, India. p. 554-559 in: *Radioactivity in the Environment, Vol. 7: The Natural Radiation Environment VII. Seventh International Symposium on the Natural Radiation Environment (NRE-VII)* (J.P. McLaughlin, S.E. Simopoulos and F. Steinhäusler, eds.). Elsevier Ltd., London, 2005.

- T34 Tokonami, S., M. Furukawa, Y. Shicchi et al. Characteristics of radon and its progeny concentrations in air-conditioned office buildings in Tokyo. *Radiat. Prot. Dosim.* 106(1): 71-76 (2003).
- T35 Tokonami, S., Q. Sun, S. Akiba et al. Radon and thoron exposures for cave residents in Shanxi and Shaanxi provinces. *Radiat. Res.* 162(4): 390-396 (2004).
- T36 Tokonami, S., T. Matsuzawa, T. Ishikawa et al. Changes of indoor aerosol characteristics and their associated variation on the dose conversion factor due to radon progeny inhalation. *Radioisotopes* 52(6): 285-292 (2003).
- T37 Tomasek, L., S.C. Darby, T. Fearn et al. Patterns of lung cancer mortality among uranium miners in West Bohemia with varying rates of exposure to radon and its progeny. *Radiat. Res.* 137(2): 251-261 (1994).
- T38 Tomasek, L. Epidemiological studies of Czech miners. *Wiss. Umwelt* 3: 163-168 (1995).
- T39 Tomasek, L., V. Placek, T. Muller et al. Czech studies of lung cancer risk from radon. *Int. J. Low Radiat.* 1(1): 50-62 (2003).
- T40 Tomasek, L. and H. Zarska. Lung cancer risk among Czech tin and uranium miners — comparison of life-time detriment. *Neoplasma* 51(4): 255-260 (2004).
- T41 Tokonami, S., Y. Ishimori, T. Ishikawa et al. Inter-comparison exercise of measurement techniques for radon, radon decay products and their particle size distributions at NIRS. *Jpn. J. Health Phys.* 40(2): 183-190 (2005).
- T42 Tokonami, S., R. Kurosawa, T. Iimoto et al. Evaluation of characteristics of passive radon monitor focussed on its transient characteristics. *J. Nucl. Sci. Technol.* 32(7): 702-712 (1995).
- T43 Tokonami, S., T. Iimoto, T. Ichiji et al. Continuous radon monitor using a two-filter method. *Radiat. Prot. Dosim.* 63(2): 123-126 (1996).
- T44 Tokonami, S., T. Iimoto, T. Ichiji et al. Integrated measurement of equilibrium equivalent radon and thoron concentrations using cellulose nitrate film. *Radiat. Meas.* 26(5): 689-699 (1996).
- T45 Tokonami, S., T. Iimoto and R. Kurosawa. Continuous measurement of the equilibrium factor F and the unattached fraction f_p of radon progeny in the environment. *Environ. Int.* 22 (Suppl. 1): S611-S616 (1996).
- T46 Tokonami, S., T. Ichiji, T. Iimoto et al. Calculation procedure of potential alpha energy concentration with continuous air sampling. *Health Phys.* 71(6): 937-943 (1996).
- T47 Tokonami, S., W. Zhuo, H. Ryuo et al. Instrument performance of a radon measuring system with the alpha-track detection technique. *Radiat. Prot. Dosim.* 103(1): 69-72 (2003).
- T48 Tokonami, S., H. Takahashi, Y. Kobayashi et al. Up-to-date radon-thoron discriminative detector for a large scale survey. *Rev. Sci. Instrum.* 76(11): 113505.1-113505.5 (2005).
- T49 Tokonami, S., K. Fukutsu, Y. Yamada et al. Particle size measurement of radon decay products using MOUDI and GSA. p. 278-280 in: *High Levels of Natural Radiation and Radon Areas: Radiation Dose and Health Effects* (T. Sugahara et al., eds.). International Congress Series 1276. Elsevier, 2005.
- T50 Taeger, D., A. Fritsch, T. Wiethege et al. Role of exposure to radon and silicosis on the cell type of lung carcinoma in German uranium miners. *Cancer* 106(4): 881-889 (2006).
- T51 Tomasek, L., S.C. Darby, A.J. Swerdlow et al. Radon exposure and cancers other than lung cancer among uranium miners in West Bohemia. *Lancet* 341(8850): 919-923 (1993).
- U2 United Nations. Sources and Effects of Ionizing Radiation. Volume I: Sources; Volume II: Effects. United Nations Scientific Committee on the Effects of Atomic Radiation, 2000 Report to the General Assembly, with scientific annexes. United Nations sales publications E.00.IX.3 and E.00.IX.4. United Nations, New York, 2000.
- U5 United Nations. Sources and Effects of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation, 1993 Report to the General Assembly, with scientific annexes. United Nations sales publication E.94.IX.2. United Nations, New York, 1993.
- U6 United Nations. Sources, Effects and Risks of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation, 1988 Report to the General Assembly, with annexes. United Nations sales publication E.88.IX.7. United Nations, New York, 1988.
- U9 United Nations. Sources and Effects of Ionizing Radiation. United Nations Scientific Committee on the Effects of Atomic Radiation, 1977 Report to the General Assembly, with annexes. United Nations sales publication E.77.IX.1. United Nations, New York, 1977.
- U16 United States Bureau of Mines. Alpha radiation exposure levels in underground uranium and other mines. *Radiol. Health Data Rep.* 9(12): 719-723 (1968).
- U17 United States Environmental Protection Agency, Office of Radiation and Indoor Air. Proposed EPA methodology for assessing risks from indoor radon based on BEIR VI. EPA, Washington, D.C. (1999).
- U18 United States Environmental Protection Agency. National residential radon survey. EPA 402/R-92-011 (1992).
- V1 Vesely, K. and J. Sada. Incidence of specific occupational disease in the Czechoslovak uranium industry. p. 1-29 in: *Proceedings of Symposium for Workers in the Mining Industry, Section for Industrial Safety and Hygiene, Czech.* Report NP-18271 (1968).
- V2 Villeneuve, P.J. and H.I. Morrison. Coronary heart disease mortality among Newfoundland fluorspar miners. *Scand. J. Work Environ. Health* 23(3): 221-226 (1997).

- V3 Vahakangas, K.H., J.M. Samet, R.A. Metcalf et al. Mutations of *p53* and *ras* genes in radon-associated lung cancer from uranium miners. *Lancet* 339(8793): 576-580 (1992).
- V4 Villeneuve, P.J., H.I. Morrison and R. Lane. Radon and lung cancer risk: an extension of the mortality follow-up of the Newfoundland Fluorspar cohort. *Health Physics* 92(2): 157-169 (2007).
- V5 Veiga, L.H., V. Melo, S. Koifman et al. High radon exposure in a Brazilian underground coal mine. *J. Radiol. Prot.* 24(3): 295-305 (2004).
- V6 Villeneuve, P., R. Lane, and H. Morrison. Coronary heart disease mortality and radon exposure in the Newfoundland fluorspar miners' cohort, 1950-2001. *Radiat. Environ. Biophys.* 46(3): 291-296 (2007).
- W1 Wagoner, J.K., V.E. Archer, F.E. Lundin Jr. et al. Radiation as the cause of lung cancer among uranium miners. *N. Engl. J. Med.* 273: 181-188 (1965).
- W2 Wagoner, J.K., V.E. Archer, B.E. Carroll et al. Cancer mortality patterns among U.S. uranium miners and millers, 1950 through 1962. *J. Natl. Cancer Inst.* 32: 787-801 (1964).
- W3 Waxweiler, R.J., R.J. Roscoe, V.E. Archer et al. Mortality follow-up through 1977 of the white underground uranium miners cohort examined by the United States Public Health Service. p. 823-830 in: *Radiation Hazards in Mining: Control, Measurement and Medical Aspects* (M. Gomez, ed.). Society of Mining Engineers, American Institute of Mining, Metallurgical and Petroleum Engineers Inc., New York, 1981.
- W4 Wiethage, T., H. Wesch, K. Wegener et al. German uranium miner study — pathological and molecular genetic findings. German Uranium Miner Study, Research Group Pathology. *Radiat. Res.* 152(6): S52-S55 (1999).
- W5 Windish, J.P. Health Hazards in the Mines of Newfoundland. III. Radiation Levels in the Workings of Newfoundland Fluorspar Limited, St. Lawrence, Newfoundland. Occupational Health Division, Department of National Health and Welfare, Ottawa, Ontario, 1960.
- W6 Windish, J.P. and H.P. Sanderson. Dust Hazards in the Mines of Newfoundland. I. Newfoundland Fluorspar Limited, St. Lawrence, Newfoundland. Occupational Health Division, Department of National Health and Welfare, Ottawa, Ontario, 1958.
- W7 Windish, J.P. and H.P. Sanderson. Dust Hazards in the Mines of Newfoundland. II. St. Lawrence Corporation of Newfoundland, Limited, St. Lawrence, Newfoundland. Occupational Health Division, Department of National Health and Welfare, Ottawa, Ontario, 1958.
- W8 Wolff, S., R. Jostes, F.T. Cross et al. Adaptive response of human lymphocytes for the repair of radon-induced chromosomal damage. *Mutat. Res.* 250(1-2): 299-306 (1991).
- W9 Wolff, S., V. Afzal, R.F. Jostes et al. Indications of repair of radon-induced chromosome damage in human lymphocytes: an adaptive response induced by low doses of X-rays. *Environ. Health Perspect.* 101 (Suppl. 3): 73-77 (1993).
- W10 Wright, E.S. and C.M. Couves. Radiation-induced carcinoma of the lung — the St. Lawrence tragedy. *J. Thorac. Cardiovasc. Surg.* 74(4): 495-498 (1977).
- W11 Wu, L.J., G. Randers-Pehrson, A. Xu et al. Targeted cytoplasmic irradiation with alpha particles induces mutations in mammalian cells. *Proc. Natl. Acad. Sci. U.S.A.* 96(9): 4959-4964 (1999).
- W12 Whittemore, A.S. and A. McMillan. Lung cancer mortality among U.S. uranium miners: a reappraisal. Technical Report No. 68. Department of Statistics, Stanford University, CA (1983). Also in *J. Natl. Cancer Inst.* 71(3): 489-499 (1983).
- W13 Wang, Z., J.H. Lubin, L. Wang et al. Residential radon and lung cancer risk in a high-exposure area of Gansu Province, China. *Am. J. Epidemiol.* 155(6): 554-564 (2002).
- W14 Wrixon, A.D., B.M.R. Green, P.R. Lomas et al. Natural radiation exposure in UK dwellings. NRPB-R190 (1988).
- W15 Woodward, A., D. Roder, A.J. McMichael et al. Radon daughter exposures at the Radium Hill uranium mine and lung cancer rates among former workers, 1952-87. *Cancer Causes Control* 2(4): 213-220 (1991).
- W16 Wang, Z. and F. Steinhäusler. Elevated indoor exposure in Chinese carbon brick and cave dwellings. *Health Phys.* 71(3): 374-378 (1996).
- W17 Wesch, H., A. Eisenmenger, K.M. Müller et al. Radiologische Erfassung, Untersuchung und Bewertung bergbaulicher Altlasten; Gesundheitliche Bewertung; Teilprojekt: Pathologie. Abschlussbericht für die Forschungsprojekte StSch 4057/4, 4118, 4242 (Februar 2004).
- W18 World Health Organization, Regional Office for Europe, Copenhagen. Indoor air pollutants: radon (Chapter 8.3). p. 209-218 in: *Air Quality Guidelines for Europe*, Second Edition. WHO Regional Publications, European Series No. 91, 2001.
- W19 Wichmann, H.E., A.S. Rosario, I.M. Heid et al. Increased lung cancer risk due to residential radon in a pooled and extended analysis of studies in Germany. *Health Phys.* 88(1): 71-79 (2005).
- W20 Wiegand, J., S. Feige, X. Qingling et al. Radon and thoron in cave dwellings (Yan'an, China). *Health Phys.* 78(4): 438-444 (2000).
- W21 Writing Group of the Summary Report on Nationwide Survey of Environmental Radioactivity Level in China. Survey of concentrations of radon and alpha potential energy of Rn daughter products in air in some regions of China (1983-1990). *Radiat. Prot.* 12(2): 164-171 (1992). (In Chinese).
- X1 Xuan, X.Z., J.H. Lubin, J.Y. Li et al. A cohort study in southern China of tin miners exposed to radon and radon decay products. *Health Phys.* 64(2): 120-131 (1993).
- Y1 Yao, S.X., J.H. Lubin, Y.L. Qiao et al. Exposure to radon progeny, tobacco use and lung cancer in a case-control study in southern China. *Radiat. Res.* 138(3): 326-336 (1994).

- Y2 Yeh, H.C. and G.M. Schum. Models of human lung airways and their application to inhaled particle deposition. *Bull. Math. Biol.* 42(3): 461-480 (1980).
- Y3 Yu, K.N., B.M.F. Lau, Z.J. Guan et al. Bronchial Rn dose survey for residences. *J. Environ. Radioact.* 54(2): 221-229 (2001).
- Y4 Yu, K.N., B.T. Wong, J.Y. Law et al. Indoor dose conversion coefficients for radon progeny for different ambient environments. *Environ. Sci. Technol.* 35(11): 2136-2140 (2001).
- Y5 Yu, K.N., T.T. Cheung, A.K. Haque et al. Radon progeny dose conversion coefficients for Chinese males and females. *J. Environ. Radioact.* 56(3): 327-340 (2001).
- Y6 Yngveson, A., C. Williams, A. Hjerpe et al. *p53* mutations in lung cancer associated with residential radon exposure. *Cancer Epidemiol. Biomarker Prev.* 8(5): 433-438 (1999).
- Y7 Yassin, S.S. and D.J. Steck. Preliminary survey of natural background radiation in the Gaza Strip in Palestine. Vol. II. p. 253-256 in: *Proceedings of the Fifth International Conference on High Levels of Natural Radiation and Radon Areas: Radiation Dose and Health Effects*, Munich, 4-7 September 2000.
- Y8 Yamada, Y., Q. Sun, S. Tokonami et al. Radon-thoron discriminative measurements in Gansu province, China, and their implication for dose estimates. *J. Toxicol. Environ. Health Part A* 69(7): 723-724 (2006).
- Y9 Yonehara, H., S. Tokonami, W. Zhuo et al. Thoron in the living environments of Japan. p. 58-61 in: *Proceedings of the Sixth International Conference on High Levels of Natural Radiation and Radon Areas: Radiation Dose and Health Effects* (T. Sugahara, Y. Sasaki, H. Morishima et al., eds.), Osaka, Japan, 6-10 September 2004. Elsevier, 2005.
- Y10 Yamada, Y., S. Tokonami, S. Zhuo et al. Rn-Tn discriminative measurements and their dose estimates in Chinese loess plateau. p. 76-80 in: *High Levels of Natural Radiation and Radon Areas: Radiation Dose and Health Effects* (T. Sugahara et al., eds.). *International Congress Series* 1276. Elsevier, 2005.
- Z1 Zhao, Y.L., C.Q. Piao, E.J. Hall et al. Mechanisms of radiation-induced neoplastic transformation of human bronchial epithelial cells. *Radiat. Res.* 155(1): 230-234 (2001).
- Z2 Zunic, Z.S., J.P. McLaughlin, C. Walsh et al. Integrated natural radiation exposure studies in stable Yugoslav rural communities. *Sci. Total Environ.* 272(1-3): 253-259 (2001).
- Z3 Zhuo, W., S. Tokonami, H. Yonehara et al. A simple passive monitor for integrating measurements of indoor thoron concentrations. *Rev. Sci. Instrum.* 73(8): 2877-2881 (2002).
- Z4 Zhuo, W. and T. Iida. Estimation of thoron progeny concentrations in dwellings with their deposition rate measurements. *J. Health Phys.* 35(3): 365-370 (2000).
- Z5 Zhuo, W., T. Iida and X. Yang. Environmental radon and thoron progeny concentrations in Fujian Province of China. *Radiat. Prot. Dosim.* 87(2): 137-140 (2000).
- Z6 Zhao, G., L. Sun, X. Ji et al. Experimental study on lung cancer induced by exposure of mine dust and radon daughters. *Radiat. Prot.* 7(3): 203-209 (1987).
- Z7 Zaborowski, W., S.D. Chambers and A. Henderson-Sellers. Ground based radon-222 observations and their application to atmospheric studies. *J. Environ. Radioact.* 76(1-2): 3-33 (2004).
- Z8 Zhuo, W., T. Iida, J. Moriizumi et al. Simulation of the concentrations and distributions of indoor radon and thoron. *Radiat. Prot. Dosim.* 93(4): 357-368 (2001).
- Z9 Zalewski, M., Z. Mnich, M. Karpińska et al. Indoor radon concentrations in Poland as determined in short-term (two-day) measurements. *Radiat. Prot. Dosim.* 95(2): 157-163 (2001).
- Z10 Zettwoog, Z.R. State-of-the-art of the alpha individual dosimetry in France. p. 321-331 in: *Radiation Hazards in Mining: Control, Measurement and Medical Aspects*, Chapter 50 (M. Gomez, ed.). Society of Mining Engineers, New York, 1982.
- Z11 Zeeb, H. World Health Organization. International Radon Project. Survey on radon guidelines, programmes and activities. Final report. WHO (2007).